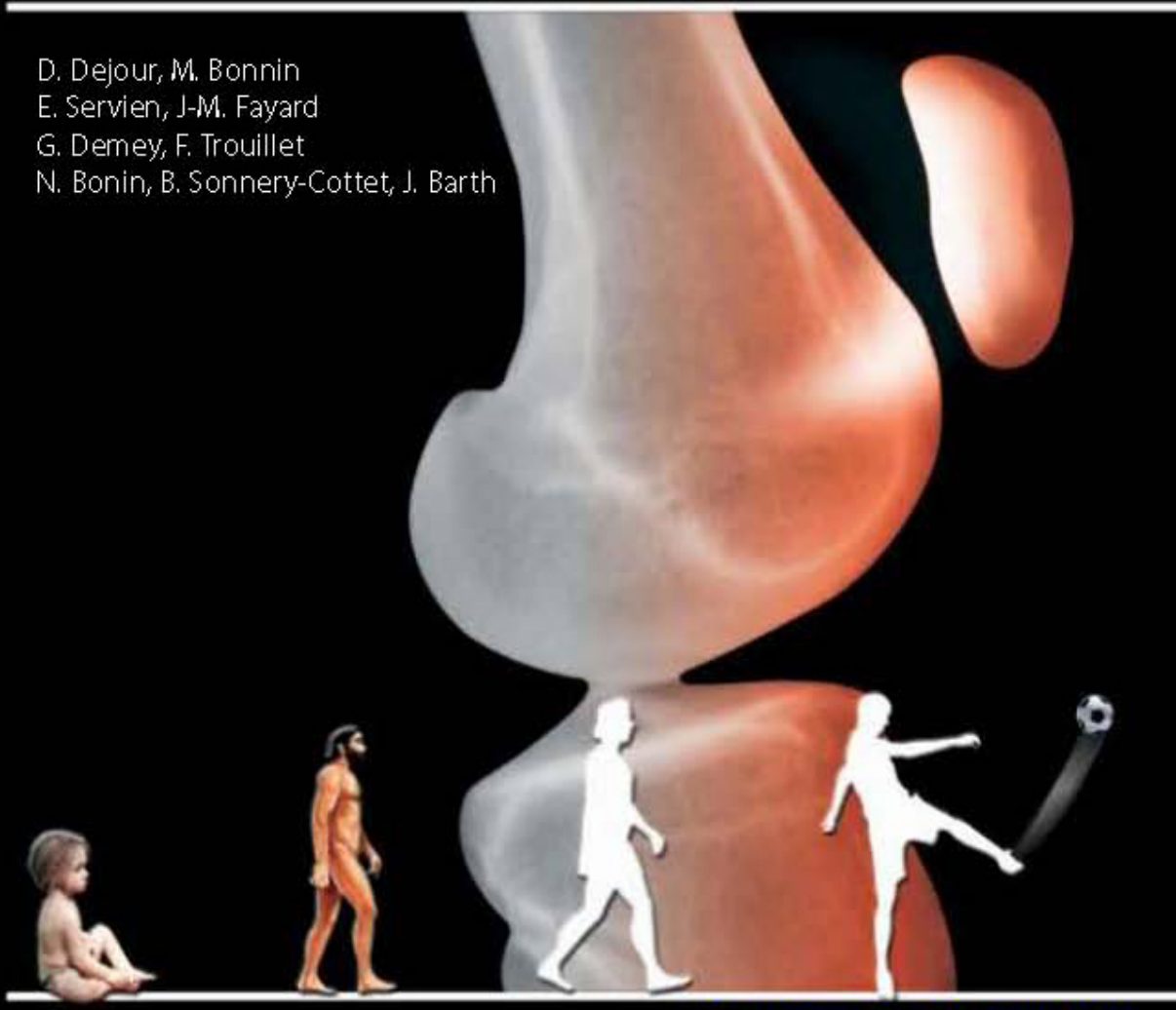
15èmes Journées Lyonnaises de Chirurgie du Genou

La Patella

D. Dejour, M. Bonnin
E. Servien, J-M. Fayard
G. Demey, F. Trouillet
N. Bonin, B. Sonnery-Cottet, J. Barth



15èmes Journées Lyonnaises de Chirurgie du Genou

La Patella

25 ans après...25 years later..

2012

1987

ALRM *Since 1969*

Past presidents : Albert Trillat, Henri Dejour, Pierre Chambat, Philippe Neyret.

SAURAMPS MEDICAL

La Patella

Ce logo a pour objet d'alerter le lecteur sur la menace que représente pour l'avenir de l'écrit, tout particulièrement dans le domaine universitaire, le développement massif du « Photocopillage ».
Cette pratique qui s'est généralisée, notamment dans les établissements d'enseignement, provoque une baisse brutale des achats de livres, au point que la possibilité même pour les auteurs de créer des œuvres nouvelles et de les faire éditer correctement est aujourd'hui menacée.
Nous rappelons donc que la reproduction et la vente sans autorisation, ainsi que le recel, sont passibles de poursuites.
Les demandes d'autorisation de photocopier doivent être adressées à l'éditeur ou au Centre français d'exploitation du droit de copie, 3, rue Hautefeuille, 75006 Paris.
Téléphone : 01 43 26 95 35

Dépôt légal :septembre 2012

I.S.B.N. : 978-2-84023-823-2
E.A.N. : 9782840238232

Imprimé en France

La Patella

D. DEJOUR, M. BONNIN, E. SERVIEN, J.M. FAYARD,
G. DEMEY, F. TROUILLET, N. BONIN,
B. SONNERY-COTTET, J. BARTH

Avec la collaboration de :

R. Afra, K.F. Almqvist, A.A. Amis, E.A. Arendt , J.N. Argenson, J.M. Aubaniac, R. Badet, M. Bakir, J. Barth, R. Bastos-Filho, P. Beaufils, J. Bérard, J.B. Berard, C. Bessiere, S. Bignozzi, P. Boisrenoult, N. Bonin, M. Bonnin, P. Byn, G.L. Camanho, J. Caton, M.M. Chaker, P. Chambat, M. Charles, V. Chassaing, S. Chomel, F. Chotel, J. Chouteau, ING F. Colle, O. Courage, D.L. Dahm, R.J. de Jong, D. Dejour, S. Delforge, G. Demey, G. Deschamps, A.A.M. Dhollander, S. Donell, V.B. Duthon, P.J. Erasmus, J.M. Fayard, J.A. Feller, P. Ferrua, C. Fink, D.C. Fithian, X. Flecher, F. Gadea, E. Gancel, Y. Glard, F. Gougeon, C. Hoser, C. Hulet, M. Jacobi, J.L. Jouve, I. Klebaner, R. Kohler, S.J. Krikler, C. Lapra, W.B. Leadbetter, S. Lippacher, N. Lopomo, S. Lustig, R.A. Magnussen, L. Malekpour, M. Marcacci, F. Mauris, C. Mayer, H. Migaud, D. Monnot, R. Mortati, N. Nakamura, M. Nelitz, P. Neyret, P.G. Ntagiopoulos, M. Odumenya, M. Ollivier, S. Özcan, J.C. Panisset, S. Parratte, G. Pasquier, D. Passeron, A. Peltier, A. Pinaroli, V. Pineau, N. Pujol, B. Quelard, H. Reichel, F. Rémy, S. Rochcongar, P.R.F. Saggin, P.B. Schoettle, E. Servien, B. Sharma, K. Shino, P. Sillanpää, B. Sonnery-Cottet, C. Tardieu, M. Thaunat, F. Trouillet, H.P.W. van Jonbergen, A. van Kampen, P. Verdonk, M. Veselko, J. Victor, A. Viste, A. Wymenga, Y. Yercan, H. Yoshikawa, S. Zaffagnini

Remerciement à Florence Bondoux (secrétariat scientifique de l'ALRM) pour la réalisation de ce livre.

SAURAMPS MEDICAL

11, boulevard Henri IV - 34000 Montpellier
E.mail : sauramps.medical@wanadoo.fr

LISTE DES AUTEURS

R. AFRA : Department of Orthopaedic Surgery, University of California San Diego

K.F. ALMQVIST : Dept. of Orthopaedic Surgery and Traumatology, Ghent University Hospital, Ghent University, De Pintelaan 185, 9000 Gent, Belgium

A.A. AMIS : Imperial College London - South Kensington Campus - London SW7 2AZ – UK

E.A. ARENDT : Professor and Vice Chair - Department of Orthopaedic Surgery - University of Minnesota - Minneapolis, MN, USA

J.N. ARGENSON : Institut du mouvement et de l'appareil locomoteur - Centre Hospitalo-Universitaire Sud - Hôpital Sainte-Marguerite - 270, bd de Sainte Marguerite - 13009 Marseille

J.M. AUBANIAC : Institut du mouvement et de l'appareil locomoteur - Centre Hospitalo-Universitaire Sud - Hôpital Sainte-Marguerite - 270, bd de Sainte Marguerite - 13009 Marseille

R. BADET : Pôle ostéo-articulaire santé et sport - 60, av. du Médipôle - 38300 Bourgoin jallieu

M BAKIR : IRM GIE Lyon Nord - Clinique Protestante - 1, chemin du Penthod - 69300 Caluire

J. BARTH : Clinique des Cèdres - 21, rue Albert Londres - 38130 Echirolles

R. BASTOS-FILHO : Hospital Federal dos Servidores do Estado, Rio de Janeiro, RJ, Brasil

P. BEAUFILS : Centre Hospitalier de Versailles - F 78150 Le Chesnay

J.B. BERARD : Service de Chirurgie orthopédique et traumatologique, Centre Hospitalier Lyon-Sud, Chemin du Grand Revoyet - 69310 Pierre-Bénite

C. BESSIERE : Centre Orthopédique Paul Santy - 24, avenue Paul Santy - 69008 Lyon

S. BIGNOZZI : 3rd Orthopaedic and Traumatology Clinic and Biomechanics Laboratory, Codivilla-Putti Research Center - Istituto Ortopedico Rizzoli - University of Bologna

P. BOISRENOULT : Centre Hospitalier de Versailles - F 78150 Le Chesnay

N. BONIN : Lyon-Ortho-Clinic ; Clinique de la Sauvegarde - 8, avenue Ben Gourion - 69009 Lyon

M. BONNIN : Centre Orthopédique Paul Santy - 24, avenue Paul Santy - 69008 Lyon

P. BYN : Maria middleAres hospital, Ghent 9000, Belgium

G.L. CAMANHO : Full Professor of the Department of Orthopaedics and Traumatology, University of São Paulo School of Medicine- Rua Oliveira Dias 61 São-Paulo-Brasil

J. CATON : Clinique Emilie de Vialar - 116, rue Antoine Charial - 69003 Lyon

M.M. CHAKER : Hôpital Femme Mère Enfant - 59 bd Pinel - 69677 Bron cedex

P. CHAMBAT : Centre Orthopédique Paul Santy - 24, avenue Paul Santy - 69008 Lyon

M. CHARLES : Department of Orthopaedic Surgery, University of California San Diego

V. CHASSAING : 67, rue de Romainvile - 75019 Paris

S CHOMEL : Service Radiologie - Clinique Protestante - 1 Chemin du Penthod - 69300 Caluire

F. CHOTEL : Hôpital Femme Mère Enfant - 59 bd Pinel - 69677 Bron cedex

J. CHOUTEAU : Cabinet de Chirurgie Orthopédique et de Chirurgie du Sport - Allée de la Mandallaz, Immeuble Le Périclès - Montée B - 74370 Metz-Tessy — Clinique Argonay - 685, route de Menthonnex - 74370 Argonay

F. COLLE : 3rd Orthopaedic and Traumatology Clinic and Biomechanics Laboratory, Codivilla-Putti Research Center - Istituto Ortopedico Rizzoli - University of Bologna

O. COURAGE : Hôpital privé de l'Estuaire - 505, rue Irène Joliot-Curie - BP 90011 - 76620 Le Havre

D.L. DAHM : Rochester Methodist Hospital, Saint-Marys Hospital - 200 First Street, SW - MN 55905 Rochester, USA

R.J. DE JONG : Dep. of Orthopedic Surgery, Radboud University Nijmegen Medical Centre, Nijmegen, The Netherlands

D. DEJOUR : Lyon-Ortho-Clinic ; Clinique de la Sauvegarde - 8, avenue Ben Gourion - 69009 Lyon

S. DELFORGE : U1075 Comete UCBN/INSERM, UFR de Médecine - Département d'Orthopédie Traumatologie - Avenue de la Côte de Nacre - 14032 Caen Cedex

G. DEMEY : Groupement Hospitalier Lyon Nord - 103, grande rue de la Croix-Rousse - Pav R - 69317 Lyon Cedex 04

G. DESCHAMPS : Centre Orthopédique - 2, rue du Pressoir - 71640 Dracy-le-Fort

A.A.M. DHOLLANDER : Dept. of Orthopaedic Surgery and Traumatology, Ghent University Hospital, Ghent University, De Pintelaan 185, 9000 Gent, Belgium

S. DONELL Consultant Orthopaedic Surgeon - Norfolk & Norwich University Hospital - Honorary Professor University of East Anglia - Norfolk & Norwich University Hospital - Colney Lane - NR4 7UY – Norwich - United Kingdom

V.B. DUTHON : Hôpitaux Universitaires de Genève - rue Gabrielle-Perret-Gentil - 1205 Genève, Suisse

P.J. ERASMUS : G3 Stellenbosch Medi Clinic - Die Boord - Stellenbosch 7600 - South Africa

J.M. FAYARD : Centre Orthopédique Paul Santy - 24, avenue Paul Santy - 69008 Lyon

J. FELLER : OrthoSport Victoria - Level 5, 89 Bridge Rd - Richmond VIC 3121 - Australia

P. FERRUA : Lyon-Ortho-Clinic ; Clinique de la Sauvegarde - 8, avenue Ben Gourion - 69009 Lyon

C. FINK : Sportsclinic Austria, Innsbruck - Olympiastr.39 - 6020 Innsbruck Austria

D.C. FITHIAN : Department of Orthopedic Surgery, Southern California Permanente Medical Group

X. FLECHER : Institut du mouvement et de l'appareil locomoteur - Centre Hospitalo-Universitaire Sud - Hôpital Sainte-Marguerite - 270, bd de Sainte Marguerite - 13009 Marseille

F. GADEA : Centre Orthopédique Paul Santy - 24, avenue Paul Santy - 69008 Lyon

E. GANCEL : Groupement Hospitalier Lyon Nord - 103, grande rue de la Croix-Rousse - Pav R - 69317 Lyon Cedex 04

Y. GLARD : Laboratoire UMR 7268 - ADéS - Anthropologie bioculturelle Droit Ethique et Santé - 13000 Marseille

F. GOUGEON : Polyclinique de la Louvière - 22 rue La Louvière - 59000 Lille

C. HOSER : Sportsclinic Austria, Innsbruck - Olympiastr.39 - 6020 Innsbruck Austria

C. HULET : U1075 Comete UCBN/INSERM, UFR de Médecine - Département d'Orthopédie Traumatologie - Avenue de la Côte de Nacre - 14032 Caen Cedex

M. JACOBI : Orthopädie am Rosenberg - St. Gallen, Suisse

J.L. JOUVE : Service d'orthopédie pédiatrique, laboratoire UMR 7268. Faculté de médecine Marseille

I. KLEBANER : U1075 Comete UCBN/INSERM, UFR de Médecine - Département d'Orthopédie Traumatologie - Avenue de la Côte de Nacre - 14032 Caen Cedex

R. KOHLER : Hôpital Edouard Herriot - Pav T bis - 5 place d'Arsonval - 69003 Lyon

S.J. KRIKLER : Coventry CV2 2DX - UK

C. LAPRA : Service Radiologie - Clinique Protestante - 1 Chemin du Penthod - 69300 Caluire

W.B. LEADBETTER : The Center for Joint Surgery and Sports Medicine - 13 Western Maryland Parkway - Suite 104 - Hagerstown, Maryland, USA

S. LIPPACHER : Department of Orthopaedics, University of Ulm, Germany - Oberer Eselsberg 45 - 89081 Ulm, Germany

N. LOPOMO : 3rd Orthopaedic and Traumatology Clinic and Biomechanics Laboratory, Codivilla-Putti Research Center - Istituto Ortopedico Rizzoli - University of Bologna

S. LUSTIG : Groupement Hospitalier Lyon Nord - 103, grande rue de la Croix-Rousse - Pav R - 69317 Lyon Cedex 04

R.A. MAGNUSSEN : Department of Orthopaedic Surgery - Sports Health and Performance Institute - The Ohio State University - Columbus, OH, USA

L. MALEKPOUR : Hôpital privé de l'estuaire - 505, rue Irène Joliot-Curie - BP 90011 - 76620 Le Havre

M. MARCACCI : 3rd Orthopaedic and Traumatology Clinic and Biomechanics Laboratory, Codivilla-Putti Research Center - Istituto Ortopedico Rizzoli - University of Bologna

F. MAURIS : Clinique des Cèdres - 21, rue Albert Londres - 38130 Echirolles

C. MAYER : Service de chirurgie orthopédique et traumatologique et médecine du sport - Centre Hospitalier Lyon Sud, Lyon

H. MIGAUD : Service de chirurgie orthopédique et traumatologique et médecine du sport - Centre Hospitalier Lyon Sud, Lyon

D. MONNOT : Centre Orthopédique Paul Santy - 24, avenue Paul Santy - 69008 Lyon

R. MORTATI : Centre Orthopédique Paul Santy - 24, avenue Paul Santy - 69008 Lyon

N. NAKAMURA : Institute for Medical Science in Sports, Osaka Health Science University - 1-9-27, Tenma, Kita-ku, Osaka city, Osaka 530-0043, Japan — Department of Orthopaedics, Osaka University Graduate School of Medicine - 2-2, Yamada-oka, Suita city, Osaka 565-0871, Japan

M. NELITZ : Department of Orthopaedics, University of Ulm, Germany - Oberer Eselsberg 45 - 89081 Ulm, Germany

P. NEYRET : Groupement Hospitalier Lyon Nord - 103, grande rue de la Croix-Rousse - Pav R - 69317 Lyon Cedex 04

P.G. NTAGIOPOULOS : Lyon-Ortho-Clinic ; Clinique de la Sauvegarde - 8, avenue Ben Gourion - 69009 Lyon

M. ODUMENYA : Coventry CV2 2DX - UK

M. OLLIVIER : Institut du mouvement et de l'appareil locomoteur - Centre Hospitalo-Universitaire Sud - Hôpital Sainte-Marguerite - 270, bd de Sainte Marguerite - 13009 Marseille

S. ÖZCAN : Service de Chirurgie orthopédique et traumatologique, Centre Hospitalier Lyon-Sud, Chemin du Grand Revoyet - 69310 Pierre-Bénite

J.C. PANISSET : Clinique des Cèdres - 21, rue Albert Londres - 38130 Echirolles

S. PARRATTE : Institut du mouvement et de l'appareil locomoteur - Centre Hospitalo-Universitaire Sud - Hôpital Sainte-Marguerite - 270, bd de Sainte Marguerite - 13009 Marseille — Service de chirurgie orthopédique et pédiatrique, Hôpital d'enfants de la Timone - 13000 Marseille

G. PASQUIER : Service de chirurgie orthopédique et traumatologique et médecine du sport - Centre Hospitalier Lyon Sud, Lyon

D. PASSERON : Centre Hospitalier de Versailles - F 78150 Le Chesnay

A. PELTIER : Hôpital Femme Mère Enfant - 59 bd Pinel - 69677 Bron cedex

A. PINAROLI : 73190 Challes-les-Eaux

V. PINEAU : U1075 Comete UCBN/INSERM, UFR de Médecine - Département d'Orthopédie Traumatologie - Avenue de la Côte de Nacre - 14032 Caen Cedex

N PUJOL : Centre Hospitalier de Versailles - F 78150 Le Chesnay

N. PUJOL : Centre orthopédique Santy, Lyon

B. QUELARD : Centre Orthopédique Paul Santy - 24, avenue Paul Santy - 69008 Lyon

H. REICHEL : Department of Orthopaedics, University of Ulm, Germany

F. RÉMY : Service de chirurgie orthopédique et traumatologique et médecine du sport - Centre Hospitalier Lyon Sud, Lyon

S. ROCHCONGAR : U1075 Comete UCBN/INSERM, UFR de Médecine - Département d'Orthopédie Traumatologie - Avenue de la Côte de Nacre - 14032 Caen Cedex

P.R.F. SAGGIN : Instituto de Ortopedia e Traumatologia de Passo Fundo - Rua Uruguai 2050 - 99010-111 Passo Fundo RS Brazil

P.B. SCHOETTLE : Gelenkzentrum Zürich

E. SERVIEN : Groupement Hospitalier Lyon Nord - 103, grande rue de la Croix-Rousse - Pav R - 69317 Lyon Cedex 04

B. SHARMA : 3rd Orthopaedic and Traumatology Clinic and Biomechanics Laboratory, Codivilla-Putti Research Center - Istituto Ortopedico Rizzoli - University of Bologna

K. SHINO : Department of Orthopaedics, Osaka University Graduate School of Medicine - 2-2, Yamada-oka, Suita city, Osaka 565-0871, Japan

P. SILLANPÄÄ : Department of Orthopaedic Surgery, Tampere University Hospital, Tampere - Teiskontie 35 - 33521 Tampere

B. SONNERY-COTTET : Centre Orthopédique Paul Santy - 24, avenue Paul Santy - 69008 Lyon

C. TARDIEU : U1075 Comete UCBN/INSERM, UFR de Médecine - Département d'Orthopédie Traumatologie - Avenue de la Côte de Nacre - 14032 Caen Cedex

M. THAUNAT : Centre Orthopédique Paul Santy - 24, avenue Paul Santy - 69008 Lyon — Orthopedic Surgery Department - André Mignot Hospital - 177, rue de Versailles - 78157 Le Chesnay

F. TROUILLET : Groupement Hospitalier Lyon Nord - 103, grande rue de la Croix-Rousse - Pav R - 69317 Lyon Cedex 04

H.P.W. VAN JONBERGEN : Dep. of Orthopedic Surgery, Deventer Hospital, Deventer, The Netherlands

A. VAN KAMPEN : Dep. of Orthopedic Surgery, Radboud University Nijmegen Medical Centre, Nijmegen, The Netherlands

P. VERDONK : Dept. of Orthopaedic Surgery and Traumatology, Ghent University Hospital, Ghent University, De Pintelaan 185, 9000 Gent, Belgium

M. VESELKO : Department of Traumatology, University Medical Center, Ljubljana, Slovenia

J. VICTOR : Dept. of Orthopaedic Surgery and Traumatology, Ghent University Hospital, Ghent University, De Pintelaan 185, 9000 Gent, Belgium

A. VISTE : Centre Hospitalier Lyon-Sud - Chirurgie orthopédique et Traumatologie - Pavillon 3A - Pierre-Bénite

A. WYMENGA : Department for Orthopedic Surgery, Sint Maartenskliniek, Nijmegen, The Netherlands

Y. YERCAN : Hüseyin Serhat - Merkez Efendi Mah Cilek Sok n° 3/4 - 45020 Manisa, Turquie

H. YOSHIKAWA : Department of Orthopaedics, Osaka University Graduate School of Medicine - 2-2, Yamada-oka, Suita city, Osaka 565-0871, Japan

S. ZAFFAGNINI : 3rd Orthopaedic and Traumatology Clinic and Biomechanics Laboratory, Codivilla-Putti Research Center - Istituto Ortopedico Rizzoli - University of Bologna

SOMMAIRE

INSTABILITÉ PATELLAIRE

LA PATELLA ARTHROSIQUE

LE SYSTÈME EXTENSEUR

ANNEXES
LA PATHOLOGIE FÉMORO-PATELLAIRE
(6e Journées Lyonnaise de Chirurgie du Genou)

PHYLOGENÈSE DE LA FÉMORO-PATELLAIRE

C. HULET, C. TARDIEU, V. PINEAU,
S. DELFORGE, I. KLEBANER, S. ROCHCONGAR

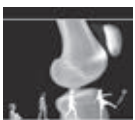
En terme d'évolution, il y a eu deux changements importants des comportements qui permettent de mieux comprendre notre anatomie. Ce sont l'abandon d'un mode de vie arboricole approximativement il y a 6 millions d'années et l'adoption d'une position dite érigée qui est complète depuis environ 3,75 millions d'années. Mieux comprendre l'analyse morpho-fonctionnelle du genou permet de rechercher l'étiologie des dysplasies fémoropatellaires et aussi la prise en charge des instabilités rotuliennes [1, 2, 3]. Pour cela, deux aspects du développement du genou sont intéressants à connaître :

- L'anatomie comparée qui illustre le lien entre forme et fonction au sein des différents groupes d'animaux selon leur mode de déplacement ou de locomotion.
- L'évaluation de cette articulation avec la phylogénie au cours de l'évolution de nos ancêtres "primates récents" avec les différents fossiles car c'est elle la mieux connue. Cela traduit le passage de la quadripédie à la bipédie par les hominidés dont "Lucy" (C. Tardieu [4]).

L'ANATOMIE COMPARÉE [4, 5]

Chez Eryops, l'ancêtre commun des reptiles, oiseaux et mammifères, qui a plus de 320 millions d'années, l'articulation du genou ne comporte pas de rotule. La patella s'est développée seulement il y a 70 millions d'années chez les oiseaux, reptiles et certains mammifères. C'est un développement tardif comparé au développement des condyles fémoraux des ligaments croisés. La salamandre, petit reptile qui se déplace par des mouvements de reptation ne possède pas de rotule. Dans la famille des reptiles, il se produit plusieurs modifications de l'anatomie du genou avec notamment l'apparition de la rotule suite aux contraintes mécaniques subies. Il se développe dans le genou, un noyau fibreux cartilagineux qui sera l'ébauche de la patella. Ainsi, le lézard et le varan ont une patella cartilagineuse et l'intensité de la contrainte subie a une incidence sur le développement de cette structure. La rotule de la lente tortue terrestre est absente et chez le crocodile, elle est très puissante.

Chez les mammifères, l'anatomie du genou est assez rudimentaire avec 2 balles rigides qui ont très peu de contact avec les glènes tibiales. Ce sont les ligaments et les ménisques qui stabilisent le tout avec des points d'insertion rapprochés pour éviter des mouvements trop importants. La forme de la fémoro-patellaire est très variable et dépend du mode de locomotion. Tardieu et Dupont [5] ont bien précisé ces différences de forme anatomique en fonction du type de déplacement chez les quadripèdes. Chez le cheval dans la famille des onguligrades, le genou est toujours placé en flexion et ne



connaît jamais l'extension complète. Il n'existe pas de continuité entre la trochlée fémorale et les condyles (fig. 1). Les chevaux dorment debout et la forme du membre inférieur est adaptée à la course avec des mouvements guidés et rapides. Les caractéristiques du genou des Digitigrade et des Plantigrades sont dans le tableau 1 et illustrées par la figure 1.

Il existe donc des variations importantes morpho fonctionnelles sensibles chez les mammifères avec parfois deux articulations dans le genou en fonction du degré d'extension atteint. En fonction de la forme de la trochlée, il est possible de dire comment marche le mammifère.

Les différentes formes de l'épiphyse distale fémorale chez les primates (Tardieu [4]) et chez différents fossiles expliquent le passage de la quadripédie à la bipédie qui est le mode de locomotion unique adopté par l'homme adulte. Valois a montré sur l'étude de l'anatomie des primates en 1913 que la morphologie de la fémoro-patellaire est dépendante du mode de locomotion. Chez tous les primates, il existe une zone de continuité entre la trochlée fémorale et les condyles fémoraux. Sur le plan de la locomotion, ils sont à la fois bipèdes et quadrupèdes. Ils sont à la fois arboricoles et terrestres.

Plus le déplacement est rapide avec un primate sauteur et léger, plus les surfaces articulaires sont étroites, allongées et creusées. La rotule est guidée et enclenchée dans la trochlée. Plus le déplacement est lent chez un primate lourd, plus la rotule et la trochlée sont plates.

A partir des différents singes : les cercopithèques (quadrupèdes arboricoles), les grands singes (chimpanzé, gorille, orang-outan, grimpeurs arboricoles), l'homme, le seul primate devenu exclusivement bipède, on peut réaliser une étude comparative sur l'ostéologie fémorale métaphysaire inférieure.

Quatre caractères osseux sont essentiels pour comprendre l'anatomie fonctionnelle du genou liée à la bipédie :

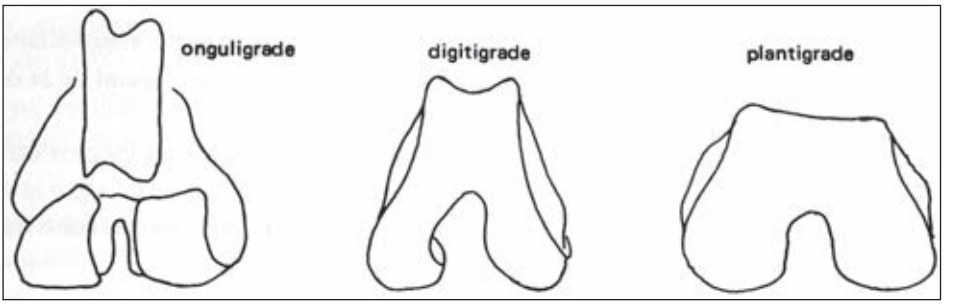
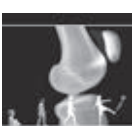


Fig. 1 : Epiphyse inférieure d'un fémur gauche d'après Tardieu [4].

Tableau 1 : Caractéristiques du genou chez différents mammifères [4, 5].

Onguligrade (Cheval)	Digitigrade (Panthère)	Plantigrade (Ours)
Trochlée étroite creusée	Trochlée plus étroite et plus creusée	Trochlée plate large sans rebord
Sillon profond entre les 2 lèvres	Rebords légèrements saillants	Mouvements de rotation et extension complète possible
Rebords saillants qui forment des bourrelets	Continuité entre la trochlée et les condyles	Continuité entre la trochlée et les condyles
Trochlée séparée complètement des condyles	Zone de transition resserrée mais encore assez large	Zone de transition plus faible



- obliquité diaphyse fémorale par rapport au plan infracondylaire,
- relief de la trochlée fémorale latérale avec surélévation latérale propre à l'homme,
- profil latéral du condyle latéral du genou,
- forme de l'épiphyse dans le plan horizontal.

L'amplitude du mouvement de rotation combinée est faible chez les cerpicothèques et importante chez les grands singes mais reste faible chez l'homme.

La saillie du rebord externe de la trochlée existe déjà à la naissance et elle est variable. Il n'a pas été retrouvé de corrélation entre le degré d'obliquité et l'importance de la saillie externe. La saillie de la berge externe de la trochlée se développe ensuite au cours de la croissance alors que la trochlée est définitivement plate chez le primate (fig. 2 à droite)

Dans une autre approche, Lovejoy [6] a comparé l'anatomie du chimpanzé et de l'homme (fig. 3 et 4).

Chez le chimpanzé qui est toujours en flexion, le quadriceps permet une compression FP nécessaire à la stabilité du genou. A l'inverse, chez l'homme en extension la contraction quadricipitale maintient une contrainte fémoro-tibiale nécessaire à la stabilité.

La forme de la patella a une certaine importance. Chez l'homme elle a deux facettes pour s'adapter successivement au relief fémoral et méniscal. Cette forme réduit le contact FP quand le genou est en flexion importante mais donnerait des contraintes FP trop importantes si la surface patellaire était trop uniforme. La rotule humaine est "multifacettes" avec une crête médiane bien caractéristique. Cette crête

Tableau 2 : Variations des caractères osseux chez les primates et l'homme [4].

Caractères osseux	Cerpicothèques Quadrupède	Grands singes Quadrupède et bipède	Homme Bipède
Obliquité diaphyse fémorale	Nulle	Nulle Genoux abductés	Elevé 5° à 15° Genoux adductés
Relief de la trochlée fémorale	Symétrique très peu creusée	Symétrique, plate Rotule monofacette	Sillon profond et lèvre latérale plus haute Rotule deux facettes
Forme condyle latéral	Circulaire	Circulaire	Elliptique
Forme épiphyse	Condyle interne plus large que latéral Epiphyse aussi longue que large	Condyle interne plus large que latéral Elargissement médio-latéral de l'épiphyse (Rectangle)	Condyle externe plus large que médial Allongement antéro-postérieur d'épiphyse (Carré)

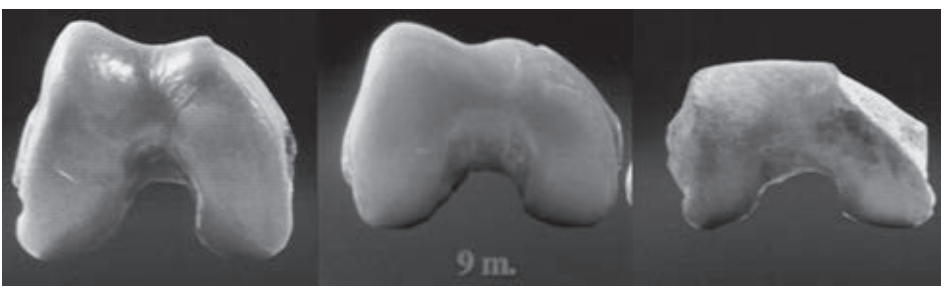


Fig. 2 : Forme de la trochlée fémorale chez un homme adulte, un fœtus de 9 mois et chez le chimpanzé d'après Tardieu [4]

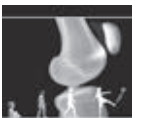


Fig. 3 : Analyse comparative de la forme de la patella d'après Owen Lovejoy [6].

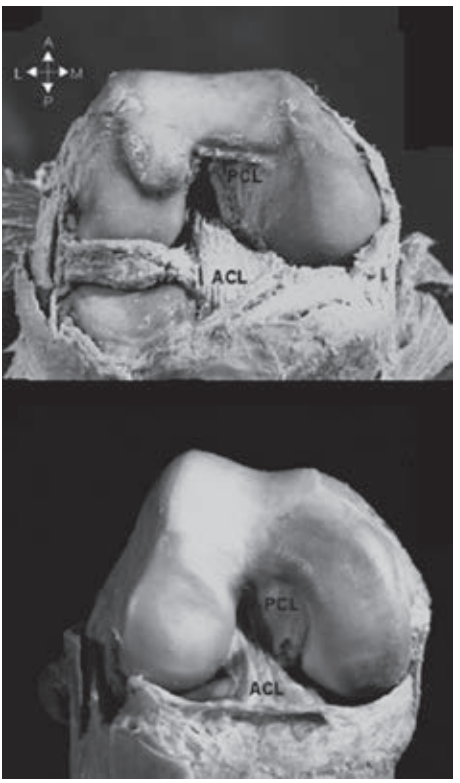


Fig. 4 : Anatomie comparée du genou du singe (en haut) et de l'homme (en bas) d'après Owen Lovejoy [6].

médiane permet une bonne adaptation à la gorge trochléenne et limite les risques de luxation. Il considère ainsi que le genou humain est tibial dominant.

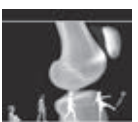
A l'inverse, la forme de la patella du gorille est plus simple, moins divisée et plus uniforme pour s'adapter au fémur pendant toute l'amplitude de la flexion. La rotule du chimpanzé est plus discoïde avec une seule surface articulaire.

Ceci permet une forte congruence FP pendant la phase de propulsion et de contact au sol. Cela réduit d'autant le stress en flexion. Le genou du chimpanzé est patella dominant.

Les dissections anatomiques de Lovejoy montrent bien les différences entre les 2 genoux :
- volume plus important du LCA chez le chimpanzé,
- la forme du condyle médial avec cette bosse spécifique chez l'homme et ce volume très important chez le singe,
- la différence de hauteur du condyle latéral avec la saillie externe plus importante,
- l'insertion du ménisque latéral qui est différente avec une insertion unique chez le singe [7].

CONCLUSION

L'anatomie comparée du genou entre les différentes espèces et l'évolution de la forme du genou entre les primates et l'espèce humaine est révélatrice de l'adaptation entre la forme et la fonction. La bipédie est un caractère unique de l'espèce humaine qui s'accompagne de changements anatomiques importants. Cela se traduit aussi par une autre caractéristique qui est l'extension complète et bloquée de l'articulation avec la rotation externe automatique en extension. L'utilisation du membre inférieur tendu en plus en permanence en flexion, donne au cours du déplacement une efficacité plus importante. Le genou de l'homme moderne bénéficie des avantages d'une longue histoire de la sélection naturelle agissant sur sa fonction devant habituellement marcher debout et courir.



Ces adaptations concernent 4 éléments importants : 1) une position distincte en valgus au cours la phase d'appui ; 2) un mécanisme génomique spécifié de la rotule de rétention réalisé par l'élévation de la lèvre du condyle latéral ; 3) un allongement et des changements dans la forme de deux condyles fémoraux qui fournissent la domination du tibia ; 4) prolongements antérieurs des deux condyles qui réduisent la force du quadriceps nécessaire pendant l'extension du genou. Trois de ces adaptations étaient déjà présentes dans les espèces ancestrales, A. Afarensis dit "Lucy".

LITTÉRATURE

[1] DEJOUR H, WALCH G, NEYRET P, ADELEINE P. Dysplasia of the femoral trochlea. *Rev Chir Orthop 1990;76: 45-54.*

[2] DEJOUR D, NOVE-JOSSERAND L, WALCH G. Patellofemoral disorders-classification and an approach to operative treatment for instability. In: Chan KM *et al.*, editor. Controversies in orthopedic sports medicine. *Hong Kong: Williams & Wilkins, 1998.*

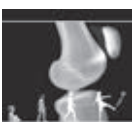
[3] SERVIEN E, AÏT SI SELMI T, NEYRET P. Subjective evaluation of surgical treatment for patellar instability. *Rev Chir Orthop 2004; 90: 137-42.*

[4] TARDIEU C. L'articulation du genou. Analyse morpho-fonctionnelle chez les Primates. Application aux Hominide's fossiles. 1983. *Paris: CNRS Editions.*

[5] TARDIEU C, DUPONT JY. The origin of femoral trochlear dysplasia: comparative anatomy, evolution, and growth of the patellofemoral joint. *Rev Chir Orthop Reparatrice Appar Mot. 2001 Jun; 87(4): 373-83.*

[6] OWEN-LOVEJOY C. The natural history of human gait and posture Part 3. *The knee Gait & Posture 25 (2007) 325-41.*

[7] JAVOIS C, TARDIEU C, LEBEL B, SEIL R, HULET C ET SOCIÉTÉ FRANÇAISE D'ARTHROSCOPIE. Comparative anatomy of the knee joint: effects on the lateral meniscus. *Orthop Traumatol Surg Res. 2009 Dec; 95(8 Suppl 1): S49-59.*



BASES EMBRYOLOGIQUES DE L'INSTABILITÉ PATELLAIRE

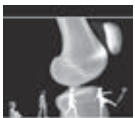
J.L. JOUVE, Y. GLARD, S. PARRATTE

La trochlée fémorale est creusée en son milieu, asymétrique, avec une berge externe plus saillante. L'engagement de la rotule sur une trochlée de plus en plus profonde et asymétrique semble être une spécificité de l'homme moderne. Cet élément est essentiel dans l'acquisition de la bipédie permanente. Néanmoins il existe une grande variabilité individuelle des caractéristiques anatomiques de la trochlée fémorale chez l'homme. La question se pose quant aux paramètres interférents dans la forme de la trochlée adulte.

Nous avons réalisé une étude biométrique sur une série de fémurs fœtaux et comparé nos résultats avec une série d'os secs squelettiques adultes déjà publiés [1]. Ce travail constitue la première évaluation biométrique statistiquement significative de la croissance fémorale durant la vie fœtale. Il vise à établir si durant les stades fœtaux précoces la morphologie des extrémités osseuses est fixée ou si au contraire elle se modifie progressivement jusqu'à atteindre la morphologie adulte. Ainsi l'observation d'un nombre significatif de sujets présentant une trochlée creusée dès les stades précoces du développement fœtal plaide en faveur de l'influence de facteurs génétiques progressivement apparus au cours de l'évolution et non de seuls facteurs mécaniques dus à l'acquisition de la marche [2, 3, 4]. Cette étude peut également permettre de mieux comprendre la genèse de certains morphotypes prédisposant à la luxation ou l'instabilité rotulienne, pathologie encore mal comprise et donnant lieu à des divergences thérapeutiques importantes.

Notre travail a consisté à réaliser une biométrie de la trochlée fémorale dans une population de fœtus des deuxième et troisième trimestres. Concernant la méthodologie, la méthode anatomique que nous avons utilisée est la même que celle utilisée dans la série de référence adulte. Les mesures ont été réalisées grâce à la réalisation des photographies numérisées selon un protocole unique. Le repérage sur ordinateur des points de référence s'est fait de façon manuelle et peut être à l'origine de biais. En revanche, la mesure informatisée est peu susceptible d'être à l'origine d'erreurs. Nous avons montré que l'outil informatique de mesure des angles et des distances était fiable et qu'il existait une bonne reproductibilité et une bonne répétabilité de la méthode de mesure.

La biométrie de la trochlée fémorale que nous avons effectuée montre des résultats similaires à ceux observés chez l'adulte. On peut dire que les 3 angles utilisés caractérisant la forme de la trochlée fémorale (Alpha, ThétaL et ThétaM)



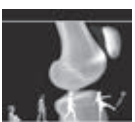
ne varient pas entre les premiers stades du développement fœtal et le stade adulte. Ceci plaide en faveur d'un déterminisme génétique de la forme de la trochlée fémorale. Malgré leur lien fonctionnel évident, l'obliquité fémorale et la trochlée fémorale ne se développeraient donc pas selon le même processus ontogénétique, puisque l'obliquité fémorale est déterminée par les contraintes mécaniques liées à la bipédie alors que la trochlée fémorale fœtale semble génétiquement déterminée.

Un autre argument en faveur du déterminisme génétique de la forme de la trochlée fémorale est donné par l'étude des formes familiales de luxation récidivante de la rotule. Les facteurs de stabilité de la rotule sont de 3 types : osseux, musculaires et capsulo-ligamentaires. Parmi les facteurs osseux, la dysplasie trochléenne est de loin le facteur le plus fréquent pouvant être la cause d'une instabilité rotulienne. L'origine génétique de la dysplasie trochléenne avait déjà été évoquée par Dejour et Walch [5] devant la fréquence des atteintes mère-fille ou frère-sœur et la bilatéralité constante de cette anomalie.

D'un point de vue anthropologique, on peut remettre l'ensemble de ces constatations dans une perspective darwinienne : en se mettant debout, l'homme impose à ses fémurs une contrainte mécanique qui les rend obliques. Une trochlée fémorale asymétrique confère un avantage sélectif aux individus qui la possèdent en luttant efficacement contre la luxation latérale de la rotule. Cette trochlée asymétrique a donc été sélectionnée et s'est imposée au cours des générations. C'est pourquoi on la retrouve chez l'homme moderne dès les stades fœtaux les plus précoces, comme un témoin de son déterminisme génétique [6].

Cependant, une telle forme de déterminisme génétique semble être l'apanage d'éléments anatomiques importants dans l'acquisition d'une fonction bipède. Dans un autre travail, nous avons effectué une biométrie de l'extrémité supérieure des mêmes pièces fémorales afin de déterminer l'antéversion et l'angle cervico-diaphysaire [7]. Les résultats sont radicalement différents que pour le fémur distal puisqu'ils mettent en évidence une modification régulière de la morphologie fémorale au cours du développement fœtal. Concernant l'angle cervico-diaphysaire, on note une diminution progressive de la valgisation fémorale tout au long de la vie fœtale et qui se continue après la naissance [8]. En revanche, l'antéversion fémorale augmente progressivement durant la vie fœtale pour atteindre un maximum lors de la naissance puis diminuer à nouveau jusque vers l'âge de 7 ans.

Il semble donc, à la lumière de ces travaux, que certains éléments de l'anatomie fœtale soient soumis à des contraintes mécaniques prédominantes sur le déterminisme génétique. Ainsi, leur morphologie est dictée par l'importance de ces contraintes. C'est le cas de l'antéversion fémorale dont l'augmentation progressive *in utero* pourrait s'expliquer par la mise en flexion plus importante des hanches au cours de la grossesse puis la mise en extension de celles-ci après l'accouchement. Cette hypothèse avait été formulée en ces termes dès 1912 par Le Damany [9] et semble se confirmer à l'occasion de nos travaux. En revanche, d'autres éléments ont une morphologie fixée génétiquement dès les premiers stades de la vie fœtale. Ils ne varient pas sauf pathologie particulière (neurologique, chromosomique). C'est le cas de la trochlée dont la profondeur est l'élément clef de la bipédie permanente. Cette condition va plaider en faveur d'une réparation chirurgicale précoce du défaut de trochlée notamment les trochlées plates. En revanche, des défauts d'antéversion fémorale susceptibles de correction durant l'enfance avec le temps et donc doivent faire considérer toute chirurgie réparatrice avec réserve.



LITTÉRATURE

[1] WANNER JA Variations in the anterior patellar groove of the human femur. *Am J Phys Anthrop., 1977; 47: 99-102.*

[2] GARRON E, JOUVE JL, TARDIEU C, PANUEL M, DUTOUR O, BOLLINI G. Etude anatomique du creusement de la trochlée fémorale chez le fœtus. *Rev Chir Orthop, 2003; 89: 407-12.*

[3] GLARD Y, JOUVE JL, GARRON E, ADALIAN P, TARDIEU C, BOLLINI G. Anatomic study of femoral patellar groove in fetus, *J Pediatr Orthop. 2005; 25: 305-8.*

[4] GLARD Y, JOUVE JL, PANUEL M, ADALIAN P, TARDIEU C, BOLLINI G. An anatomical and biometrical study of the femoral trochlear groove in the human foetus. *J Anat, 2005; 25: 305-8.*

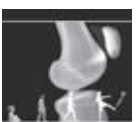
[5] DEJOUR H, WALCH G. La dysplasie de la trochlée fémorale. *Rev Chir Orthop 1990; 76: 45-54.*

[6] TARDIEU C, DUPONT JY. Evolution et croissance de l'articulation fémoro-patellaire. Origine des dysplasies de la trochlée fémorale: anatomie comparée, 383. *Rev Chir Orthop, 2001; 87: 373.*

[7] JOUVE JL, GLARD Y, GARRON E, PIERCECCHI MD, DUTOUR O, TARDIEU C, BOLLINI G. Anatomical study of the proximal femur in the foetus. *J Pedatr orthop B, 2005; 14: 105-10.*

[8] TARDIEU C, GLARD Y, GARRON E, BOULAY C, JOUVE JL, DUTOUR O, BOETSCH G, BOLLINI G. Relationship between formation of the femoral bicondylar angle and trochlear shape: independence of diaphyseal and epiphyseal growth. *Am J Phys Anthropol. 2006; 13: 491-500.*

[9] LE DAMANY P. La luxation congénitale de hanche, *1912 ; Paris : Felix Alcan.*



IMAGING IN PATELLOFEMORAL INSTABILITY

P.R.F. SAGGIN, P. FERRUA,
P.G. NTAGIOPOULOS, D. DEJOUR

Imaging in patellofemoral instability must identify the 4 classic factors implied in the genesis of the instability – trochlear dysplasia, patella alta, abnormal tibial tubercle – trochlear groove distance (TT-TG) and patellar tilt (excessive patellar tilt with medial ligamentous disruption) – specially in the chronic setting. In the acute cases, imaging is sometimes the only element to provide the diagnosis.

TROCHLEAR DYSPLASIA

Trochlear dysplasia is the most important factor implied in the genesis of patellar instability [1] since the femoral sulcus is not sufficient to provide the osseous restraint to the patella.

Standard lateral X-ray films with perfect superimposition of the posterior medial and lateral femoral condyles are the key to the diagnosis of dysplasia. The crossing sign is typically found in this projection and represents the point where trochlea becomes flat (the bottom of the groove reaches the height of the facets) [1, 2]. Additional findings include the double-contour sign (representing the hypoplastic medial facet found posterior to the lateral one) and the supratrochlear spur (found in the superolateral aspect of the trochlea) [3, 4] (fig. 1).

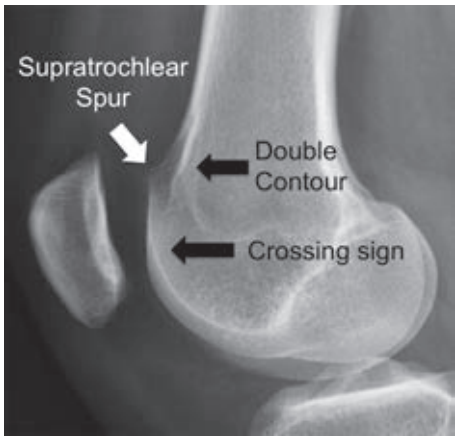
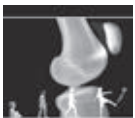


Fig. 1: Trochlear dysplasia features on the lateral view.

Axial X-ray views performed in 45° of knee flexion allow the measurement of the sulcus angle [5]. Normal mean value is 138° (SD±6) [6]. Angles above 150° are found in trochlear dysplasia. An important issue when analyzing axial views is that X-rays obtained with higher flexion angles image the lower part of the trochlea, frequently missing the dysplasia present in its upper portion [7]. For this reason, images obtained at 30° of flexion are preferred.



CT and MRI have the ability to image the entire trochlea in sequential cuts, allowing better visualization of the dysplastic upper part. Frequently, the dysplasia found in these modalities is missed on X-ray axial views. Comparing CT and MRI images, bony and cartilaginous trochlear anatomy do not match perfectly [8], but this looses importance in the dysplasia setting, where flat or convex bone will be covered by analogous cartilage.

Carillon *et al.* [9] investigated the lateral trochlear inclination angle on axial MRI cuts (LTI, calculated by means of a line tangential to the subchondral bone of the posterior aspect of the two femoral condyles crossed with a line tangential to the subchondral bone of the lateral trochlear facet) in healthy and patellar instability patients. A significant difference between groups was recorded. The mean value in patellar instability patients was 6.17° while in the control group it was 16.9°. Choosing 11° as the threshold value for LTI, results were excellent in discrimination between the two groups with sensitivity of 93%, specificity of 87% and an accuracy of 90%.

Based primarily on X-ray lateral views, and helped by CT or MRI axial cuts, one can classify trochlear dysplasia in four types (D. Dejour) [3, 4, 10]:

- ***Type A:*** presence of crossing sign in lateral true view. The trochlea is shallower than normal, but still symmetric and concave.
- ***Type B:*** crossing sign and trochlear spur. The trochlea is flat or convex in axial images.
- ***Type C:*** presence of crossing sign and the double-contour sign on the lateral view, representing the medial hypoplasic facet. There is no spur, and in axial views, the lateral facet is convex and the medial hypoplasic.
- ***Type D:*** crossing sign, supratrochlear spur and double-contour sign. In axial views, there is clear asymmetry of the facets height, also referred as a cliff pattern.

Lippacher *et al.* [11] analyzed intraobserver and interobserver agreements of radiographic and MRI-based D. Dejour's classification. They concluded that better overall agreement was found for a 2-grade analysis on MRI scans (type A, low grade, versus types B, C and D combined, representing high grade dysplasia), and that lateral radiographs tended to underestimate the severity of dysplasia compared with axial MRI views.

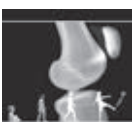
PATELLA ALTA (PATELLAR HEIGHT)

Patella alta refers to an abnormally high riding patella that engages the trochlear groove later in flexion, increasing the patellar "free" arch of movement and facilitating dislocation.

As in trochlear dysplasia, X-ray lateral views are the key to the diagnosis of the patellar height. Several methods of measurement (and diagnosis) using the tibia as reference have been described, the three main are (fig. 5):

- *Caton and Deschamps* [12]: is the ratio between the distance from the lower edge of the patellar articular surface to the anterosuperior angle of the tibia outline (AT), and the length of the articular surface of the patella (AP). A ratio (AT/AP) of 0.6 and smaller determines patella infera, and a ratio greater than 1.2 indicates patella alta.
- *Insall and Salvatti* [14]: is the ratio between the length of the patellar tendon (LT) and the longest sagittal diameter of the patella (LP). Insall determined that this ratio (LT/LP) is normally 1. A ratio smaller than 0.8 indicates a patella infera and greater than 1.2 patella alta.
- *Blackburne-Peel* [15]: is the ratio between the length of the perpendicular line drawn from the tangent to the tibial plateau until the inferior pole of the articular surface of the patella (A) and the length of the articular surface of the patella (B). The normal ratio (A/B) was defined as 0.8. In patella infera it is smaller than 0.5, in patella alta greater than 1.0. (fig. 2).

Patellar height using the tibia as reference can also be measured on MRI. Miller *et al.* [16] applied the Insall-Salvati method to 46 knees comparing MRI and radiographs. Good-to-excellent correlation between the values was found, and they concluded that patellar height can be reliably assessed on sagittal MR imaging



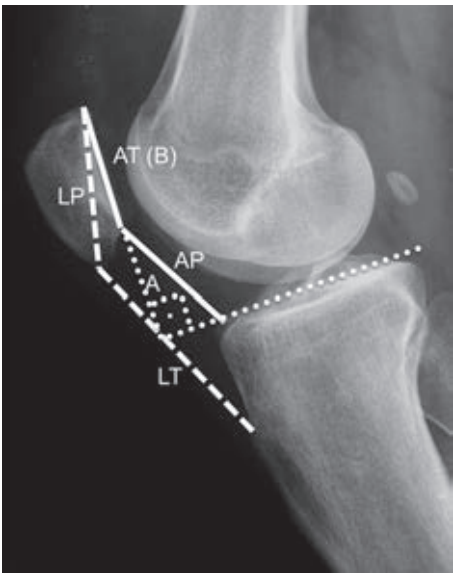


Fig. 2: Patellar height measurement methods (using the tibia as reference) on the lateral view. Caton and Deschamps (AT/AP), Insall and Salvatti (LT/LP) and Blackburne-Peel (A/B).

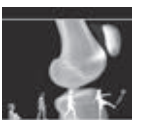
using the patellar tendon: patella ratio. On sagittal MR imaging, patella alta is suggested at values greater than 1.3. Neyret *et al.* [17] measured with radiographies and MRI the patellar tendon length in 42 knees with history of patellar dislocation and 51 control knees. They concluded that patella alta is caused by a long patellar tendon rather than by its abnormal insertion into the tibia. Additionally, they did not found any significant difference between X-ray and MRI tendon length measurements.

Other methods of measuring patellar height use the trochlea as the reference. Bernageau [18] described a method on lateral X-rays with the knee in extension and the quadriceps contracted. If the inferior edge of the articular surface of the patella (R) is more than 6mm above the superior limit of the trochlea (T) there is patella alta, and if R is more than 6mm beneath T there is patella baja. Biedert and Albrecht [19] described the patellotrochlear index on sagittal cuts of MRI, performed with the knees in extension, the foot 15° externally rotated and the quadriceps consciously relaxed. To calculate the index, first we must measure the length of the articular cartilage of the patella (baseline patella: BLp). The second measure is the length from the trochlear most superior aspect to the most inferior part of the trochlea facing the patellar articular cartilage (BLt). The ratio BLt/BLp is calculated in percentages, and values above 50% indicate patella baja while values inferior to 12,5% indicate patella alta.

TIBIAL TUBERCLE – TROCHLEAR GROOVE DISTANCE (AND TORSIONAL MEASURES)

TT-TG is a simple way to measure the valgus (lateralizing) forces acting on the patella. This distance is able to quantify the coronal alignment of the extensor mechanism, or what is called in clinical evaluation the "Q-angle". Originally, TT-TG has been described by Goutallier and Bernageau [20] on X-ray axial views, but it was popularized as the distance from the bottom of the most proximal part of the trochlear groove to proximal part of the tibial tubercle, measured with two CT superimposed cuts and expressed in millimeters [1]. The first cut (reference cut) is through the proximal trochlea, identified by a slight condensation of the lateral facet and by the shape of the notch (rounded, looks like a roman arch). The second cut goes through the proximal part of the tibial tubercle. The deepest point of the trochlear groove and the central point of the tibial tubercle are projected on a line tangential to the posterior femoral condyles and the distance from each other is measured. The normal mean value in a control population is 12mm; in the population with objective patellar dislocation the value is superior to 20mm in 56% of the cases [1]. Values above 20mm are considered abnormal.

Another important contribution of CT produced by the superimposition of images is the assessment of torsional deformities, such as femoral anteversion and external tibial torsion. Femoral anteversion is increased in patients with instability (15,6±9 vs. 10,8±8,7 in normal



knees), although some overhang of values may exist. Combined with tilt and TT-TG, these constitute the Lyon protocol for CT analysis [1] (fig. 3).

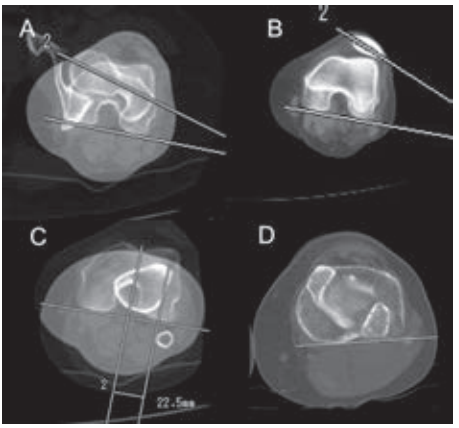


Fig. 3: The Lyon Protocol for CT Scan analysis. A: femoral anteversion; B: external patellar tilt; C: tibial tubercle-trochlear groove distance (TT-TG); D: external tibial torsion.

Schoettle *et al.* [21] evaluated the reliability of the TT-TG on MRI compared to CT scan in 12 knees with patellofemoral instability or anterior knee pain. The mean TT-TG referenced on bony landmarks was 14.4±5.4mm on CT scans, and 13.9±4.5mm on MR images. The mean TT-TG referenced on cartilaginous landmarks was 15.3±4.1mm on CT scans, and 13.5±4.6mm on MR images. They found excellent interperiod (bony vs. cartilaginous TT-TG), and intermethods (CT vs. MRI measurement) reliabilities, 91 and 86% respectively.

PATELLAR TILT (AND SUBLUXATION)

Patellar tilt and subluxation refers to the abnormal position of the patella in relation to the trochlear groove. While tilt means increased lateral inclination of the transverse diameter of the patella, subluxation refers primarily to abnormal mediolateral displacement of the patella in relation to the trochlea. Whether cause or consequence of instability, they must be considered for diagnosis and adequate treatment of instability.

On the lateral view, the shape of the patella is dependent on its tilt. Normally, the lateral facet is anterior to the crest. Mild tilt occurs when both lines (lateral facet and crest) are superimposed, and severe tilt is when the crest is anterior to the lateral facet [2].

Methods of evaluating tilt and subluxation have been described for x-rays axial views:

1) The congruence angle is measured on X-rays at 45° of knee flexion. After measuring the sulcus angle (used to access trochlear shape), two other lines are drawn from its vertex: one bisecting the sulcus angle (reference line) and another to the apex of the patella. The angle between these two lines is the congruence angle, considered positive if the line to the patellar apex is lateral to the reference line. Average congruence angle is -6° (SD±11°), and measures primarily subluxation [6] (fig. 4).

2) The lateral patellofemoral angle is formed by one line connecting the highest points of the medial and lateral facets of the trochlea and another tangent to the lateral facet of the patella, drawn on 20° of knee flexion axial views (Laurin). In normal knees this angle should open laterally (except in 3% in which it is parallel). It demonstrates primarily tilt [22, 23].

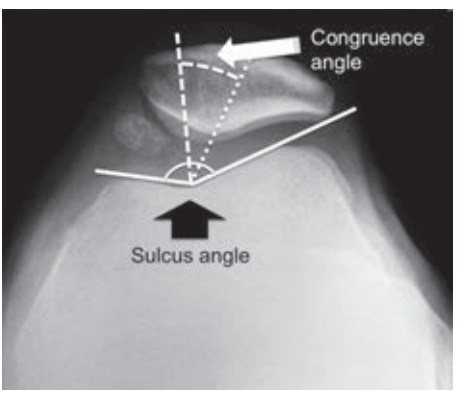


Fig. 4: The sulcus and the congruence angles.

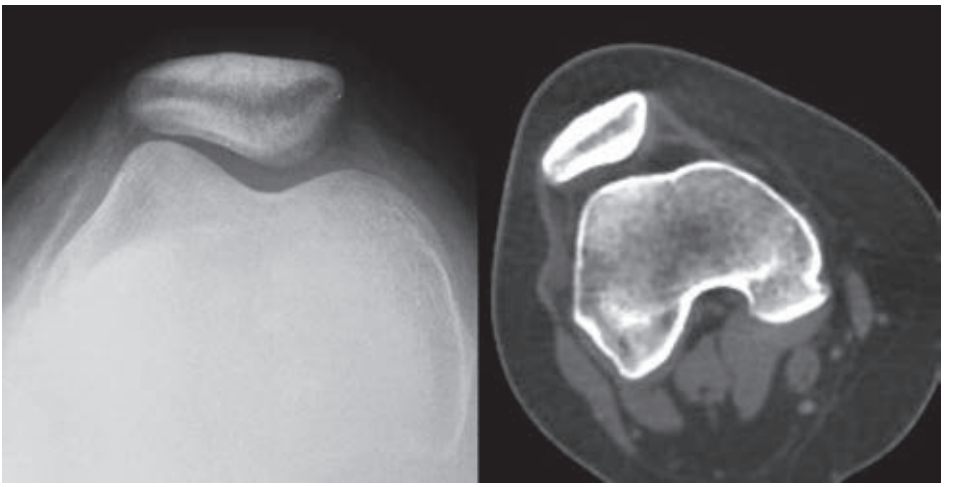


Fig. 5: Patellar tilt and trochlear dysplasia were clearly depicted on axial CT imaging (at full extension), but missed on the axial X-ray.

3) The patellofemoral index is the ratio (M/L) between the thickness of the medial joint space (M) and the lateral joint space (L), measured on 20° axial views (Laurin). It should measure 1.6 or less [22, 23].

CT scans allow tilt measurements in complete extension, which increases sensitivity because as the knee flexes, trochlear engagement of the patella reduces or corrects tilt and subluxation (fig. 5). Another important contribution of CT scans is that they allow tilt measurements to be performed with a constant reference – the posterior femoral condyles (versus the variable trochlear shape in the instability population observed in X-rays). According to Lyon's Protocol [1] patellar tilt is the angle formed by the transverse axis of the patella and a tangent to the posterior femoral condyles. It must be measured with and without quadriceps contraction, and this can be accomplished either with two superimposed cuts or with a single cut that images both references. Values above 20° are considered abnormal.

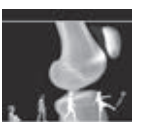
In H. Dejour's study, 83% of the objective patellar dislocation group have patellar tilt superior to 20° compared to 3% in the reference normal group. If instead of using only the relaxed quadriceps measure a mean is calculated between the measures performed relaxed and in contraction, and the threshold value remain the same, sensitivity and specificity are improved. Ninety percent of the objective patellar dislocation population have presented values over this, while the same remains true for only 3% of controls [1].

ACUTE DISLOCATIONS

Imaging of acute dislocations is useful to confirm the diagnosis and define treatment.

X-rays are useful to identify gross instability and incongruence. Antero-posterior, lateral and axial views are complementary and must be performed. Fragments of the patella or the lateral femoral condyle can be identified after acute dislocations and may indicate surgical treatment. CT findings in acute dislocations are similar to the X-ray ones, but with increased accuracy. Smaller osseous fragments can be identified and better measured.

Patellar dislocation may not be suspected before MRI examination in up to 50% of cases [24]. MR imaging is particularly helpful in



acute dislocations recognition and evaluation of associated lesions. The acute findings include [25-28] (fig. 6):

- Lateral femoral condyle contusion and⁄or osteochondral lesion;
- Medial patellar facet contusion and⁄or osteochondral lesion, sometimes with osteochondral fragment avulsion;
- Injury of the medial retinaculum at its patellar attachments or mid-substance; Tearing of the distal belly of the *vastus medialis obliquus*;
- Injury of the medial patellofemoral ligament at its femoral origin;
- Patellar tilt and subluxation;
- Joint effusion.

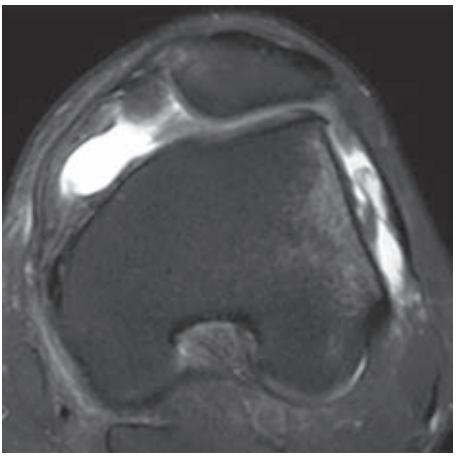


Fig. 6: MRI performed after acute dislocation. The medial retinaculum is torn and contusions of the medial patellar facet and lateral condyle are evident.

LITERATURE

[1] DEJOUR H, WALCH G, NOVE-JOSSERAND L, GUIER C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc. 1994; 2(1): 19-26.*

[2] MALDAGUE B, MALGHEM J. Significance of the radiograph of the knee profile in the detection of patellar instability. Preliminary report. *Rev Chir Orthop Reparatrice Appar Mot. 1985;71 Suppl 2: 5-13.*

[3] DEJOUR D, REYNAUD P, LECOULTRE B. Douleurs et instabilité rotulienne, Essai de Classification. *Médecin et Hygiène. Juillet. 1998; 1466-71.*

[4] DEJOUR D, SAGGIN P. The sulcus deepening trochleoplasty-the Lyon's procedure. *Int Orthop. 2010 fev; 34(2): 311-6.*

[5] BRATTSTROEM H. Shape of the Intercondylar Groove Normally and In Recurrent Dislocation of Patella. A Clinical And X-Ray-Anatomical Investigation. *Acta Orthop Scand Suppl. 1964;68: Suppl 68: 1-148.*

[6] MERCHANT AC, MERCER RL, JACOBSEN RH, COOL CR. Roentgenographic analysis of patellofemoral congruence. *J Bone Joint Surg Am. 1974 out; 56(7): 1391-6.*

[7] DAVIES AP, BAYER J, OWEN-JOHNSON S, SHEPSTONE L, DARRAH C, GLASGOW MM, *et al.* The optimum knee flexion angle for skyline radiography is thirty degrees. *Clin Orthop Relat Res 2004 jun;(423): 166-71.*

[8] STÄUBLI HU, DÜRRENMATT U, PORCELLINI B, RAUSCHNING W. Anatomy and surface geometry of the patellofemoral joint in the axial plane. *J Bone Joint Surg Br. 1999 maio; 81(3): 452-8.*

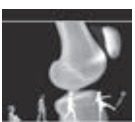
[9] CARRILLON Y, ABIDI H, DEJOUR D, FANTINO O, MOYEN B, TRAN-MINH VA. Patellar instability: assessment on MR images by measuring the lateral trochlear inclination-initial experience. *Radiology 2000 ago; 216(2): 582-5.*

[10] DEJOUR D, LE COULTRE B. Osteotomies in patello-femoral instabilities. *Sports Med Arthrosc 2007 mar;15(1): 39-46.*

[11] LIPPACHER S, DEJOUR D, ELSHARKAWI M, DORNACHER D, RING C, DREYHAUPT J, *et al.* Observer Agreement on the Dejour Trochlear Dysplasia Classification: A Comparison of True Lateral Radiographs and Axial Magnetic Resonance Images. *The American Journal of Sports Medicine [Internet]. 2012 jan 11 [citado 2012 fev 12]; Available de: http://www.ncbi.nlm.nih.gov/pubmed/22238057.*

[12] CATON J. Method of measuring the height of the patella. *Acta Orthop Belg. 1989; 55(3): 385-6.*

[13] CATON J, DESCHAMPS G, CHAMBAT P, LERAT JL, DEJOUR H. Patella infera. Apropos of 128 cases. *Rev Chir Orthop Reparatrice Appar Mot. 1982; 68(5): 317-25.*



[14] INSALL J, SALVATI E. Patella position in the normal knee joint. *Radiology. 1971 out; 101(1): 101-4.*

[15] BLACKBURNE JS, PEEL TE. A new method of measuring patellar height. *J Bone Joint Surg Br. 1977 maio; 59(2): 241-2.*

[16] MILLER TT, STARON RB, FELDMAN F. Patellar height on sagittal MR imaging of the knee. *AJR Am J Roentgenol. 1996 ago; 167(2): 339-41.*

[17] NEYRET P, ROBINSON AHN, LE COULTRE B, LAPRA C, CHAMBAT P. Patellar tendon length- the factor in patellar instability? *Knee. 2002 fev; 9(1): 3-6.*

[18] BERNAGEAU J, GOUTALLIER D, DEBEYRE J, FERRANÉ J. New exploration technic of the patellofemoral joint. Relaxed axial quadriceps and contracted quadriceps. *Rev Chir Orthop Reparatrice Appar Mot. 1975;61 Suppl 2: 286-90.*

[19] BIEDERT RM, ALBRECHT S. The patellotrochlear index: a new index for assessing patellar height. *Knee Surg Sports Traumatol Arthrosc. 2006 ago; 14(8): 707-12.*

[20] GOUTALLIER D, BERNAGEAU J, LECUDONNEC B. The measurement of the tibial tuberosity. Patella groove distanced technique and results (author's transl). *Rev Chir Orthop Reparatrice Appar Mot. 1978 ago; 64(5): 423-8.*

[21] SCHOETTLE PB, ZANETTI M, SEIFERT B, PFIRRMANN CWA, FUCENTESE SF, ROMERO J. The tibial tuberosity-trochlear groove distance; a comparative study between CT and MRI scanning. *Knee. 2006 jan; 13(1): 26-31.*

[22] LAURIN CA, DUSSAULT R, LEVESQUE HP. The tangential x-ray investigation of the patellofemoral joint: X-ray technique, diagnostic criteria and their interpretation. *Clin Orthop Relat Res 1979 out;(144): 16-26.*

[23] LAURIN CA, LÉVESQUE HP, DUSSAULT R, LABELLE H, PEIDES JP. The abnormal lateral patellofemoral angle: a diagnostic roentgenographic sign of recurrent patellar subluxation. *J Bone Joint Surg Am. 1978 jan; 60(1): 55-60.*

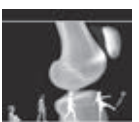
[24] LANCE E, DEUTSCH AL, MINK JH. Prior lateral patellar dislocation: MR imaging findings. *Radiology 1993 dez; 189(3): 905-7.*

[25] DIEDERICHS G, ISSEVER AS, SCHEFFLER S. MR imaging of patellar instability: injury patterns and assessment of risk factors. *Radiographics 2010 ago; 30(4): 961-81.*

[26] ELIAS DA, WHITE LM, FITHIAN DC. Acute lateral patellar dislocation at MR imaging: injury patterns of medial patellar soft-tissue restraints and osteochondral injuries of the inferomedial patella. *Radiology. 2002 dez; 225(3): 736-43.*

[27] KIRSCH MD, FITZGERALD SW, FRIEDMAN H, ROGERS LF. Transient lateral patellar dislocation: diagnosis with MR imaging. *AJR Am J Roentgenol 1993 jul; 161(1): 109-13.*

[28] VIROLAINEN H, VISURI T, KUUSELA T. Acute dislocation of the patella: MR findings. *Radiology 1993 out;189(1): 243-6.*



WHAT SHOULD WE THINK ABOUT OR LOOK AT, IN CASE OF PATELLA DISLOCATION IN CHILDREN?

F. CHOTEL, A. PELTIER, R. KOHLER, J. BÉRARD

INTRODUCTION

Patella dislocation had multiples facets. Although considered a spectrum of disease related to episodic or recurrent patella instability regularly seen in older patient, congenital and habitual dislocation of the patella are now understood as two distinct entities. A genetic etiology is supported by many associated syndromes and clinical findings seen in accordance with congenital dislocation of the patella. These pathologies are rare and more often encountered in pediatric orthopedic practice.

Congenital patellar aplasia or hypoplasia belongs to another heterogeneous group of lower limb malformations (with sometime patella dislocation) associated with multiple genetic disorders.

The aim of this paper is to **highlight these associated pathologies that could impact the patient management** and try to answer to the question: what should we think about or look at in case of patella dislocation in children?

EPISODIC OR RECURRENT PATELLA INSTABILITY

This is the more benign and common expression of the disease usually seen in healthy pre-or adolescent. Classical predominance in children or adolescent girls could suggest association with benign local hyperlaxity. But clinicians should also be aware of the **possible association with occult and more serious general joint hypermobility**. This association sometime needs others investigations looking after collagen abnormality or muscular disease that could facilitate failure and recurrence after stabilization surgery.

Joint hypermobility, is often confirmed by a score of four or more (for adult) and six or more (for children) on the nine-point **Beighton scale** [10], one point is scored for each of the following (fig. 1):

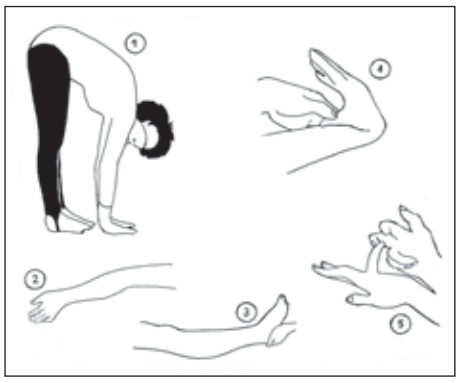
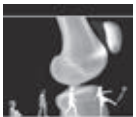


Fig. 1: Beighton criteria in order to detect joint hypermobility.



- passive dorsiflexion of each fifth finger greater than 90°,
- passive apposition of each thumb to the flexor surface of the forearm,
- hyperextension of each elbow greater than 10°,
- hyperextension of each knee greater than 10°,
- ability to place the palms on the floor with the knees fully extended.

Figure 2 gives a proposition of algorithm for diagnosis approach in children with hyperlaxity. Typical examples of these pathologies are:

1) Ehlers-Danlos syndrome (EDS) is a heterogeneous group of connective tissue disorders characterized by hyperextensibility, delayed wound healing, joint hypermobility, thin skin, easy bruising, tissue fragility, "cigarette-paper" scarring over bony prominences, mitral valve prolapse, and other findings. There are 6 main types of EDS but EDS Type III is the main type associated with joint or patellar dislocation. Regardless of presentation as a chief concern or an incidental finding, clinicians should be aware that the prominent skin findings of EDS are cutaneous signs of an important systemic disorder.

2) Joint hypermobility syndrome (JHS) is an emerging pathology. This heritable disorder of the connective tissues is characterized by hypermobility, often affecting multiple joints, and musculoskeletal pains in the absence of systemic inflammatory joint disease such as rheumatoid arthritis. As yet, the gene defect underlying this condition remains unknown, and the diagnosis is based upon clinical signs and symptoms. The phenotype is similar to Ehlers-Danlos type III syndrome hypermobility type (same condition?). Under recognized and sometimes dismissed as a pathologic entity, this condition has no definitive treatment and therefore poses a management challenge. JHS have impaired proprioception, so that specific rehabilitation program leads not only to symptomatic improvement, but also to demonstrable enhancement of objective parameters such as proprioception [4].

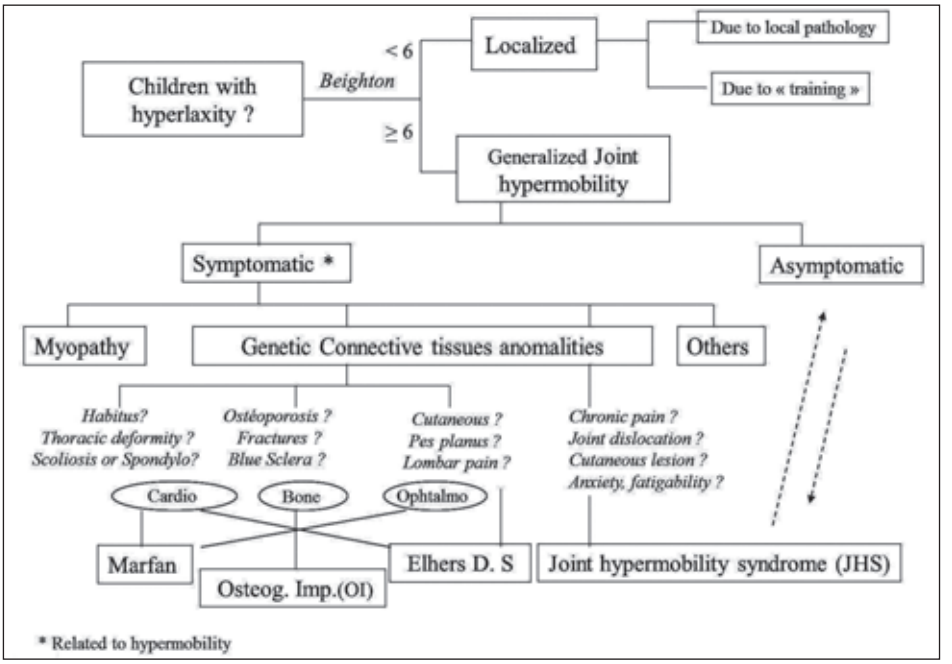
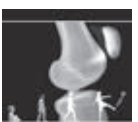


Fig. 2: Proposition of algorithm for diagnosis approach in children with hyperlaxity (Chotel French pediatric societies Paris 2010). Genetic connective tissue diseases are important to consider according to associated symptoms such as: scoliosis, thoracic deformity, pes planus, multiple fractures history…



CONGENITAL AND HABITUAL DISLOCATION OF THE PATELLA

Congenital (or persistent lateral) dislocation of the patella is the most severe form of the pathology spectrum. It is presents in early infancy and could result in early malrotation and shortened extensor mechanism. This entity must be differentiated from habitual (or obligatory) dislocation of the patella which often presents later in childhood and find spontaneous reduction of the patella with flexion and extension of the knee.

More common associated syndromes are:

1) Down syndrome is one of the most common chromosomal abnormalities in human, occurring in 1 in 700-1000 live births. A variety of orthopedic manifestations can occur in relation to ligamentous laxity, muscular hypotonia, and joint hypermobility: cervical spine instability, scoliosis, foot disorder (pes planus, metatarsus), hip disorder, and genu valgum and patella dislocation (10 to 20% of patients) [1]. Life expectancy has improved mainly for advances in the management of cardiac anomalies, infections, and leukemia.

Surgical intervention in children with Down syndrome has a high risk of complications, particularly infection and wound healing problems. Careful anesthetic airway management is needed because of the associated risk of cervical instability.

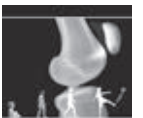
2) Larsen's syndrome is a disease of generalized defect in collagen. This rare and severe syndrome is characterized by flat, hyperteloric facies, multiple dislocations of the joints, club foot deformity, and long cylindrical fingers. Many of these patients died of early complications secondary to dislocation of the cervical spine, tracheomalacias, heart disease, and severe respiratory infection. These children **pose a problem of planning early treatment**. Clubfeet must be treated conservatively early, and operation should be postponed until after knee deformities (dislocated knee or patella) are corrected by plaster cast and/or early operation. Unilateral hip dislocations should be treated surgically in the second year of life. Treatment of bilateral hip dislocations is best neglected. Finally, the spinal status of these children must be monitored throughout their life [6]. Differential diagnosis is tricho-rhinophalangeal syndrome (associated with laxity of the skin and joints) which can be mistaken for the Larsen syndrome.

3) Diastrophic dysplasia and recessive multiple epiphyseal dysplasia (rMED) are non-lethal conditions of a family of the more common generalized skeletal dysplasia. **Double-layered patella (DLP) or "multilayered" patella**, a form of partite patella consisting of anterior and posterior components, is hardly suggestive for this diagnosis. When symptomatic, surgical fusion of the patellar fragments in DLP using threaded screw has been suggested [7].

4) Rubinstein-Taybi syndrome (RTS) is a well-defined complex of congenital malformations characterized by mental and growth retardation, broad thumbs, broad big toes, and typical face. Associated patellar instability demands early recognition and treatment to prevent potentially catastrophic gait disturbances [8].

5) Beals-Hecht syndrome (BHS) also known as congenital contractural arachnodactyly, is caused by a defect in fibrillin as in Marfan syndrome. This syndrome is characterized by a multitude of clinical findings including **arachnodactyly, narrow body habitus**, scoliosis, congenital contractures, and external ear deformities. Restrictive lung disease may be associated with the severe scoliosis and thoracic cage abnormalities in this syndrome.

6) Ellis van Creveld syndrome (EVCS) is a rare chondroectodermal dysplasia, genetically transmitted with a recessive autosomal pattern, which involves the skeletal system (valgus deformity with lateral dislocation of the patella, polydactyly), nails and teeth [11]. In about 50 to 60 percent of cases, the affected individuals show **cardiac abnormalities**. Aortic atresia, hypoplasia of the ascending aorta or of the left ventricle is also reported. About half of the patients die in the childhood due to cardio-respiratory complications.



7) And many others diseases will not be detailed here: arthrogryposis, myelomenigocele, chondroosteodystrophy…

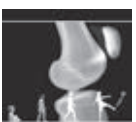
PATELLA APLASIA-HYPOPLASIA

Patella aplasia-hypoplasia is a rare condition characterized by the congenital absence or marked reduction of the patella (but dislocation is also possible). Aplasia or hypoplasia occurs classically in nail-patella syndrome, small patella syndrome, and several other syndromes (Table 1), but can also very rarely be isolated [9]. Most common syndromes are:

1) Nail-patella syndrome (NPS), also called hereditary osteo-onychodysplasia, is a rare hereditary autosomal dominant disorder resulting from a heterogenous loss of function in the LMXB1 gene on chromosome 9q34. The clinical manifestations are extremely variable in both frequency and severity. Patients have a characteristic tetrad of pathologic symptoms including aplasia/hypoplasia patellas and iliac horns (fig. 3), **fingernail dysplasia, and radial head dislocation** (fig. 4) [12].

Table 1: Differential Diagnoses of Nail-Patella Syndrome according to Sweeney [12].

Syndrome	Similarities	Differences	References
Small patella syndrome (SPS)	Small or absent patellae Recurrent patella dislocations Pelvic anomalies	Defective ossification at the ischiopubic junction - Ischial hypoplasia Infra-acetabular «axe-cut» notch No nail changes No elbow changes No renal involvement No ocular involvement	OMIM 147891
Patella aplasia-hypoplasia (PTLAH)	Hypoplasia of the patella or Isolated aplasia	No nail changes No elbow changes No renal involvement No ocular involvement	OMIM 168860
Familial recurrent dislocation of the patella	Familial tendency toward patella dislocation		OMIM 169000
Meier-Gorlin syndrome (MGS)	Absent patellae Dislocation of the radial head	Microtia Markedly short stature Delayed bone age Characteristic facial appearance	OMIM 224690
Genitopatellar syndrome (GPS)	Absent patellae Renal anomalies Flexion deformities of the knees and hips Club foot	Hypoplasia of the ischia and iliac bones Genital anomalies Facial dysmorphism Microcephaly Intellectual disability Multicystic kidneys or hydronephrosis Renal manifestations	OMIM 606170
Trisomy 8 mosaicism	Hypoplastic patellae Limited elbow supination Abnormal nails	Significant learning difficulties Variable facial dysmorphism Camptodactyly and progressive joint restriction, usually of the fingers and toes	Jones [1997]
Coffin-Siris syndrome (CSS)	Absence or hypoplasia of the nails and patellae Elbow dislocation	Nail hypoplasia, usually affecting the little finger nails Facial dysmorphism	OMIM 135900
RAPADILINO syndrome (Rothmund-Thomson syndrome)	Radial defects Absent or hypoplastic patellae Dislocated joints	Cleft palate Facial dysmorphism Short stature Radial defects, including absent or hypoplastic thumbs and radii	OMIM 266280



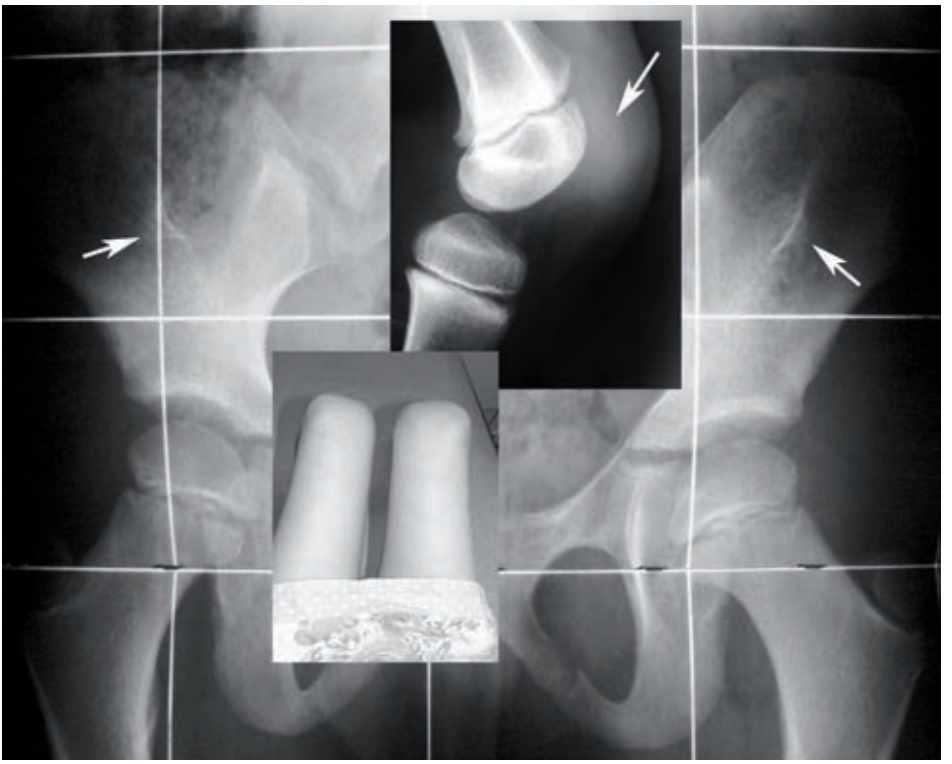
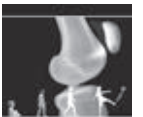


Fig. 3: Nail patella syndrome (NPS) lower limb signs: hypoplasic and dislocated patella (central). Iliac horns are bilateral accessory outgrowths consisting of cortex and medulla continuous with the iliac bone. They are located at the site of attachment of the gluteus medius muscles and project posterolaterally. They are asymptomatic, frequently palpable, and need not be treated. Iliac horns are the pathognomonic feature of Nail-patella syndrome; they occur in approximately 80% of cases and are observed only in this condition.

Fig. 4: Nail patella syndrome (NPS) upper limb signs: Radial head dislocation according to Storen lign (in black) the radial axis does not cut the lateral condyle epiphysis (left). Finger nail involvement lessens in the more ulnar-sided rays, being more severe in the thumb. The nail changes may be limited to triangular lunulae a characteristic feature of NPS (right).



Soft-tissue changes (glaucoma) and **renal involvement** (30-50%) have also been associated with the syndrome. Nephropathy secondary to long-standing proteinuria ultimately affects mortality more so than any musculoskeletal problem. After making diagnosis, orthopedic surgeon must refer the patient to GP for further evaluation. Blood pressure measurement and urinalysis to screen for renal disease are recommended and patient should be referred to a nephrologist if any abnormalities. Screening for **glaucoma** should be referred to an ophthalmologist. Regular surveillance of the syndrome is required.

Because of similarities between syndromes, differential diagnoses of NPS are sometime clearly difficult! (Table 1).

2) Small patella syndrome (SPS) also called ischiopatellar dysplasia, coxopodo patellar syndrome, or Scott-Taor syndrome, is a rare autosomal dominant disorder. This bone dysplasia is characterized by patellar a/hypoplasia and pelvic anomalies, including **bilateral absent or delayed ossification of the ischiopubic junction and infra-acetabular axe cut notches** (fig. 5). Other major signs are a wide gap between the first and second toes, short fourth and fifth rays of the feet, and pes planus. Various other skeletal anomalies have been reported, such as elongated femoral necks, flattened and widened proximal femoral epiphyses, hypoplasia of the lesser trochanter, and tarsal anomalies. At contrary to NPS, no nails change, iliac horn, elbow anomaly, or renal disease is associated with this condition [3].

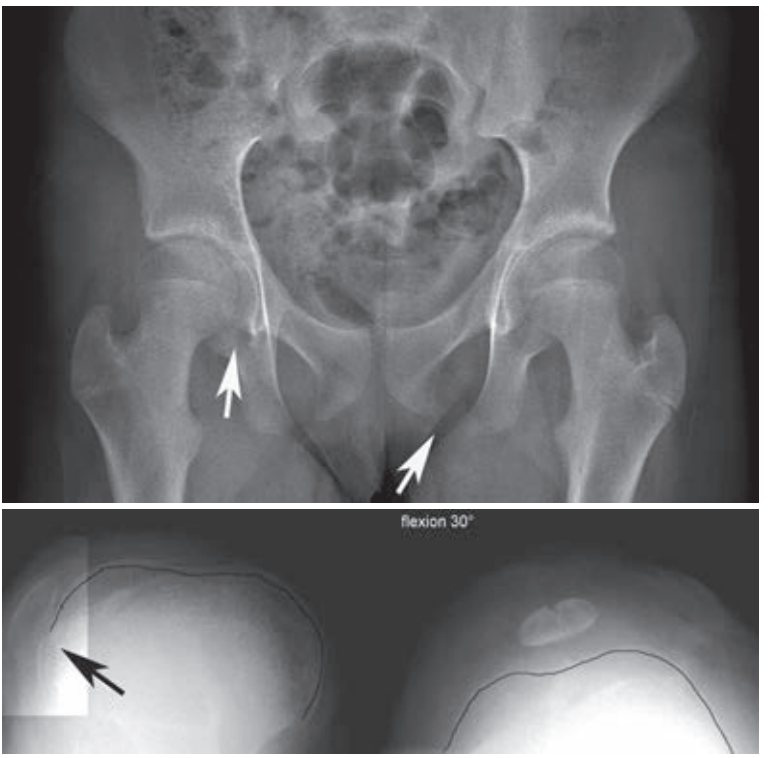
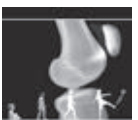


Fig. 5: Small patella syndrome (SPS): pelvic anomalies on AP X-ray, including bilateral absent ossification of the ischiopubic junction (arrow), infra-acetabular axe cut notches (arrow) and elongated femoral necks (up). Permanent patellar dislocation on the left side not yet treated, right side had been reduced surgically after lateral release, Judet quadriceps desinsertion, Grammont procedure, MPFL reconstruction and patelloplasty… (down).



3) Genito-patellar syndrome (GPS) is one of the syndromes described in the last decade. This probable autosomal recessive inheritance is characterized by aplasia/hypoplasia patellae, agenesis of the corpus callosum, microcephaly, mental retardation, extremity flexion contractures, skeletal anomalies, urogenital anomalies, and characteristic facies [13].

4) The Meier-Gorlin syndrome (MGS) or ear, patella, short stature syndrome is a rare autosomal recessive disorder, characterized by the association of aplasia/hypoplasia of the patellae, bilateral microtia, and severe pre- and postnatal growth retardation with short stature [2].

5) Coffin-Siris syndrome (CSS) can be confusing with NPS because of many similarities (Table 1). The most frequent findings include some degree of mental retardation or developmental delay, "coarse" facial appearance, feeding difficulties, frequent infections, and patellar a/hypoplasia, fifth fingernails and fifth distal phalanges [5].

6) Isolated Patella aplasia-hypoplasia (PTLAH) is a rare genetic defect usually characterized by isolated congenital absence or marked reduction of the patella [9].

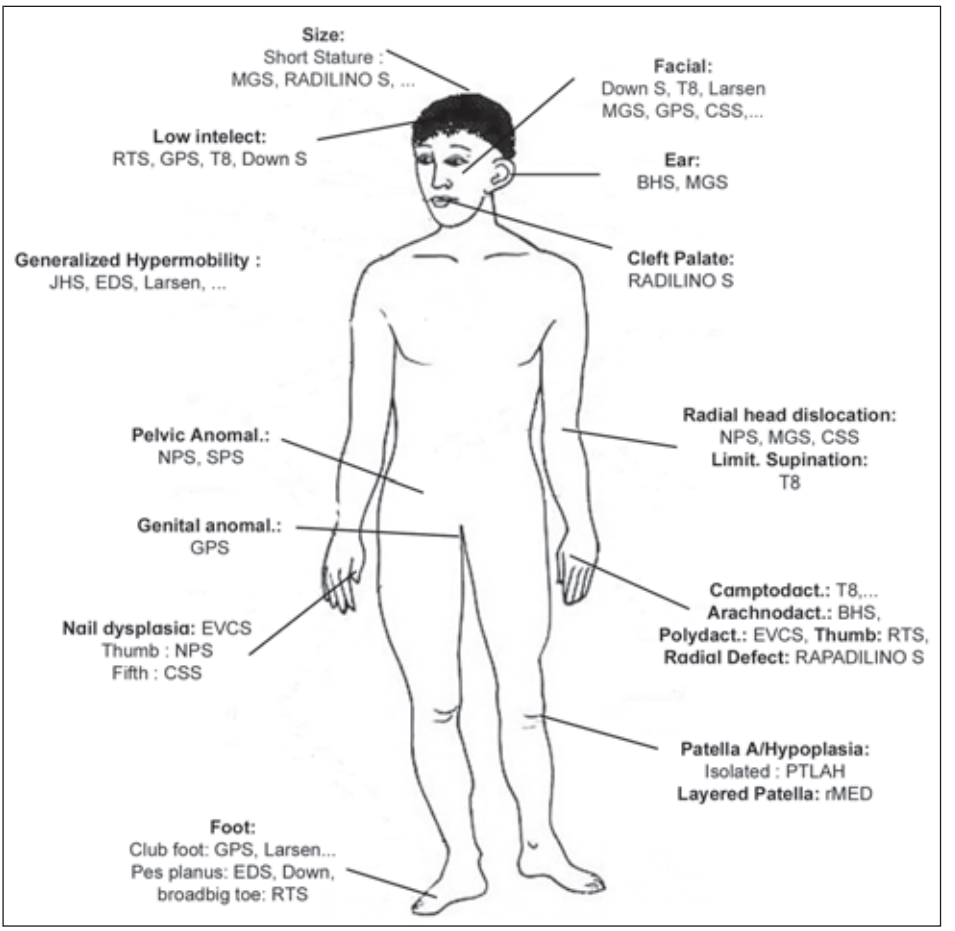


Fig. 6

CONCLUSION

Separation of syndromes associated with congenital dislocation and syndromes associated with patellar aplasia or hypoplasia is artificial and used here only for a didactic point of view. When dealing with these pathology, careful attention must be paid during clinical exam in order to detect associated syndromes that could impact the management of the patient. Permanent dislocation in younger age can be related with **many associated syndromes**; whereas episodic dislocation is often isolated but should consider connective tissues pathologies when generalized hypermobility is noticed. The figure 6 summarizes where to look at when a clinician deal with patellar dislocation, aplasia or hypoplasia.

In case of familial history or small patella, a genetic disorder is suspected: parent's assessment, pelvis X-ray will be of major diagnosis value (NPS, SPS and GPS).

And so the orthopedist surgeon will sometime have to refer the patient to other specialist such as geneticist, ophthalmologist, cardiologist, or nephrologist…

LITERATURE

[1] BETTUZZI C, LAMPASI M, MAGNANI M, *and al.* Surgical treatment of patellar dislocation in children with Down syndrome: a 3- to 11-year follow-up study. *Knee Surg Sports Traumatol Arthrosc 2009; 17: 334-40.*

[2] BONGERS EM, OPITZ JM, FRYER A, *and al.* Meier-Gorlin syndrome: report of eight additional cases and review. *Am J Med Genet. 2001; 102: 115-24.*

[3] BONGERS EM, VAN BOKHOVEN H, VAN THIENEN MN, *and al.* The small patella syndrome: description of five cases from three families and examination of possible allelism with familial patella aplasia-hypoplasia and nail-patella syndrome. *J Med Genet 2001; 38: 209-14.*

[4] FERRELL WR, TENNANT N, STURROCK RD, *and al.* Amelioration of symptoms by enhancement of proprioception in patients with Joint hypermobility syndrome. *Arthritis and Rheumatism 2004; 50: 332-28.*

[5] FLECK BJ, PANDYA A, VANNER L, *and al.* Coffin-Siris syndrome: review and presentation of new cases from a questionnaire study. *Am J Med Genet. 2001; 99: 1-7.*

[6] LAVILLE JM, LAKERMANCE P, LIMOUZY F. Larsen's syndrome: review of the literature and analysis of thirty-eight cases. *J Pediatr Orthop. 1994;14: 63-73.*

[7] LENG HC, FOSTER P, TEMPLETON PA. Fusion of double-layered patella using a single small fragment partially threaded screw: a case report. *J Pediatr Orthop 2011; 31: 9-12.*

[8] MEHLMAN CT, RUBINSTEIN JH, ROY DR. Instability of the patellofemoral joint in Rubinstein-Taybi syndrome. *J Pediatr Orthop. 1998; 18: 508-11.*

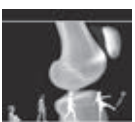
[9] NOMURA E, INOUE M, KOBAYASHI S. Bilateral recurrent patellar dislocation in a patient with isolated patella aplasia-hypoplasia. *Arthroscopy. 2007; 23: 1136.e1-4.*

[10] REMVIG L, JENSEN DV, WARD RC. Are diagnostic criteria for general joint hypermobility and benign joint hypermobility syndrome based on reproducible and valid tests ? A review of the literature. *J Rheumatol. 2007; 34:798-803.*

[11] SHIBATA T, KAWABATA H, YASUI N, *and al.* Correction of knee deformity in patients with Ellis-van Creveld syndrome. *J Pediatr Orthop B. 1999; 8: 282-4.*

[12] SWEENEY E, HOOVER-FONG JE, MCINTOSH I. Nail-Patella Syndrome. In: Pagon RA, Bird TD, Dolan CR, Stephens K, Adam MP, editors. GeneReviews™. *Seattle (WA): University of Washington, Seattle; 1993-2003 [updated 2009].*

[13] TO M, NEGANDHI R, CHEUNG K, *and al.* Genitopatellar syndrome: a case report of a rare entity with 11 years of follow-up. *J Pediatr Orthop B 2012 May 6.*



ANATOMICAL POSITIONING OF THE MEDIAL PATELLOFEMORAL LIGAMENT IN CHILDREN

M. NELITZ, S. LIPPACHER

INTRODUCTION

Lateral patellar dislocation is a common knee injury in the skeletally immature adolescent with a high recurrence rate in patients younger than 14 years [5, 9, 10, 12, 24]. The stabilising role of the MPFL as the main restraining force to lateral displacement of the patella has been emphasized by many authors [2, 4, 20]. In adults reconstruction of the patellofemoral ligament has shown good results [1, 4, 7, 21]. Since it has been shown historically in children, that operative procedures like lateral release, medial reefing and the Roux-Goldthwait procedure have a high failure rate reconstruction of the MPFL has been advocated in skeletally immature patients as well [19]. Due to the adjacent physis different non anatomical techniques have been described for children [6, 12, 24]. But many authors emphasized the need of an anatomical reconstruction with tightening of the MPFL in knee flexion when the femoral origin is placed too proximally [4, 13, 25, 26]. Additionally several authors have shown, that the femoral insertion of the MPFL is distal to the femoral physis [3, 14, 15, 16, 18]. As the importance of an anatomical repair respecting the femoral and patellar insertion of the ligament has been proven, this technical report describes the technique of an anatomical, physeal-sparing reconstruction of the MPFL in children with open growth plates.

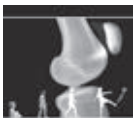
METHODS

Before surgery every patient's knee was examined clinically under anesthesia and a diagnostic arthroscopy is performed to rule out intraarticular pathology.

Following the diagnostic arthroscopy an oblique incision is made along the pes anserinus. After exposing the fascia the gracilis tendon is harvested proximally using a tendon stripper. Distally the tendon is sharply detached from the tibia. The tendon is prepared with a Vicryl suture on both ends and stored within a moist swab.

A longitudinal incision is made over the medial proximal two-thirds of the patella. The medial border of the patella is exposed subperiosteally avoiding injury of the joint capsule. A 4-mm drill is used to create a V-shaped tunnel at the superomedial half of the patella with sufficient distance between tunnels to avoid fracturing. The graft can then be inserted into the tunnel forming a loop through the patella (fig. 1).

By blunt dissection the interval between the capsule and the vastus medialis obliquus is developed to the femoral insertion of the MPFL. Using the indirect radiographic method described by Schöttle *et al.* [22] the anatomical femoral insertion of the MPFL is identified



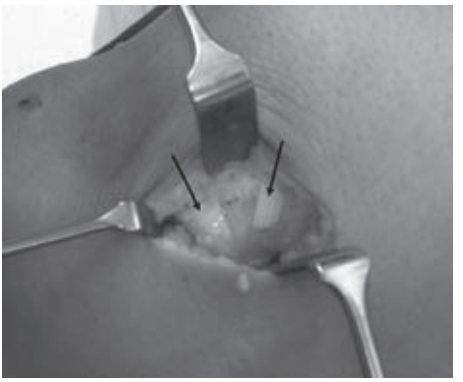


Fig. 1 : Double bundle insertion of the graft (arrows) reconstructs the wide span of the patellar insertion of the native MPFL.

under fluoroscopic control. A guide pin is placed at the femoral insertion. Fluoroscopy is used to confirm the correct placement of the guide-pin sparing the distal femoral physis. Due to the concave curvature of the distal femoral physis, the lateral radiograph alone can be misleading for the determination of the relation between the distal femoral physis and the medial patellofemoral ligament [11, 18]. The cross-reference onto an AP view shows that the same point that is projected on or proximal to the physis on the lateral view is distal to the physis on the AP view (fig. 2). After meticulous verification of the entry-point the guide-pin is drilled to the lateral epicondyle distal to the physis (fig. 4). Then a medial blind tunnel is drilled along the guide pin to accommodate a double thickness of graft to an adequate depth to allow optimal graft tensioning.

The graft is then pulled between the second and third layer to the femoral insertion point (fig. 3).

A locking suture is passed through the trans-epicondylar axis pulling the graft into the medial tunnel. The knee is cycled several times from full flexion to full extension with the graft under tension. In this way, the graft is prestretched. The graft is then secured within the medial condyle tunnel using a bioresorbable interference screw with the knee flexed to 30° (fig. 5).

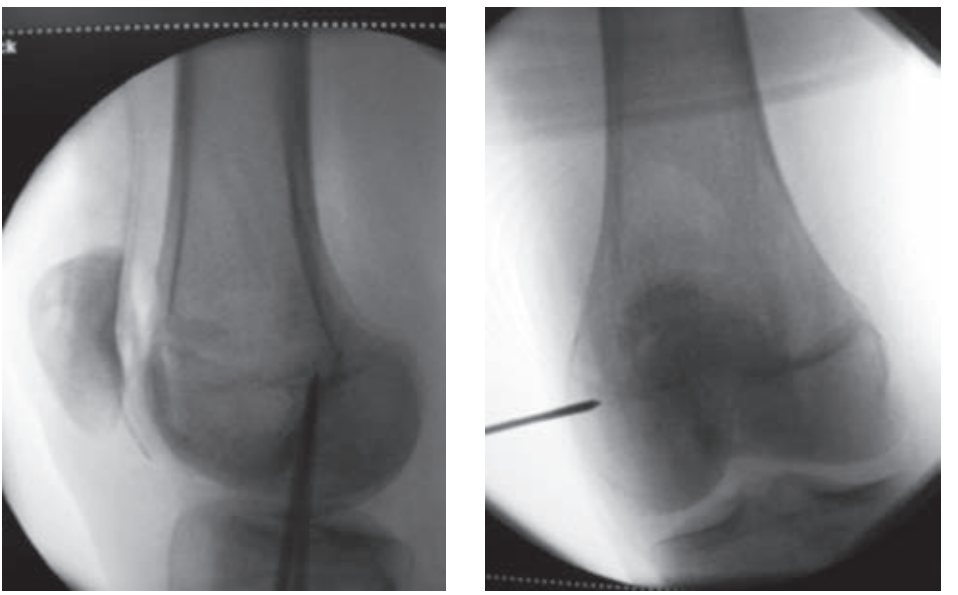


Fig. 2 : Intraoperative cross-reference of the physis on the lateral view onto an ap view shows that the same point that is projected on or proximal to the physis on the lateral view is distal to the physis on the AP view.

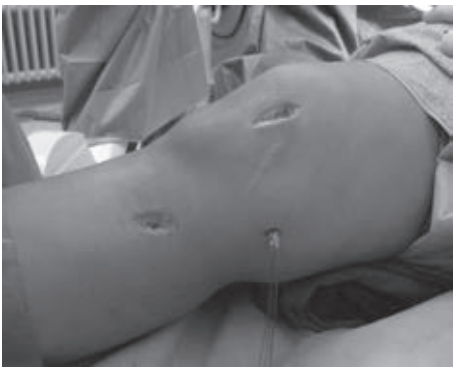


Fig. 3 : The free ends of the graft are pulled between the second and third layer to the femoral insertion point.

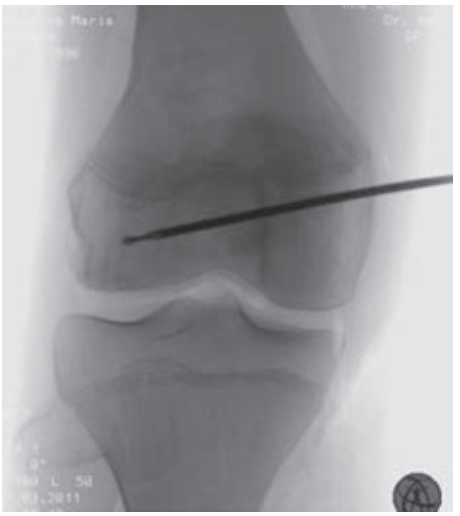


Fig. 4 : After identification of the entry-point the guide-pin is drilled to the lateral condyle strictly distal to the physis.

The aponeurosis of the VMO is sutured back to the patella using Vicryl, with further closure of subcutaneous tissues and skin. Routine dressings and bandages are applied.

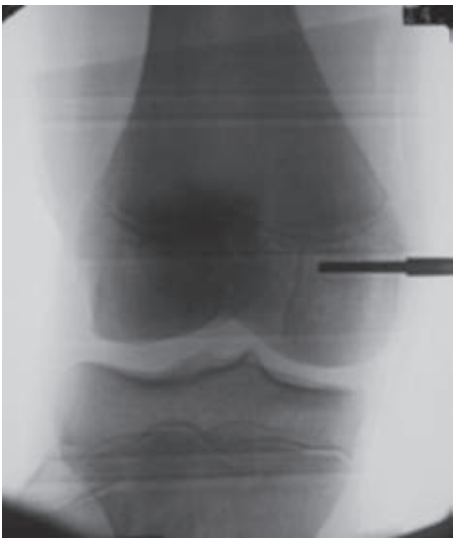


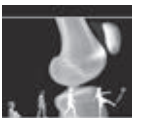
Fig. 5 : Fixation of the graft within the medial condyle tunnel using a bioresorbable interference screw.

REHABILITATION

Postoperatively partial weight-bearing using crutches was allowed. Daily physiotherapy with active and passive flexion and extension exercises of the knee, strengthening of the vastus medialis muscle and straight leg-raising exercises were recommended. Full weight-bearing was allowed at two weeks and return to sport was allowed at the third postoperative month.

DISCUSSION

Whereas there are numerous publications about anatomical reconstruction of the MPFL in adults to our knowledge this is the first report about anatomical reconstruction of the MPFL considering the relation of its femoral insertion to the distal femoral physis in children.



Deie *et al.* have [12] described good results after non anatomic MPFL reconstruction in children between 6 and 10 years. They have chosen the posterior one-third of the proximal attachment of the MCL as the site for pulley.

Brown *et al.* [6] described a technique with combined medial patellofemoral ligament and medial patellotibial ligament reconstruction leaving the insertion of the semitendinosus tendon intact. They sutured the free limb to the MCL as proximal as possible. Both techniques can produce an inadvertent distalisation of the patella, even if it is not needed.

Sillanpää *et al.* [24] described a technique using a free graft wrapped around the adductor magnus tendon.

None of the described techniques is strictly anatomical, as they use the femoral insertion of the MCL or of the adductor magnus tendon as a reference for the femoral insertion of the MPFL. A detailed anatomical study by Baldwin [3] has shown that the adductor tubercle provides exclusive attachment for the adductor magnus tendon and the medial epicondyle provides exclusive attachment for the MCL, whereas the insertion of the MPFL is found in a groove between these two landmarks. Using these non anatomical techniques it is furthermore difficult to control the accurate tension of the graft during fixation.

The need for an anatomical reconstruction is now widely accepted. If the femoral origin is placed too proximally tightening of the MPFL in knee flexion with concomitant increased contact stress can occur [13, 25, 26]. Camp *et al.* [8] found the failure to restore the anatomical femoral insertion to be a main risk factor for the failure of MPFL reconstruction.

Several studies have shown that the insertion of the MPFL is typically distal to the femoral physis [3, 14, 15, 16, 18]. On MRI scan, Kepler *et al.* [14] measured the distance between the MPFL insertion onto the distal femur and the medial distal femoral growth plate or physeal scar. The femoral MPFL insertion averaged 5mm distal to the femoral growth plate. A radiographic study using the radiographic landmarks desribed by Schöttle *et al.* [22] confirmed the results of Kepler *et al.* [14]. The authors have shown that the median origin of the MPFL as seen on the AP view averaged 6.4mm (2.9-8.5mm) distal to the femoral physis [18].

For patients with open physis this means that an insertion proximal to the physis has to be strictly avoided as it can create increased medial patellofemoral pressure [13].

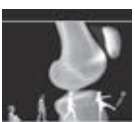
In summary the technique described in this study has two major advantages. First it reconstructs the anatomy of the MPFL and at the same time it is respecting the distal femoral physis. To avoid injury of the physis the femoral insertion as well as the direction of the blind hole have to be checked radiographically on lateral and AP view. As the femoral insertion of the MPFL is distal to the physis the bone tunnel has to be strictly in the epiphysis. Secondly the technique desribed in the present study technique uses a sling through the proximal half of the patella, which recreates the double-bundle structure of the MPFL and decreases patellar rotation compared to single-point fixation [2, 23].

CONCLUSION

This is the first report of a minimal invasive procedure for anatomical reconstruction of the MPFL in children with open growth-plates. This technique considers the fact that the femoral insertion of the MPFL is distal to femoral physis. As a too proximal insertion of the graft proximal to the physis can cause unintentional tightening of the MPFL in knee flexion this has to be considered during the reconstruction of the MPFL in skeletally immature patients.

ABSTRACT

Recurrent lateral patellar dislocation is a common knee injury in the skeletally immature adolescent. In adults anatomical reconstruction of the MPFL is recommended, but due to the



open physis operative therapy in children is more challenging. We present a minimal invasive technique for anatomical reconstruction of the MPFL in children considering the important fact, that the femoral insertion is distal to the femoral physis. Since the failure to restore the anatomical femoral insertion is the main risk factor for failure of MPFL reconstruction, an insertion proximal to the physis has to be strictly avoided.

LITERATURE

[1] AHMAD CS, BROWN GD, STEIN BS. The docking technique for medial patellofemoral ligament reconstruction: surgical technique and clinical outcome *Am J Sports Med 37 (2009): 2021-7.*

[2] AMIS AA, FIRER P, MOUNTNEY J, SENAVONGSE W, THOMAS NP Anatomy and biomechanics of the medial patellofemoral ligament. *Knee 2003. 10:215-20.*

[3] BALDWIN JL The anatomy of the medial patellofemoral ligament. *Am J Sports Med 2009. 37: 2355-61.*

[4] BICOS J, FULKERSON JP, AMIS A Current concepts review: the medial patellofemoral ligament. *Am J Sports Med 2007. 35: 484-92.*

[5] BITAR AC, DEMANGE MK, DÈLIA CO, CAMANHO GL Traumatic Patellar Dislocation: Nonoperative treatment compared with MPFL reconstruction using patellar tendon. *Am J Sports Med epub ahead of print 2011.*

[6] BROWN GD, AHMAD CS Combined medial patellofemoral ligament and medial patellotibial ligament reconstruction in skeletally immature patients. *J Knee Surg 2008. 21: 328-32.*

[7] BUCKENS CF, SARIS DB Reconstruction of the medial patellofemoral ligament for treatment of patellofemoral instability: a systematic review. *Am J Sports Med 2010. 38: 181-8.*

[8] CAMP CL, KRYCH AJ, DAHM DL, LEVY BA, STUART MJ Medial patellofemoral ligament repair for recurrent patellar dislocation. *Am J Sports Med 2010. 38: 2248-54.*

[9] CASH JD, HUGHSTON JC Treatment of acute patellar dislocation. *Am J Sports Med 1988. 16: 244-49.*

[10] COLVIN AC, WEST RV Patellar instability. *J Bone Joint Surg Am 2008. 90: 2751-62.*

[11] CRAIG JG, CODY DD, VAN HOLSBEECK M The distal femoral and proximal tibial growth plates: MR imaging, three-dimensional modeling and estimation of area and volume. *Skeletal Radiol 2004. 33: 337-40.*

[12] DEIE M, OCHI M, SUMEN Y, YASUMOTO M, KOBAYASHI K, KIMURA H Reconstruction of the medial patellofemoral ligament for the treatment of habitual or recurrent dislocation of the patella in children. *J Bone Joint Surg Br 2003. 85:887-90.*

[13] ELIAS JJ, COSGAREA AJ Technical errors during medial patellofemoral ligament reconstruction could overload medial patellofemoral cartilage: a computational analysis. *Am J Sports Med 2006. 34: 1478-85.*

[14] KEPLER CK, BOGNER EA, HAMMOUD S, MALCOLMSON G, POTTER HG, GREEN DW Zone of injury of the medial patello-femoral ligament after acute patellar dislocation in children and adolescents. *Am J Sports Med 2011. epub ahead of print.*

[15] LADD PE, LAOR T, EMERY KH, SALISBURY SR, PARIKH SN Medial collateral ligament of the knee on magnetic resonance imaging: does the site of the femoral origin change at different patient ages in children and young adults? *J Pediatr Orthop 2010. 30: 224-30.*

[16] LAPRADE RF, ENGEBRETSEN AH, LY TV, JOHANSEN S, WENTORF FA, ENGEBRETSEN L The anatomy of the medial part of the knee. *J Bone Joint Surg Am 2007. 89: 2000-10.*

[17] MELEGARI TM, PARKS BG, MATTHEWS LS Patellofemoral contact area and pressure after medial patellofemoral ligament reconstruction. *Am J Sports Med 2008. 36: 747-52.*

[18] NELITZ M, DORNACHER D, DREYHAUPT J, REICHEL H, LIPPACHER S The relation of the distal femoral physis and the medial patellofemoral ligament. *Knee Surg Sports Traumatol Arthrosc 2011. 19: 2067-71.*

[19] NELITZ M, THEILE M, DORNACHER D, WÖLFLE J, REICHEL H, LIPPACHER S Analysis of failed surgery for patellar instability in children with open growth plates. *Knee Surg Sports Traumatol Arthrosc 2012. 20: 822-28.*

[20] NOMURA E, HORIUCHI Y, KIHARA M Medial patellofemoral ligament restraint in lateral patellar translation and reconstruction. *Knee 2000. 7:121-27.*

[21] RONGA M, OLIVA F, LONGO UG, TESTA V, CAPASSO G, MAFFULLI N Isolated medial patellofemoral ligament reconstruction for recurrent patellar dislocation. *Am J Sports Med 2009. 37:1735-42.*

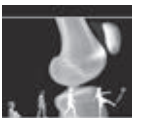
[22] SCHOETTLE PB, SCHMELING A, ROSENSTIEL N, WEILER A Radiographic landmarks for femoral tunnel placement in medial patellofemoral ligament reconstruction. *Am J Sports Med 2007. 35: 801-4.*

[23] SCHOETTLE PB, HENSLER D, IMHOFF AB Anatomical double-bundle reconstruction with an aperture fixation. *Knee Surg Sports Traumatol Arthrosc 2010. 18: 147-51.*

[24] SILLANPÄÄ PJ, MÄENPÄÄ HM, ARENDT EA Treatment of lateral patella dislocation in the skeletally immature athlete. *Oper Tech Sports Med 2010. 18:83-92.*

[25] SMIRK C, MORRIS H The anatomy and reconstruction of the medial patellofemoral ligament. *Knee 2003. 10: 221-27.*

[26] STEENSEN RN, DOPIRAK RM, MCDONALD WG The anatomy and isometry of the medial patellofemoral ligament: implications for reconstruction. *Am J Sports Med 2004. 32: 1509-13.*



PHYSEAL-SPARING MPFL RECONSTRUCTION IN CHILDREN: experience of the pediatric orthopaedic department of Lyon

F. CHOTEL, A. PELTIER, A. VISTE, M.M. CHAKER, J. BÉRARD

INTRODUCTION

There is a very high rate of recurrent instability after primary acute patellar dislocation in children with or without early repair medial structures [13]. More than 100 procedures reported for patella stabilization… This fact presumes that no single technique is superior for this multifaceted condition. More recently, some authors highlighted the anatomy of mediopatello femoral ligament and described anatomic reconstruction of this structure [5, 14, 15]. Since 2000, there is growing interest in exchanging the myriad of nonanatomic extensor mechanism reconstructions for more anatomic procedures based on restitution of the MPFL in adults [1].

Few data are available about MPFL reconstruction in children since the first experience published [2], except some recent and short series [7, 12, 17]. The aim of this study was to report our experience with MPFL reconstruction in children and to describe our evolution toward a personal and new procedure for MPFL reconstruction on skeletally immature patients and adolescents.

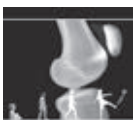
PATIENTS AND METHOD

Indication

Great majority of permanent and habitual dislocations have to be surgically corrected as soon as possible. Stabilization is indicated in case of episodic dislocation after major objective patella instability with 2 or more patella dislocations, trochlear dysplasia and apprehension test +.

Our global strategy

Except MPFL reconstruction which is systematically part of the treatment, patella stabilisation surgery in our unit is a "à la carte surgery" [4]. We are concerned about patella alta and this condition should be treated during the same time but anteriorly to the MPFL reconstruction in order to keep a satisfied graft isometry. Lateral release is performed "on demand" only when lateral retinaculum is very tight. For skeletally immature patient soft tibial tubercule transfer according to Grammont is associated when the Q angle was important or



TAGT distance > to 20mm [6, 8]. In such situation and for adolescents with closed growth plate, a classical tibial tubercule osteotomy is used. More rarely, and for adolescents a trochleoplasty according to Dejour is associated. In younger children, and when dealing with permanent patella dislocation which combine short quadriceps, a Judet quadriceps liberation is associated.

Surgical procedures

Since 2007, we perform MPFL reconstruction in children. Initially we used Deie technique for skeletally immature patients [2] and Fithian technique for adolescents [1] (fig. 1). This preliminary experience about 13 cases has been reported during EPOS meeting in 2010 [3] (Results).

In the mid-2010, we develop an anatomic double bundle physeal-sparing technique using a free semitendinosus tendon. This technique will be reported here (fig. 2).

This technique is mixing Fithian method and a modified Deie method (fig. 1). It is conducted under general anaesthesia and continuous loco-regional analgesia. The patient is in supine position. The operated knee is placed at 80° flexion with a foot support distally and a support on the lateral aspect of the thigh allows stability during surgery. A non-sterile tourniquet is placed as proximal as possible.

The procedure starts with semitendinosus transplant harvest. A longitudinal and oblique 2cm skin incision is made medial to the tibial tubercule. The semitendinosus is identified, and its tendon is harvested proximally with a tendon stripper first (fig. 2A-B), while it distal insertion is also separated. A running 2-0 resorbable crisscross suture is placed in both end of the free tendon.

Two additional 2cm longitudinal incisions are made at the superomedial border of the patella and over the medial epicondyle. Through the epicondyle approach, the medial collateral ligament is dissected on an O'shaughnessy dissecting forceps (fig. 2D). A space is created between 2/3 anterior and 1/3 posterior of MCL proximal fiber. The tendon of semitendinosus is transferred to the patella using the posterior one-third of the femoral insertion of the medial

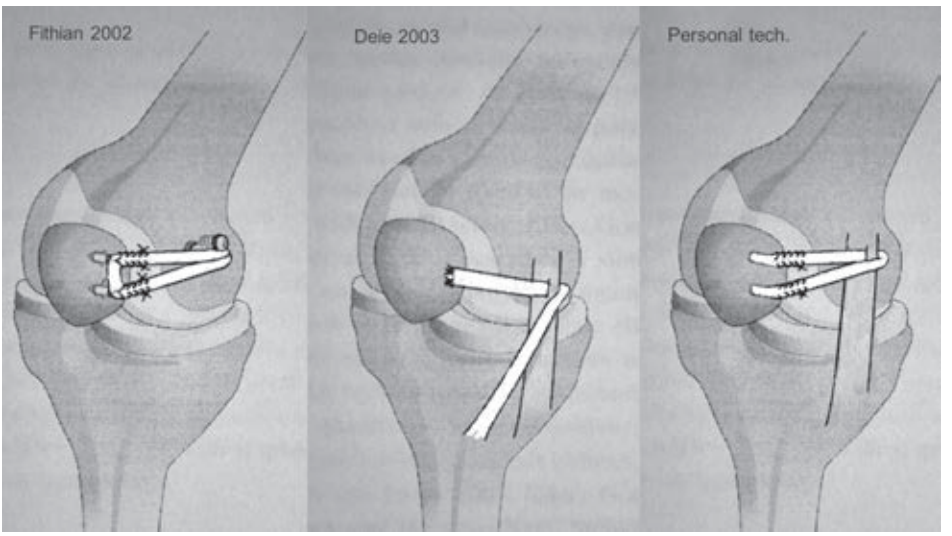
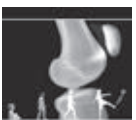


Fig. 1: Illustration of Fithian, Deie and personal technique for MPFL reconstruction in children and adolescent.



collateral ligament as a pulley as described by Deie [2] (fig. 2E). The three different layers of the medial patellar system were dissected: the first corresponds to the superficial retinaculum, the second to the MPFL and MCL and the third to the knee capsule. The tendon bundles were transferred from the pulley to the patella locating in the second layer taking care they were not twisting each other.

Through the patella approach, two transversal bone tunnels are drilled with the size of the semitendinosus tendon (fig. 2C). Each graft bundle is passed through the patella tunnels [1]. After the graft is passed in the tunnels (fig. 2F), it is passed back and sutured to itself pulling the patella medially. Tensionning the graft is performed with the knee in 30° of flexion. The correct among of tension prevents lateral subluxation without causing medial subluxation or excessive medial compression. Patella tracking and stability are tested throught the range of knee motion. In full extension, the patella slightly moves medially (favorable non isometry).

Selected associated procedures are performed during the same time *(Cf. global strategy)*.

The tourniquet is deflated and after accurate hemostasis, an intra-articular drainage is or no associated. The wounds are closed with interrupted subcutaneous sutures and absorbable running subcuticular sutures. Elastic bandage wrap and postoperative immobilization amovible brace with 10° flexion are used.

Postoperative care: full weight-bearing is allowed immediately. Early active range of motion exercices are started as tolerated. Immediate continuous passive motion is associated. A return to sport and full activities is allowed after 4 to 6 months.

Method

Results were assessing with Kujala scoring system. Objective correction of the patella tilt was measured with CT scan preoperatively and postoperatively 1 year following surgery.

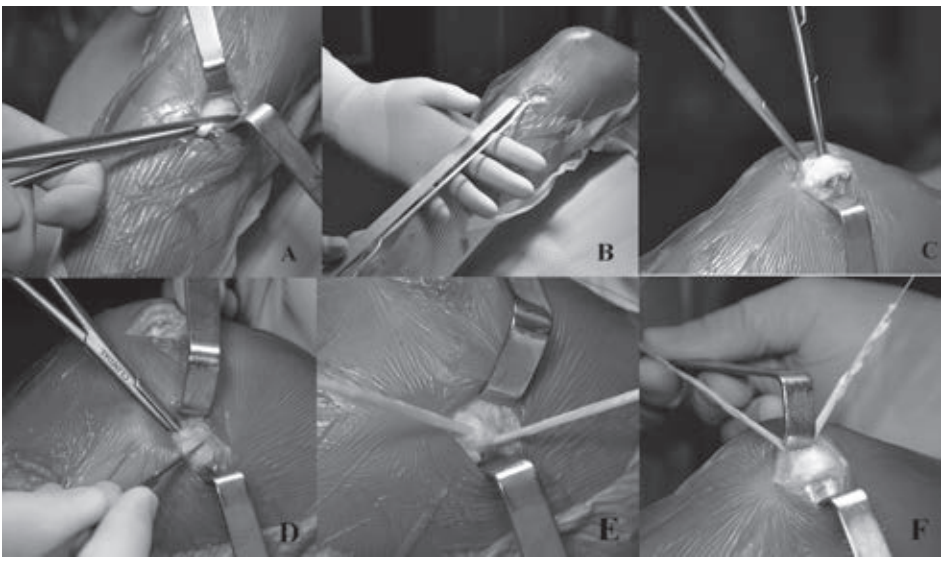
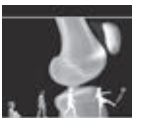


Fig. 2: Details and intraoperative views of different steps for MPFL reconstruction in our unit.



RESULTS

Initial experience with MPFL

This is about the 13 first patients (series presented at 2010 EPOS meeting) [3]. No permanent or habitual dislocation was included in this study. Inclusion criteria were: ***i)*** major objective patella instability with 2 or more patella dislocations, ***ii)*** all trochlear dysplasia/apprehension test + (fig. 3), ***iii)*** no previous surgery.

After a mean of 10.5 months (range, 3-23) follow-up after surgery, no recurrent episodes of dislocation or subluxation were reported. Mean Kujala score was of 90.2 (range, 84-99) at latest follow-up. For all patients the moving patellar apprehension test was positive before and negative after surgery. A firm end point to lateral patellar translation was noticed in all patients at latest follow-up (fig. 4).

Objective assessment with CT noted that the patellar tilt on relaxed quadriceps was significantly improved from 28° preoperatively (range, 16-41) to 16° at follow-up (range, 7-32). The patellar tilt on contracted quadriceps was significantly improved from 35° preoperatively (range, 21-52) to 24.6° at follow-up (range, 11-48) (fig. 5).

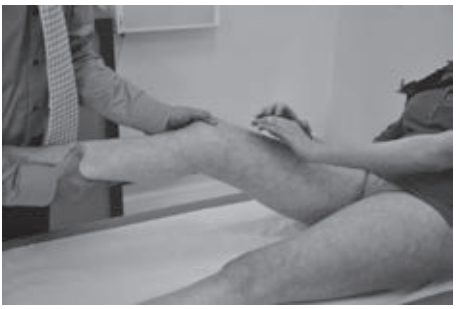


Fig. 3: Typical positive moving patellar apprehension test (look at patient's hands).

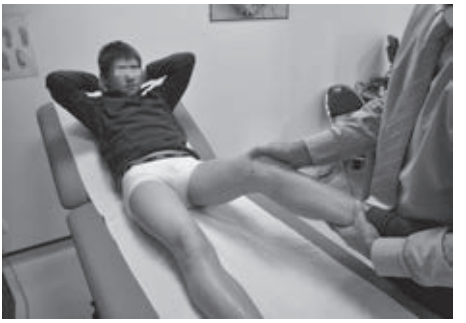


Fig. 4: In our experience, moving patellar apprehension test (or Smilie test) was always find negative post-operatively in the series treated with MPFL reconstruction.

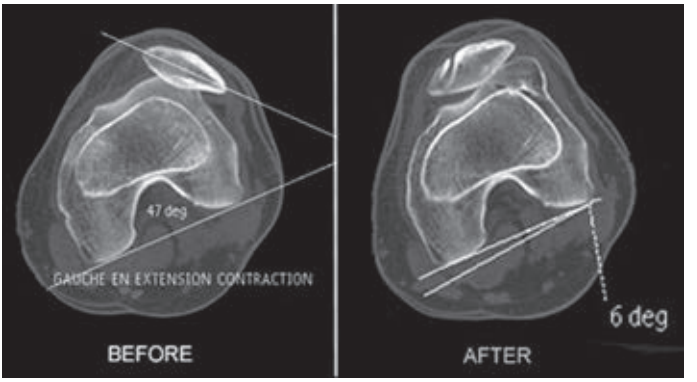


Fig. 5: Patellar tilt during contracted Q and CT scan. Before and after MPFL reconstruction in a young child with habitual patella dislocation. The patella tunnel can be noticed.

Preliminary results with our new physeal-sparing method

These are, for part, included in the prospective study of symposium SFA 2012. In the overall series, there was no recurrence of dislocation after surgery. Radiological assessment showed that the congruence angle, the tilt angle and the lateral shift radio were restored to normal. More detailed results will be presented during the meeting.

DISCUSSION

The exact relation of MPFL and the distal femoral physis is still controversial; some authors find it proximal and others distal to the physis [10, 16]. Anatomic femoral insertion is probably very close to the growth plate and reconstruction should avoid femoral tunnel that could generate growth disturbance. The importance of an anatomically positioned MPFL reconstruction has been highlighted as non-anatomic attachments caused significant loss of isometry.

Physeal-sparing technique of MPFL reconstruction using the posterior one-third of the femoral insertion of the medial collateral ligament as a pulley, have been described by Deie in 2003 [2]. But in his technique the semitendinosus was still attached distally on the tibia and the reconstruction did use a single bundle. We believe that a single bundle offer low resistance in an adolescent knee and is less anatomic than a double bundle. In mid-2010, we started to detach the semitendinosus distally keeping the principle of the pulley, and now this procedure is used whatever is the skeletal maturity.

CONCLUSION

Anatomic MPFL reconstruction is possible in children and adolescent. In preliminary study, the patellar tilt could be efficiently improved by MPFL reconstruction and these results correlated with Kujala score. Middle and long term results still have to be evaluated.

LITERATURE

[1] DAVIS DK, FITHIAN DC. Techniques of medial retinacular repair and reconstruction. *Clin Orthop Relat Res. 2002; 402: 38-52.*

[2] DEIE M, OCHI M, SUMEN Y *and al.* Reconstruction of the medial patellofemoral ligament for the treatment of habitual or recurrent dislocation of the patella in children. *J Bone Joint Surg Br. 2003; 85: 887-90.*

[3] CHOTEL F, VISTE A, CHAKER M, *and al.* Outcomes of MPFL reconstruction for patellar instability in children and adolescent. Objective assessment with CT scans and preliminary results. *J Child Orthop 2010;4: S31.*

[4] DEJOUR H, WALCH G, NEYRET P, ADELEINE P. Dysplasia of the femoral trochlea. *Rev Chir Orthop Reparatrice Appar Mot. 1990; 76: 45-54.*

[5] FELLER JA, FEAGIN JA Jr, GARRETT WE Jr. The medial patellofemoral ligament revisited: an anatomical study. *Knee Surg Sports Traumatol Arthrosc. 1993; 1: 184-6.*

[6] GARIN C, CHAKER M, DOHIN B *and al.* Permanent, habitual dislocation and recurrent dislocation of the patella in children: surgical management by patellar ligamentous transfer in 50 knees. *Rev Chir Orthop Reparatrice Appar Mot. 2007;93: 690-700.*

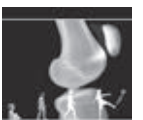
[7] GIORDANO M, FALCIGLIA F, AULISA AG, *and al.* Patellar dislocation in skeletally immature patients: semitendinosous and gracilis augmentation for combined medial patellofemoral and medial patellotibial ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc 2011 Nov 25.*

[8] GRAMMONT PM, LATUNE D, LAMMAIRE IP. Treatment of subluxation and dislocation of the patella in the child. Elmslie technic with movable soft tissue pedicle (8 year review). *Orthopade 1985;14: 229-38.*

[9] KEPLER CK, BOGNER EA, HAMMOUD S, *and al.* Zone of injury of the medial patellofemoral ligament after acute patellar dislocation in children and adolescents. *Am J Sports Med. 2011; 39: 1444-9.*

[10] NELITZ M, DORNACHER D, DREYHAUPT J, *and al.* The relation of the distal femoral physis and the medial patellofemoral ligament. *Knee Surg Sports Traumatol Arthrosc. 2011;19: 2067-71.*

[11] NELITZ M, THEILE M, DORNACHER D, *and al.* Analysis of failed surgery for patellar instability in children with open growth plates. *Knee Surg Sports Traumatol Arthrosc. 2012; 20: 822-8.*



[12] NIETOSVAARA Y, PAUKKU R, PALMU S, *and al.* Acute patellar dislocation in children and adolescents. Surgical technique. *J Bone Joint Surg Am. 2009 ; 91 Suppl 2: 139-45.*

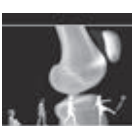
[13] PALMU S, KALLIO PE, DONELL ST, *and al.* Acute patellar dislocation in children and adolescents: a randomized clinical trial. *J Bone Joint Surg Am. 2008; 90: 463-70.*

[14] PHILIPPOT R, CHOUTEAU J, WEGRZYN J, *and al.* Medial patellofemoral ligament anatomy: implications for its surgical reconstruction. *Knee Surg Sports Traumatol Arthrosc. 2009; 17: 475-9.*

[15] SERVIEN E, FRITSCH B, LUSTIG S, *and al.* In vivo positioning analysis of medial patellofemoral ligament reconstruction. *Am J Sports Med. 2011; 39: 134-9.*

[16] SHEA KG, GRIMM NL, BELZER J, *and al.* The relation of the femoral physis and the medial patellofemoral ligament. *Arthroscopy 2010; 26: 1083-7.*

[17] YERCAN HS, ERKAN S, OKCU G, *and al.* A novel technique for reconstruction of the medial patellofemoral ligament in skeletally immature patients. *Arch Orthop Trauma Surg. 2011; 131: 1059-65.*



ACUTE PATELLAR DISLOCATION: WHICH EXAMINATION FOR WHICH MPFL LESION?

J.A. FELLER

Given that the medial patellofemoral ligament is the primary soft tissue restraint preventing lateral patellar dislocation [1, 2], it is not surprising that it is usually disrupted to some degree when the patella dislocates laterally. In the setting of acute dislocation, the reported rate of injury to the MPFL is up to 100% [3, 4]. The ligament appears to have a poor capacity to heal back to a near normal structure. It tends to heal in an elongated fashion and is thus often noted to be attenuated in the setting of recurrent patellar instability.

The precise anatomy of the MPFL has been extensively studied [5-11]. The patellar attachment is broader than the femoral attachment and extends over the proximal one half to two thirds of the medial border of the patella. The exact location of the femoral attachment has been variably described but is generally agreed to be in the region of the medial femoral epicondyle to the adductor tubercle.

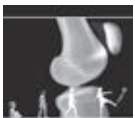
It has been reported that the MPFL is frequently disrupted at more than one site [12], particularly in children and adolescents [4]. However, the femoral attachment is the most common location of injury and occurs in up to 97% of cases of acute patellar dislocation [13]. The next most common site is the femoral attachment. This is relevant in terms of the location of tenderness in the acute setting.

Physical examination of the MPFL specifically is essentially comprised of palpation for tenderness, assessment of laxity of the MPFL, and identification of patellar apprehension. Ancillary testing includes assessment of generalized ligamentous laxity and the identification of predisposing factors for recurrent patellar instability.

The identification of tenderness is really only relevant to the acute patellar dislocation. No particular site is specific for MPFL injury, but tenderness in the region of the medial femoral epicondyle is most common [3]. However this may also be due to an injury to the medial collateral ligament, which may be co-existent and which will usually be associated with pain when a valgus stress is applied to the knee.

Tenderness at the medial border of the patella may be associated with injury to MPFL, but can also reflect capsular injury, tearing of the insertion of vastus medialis, synovial injury and associated haemorrhage, and osteochondral injury of the patella. Tenderness over the mid portion of the MPFL may similarly reflect MPFL injury as well as capsular injury.

MPFL insufficiency will result in a decreased resistance to lateral translation of the patella. Although the slope of the lateral facet of the trochlear groove provides the principal resistance to lateral translation [2], the MPFL



provides the most soft tissue restraint. Senavongse et al showed that division of the medial retinacular structures decreased the force required to laterally displace the patella by up to 50% [2]. The effect was significant between 0% and 20%, but was most marked at 0%.

Clinically, this can be assessed by measuring how far laterally the patella can be displaced, relative to the trochlear groove. Traditionally the patella is divided into four segments, each 25% of the width. The number of quadrants by which the patella can be displaced is recorded [14]. Two to three (25 to 50%) is considered normal. One or less represents tightness and three or more represents excessive laxity. The end point can also be assessed with a soft end point being suggestive of extensive disruption of the MPFL.

Attempts have been made to more accurately quantify the amount of lateral displacement, but there is considerable variation in the reported results. Kujala *et al.* reported a mean total mediolateral patellar translation of 31mm in healthy volunteers with the knee extended [15]. Joshi and Heatley reported a range of lateral patellar mobility of 8.3 to 19.6mm in women and 9 to 18.6mm in men [16]. Overall, they suggested a normal range of 8 to 20mm. Skalley *et al.* measured lateral patellar displacement with the knee in extension and at 35 degrees flexion, using both manual displacement and a mechanical "patellar pusher" [17]. They found manual displacement to be less variable and reported mean displacement values of 5.4mm in extension and 10mm in flexion.

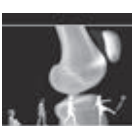
The variability in reported norms suggests that the measurement can be affected by a number of factors. This is also evident from the relatively poor reliability reported by Smith *et al.* [18]. They reported weighted Kappa indices of 0.43 ($p<0.01$) and 0.11 ($p=0.21$) for intra- and inter-observer reliability respectively. Similar concerns have been expressed by other authors [19, 20]. Difficulty in detecting MPFL deficiency in patients with patellar instability was also described by Garth *et al.* [21].

The size, specifically the width, of the patella will affect how the absolute distance is recorded. The starting point of the patella will also have an effect. It is in important to have the patella centrally placed in the trochlea before the lateral displacement is recorded. This can be difficult in the presence of a very flat trochlea. Fulkerson has noted that the impression of lateral displacement can be accentuated by inadvertent lateral tilting of the patella [22]. This can be countered by ensuring that the patella is kept aligned in a coronal plane throughout the maneuver.

The amount of force used will also have an impact, as will the relaxation of the patient. The test needs to be performed gently. In the acute setting, the point at which the patella is grasped on the medial side should be away form any tender site. In both the acute and chronic situations apprehension as the patella is displaced laterally may cause the patient to tense and contract their quadriceps muscle, thereby resisting movement of the patella.

Perhaps the most important consideration is the angle of knee flexion at which the test is performed. Testing the knee in extension reduces or eliminates the effect of the bony morphology of the patellofemoral joint, particularly in the presence of patella alta and trochlear dysplasia. It is also the angle at which the effect of the MPFL appears to be maximal [2]. However, it also makes it more difficult to determine the correct starting point for the patella. Testing with the knee in 20 degrees flexion, on the other hand, tests the MPFL at the angle at which the patella is most susceptible to dislocation. This is, however, offset to some degree by the effect of the slope of the lateral facet of the trochlea, which is greater the more the knee is flexed.

Testing the lateral displacement of the patella will at the same time give an indication of apprehension on the part of the patient. The apprehension test is well known and is regarded as a good indicator of persistent patellar instability.



Ahmad *et al.* described what they called the moving patellar apprehension test [23]. Essentially, a lateral displacement force is applied to the patellar as the knee is taken from extension into 90 degrees flexion and back to extension. The patient is observed for apprehension. In the second part of the test the knee is taken through the same range of motion, but with a medial displacement force applied to the patellar, which should eliminate apprehension in the patient with an incompetent MPFL. Compared to the ability to dislocate the patella under anesthesia, the moving patellar apprehension test was found to be highly sensitive, and both accurate and specific.

It is important to consider the amount of lateral translation of the patellar in terms of the overall ligamentous laxity of the patient. Generalised ligamentous laxity is assessed using the signs described by Beighton and Horan [24]. These are passive dorsiflexion of the little finger beyond 90 degrees with the forearm flat on a table, passive opposition of the thumb to the flexor aspect of the forearm, hyperextension of the elbow beyond 10 degrees, hyperextension of the knee beyond 10 degrees, and forward flexion of the trunk so that the palms of the hands can be rested easily on the floor. They can be scored by assigning a score of zero (absent) or one (present) to each of the tests that can be performed bilaterally as well as to trunk flexion [25]. A total score of 4 or more out of 9 is considered indicative of generalized ligamentous laxity.

In summary, the MPFL is almost always injured in an acute patellar dislocation. The most frequent site is the femoral attachment and this is often associated with tenderness. The second most frequent site is the patellar attachment, but multiple sites of injury have also been reported, especially in children and adolescents. Lateral patellar glide can be assessed with the knee in extension and also in 20 to 30 degrees flexion. Displacement of more than 50% of the width of the patella is considered abnormal. Patellar apprehension can be assessed at the same time. Consideration should also be given to the presence of generalized ligamentous laxity.

LITERATURE

[1] CONLAN T, GARTH WPJ, LEMONS JE. Evaluation of the medial soft-tissue restraints of the extensor mechanism of the knee. *J Bone Joint Surg Am. 1993rd ed. 1993 May; 75(5): 682-93.*

[2] SENAVONGSE W, AMIS AA. The effects of articular, retinacular, or muscular deficiencies on patellofemoral joint stability. The Journal of bone and joint surgery. *British volume. 2005th ed. 2005 Apr. 87(4): 577-82.*

[3] SALLAY PI, POGGI J, SPEER KP, GARRETT WE. Acute dislocation of the patella. A correlative pathoanatomic study. *Am J Sports Med. 1996 Jan. 24(1): 52-60.*

[4] BALCAREK P, WALDE TA, FROSCH S, SCHÜTTRUMPF JP, WACHOWSKI MM, STÜRMER KM, *et al.* Patellar dislocations in children, adolescents and adults: a comparative MRI study of medial patellofemoral ligament injury patterns and trochlear groove anatomy. *Eur J Radiol. 2011 Sep.; 79(3): 415-20.*

[5] MOCHIZUKI T, NIMURA A, TATEISHI T, YAMAGUCHI K, MUNETA T, AKITA K. Anatomic study of the attachment of the medial patellofemoral ligament and its characteristic relationships to the vastus intermedius. *Knee Surg Sports Traumatol Arthrosc. 2012 Apr. 11.*

[6] AMIS AA, FIRER P, MOUNTNEY J, SENAVONGSE W, THOMAS NP. Anatomy and biomechanics of the medial patellofemoral ligament. *Knee. 2003 Sep.; 10(3): 215-20.*

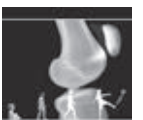
[7] FELLER JA, FEAGIN JA, GARRETT WE. The medial patellofemoral ligament revisited: an anatomical study. *Knee Surg Sports Traumatol Arthrosc. 1993;1(3-4): 184-6.*

[8] NOMURA E, INOUE M, OSADA N. Anatomical analysis of the medial patellofemoral ligament of the knee, especially the femoral attachment. *Knee Surg Sports Traumatol Arthrosc. 2005 Oct.; 13(7): 510-5.*

[9] SMIRK C, MORRIS H. The anatomy and reconstruction of the medial patellofemoral ligament. *Knee. 2003 Sep.; 10(3): 221-7.*

[10] STEENSEN RN, DOPIRAK RM, MCDONALD WG. The anatomy and isometry of the medial patellofemoral ligament: implications for reconstruction. *Am J Sports Med. 2004 Sep.; 32(6): 1509-13.*

[11] WARREN LF, MARSHALL JL. The supporting structures and layers on the medial side of the knee: an anatomical analysis. *J Bone Joint Surg Am. 1979 Jan.; 61(1): 56-62.*



[12] ELIAS DA, WHITE LM, FITHIAN DC. Acute lateral patellar dislocation at MR imaging: injury patterns of medial patellar soft-tissue restraints and osteochondral injuries of the inferomedial patella. *Radiology 2002 Dec.; 225(3): 736-43.*

[13] SALLAY PI, FRIEDMAN RL, COOGAN PG, GARRETT WE. Hamstring Muscle Injuries Among Water Skiers: Functional Outcome and Prevention. *Am J Sports Med. 1996 Mar. 1;24(2): 130-6.*

[14] KOLOWICH PA, PAULOS LE, ROSENBERG TD, FARNSWORTH S. Lateral release of the patella: indications and contraindications. *Am J Sports Med. 1990 Jul.; 18(4): 359-65.*

[15] KUJALA UM, KVIST M, OSTERMAN K. Knee injuries in athletes. Review of exertion injuries and retrospective study of outpatient sports clinic material. *Sports Med. 1986 Nov.; 3(6): 447-60.*

[16] JOSHI RP, HEATLEY FW. Measurement of coronal plane patellar mobility in normal subjects. *Knee Surg Sports Traumatol Arthrosc. 2000; 8(1):40-5.*

[17] SKALLEY TC, TERRY GC, TEITGE RA. The quantitative measurement of normal passive medial and lateral patellar motion limits. *Am J Sports Med. 1993 Sep.; 21(5): 728-32.*

[18] SMITH TO, CLARK A, NEDA S, ARENDT EA, POST WR, GRELSAMER RP, *et al.* The intra- and inter-observer reliability of the physical examination methods used to assess patients with patellofemoral joint instability. *Knee 2011 Jun. 27.*

[19] TOMSICH DA, NITZ AJ, THRELKELD AJ, SHAPIRO R. Patellofemoral alignment: reliability. *The Journal of orthopaedic and sports physical therapy. 1996 Mar.; 23(3): 200-8.*

[20] WATSON CJ, LEDDY HM, DYNJAN TD, PARHAM JL. Reliability of the lateral pull test and tilt test to assess patellar alignment in subjects with symptomatic knees: student raters. *The Journal of orthopaedic and sports physical therapy. 2001 Jul.; 31(7): 368-74.*

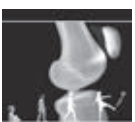
[21] GARTH WP, DICHRISTINA DG, HOLT G. Delayed proximal repair and distal realignment after patellar dislocation. *Clin Orthop Relat Res 2000 Aug.;(377): 132-44.*

[22] FULKERSON JP, BUUCK DA. Disorders of the patellofemoral joint. *2004. p. 374.*

[23] AHMAD CS, McCARTHY M, GOMEZ JA, SHUBIN STEIN BE. The moving patellar apprehension test for lateral patellar instability. *Am J Sports Med. 2009 Apr.; 37(4): 791-6.*

[24] BEIGHTON P, HORAN F. Orthopaedic aspects of the Ehlers-Danlos syndrome. The *Journal of bone and joint surgery. British volume. 1969 Aug.; 51(3): 444-53.*

[25] GRAHAME R, BIRD HA, CHILD A. The revised (Brighton 1998) criteria for the diagnosis of benign joint hypermobility syndrome (BJHS). *J Rheumatol. 2000 Jul.; 27(7): 1777-9.*



PRIMARY PATELLAR DISLOCATION AND MEDIAL PATELLOFEMORAL LIGAMENT INJURY

P. SILLANPÄÄ

First-time (primary) patellar dislocation commonly occurs in the young physically active population [1] and is associated with a high rate of recurrent patellar instability. Depending on the patient cohort, 44% to 70% patients sustain recurrent dislocations [2-4]. In patellar dislocation, the primary stabilizer, the MPFL, is frequently torn. A total or partial MPFL disruption can be seen by magnetic resonance imaging (MRI) [5, 6]. Biomechanical studies [7-9] have shown that the MPFL is the major ligamentous restraint against lateral patellar dislocation. The MPFL extends from the medial margin of the patella and attaches firmly to the femur between the adductor tubercle and the medial epicondyle [10, 11] The MPFL is estimated to contribute 50 to 60% of the restraining force against lateral patellar displacement [7-9] MPFL injury can be diagnosed using MRI [5, 6, 12, 13]. Primary patellar dislocations quite often involve an osteochondral fracture that may require surgical fixation [2, 14]. The significant variation in the injury pattern and other associated factors such as alignment, dysplasia and osteochondral injury can make treatment challenging. The majority of primary dislocations can be managed nonsurgically, although occasionally surgery is warranted and a thorough evaluation of each case is required [15]. The optimal strategy is not yet established.

When the patella dislocates laterally, the medial patellar restraints are injured, particularly the MPFL [1, 9, 16]. The force required to dislocate the patella most likely depends on the individual patellofemoral morphology [1, 17]. The incidence of primary patellar dislocation is reported to range from 6 to 112 per 100,000 persons depending on the age of the population [1, 16, 18-20]. The mechanism of injury is reported to be knee valgus stress and internal rotation of the femur with the foot fixed on the ground [1, 16, 17, 21]. The risk factors for primary patellar dislocation are tall height and excess weight (Table 1) [1]. The risk factors for recurrent patellar instability are well described and include anatomic predisposing factors; trochlear dysplasia, patella alta, variations of limb alignment, connective tissue laxity, and insufficiency of previously injured medial restraints [3, 13, 22].

Primary patellar dislocation has certain important signs that affect decision-making between surgical and nonsurgical methods. Radiographs are needed to evaluate patellar position and assess osteochondral fractures. Sagittal and anteroposterior views with axial Merchant [24] are used. MRI is recommended to assess the cartilage more precisely [25]. MPFL injury location can be assessed reliably only by MRI [6]. MPFL injuries are classified

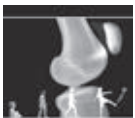


Table 1: Risk factors for acute traumatic primary patellar dislocations among military conscripts (median and standard deviation). Reprinted with permission of Wolters Kluwer Health (Sillanpaa P, Mattila VM, Iivonen T, et al. Incidence and risk factors of acute traumatic primary patellar dislocation. Med Sci Sports Exerc. 2008;40: 606-611.) [1]

Risk factor	Acute traumatic primary patellar dislocation (n=75)	Healthy controls (n=130,421)	p-value
Sex - Male - Female	 73 2	 128,642 1,992	0.283
Age (y)	19.8 (0.8)	20.0 (1.3)	0.287
Height (cm)	180.3 (7.2)	178.5 (6.7)	0.031*
Weight (kg)	77.2 (4.3)	73.2 (12.7)	0.014*
Muscle strength	16.4 (3.5)	16.4 (3.6)	0.960
Run test (m)	2500 (371)	2520 (355)	0.703
Body mass index	23.7 (3.8)	22.9 (3.5)	0.105

* considered significant with p-value < 0.05

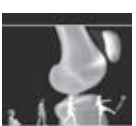
in three categories based on location: at the level of the MPFL patellar insertion, at the midsubstance of the MPFL and medial retinaculum, and at the femoral origin of the MPFL (Table 2) [6, 12]. Therefore, MRI is recommended in cases of primary dislocation to verify the diagnosis, evaluate additional injuries, and, importantly, describe the anatomic factors of the patellofemoral joint [6, 26-30].

Prospective studies report variable results after surgical treatment for patellar dislocation [3]. Two prospective randomized studies described better patellar stability after MPFL repair compared with conservative treatment [5, 31]. However, only one study described clinically significant improvement in subjective outcome [31]. The recent study included MPFL reinsertion with anchors [31], showing more favorable results with surgical treatment. All of the prospective randomized studies utilized different kinds of MPFL repair [5, 21, 23, 31, 33]. To date, no study has compared MPFL reconstruction to nonoperative treatment in a prospective and randomized study setting.

MPFL repair by sutures is not better than nonsurgical treatment, and does not decrease recurrent instability rate in skeletally immature children and do not improve subjective results in adults [5, 21, 23]. Acute arthroscopic MPFL repair is also not superior to nonsurgical management [12]. Arthroscopic repair is likely an insufficient method to approach all the MPFL injury locations. Delayed repair is usually not targeted to the previous injury location and is therefore not useful [32-34].

Table 2: Classification on medial patellofemoral ligament (MPFL) injuries based on injury location and reported incidence in the literature [6, 26-30]

MPFL injur classification	Anatomical description	Proportion in primary dislocations	Mean reported incidence
Patellar	MPFL patellar attachment	13-76%	54%
Midsubstance	MPFL midsubstance (region between patellar and femoral attachments)	0-30%	12%
Femoral	MPFL femoral attachment	12-66%	34%



The clinical importance of an MPFL injury location was evaluated in one study [28]. In that study, MPFL disruption at the femoral attachment was related to more frequent subsequent instability than patellar or midsubstance MPFL injuries [28]. The study is limited by its retrospective nature and the use of only male subjects. MPFL patellar attachment injury was recently reported to be at least as common as femoral attachment injury [6, 27, 29, 30]. Midsubstance MPFL disruption as an independent injury location is less common [6, 28]. Partial tears or wavy features of the midsubstance MPFL structure are commonly seen in cases of patellar or femoral attachment MPFL disruption [6, 30] Patellar MPFL injury may include an osteochondral avulsion fracture, in which the MPFL structure is relatively intact and attached to the avulsed bone from the medial margin of the patella [30]. In some cases, articular cartilage involvement is seen [14] (fig. 1). Based on these previous findings, MPFL repair may be considered more unreliable than MPFL reconstruction, in terms of providing sufficient medial soft tissue stability.

MPFL patellar or femoral attachment injury can be surgically reinserted with satisfying results and may lead to a better outcome than nonsurgical treatment [31], although some controversy exists in the results of prospective studies [5, 21, 23, 33]. MPFL midsubstance injuries seem to not benefit from acute repairs [28]. MPFL injury at the femoral or patellar attachment can be repaired with sutures or suture anchors (fig. 2) [31]. Midsubstance MPFL injury is difficult to repair adequately and repair is not recommended [5, 21]. Midsubstance MPFL injury should be repaired only in rare cases with extensive VMO fascial disruption in a high-energy dislocation. Patellar attachment MPFL injury can be classified as a ligamentous or bony avulsion from the medial margin of the patella [28]. A third type includes an osteochondral fragment with articular cartilage involvement from the medial patella (fig. 1). According to a retrospective study, ligamentous patellar MPFL avulsion is not associated with an increased rate of recurrent instability compared with femoral MPFL avulsion injury with similar nonsurgical management [28]. Articular cartilage involvement can be considered an indication for surgery, and cartilage defects should be repaired by reduction and fixation of the fragment [14, 15].

In some cases, the MPFL may be injured in two locations [6]. Most likely, MPFL patellar or femoral attachment disruption can be accompanied by midsubstance total or partial tear. Therefore, MPFL reconstruction may be

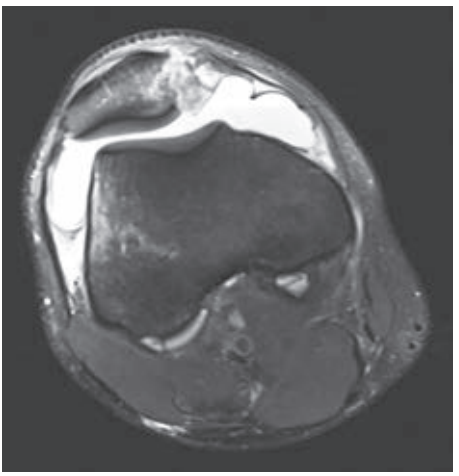


Fig. 1: Medial osteochondral avulsion fracture with articular surface involvement (at the patellar insertion of the medial patellofemoral ligament). Axial proton-density magnetic resonance image.

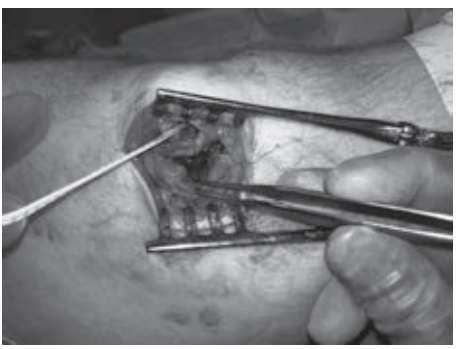
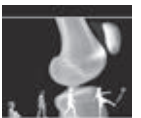


Fig. 2: Medial patellofemoral ligament patellar attachment injury with avulsion fracture from the medial patellar margin. Suture anchors have been inserted to the patella for injury repair.



more reliable surgical method than MPFL repair, which typically address only single injury location. MPFL reconstruction requires experience in patellofemoral surgery and may be considered technically more demanding than repair. Current evidence for MPFL repair is relatively limited compared to that for MPFL reconstruction. MPFL repair cannot be performed without MRI verification of the injury location and repair is inappropriate if performed later in cases of chronic patellar instability [32]. Repair is perhaps best indicated in acute cases with MRI-verified patellar or femoral attachment MPFL disruption within a few weeks of the injury. An additional indication for surgical repair could be an osteochondral fragment from the medial patella with simultaneous patellar attachment MPFL avulsion (fig. 1).

In conclusion, primary patellar dislocation leads to MPFL injury. MPFL injuries can occur in different locations and may be combined with an osteochondral avulsion fracture of the medial patellar margin or an impact fracture, which should be treated with initial fixation of the fragment. MRI is recommended for assessment of the MPFL injury and the exclusion of additional injuries. Current evidence supports nonoperative management for primary patellar dislocation in majority of the cases. Surgery should probably be considered for MPFL patellar or femoral attachment disruptions in cases with one or more dysplastic features, especially if patella is highly unstable after dislocation. In some cases, osseous corrections may be needed, but the majority will stabilize with MPFL reconstruction alone. Due to the complex nature of patellar instability, treatment of primary patellar dislocation is challenging.

ABSTRACT

Primary patellar dislocation results in medial patellofemoral ligament (MPFL) injury. MPFL is the major soft tissue stabilizer of the patella and MPFL injury may lead to recurrent patellar instability. The variation in location of injury of the MPFL and the presence of an osteochondral fracture produces challenges in clinical decision making between nonoperative and operative treatment, including the surgical modality, to repair or reconstruct the MPFL. Current evidence suggests that not all primary dislocations should undergo the same treatment. MPFL reconstruction may theoretically be more reliable than repair. Femoral attachment MPFL injury has been reported to be a risk factor for recurrent patellar instability.

REFERENCES

[1] SILLANPAA P, MATTILA VM, IIVONEN T, *et al.* Incidence and risk factors of acute traumatic primary patellar dislocation. *Med Sci Sports Exerc. 2008; 40: 606-11.*

[2] STEFANCIN JJ, PARKER RD. First-time traumatic patellar dislocation: a systematic review. *Clin Orthop Relat Res. 2007; 455: 93-101.*

[3] SMITH TO, SONG F, DONELL ST, *et al.* Operative versus non-operative management of patellar dislocation. A meta-analysis. *Knee Surg Sports Traumatol Arthrosc. 2011;19: 988-98.*

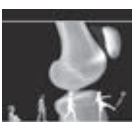
[4] HING CB, SMITH TO, DONELL S, *et al.* Surgical versus non-surgical interventions for treating patellar dislocation. *Cochrane Database Syst Rev. 2011; 11: CD008106.*

[5] SILLANPAA PJ, MATTILA VM, MAENPAA H, *et al.* Treatment with and without initial stabilizing surgery for primary traumatic patellar dislocation. A prospective randomized study. *J Bone Joint Surg Am. 2009; 91: 263-73.*

[6] ELIAS DA, WHITE LM, FITHIAN DC. Acute lateral patellar dislocation at MR imaging: injury patterns of medial patellar soft-tissue restraints and osteochondral injuries of the inferomedial patella. *Radiology. 2002; 225: 736-43.*

[7] CONLAN T, GARTH WP Jr, LEMONS JE. Evaluation of the medial soft-tissue restraints of the extensor mechanism of the knee. *J Bone Joint Surg Am. 1993; 75: 682-93.*

[8] DESIO SM, BURKS RT, BACHUS KN. Soft tissue restraints to lateral patellar translation in the human knee. *Am J Sports Med. 1998; 26: 59-65.*



[9] HAUTAMAA PV, FITHIAN DC, KAUFMAN KR, *et al.* Medial soft tissue restraints in lateral patellar instability and repair. *Clin Orthop. 1998: 174-82.*

[10] LAPRADE RF, ENGEBRETSEN AH, LY TV, *et al.* The anatomy of the medial part of the knee. *J Bone Joint Surg Am. 2007; 89: 2000-10.*

[11] WARREN LF, MARSHALL JL. The supporting structures and layers on the medial side of the knee: an anatomical analysis. *J Bone Joint Surg Am. 1979; 61: 56-62.*

[12] SILLANPAA PJ, MAENPAA HM, MATTILA VM, *et al.* Arthroscopic surgery for primary traumatic patellar dislocation: a prospective, nonrandomized study comparing patients treated with and without acute arthroscopic stabilization with a median 7-year follow-up. *Am J Sports Med. 2008; 36: 2301-9.*

[13] DEJOUR H, WALCH G, NOVE-JOSSERAND L, *et al.* Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc. 1994; 2: 19-26.*

[14] SLABAUGH MA, HESS DJ, BAJAJ S, *et al.* Management of Chondral Injuries Associated with Patellar Instability. *Oper Tech Sports Med. 2010;18 115-22.*

[15] MEHTA VM, INOUE M, NOMURA E, *et al.* An algorithm guiding the evaluation and treatment of acute primary patellar dislocations. *Sports Med Arthrosc. 2007; 15: 78-81.*

[16] COLVIN AC, WEST RV. Patellar instability. *J Bone Joint Surg Am. 2008; 90: 2751-62.*

[17] NIKKU R, NIETOSVAARA Y, AALTO K, *et al.* The mechanism of primary patellar dislocation: trauma history of 126 patients. *Acta Orthop. 2009; 80: 432-4.*

[18] HSIAO M, OWENS BD, BURKS R, *et al.* Incidence of acute traumatic patellar dislocation among active-duty United States military service members. *Am J Sports Med. 2010; 38: 1997-2004.*

[19] FITHIAN DC, PAXTON EW, STONE ML, *et al.* Epidemiology and natural history of acute patellar dislocation. *Am J Sports Med. 2004; 32: 1114-21.*

[20] NIETOSVAARA Y, AALTO K, KALLIO PE. Acute patellar dislocation in children: incidence and associated osteochondral fractures. *J Pediatr Orthop. 1994;14: 513-15.*

[21] NIKKU R, NIETOSVAARA Y, AALTO K, *et al.* Operative treatment of primary patellar dislocation does not improve medium-term outcome: A 7-year follow-up report and risk analysis of 127 randomized patients. *Acta Orthop. 2005; 76: 699-704.*

[22] MÄENPAA H, LEHTO MU. Patellar dislocation has predisposing factors. A roentgenographic study on lateral and tangential views in patients and healthy controls. *Knee Surg Sports Traumatol Arthrosc. 1996; 4: 212-16.*

[23] PALMU S, KALLIO PE, DONELL ST, *et al.* Acute patellar dislocation in children and adolescents: a randomized clinical trial. *J Bone Joint Surg Am. 2008; 90: 463-70.*

[24] MERCHANT AC, MERCER RL, JACOBSEN RH, *et al.* Roentgenographic analysis of patellofemoral congruence. *J Bone Joint Surg Am. 1974; 56: 1391-6.*

[25] SMITH TO, DREW BT, TOMS AP, *et al.* Accuracy of magnetic resonance imaging, magnetic resonance arthrography and computed tomography for the detection of chondral lesions of the knee. *Knee Surg Sports Traumatol Arthrosc. 2012.*

[26] ZAIDI A, BABYN P, ASTORI I, *et al.* MRI of traumatic patellar dislocation in children. *Pediatr Radiol. 2006; 36: 1163-70.*

[27] WEBER-SPICKSCHEN TS, SPANG J, KOHN L, *et al.* The relationship between trochlear dysplasia and medial patellofemoral ligament rupture location after patellar dislocation: an MRI evaluation. *Knee 2011; 18: 185-8.*

[28] SILLANPAA PJ, PELTOLA E, MATTILA VM, *et al.* Femoral avulsion of the medial patellofemoral ligament after primary traumatic patellar dislocation predicts subsequent instability in men: a mean 7-year nonoperative follow-up study. *Am J Sports Med. 2009; 37: 1513-21.*

[29] KEPLER CK, BOGNER EA, HAMMOUD S, *et al.* Zone of injury of the medial patellofemoral ligament after acute patellar dislocation in children and adolescents. *Am J Sports Med. 2011; 39: 1444-49.*

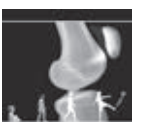
[30] GUERRERO P, LI X, PATEL K, *et al.* Medial patellofemoral ligament injury patterns and associated pathology in lateral patella dislocation: an MRI study. *Sports Med Arthrosc Rehabil Ther Technol. 2009;1: 17.*

[31] CAMANHO GL, VIEGAS ADE C, BITAR AC, *et al.* Conservative versus surgical treatment for repair of the medial patellofemoral ligament in acute dislocations of the patella. *Arthroscopy. 2009; 25: 620-5.*

[32] ARENDT EA, MOELLER A, AGEL J. Clinical outcomes of medial patellofemoral ligament repair in recurrent (chronic) lateral patella dislocations. *Knee Surg Sports Traumatol Arthrosc. 2011;19: 1909-14.*

[33] CHRISTIANSEN SE, JAKOBSEN BW, LUND B, *et al.* Isolated repair of the medial patellofemoral ligament in primary dislocation of the patella: a prospective randomized study. *Arthroscopy. 2008; 24: 881-7.*

[34] CAMP CL, KRYCH AJ, DAHM DL, *et al.* Medial patellofemoral ligament repair for recurrent patellar dislocation. *Am J Sports Med. 2010; 38: 2248-54.*



ACUTE PATELLAR DISLOCATION. MINI- BATTLE- CONSERVATIVE TREATMENT VERSUS SURGICAL TREATMENT

G.L. CAMANHO

INTRODUCTION

Acute dislocation of the patella is an infrequent injury accounting for only 2 to 3% of cases of knee trauma [1]. Its treatment was classically always conservative until the medial patello-femoral ligament (MPFL) was described and became known. This stimulated some authors to study repair or reconstruction of this ligament as a treatment for acute dislocation of the patella.

In the literature, few studies reported on surgical treatment for acute dislocation of the patella before the MPFL became known. In 1978, Boring and O'Donoghue [2] reported suturing the medial retinaculum in 18 patients who evolved without recurrence of their acute dislocations.

Conservative treatment leads to recurrence rates ranging from 15 to 44% [1, 3].

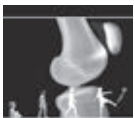
We started our work on the MPFL by seeking data through an anatomical and biomechanical study [4]. From this, we assessed our results that had been obtained from conservative treatment and then we analyzed repair and reconstruction of the MPFL.

ANATOMICAL STUDIES ON THE MEDIAL PATELLO-FEMORAL LIGAMENT

We began our studies on the MPFL in the 1990s, with an anatomical and arthroscopic study on 15 cadavers in order to identify it. We found the MPFL in all the specimens and described it anatomically and under arthroscopic viewing [4].

We found that there was a variable femoral insertion, which was always close to the epicondyle and to the tubercle of the adductors, but with variation in its width and size. The origin in the patella was, in all cases, at the transition between the proximal and middle thirds. We did not observe any significant variations in the insertions in the patella (fig. 1).

Our biomechanical studies demonstrated that the tensile strength of the MPFL was around 80 N, and that in resistance tests under axial traction, rupture occurred in the substance of the ligament and at its femoral insertion. It did not occur at the patellar insertion in any case in the series that we studied.



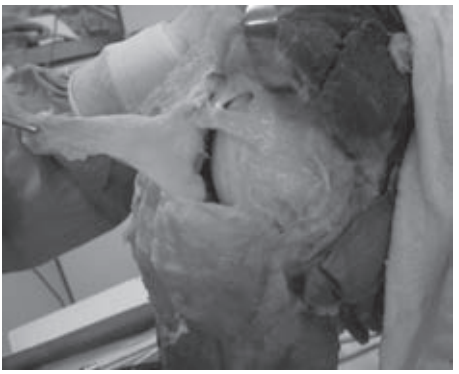


Fig. 1 : Medial patellofemoral ligament deinserted from the femur while maintained in its patellar insertion.

In studies on lateral traction that we conducted in order to determine the best fixation position for MPFL reconstruction using the patellar tendon, we found that although the ligament was not isometric, the best fixation position was at 60 degrees of flexion, at which there was less lateralization of the patella.

CLINICAL EXPERIENCE

Non-randomized study

After gaining anatomical knowledge on the MPFL, we moved on to study the evolution of patients with acute patellofemoral dislocation. In a non-randomized manner, we analyzed the evolution of 16 patients who had been treated conservatively and 17 who had been treated surgically [5]. The study was conducted in a public hospital, among patients of low social condition, which caused follow-up difficulties, especially among the patients who had been treated conservatively.

Conservative treatment

This treatment was provided for 16 patients who had been radiographically examined in anteroposterior, lateral and axial patellar views. These radiographs allowed us to investigate the presence of factors that might predispose these patients to patellofemoral instability. In accordance with Dejour *et al.* [6], we took into consideration three predisposing factors: flat trochlea on lateral or axial x-rays and high patella on lateral X-rays. In this group, 14 patients had at least one predisposing factor.

The conservative treatment was administered in the following manner:

1) Aspiration of hemarthrosis when present and voluminous
2) Immobilization for three weeks
3) Rehabilitation program until movement and muscle strength had been recovered

The results showed that after a minimum follow-up of 26 months (mean of 36.5), we had eight patients with recurrent episodes of dislocation and six patients with complaints of some degree of instability.

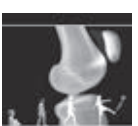
Surgical treatment

The surgical treatment was administered to 17 patients. All these patients underwent magnetic resonance imaging (MRI) in order to diagnose the lesion. We analyzed the MRI and decided whether the lesion was in the patella or in the femur. This decision was made by one of the surgeons.

Nine patients had a MPFL at the patellar insertion. In these cases, the ligament was repaired by reinserting the ligament in the patella by means of direct suturing or under arthroscopic viewing (fig. 2).

In seven patients, the avulsion of the MPFL was interpreted as if it was in the femur, and the ligament was reinserted using anchors.

The results showed that there were no episodes of dislocation, but three patients reported instability with symptoms of subluxation, in the group in which the MPFL had been reinserted into the femur.



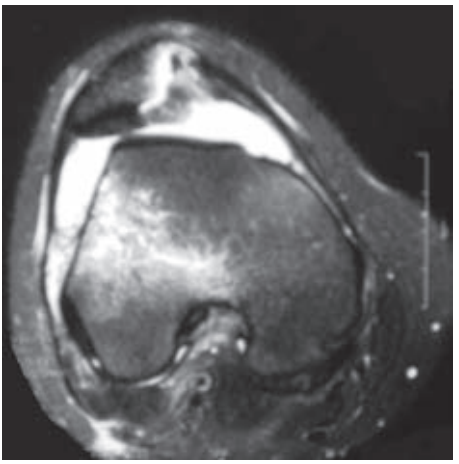


Fig. 2 : Magnetic resonance imaging demonstrating patellar dislocation with signs of bruising of the lateral femoral condyle and fracturing due to tearing of the medial edge of the patella.

The Kujala score was 69 in the conservatively treated group and 92 in the surgically treated group.

Our results from conservative treatment greatly resembled those of Maenpaa and Letto [3], who found that 44% of their patients presented recurrent dislocation over a 13-year follow-up period.

In analyzing this series, we found that the conservative treatment did not follow any pattern. The patients' adherence to use of immobilization and physiotherapy was variable. The patients who were treated surgically presented worse results when their deinsertions occurred in the ligament substance and in the femoral insertion. This result is in agreement with the observations of Sillanpaa *et al.* [7], in a study on deinsertions of the MPFL in the femur, who also considered that these had a worse prognosis.

The better results from surgical treatment than from conservative treatment, with absence of recurrent dislocation, were in agreement with those of Nomura *et al.* [8], Ahmad *et al.* [9] and Sallay *et al.* [10].

Randomized study

We organized a second series, now in the form of a randomized study, in which one group of orthopedists had the assignment of guiding and following up conservative treatment and other group, surgical treatment. Radiography and MRI were performed on all the patients, who were then divided into two groups [11].

The surgical treatment consisted of a MPFL reconstruction technique using a 0.5cm strip from the medial patellar tendon, which was kept inserted in the proximal third of the patella (fig. 3) and was fixed in the region between the medial femoral epicondyle and the tubercle of

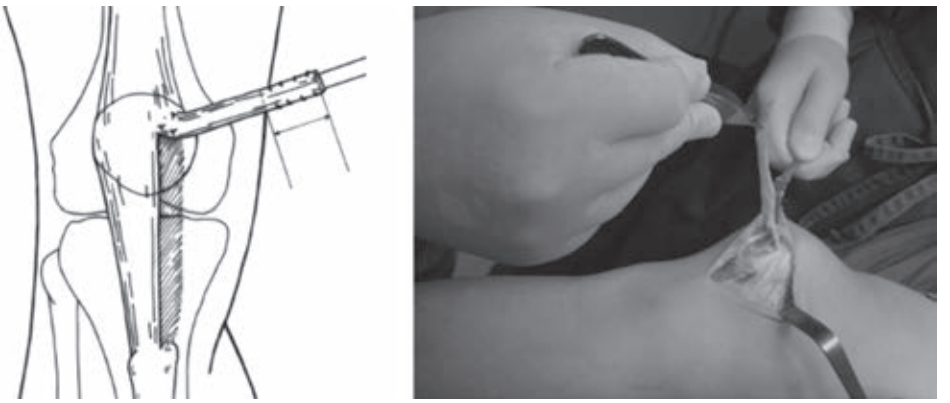
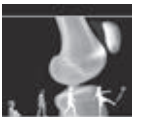


Fig. 3 : Harvesting of 0.5cm graft from the patellar ligament after its deinsertion from the tibia.



the adductors using an interference screw or anchors, with the knee at 60 degrees of flexion. The vastus medialis muscle was dissected and sutured to the new ligament in all cases [12] (fig. 4).

We treated 20 patients conservatively, with at least two years of follow-up, and 21 patients with MPFL reconstruction, also with a minimum follow-up of two years.

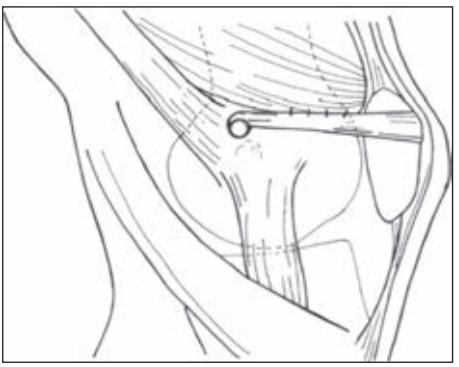


Fig. 4 : Diagram showing insertion of the graft from the patellar tendon, while maintaining its origin in the proximal third of the patella, and demonstrating the suturing of the vastus medialis muscle of the neo-ligament.

The conservative treatment consisted of intermittent use of a brace for three weeks, along with a rehabilitation program that continued until the patient's range of motion and muscle strength had been restored. The mean duration of these patients' treatment was three months. Among the results from this conservative treatment, there were five patients with recurrent dislocation and three patients with complaints of instability.

We performed MPFL reconstruction on 21 patients, using a previously described technique [12] in all cases. After a minimum follow-up of two years, there were no cases of recurrence and no complaints of instability in this group. Our results were concordant with those of Sillanpaa *et al.* [13].

We took occurrences of a high patella and signs of a flat trochlea on true lateral and axial radiographs to be predisposing factors [6]. However, in this group of 41 patients, the presence of predisposing factors did not affect the results, with regard to recurrence of dislocation or complaints of instability.

We used the Kujala score and observed that patients treated surgically had better scores. With regard to predisposing factors, there was a difference in quantitative Kujala scores, which were worse in patients with predisposing factors, especially in cases of a flat trochlea.

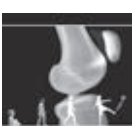
COMMENTS

We described and studied the MPFL at the end of the 1990s. Subsequently, we decided to assess our results from conservative treatment for acute dislocation of the patella, which was the method of choice until that time.

From a retrospective study on 16 patients, we found that there was a high percentage of recurrence. We chose to operate the patients with acute dislocation of the patella, thus repairing the lesions when this was anatomically possible.

In that initial series, we reinserted the MPFL in the patella when we judged that the lesion was closer to the patella, or in the femur when we judged that the lesion was closer to the femoral insertion. There were cases in which the lesion was diffuse and it was difficult to distinguish its real anatomical site. We found that there was a significant improvement in the results, in comparison with conservative treatment. A further group presented complaints of instability in situations of reinsertion in lesions due to deinsertion close to the femur. We therefore developed a reconstruction technique that used a medial strip from the patellar tendon, for cases in which the lesion was diffuse.

We began a series of 41 patients with a multicenter randomized study in which the patients who were treated conservatively were



followed up by the same group of orthopedists. The patients who were operated always underwent ligament reconstruction using the same technique, always performed by the same surgeons.

The results demonstrated that conservative treatment led to instability or recurrence in 40% of the cases, while surgical treatment did not lead to any recurrences or complaints of instability.

CONCLUSION

We conclude that in active patients, acute dislocation of the patella should be treated surgically, and that the method of choice is MPFL reconstruction. In cases of deinsertion of the MPFL from the patella, with a clear diagnosis using MRI, the ligament can be reinserted in the patella.

LITERATURE

[1] STEFANCIN JJ, PARKER RD. First time traumatic patelar dislocation:a systematic review. *Clin Orthop Relat Res. 2007;(455): 93-101.*

[2] BORING TH, O'DONOGHUE DH. Acute patellar dislocation:Results of immediate surgical repair. *Clin Orthop Relat Res. 1978;(136): 182-5.*

[3] MAENPAA H, LETTO MU. Patellar dislocation:the long term results of non-operative management in 100 patients. *Am J Sports Med. 1997; 25(2): 213-37.*

[4] CAMANHO GL, VIEGAS AC. Estudo anatomico e artroscopico do ligamento femoro patellar medial. *Acta Ortop Bras. 2003: 11(3); 145-9.*

[5] CAMANHO GL, VIEGAS AC, BITAR AC, DEMANGE MK, HERNANDEZ AJ. Conservative versus surgical treatment for repair of the medial patellof emoral ligament in acute dislocation of the patela. *Arthroscopy. 2009: 25(6): 620-5.*

[6] DEJOUR H, WALCH G, NOVE-JOSSERAND L, GUIER C. Factors of patellar instability ;an anatomical radiographic study. *Knee Surg Sports Traumatol Arthrosc. 1994;2(1): 19-26.*

[7] SILLANPPA PJ, PEITOLA E, MATTILE VM, KIURU M, PIHLAJAMAKI H. Femoral avulsion of the medial patellofemoral ligament after primary traumatic patellar dislocation predicts subsequent instability in men: a mean 7-years nonoperative follow up study. *Am J Sports Med. 2009, 37(8); 1513-21.*

[8] NOMURA E, INOUE M, OSADA N. Augmented repair of avulsion-tear type medial patellofemoral ligament injury in acute patellar dislocation. *Knee Surg Sports Traumatol Arthrosc. 2005; 13(5): 340-51.*

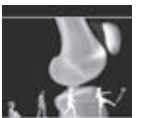
[9] AHMAD CS, STEIN BE, MATUZ D, HENRY JH. Immediate surgical repair of the medial patellar stabilizers for acute dislocation. A review of eight cases. *Am J Sports Med. 2000; 28(6): 804-10.*

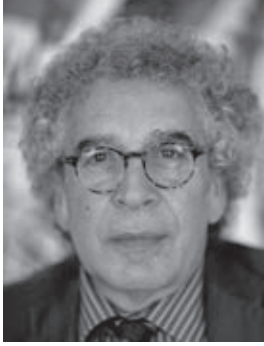
[10] SALLAY PI, POGGI J, SPEER KP, GARRET WE. Acute dislocation of the payella: an pathoanatomic study. *Am J Sports Med. 1996; 24(1): 52-60.*

[11] BITAR AC, DEMANGE MK, D'ELIA CO, CAMANHO GL. Traumatic Patellar dislocation: nonoperative treatment compared with MPFL reconstruction using patelar tendon. *Am J Sports Med. 2012; 40(1): 114-22.*

[12] CAMANHO GL, BITAR AC, HERNANDEZ AJ, OLIVI R. Medial patello femoral ligament: a novel technique using the patellar ligament. *Arthroscopy. 2007; 23(1): 108.e1-4.*

[13] SILLANPAA PJ, MATTILE VM, KIURU M, VISURI T, PIHLAJAMAKI H. Treatment with and withut initial stabilizing surgery for primary traumatic patellar dislocation. A prospective randomized study. *J Bone Joint Surg Am. 2009; 91(2); 263-73.*





HISTOIRE D'INDEX

J. CATON, G. DESCHAMPS

INTRODUCTION

Externe, interne puis assistant chef de clinique, je me suis toujours intéressé aux problèmes rotuliens et étais très intrigué par le terme de *Patella Baja*, néologisme barbare mélange de latin et d'Espagnol dont nous pensons que l'appellation *Patella Infera* est plus appropriée, G. Deschamps en a d'ailleurs fait sa thèse [13].

HISTORIQUE

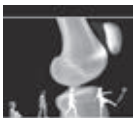
Au sein du service, dans les années 70, la hauteur rotulienne était appréciée selon l'ancienne méthode de Blumensaat (fig. 4) (1938) aussi lorsque mon patron Albert Trillat me proposa comme sujet de thèse en 1976 "les ruptures du système extenseur du genou, fracture de la rotule exceptée" (fig. 1) [8], je me suis trouvé confronté à ce problème de définition d'une bonne hauteur rotulienne fiable, ce que ne me permettait pas les mesures plus anciennes compte tenu de la qualité assez médiocre des radiographies du genou à l'époque, pas toujours de profil, développées à la main et avec un degré de flexion aléatoire de 0 à 90°.

Mes amis Gilles Melere et François Lecuire s'étaient trouvés également dans cette situation dans leurs thèses respectives sur "les fractures des plateaux tibiaux (thèse Lyon 1976) et sur le genu recurvatum et son traitement par ostéotomie tibiale" (thèse Lyon 1976).

Aussi, après avoir examiné les 130 dossiers sélectionnés pour ma thèse provenant essentiellement des services de A. Trillat, H. Dejour, G. Bousquet et C.R. Michel, mon premier travail fut, devant l'impossibilité d'utiliser soit la méthode de Blumensaat (pas de profil strict et flexion variable) soit la méthode d'Insall et Salvati (TTA pas toujours visible et flexion variable supérieure à 30°) de trouver une nouvelle méthode remplissant au moins 2 conditions : repère fixe indépendant de la qualité des radios et indépendant du degré de flexion du genou entre 10 et 80°. Ceci me conduisit à une première méthode de mesure avec prise en compte de la longueur rotulienne (idem Insall et Salvati) et un repère fixe du tibia (bord antéro-supérieur) la notion de rapport effaçant l'influence du degré de flexion (fig. 2).

Ceci me permit également de définir la notion de rotule haute et de rotule basse à partir des données de ce nouvel index ce que ne permettait ni la méthode de Blumensaat, ni celle d'Insall et Salvati qui n'avaient pas encore défini de hauteurs seuils.

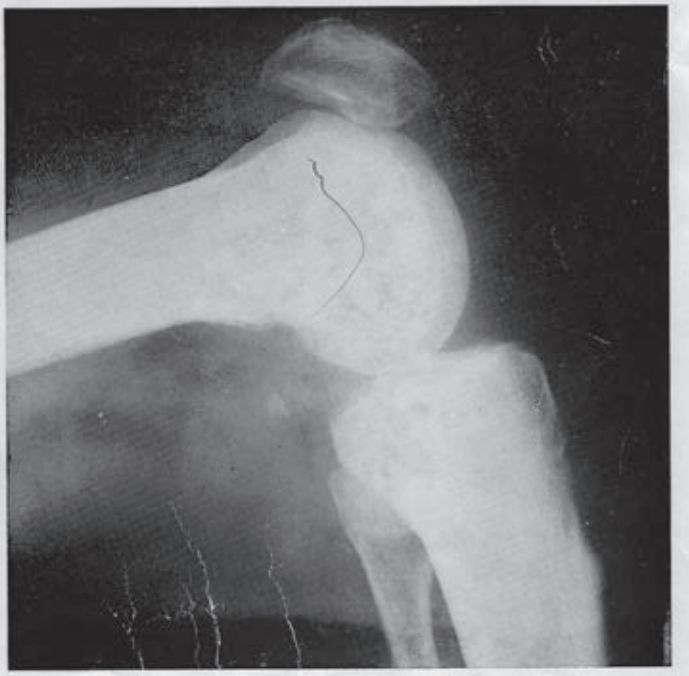
Selon cet index (fig. 2) la hauteur rotulienne était pour moi normale si la distance PT (pointe de la rotule bord antéro-supérieur du tibia) était



Jacques CATON
Interne Médaille d'Or des Hôpitaux de Lyon

LES RUPTURES DU SYSTEME EXTENSEUR DU GENOU

(Fractures de la rotule exceptées)
à propos de 130 observations

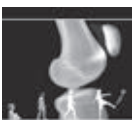


Travail de la Clinique de Chirurgie Orthopédique et Traumatologique de Lyon
Professeur A. TRILLAT

du Service d'Orthopédie Traumatologie d'urgence de Lyon
Professeur Agrégé H. DEJOUR

du Service d'Orthopédie Traumatologie de Saint-Étienne
Professeur Agrégé G. BOUSQUET

Fig. 1 : Couverture thèse J. Caton Lyon 1977.

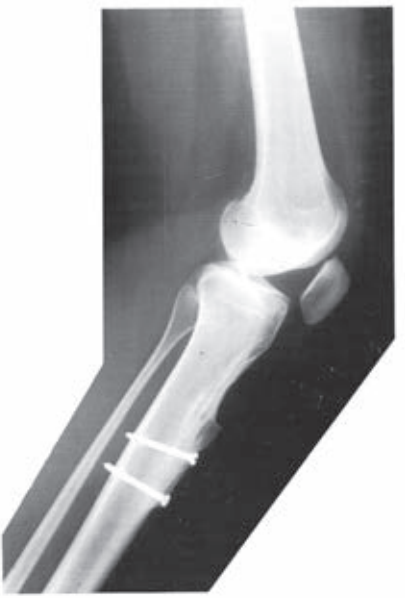


Gérard DESCHAMPS
Interne des Hôpitaux de Lyon

LES ROTULES BASSES

ETUDE ETIOPATHOGENIQUE-CLINIQUE ET PROPOSITIONS THERAPEUTIQUES

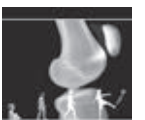
A PROPOS DE 131 OBSERVATIONS



Travail de la Clinique de Chirurgie Orthopédique
et de Traumatologie

Professeur H. DEJOUR
Docteur J. L. LERAT

Fig. 3 : Couverture thèse G. Deschamps, Lyon 1981.



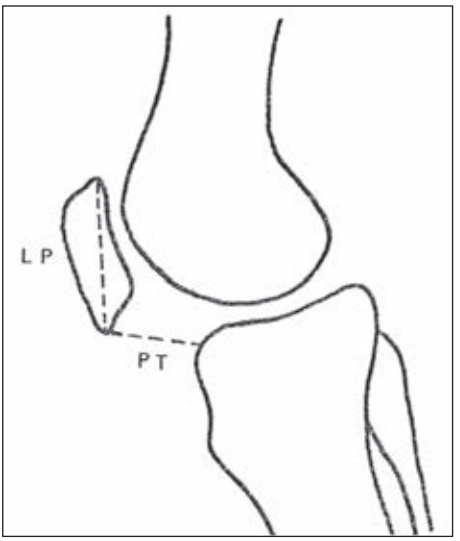


Fig. 2 : Index de Caton (1re génération) extrait de sa thèse sur les ruptures du système extenseur du genou, Lyon 1981.

égale à la moitié de la longueur rotulienne LP, la rotule étant haute si PT/LP était > ou égale à 1 et basse si PT/LP était < ou égale à 0,3 [8].

Pour la première fois nous pouvions quantifier ces données et les corrections afférentes pour mettre la rotule à un niveau normal après une rotule haute ou basse.

Ma thèse passée en mai et mon travail fini je vois publier dans le JBJS(B) de juin la méthode de Blackburne et Peel [3] assez similaire à celle que je venais de décrire dans ma thèse avec, comme référence, le plateau tibial. Blackburne et Peel éliminaient également la pointe rotulienne, facteur possible d'erreur lorsqu'elle était pathologique. Dans le service nous nous intéressions aussi à ce problème et ainsi lorsque je fus chargé de diriger la thèse de Gérard Deschamps [13] sur les rotules basses (*Patella Infera*) nous nous accordions pour être encore plus précis dans l'index que j'avais défini en éliminant la pointe rotulienne que nous appelions le "nez rotulien", définissant ainsi un nouveau rapport plus articulaire de la rotule ; le point P était transformé en point A (bord inférieur de la surface articulaire) conservation du point T et un rapport AT/AP égal à 1. La rotule étant basse à partir d'un index inférieur ou égal à 0,8 et haute si ce rapport était supérieur ou égal à 1 (fig. 4).

Ainsi est né l'index de Caton et Deschamps utilisé maintenant par une grande majorité de chirurgiens orthopédistes adultes ou enfants [21] dans le monde entier depuis plus de trente ans.

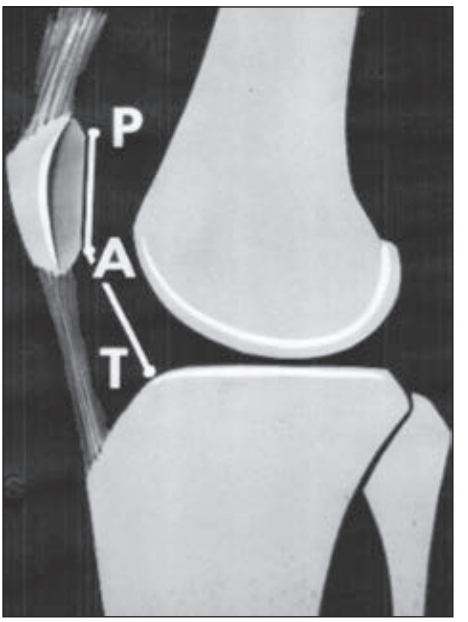
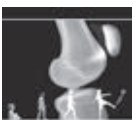


Fig. 4 : Méthode de Caton et Deschamps.

ETUDE CRITIQUE DE LA MESURE DE LA HAUTEUR DE LA ROTULE

La première méthode utilisée a été la méthode de Jansen (1929). Il s'agissait d'une méthode clinique, la pointe de la rotule devant se trouver à un travers de doigt au-dessus de l'interligne du genou de face. La première méthode chiffrée a été la méthode de Boon-Itt (1930) [5]. Cette méthode trop complexe n'a malheureusement jamais été appliquée. Ces méthodes peuvent se décliner en méthodes ayant comme repère la trochlée fémorale (méthodes de Blumensaat – Laurin [7] – Bernageau [1] et en



méthodes ayant comme repère l'extrémité supérieure du tibia (méthodes d'Insall & Salvati [15] – Blackburne & Peel [3] – De Carvalho [12] – Caton & Deschamps). Il existe par ailleurs des méthodes mixtes.

Méthode ayant comme repère la trochlée fémorale

a) La méthode la plus utilisée jusque dans les années 70 a été la méthode de Blumensaat [4] décrite en 1933. Pour Blumensaat, la pointe de la rotule devait affleurer la ligne intercondylo-trochléenne à 30° de flexion sur une radiographie du genou de profil. Cette méthode reste relativement imprécise car la ligne intercondylo-trochléenne peut varier. En effet, l'angle de Brattstrom [6] déterminé par l'axe du fémur de profil et la ligne intercondylo-trochléenne (angle U) peut varier de 27 à 60°.

b) En 1977, C.A. Laurin [17] a décrit une méthode de mesure de hauteur de la rotule avec également, comme repère, le fémur. Néanmoins, cette méthode qui nécessite une radiographie du genou de profil à 90° de flexion a peu de valeur sur le plan thérapeutique.

c) La méthode de J. Bernageau [1] est une méthode qui permet de faire une analyse sémiologique tout à fait intéressante des rapports de la rotule avec la trochlée. Cette méthode décrite en 1984 précisait que la rotule était haute lorsque le bord inférieur de la surface articulaire de la rotule était plus haut que le bord supérieur de la trochlée de plus de 6 mm et que la rotule était basse lorsque ce point R est plus bas que la trochlée, plus 6 mm. Cette méthode, tout à fait informative sur le plan clinique, est difficile à utiliser sur le plan thérapeutique, notamment dans le traitement chirurgical des rotules hautes et des rotules basses et par ailleurs s'avère difficile, voire impossible, dans le cas de dysplasies trochléennes sévères.

d) Enfin, la récente méthode de Biedert et Albrecht (2006) [2].

Méthode ayant comme repère le tibia

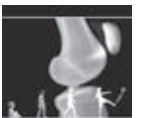
a) *Méthode d'Insall & Savati* [15]. Cette méthode écrite en 1971 établit un rapport entre la longueur de la rotule dans sa plus grande diagonale et la longueur du tendon rotulien (LP/LT). Cette méthode nécessite un profil strict du genou à 30° de flexion, une bonne visualisation de la tubérosité tibiale antérieure. Elle est donc peu utilisable lorsqu'il y a eu un traitement chirurgical au niveau de l'extrémité supérieure du tibia. Cette méthode a été modifiée par Gresalmer [14] en 1992 de façon à éliminer les problèmes secondaires à des modifications de la pointe de la rotule.

b) *Méthode de Caton* (cf. historique). Cette méthode a été décrite en 1977 [8] et a été modifiée en 1981 (technique de Caton & Deschamps [9-10-13]) (fig. 4) de façon à éliminer les artefacts secondaires à la pointe de la rotule.

Le rapport AT/AP (AP = longueur articulaire de la rotule et AT = distance entre le bord inférieur de la surface articulaire de la rotule et le bord antéro-supérieur du tibia est normalement égal à 1 et est identique chez l'homme et chez la femme). AT/AP = 0,96 ± 0,134 chez l'homme et 0,99 ± 0,129 chez la femme.

On parle de rotule haute lorsque le rapport AT/AP (index de Caton & Deschamps) est supérieur ou égal à 1,2 et de rotule basse (patella infera ou patella "baja") lorsque le rapport AT/AP est inférieur ou égal à 0,8, un rapport inférieur ou égal à 0,6 étant le seuil que nous admettons pour une correction chirurgicale.

c) *Blackburne & Peel* [3]. Méthode décrite en 1977. Cette méthode fait le rapport entre la distance du bord inférieur de la rotule et la prolongation de la droite déterminée par les plateaux tibiaux. Cette méthode nécessite une radio du genou de profil strict en flexion à 30°. Par ailleurs, cette distance peut varier outre le degré de flexion du genou, du fait de la variation anatomique de l'inclinaison des plateaux tibiaux (pente).



d) *Méthode de Linclau* 1984 [19] et de de Carvalho [12] en 1985. Ces méthodes sont strictement identiques à la méthode décrite par J. Caton et G. Deschamps.

Méthodes mixtes

a) *Méthode de Picard et de Saragaglia* en 1997 [20]. Cette méthode tient compte à la fois du bord supérieur de la trochlée et du bord inférieur du plateau tibial.

b) *Méthode de Leung et Wai.* (1996) IO (Chine) [18].

c) *Méthode de Koshino et Sugimoto* 1989 uniquement chez l'enfant (IPO) Japon [16].

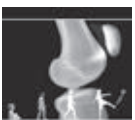
d) *Méthode de Chareancholvanich et Narkbunnam* (2012) *Io Thaïlande* [11], évaluée en extension complète ou en flexion, ce qui pour nous fausse la mesure de hauteur, car le système extenseur est détendu à 0 %.

CONCLUSION

Une bonne méthode de mesure de la rotule doit toujours être fiable. Elle doit être indépendante de la qualité des radiographies, indépendante du degré de flexion du genou entre 10 et 80°, indépendante de la taille du genou et de l'agrandissement radio, ce qui est possible lorsque cette méthode est basée sur un rapport. Enfin, elle doit être indépendante de la tubérosité tibiale antérieure, de ses modifications et des modifications rotuliennes et indépendantes de l'âge des sujets. Elle doit être possible à la fois chez les adultes et les enfants, à partir du moment où la rotule est visible et doit permettre de programmer chirurgicalement une modification précise de la hauteur rotulienne en cas d'anomalie de hauteur (fig. 5-6-7).

La méthode que nous avons décrite (J. Caton et G. Deschamps) (fig. 4) nous semble répondre à tous ces critères.
Elle a d'ailleurs été considérée également comme la meilleure méthode chez l'enfant par Camille Thevenin-Lemoine *et al.* [21].
Cependant bien qu'elle soit universelle, adulte et enfant, pour le chirurgien il faut adapter cette mesure pour les prothèses totales du genou du fait de la perte du repère tibial. Pour cela, en cas de prothèse totale, nous utilisons notre méthode classique pour l'évaluation de la hauteur rotulienne en préopératoire et une méthode comparative modifiée avec détermination d'un nouveau point T en pré- et post-op afin de savoir si nous avons changé la hauteur rotulienne après l'intervention. Ce nouveau point T' doit être déterminé à partir d'éléments fixes non modifiés par l'acte, à savoir la tête du péroné et l'axe tibial (fig. 8).

Fig. 5 : J. Caton, G. Deschamps aux journées caribéennes d'orthopédie.



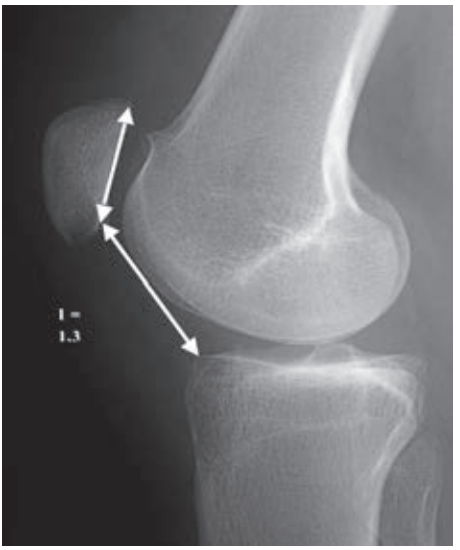


Fig. 6 : Rotule haute avec dysplasie de trochlée AT/AP = 1,3.

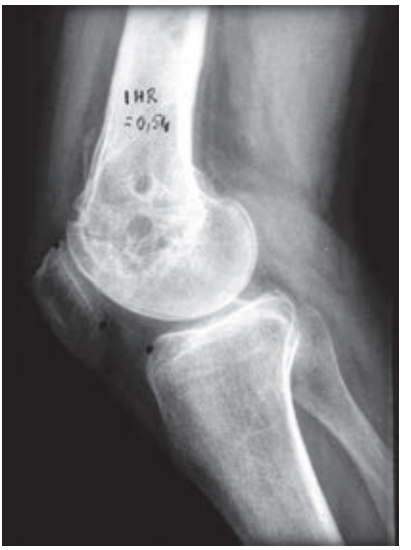


Fig. 7 : Patella infera d'origine quadricipitale avec index de Caton et Deschamps à 0,54. Séquelle d'infection du genou après ostéosynthèse et algodystrophie.

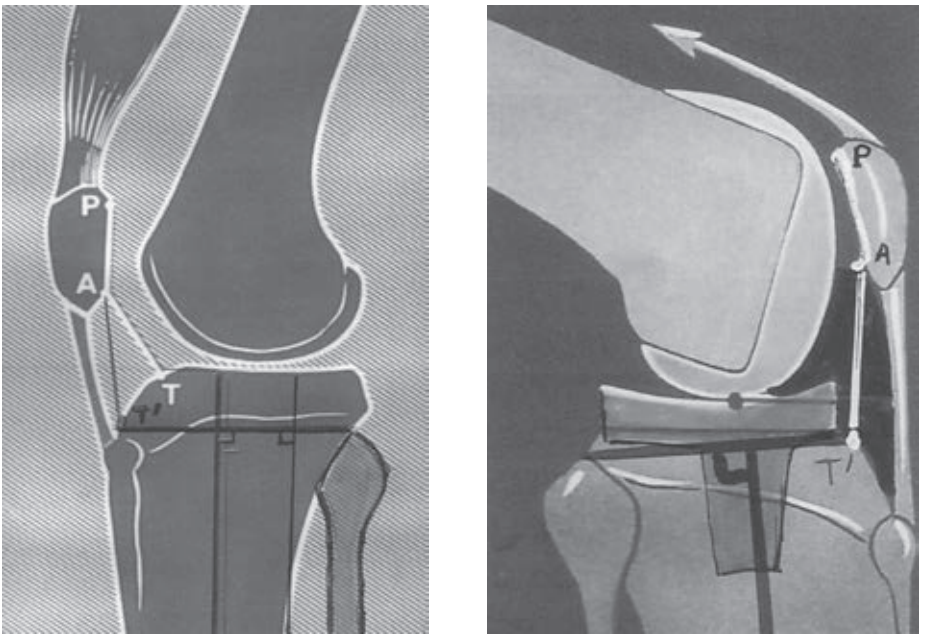


Fig. 8 : Index J. Caton et G. Deschamps modifié, pour la mesure des variations des hauteurs rotuliennes pour les PTG.

A VALUABLE RATIO FOR PATELLAR VERTICAL HEIGHT

THE CATON – DESCHAMPS INDEX

INTRODUCTION

Since 1977 after our medical thesis in Lyon about tears of the knee extensors (patella fractures excluded) we have been using our assessment index modified in 1981 after the medical thesis of Gerard Deschamps about patella infera and known as Caton - Deschamps index.

OUR INDEX

The method (fig. 4) consists of plotting the distance between the inferior edge of the patellar joint surface and the antero-superior angle of the tibia on sagittal X-rays (AT) and the joint length of the patella (AP).

The value ratio AT/AP is normally equal to 1 (0.96 ± 0.134 for men and 0.99 ± 0.129 for women). When the ratio is equal or superior to 1.2 it is defined as patella alta and when the ratio is less than or equal to 0.8 it is a patella infera but we do operation for patella infera when the CD ratio is inferior or equal to 0.6.

CONCLUSION

In our opinion the most accurate diagnosis method for patellar height variations or patellar instabilities is the Caton-Deschamps index because this ratio relies on readily identifiable and reproducible anatomical landmarks and is possible regardless of the quality of the X-rays, knee size, radiological enlargement or position of the tibial tubercle or patellar modification and knee flexion between 10° and 80°/ used to adults and children and its calculation quantified the necessary surgical correction of patella alta or patella infera.

REFERENCES

[1] BERNAGEAU J, GOUTALLIER D. Examen radiologique de l'articulation fémoro-patellaire. In : l'actualité rhumatologique, de Seze et coll. *Expansion Scientifique Française, Paris, 1984.*

[2] BIEDERT RM, ALBRECHT S. The patella trochlear index: a new index for assessing patella height 2006. *Knee surg sports traumatol arthrose 14:707-12.*

[3] BLACKBURNE JS, PEEL TE. A new method of measuring patella height. *J Bone Joint Surg (Br), 1977, 59, 241-2.*

[4] BLUMENSAAT C. Die lageabweichungen und Vezzenkungen der Kniescheibe Ergebn *Chir. Orthop, 1938, 31. 149-229.*

[5] BOON-ITT SB. The normal position of the patella. *Am J Roentgenol, 1930, 24, 389-94.*

[6] BRATTSTROM H. Shape of the intercondylar groove normally and in recurrent dislocation of the patella. *Acta Orthop Scand, 1964, suppl 68, 1-148.*

[7] BRATTSTROM H. Patella alta in non-disclocating knee joints. *Acta Orthop Scand. 1970, 41, 578.*

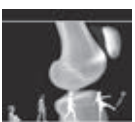
[8] CATON J. Les ruptures du système extenseur du genou (fractures de la rotule exceptées). A propos de 130 observations. *Thèse médecine 1977. Lyon.*

[9] CATON J, DESCHAMPS G, CHAMBAT P, LERAT JL, DEJOUR H. Les rotules basses (Patellae inferae) – A propos de 128 observations. *Rev Chir Orthop 1982, 317-25.*

[10] CATON J. Méthode de mesure de la hauteur de la rotule. *Acta Orthop Belg 1989, 55. 385-6.*

[11] CHAREANCHOLVANICH K, NARKBUNNAM R. Novel method of measuring patellar height ratio using to distal femoral reference point *2012 int orthop. 36. 749-53.*

[12] DE CARVALHO A, HOLST ANDERSEN A, TOPP S, JURIK AG. A method for assessing the height of the patella, *1985, Int Orthop 9:195-7.*



[13] DESCHAMPS G. Les rotules basses *thèse Lyon 1981.*

[14] GRELSAMER RS, MEADOWS S. The modified Insall Salvati ratio for assessment of patellar height. *Clin Orthop 1992, 282. 170-6.*

[15] INSALL J, SALVATI E. Patella position in the normal knee joint. *Radiology, 1971, 101, 101-4.*

[16] KOSHINO T, SUGIMOTO K. New measure of patellar height in the knee of children using the epiphyseal line midpoint. *1989, J Pediatr Orthop 9: 216-8.*

[17] LAURIN CA. The investigation of the patello femoral joint. *J Bone Joint Surg (Br), 1977, 59, 107.*

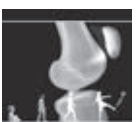
[18] LEUNG YF, WA YL, LUNG YC. Patella alta in southern China: a new method of measurement. *1996, Int Orthop 20: 305-10.*

[19] LINCLAU L, DOKTER G. Iatrogénic palla "Baja", *1984, Acta. Orthop. Belg. 50: 75-80.*

[20] PICARD F, SARAGAGLIA D, MONTBARBON E. Morphometric study of the femoro patellar joint on lateral X-rays, *1997, Rev Chir Orthop 83: 104-11.*

[21] THEVENIN-LEMOINE C, FERRAND M, COURVOISIER A, DAMSIN JP *et al.* Is the Caton-Deschamps index a valuable ratio to investigate patellar height in children? *2011, J Bone Joint Surg Am 93: 35.*

[22] TRILLAT A, LECLERC P. Anomalies de hauteur de la rotule. Patella alta-patella baja. 3e journées Lyonnaises du Genou, Lyon 1973. (*Monographie) SIMEP Editions, Lyon, 1973.*



MEDIAL TRANSFER OF THE ANTERIOR TIBIAL TUBEROSITY HISTORICAL CONSIDERATIONS

P. NEYRET, R.A. MAGNUSSEN, E. SERVIEN, S. LUSTIG, G. DEMEY, V.B. DUTHON

INTRODUCTION

In the Lyon school after the "6e Journées Lyonnaises de Chirurgie du Genou" entitled "Pathologie Fémoro-Patellaire", hold in Lyon under the direction of H. Dejour and G. Walch, we considered two periods.

BEFORE 1987

In the past, many surgical techniques were proposed to manage patellofemoral disorders as patellectomy, Maquet, patellectomie+ Maquet, Hauser, Goldwaith. At this time, simple biomechanical theory including the force Fq was used to understand patellar dislocation and clinical evaluation of the lateral implantation of the ATT was very limited (Q angle, baïonnette sign).

The Roux Elmslie Trillat period

In order to correct the lateral implantation of the ATT, a medialisation of the ATT was proposed. In 1959, the first results of the medialisation of the ATT were reported in the Jaqueline Ledeuil thesis (A. Trillat department). The history of the publications of the Elmslie technique is really special. According to an original letter written by A. Trillat himself and given by G Gacon. So "quae sunt Caesaris, Caesari"!

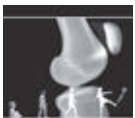
A. Trillat wrote :

"Dear Friend,

Cesar Roux has published in 1888 in the "Revue de chirurgie" the original technique that did not change a lot. It's a part of the tendon, keeping the insertion of the patellar tendon on the tibial tuberosity and then transfer medially. Very different from the Hauser's technique.

On 1944 visiting London I met Jim Seddon, a pupil of Elmslie, who explained me what Elmslie was doing in order to realign the extensor mechanism. This author had never published anything about it. At this time, I was not aware Roux's publication and when I came back to France, I named this technique the "Elmslie's technique" and this terminology was spread out very quickly.

Fifteen years later, I finally discovered the Roux's publication (100% identical to the Elmslie technique). I imagine that Elmslie also found this publication. He had never referred to him due to the fact, I suspect, that Elmslie



thought it was his original idea from his point of view. But when he realized that Roux had published it in a review, at this time internationally well known, he just decided to continue to perform this procedure and not to publish it.
Anyway it was a very shirt series.

To summarize:
- *1882 Roux: only one case performed on a 13 years old girl followed-up for 3 months,*
- *Elmslie discovered perhaps later on the publication. Few cases,*
- *Only known by Seddon and me,*
- *Wrong Christening of the operation,*
- *Fast dissemination due to publications and "Journées du Genou",*
- *Wrong knowledge of the origin,*
- *No possibility to change the Christening name,*
- *No interest to add my name.*

That's all I can say
I wish you a happy new year.
A Trillat"

The technique of the Elmslie-Trillat

The technique of Elmslie-Trillat is precise and every detail must be respected. It includes:
- Vertical paramedial approach
- Osteotomy :
  - 2 cuts: the first (medial) is vertical and the second (lateral) is horizontal,
  - length of the piece of bone detached: 6-8cm,
  - deep enough to include also cancellous bone,
  - the base of the osteotomy is bent (osteoclasy).
- Fixation: 1 oblique 4.5mm AO screw

In distal transfer of the ATT, the ATT is totally detached. The fixation is ensured by 2 horizontal 4.5mm AO screws.

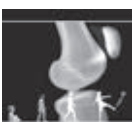
AFTER 1987

In our school, the "modern" period started with the *"Journées Lyonnaises de Chirurgie du Genou"* in 1987, where a strict difference was established between patients who had had one or several patellar dislocations, and the group of patients who complained anterior knee pain without morphological factors of patellar instability. In this last group, after 1987, there is no indication anymore for a medial transfer of the ATT. Three theses were published at this period by A. Mironneau, C. Levigne and G. Py.

At this period, Bernageau and Goutallier defined the TT-TG (Tibial Tubercule – Trochlear Groove distance) that measured the lateral implantation of the ATT and external femorotibial rotation of the knee. The Lyon group validated the normal value (16mm±4mm) of the TT-TG, knee in extension. Then a medial transfer of the ATT was recommended in episodic patellar dislocation patient with a TT-TG superior to 20mm. An individualized management of patellofemoral instabily was proposed and the "menu à la carte" was defined. We treated the patients according this algorythm after 1987.

Elvire Servien reported the results of the ATT transfert in 2003 considering a group of 130 patients (174 knees, operated between 1988 and 1999). The follow-up was 2 to 13 years. The subjective IKDC score was 77.2 (range 45.9-95.4). 94% of the patients were very satisfied or satisfied. The post-operative recurrent dislocation rate was 4.5%.

In this series, distalization of the ATT was performed in patients with relatively normal TT-TG. E. Servien found that pure distalization of the tibial tubercle resulted also in some medialization of the patella (4mm average). The conclusions were that:
- Medialization and distalization was indicated in patients with significant patella alta and lateralized tibial tubercle (high TT-TG).
- Medialization was primarily indicated for patients with tibial tubercle lateralization. Some authors report that isolated medialization results in patellar tendon shortening as well (perhaps due to scar tissue formation). We haven't noted shortening of the patellar tendon in our center provided post-operative mobilization begins immediately.



A second study performed in the A. Trillat center showed that the measurement of the TT-TG after an osteotomy of the ATT is not simple. "Is TT-TG reliable after medialization of the ATT?"

Since 1987, osteotomy of the ATT is proposed in many others surgical situations:
- in the management of patello-femoral osteo-arthritis (G. Py thesis and more recent results were reported by Ferran Montserrat),
- in the approach of the lateral compartment during lateral unicompartimental knee arthro-plasty,
- in the approach of some total knee arthroplasty (patella infera, stiffknee…),
- in some revision of total knee arthroplasty .

CONCLUSIONS

If in the past, osteotomy of the ATT was almost always performed to treat episodic patellar instability. Nowadays, the proximal gestures seem to become more and more frequent. Nevertheless in some indications when morphological abnormalities are obvious, we continue to perform an ATT osteotomy according to the indications defined in 1987 and the technique described by "Elmslie-Trillat… Roux".

The authors have no potential conflict of interest in this presentation.

LITERATURE

DEJOUR H, WALCH G, NEYRET P, *et al.* Trochlea dysplasia. *Rev Chir Orthop 1990; 76:45.*

ESCALA J, MELLADO J, OLONA M, *et al.* Objective patellar instability: MR-based quantitative assessment of potentially associated anatomical features. *KSSTA 2006;4: 264-72.*

FAYARD JM, SERVIEN E, LUSTIG S, *et al.* Le transfert de la tubérosité tibiale antérieure favorise-il la survenue de l'arthrose fémorotibiale interne à long terme? *Rev Chir Orthop 2009;95: 229-30.*

FITHIAN D, NEYRET P, SERVIEN E. Patella instability: The Lyon experience. *Tech Knee Surg 2007;6: 112-23.*

GOUTALLIER D, BERNAGEAU J, LECUDONNEC B. The measurement of the tibial tuberosity. Patella groove distanced technique and results. *Rev Chir Orthop 1978; 64(5): 423-8.*

LUSTIG S, SERVIEN E, BIEDERT R, *et al.* Isolated arthrosis of the patellofemoral joint in younger patients (<50 years). *Ortopade 2008; 37(9): 848-57.*

MALGHEM J, MALDAGUE B. Apport du cliché de profil du genou dans le dépistage des instabilités rotuliennes. Rapport préliminaire. *Rev Chir Orthop 1985; 71:5-15.*

MALGHEM J, MALDAGUE B, LECOUVET F, *et al.* Plain radiography of the knee: the articular surfaces. *J Radiol 2008; 89(5): 692-7.*

MAYER C, MAGNUSSEN RA, SERVIEN E, *et al.* Patellar Tendon Tenodesis in Association With Tibial Tubercle Distalization for the Treatment of Episodic Patellar Dislocation With Patella Alta. *Am J Sports Med 2011; 40(2): 346-51.*

NEYRET P, ROBINSON AHN, LE COULTRE B, *et al.* Patellar tendon length - the factor in patellar instability? *The Knee 2002;9 : 3-6.*

NEYRET P, DEMEY G, SERVIEN E, *et al.* Traité de chirurgie du genou. *Elsevier-Masson 2012. Issy-les-Moulineaux.*

PANNI S, CERCIELLO S, MAFFULI N, *et al.* Patellar shape can be a predisposing factor in patellar instability. *Knee Surg Sports Traumatol Arthrosc 2011;19: 663-70.*

PIEDADE S, PINAROLI A, SERVIEN E, *et al.* Tibial tubercle osteotomy in primary total knee arthroplasty: a safe procedure or not? *The Knee 2008;15 : 439-50.*

PIEDADE S, PINAROLI A, SERVIEN E, *et al.* Revision after early aseptic failures in primary total knee arthroplasty. *Knee Surg Sports Traumatol Arthrosc 2009; 17(3): 248-53.*

PINAROLI A, PIEDADE S, SERVIEN E, *et al.* Intraoperative fractures and ligament tears during total knee arthroplasty. A 1795 posterostabilzed TKA continuous series. *Orthopaedics & Traumatology: Surgery & Research, 2009; 95(3): 183-9.*

ROUX C. Luxation habituelle de la rotule. Traitement opé-ratoire. *Rev. Chir. 1888; 8; 682-689.*

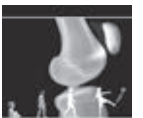
SERVIEN E, AIT SISELMI T, NEYRET P. Study of the patellar apex in objective patellar dislocation. *Rev Chir Orthop 2003;89 : 605-12.*

SERVIEN E, AIT SI SELMI T, NEYRET P. Subjective evaluation of surgical treatment for patellar instability. *Rev Chir Orthop 2004;90: 137-42.*

SERVIEN E, VERDONK P, NEYRET P. Tibial tuberosity transfer for episodic patellar dislocation. *Sports Med Arthrosc Rev 2007;98: 364-72.*

SERVIEN E, PIEDADE S, LUSTIG S, *et al.* Distal Realignements Techniques. *Chirurgiche in Ortopedia e Traumatologia 2007;5(3): 129-33.*

SERVIEN E, FRITSCH B, LUSTIG S, *et al.* In vivo positioning analysis of medial patellofemoral ligament reconstruction. *Am J Sports Med 2011;39(1): 134-9.*



HOW THE ELMSLIE-TRILLAT PROCEDURE HAS BEEN USED IN EUROPE OR US?

A. WYMENGA

The Elmslie-Trillat procedure is a tuberosity medialisation leaving the distal part of the tubercle attached on a periostial hinge and was reported by Trillat in 1964 (ref) Trillat credited the English orthopedic surgeon Elmslie for the technique who never published his technique himself (fig. 1).

A medial realignment of the extensor mechanism was first reported by Cesar Roux in 1888 (ref) (fig. 2).

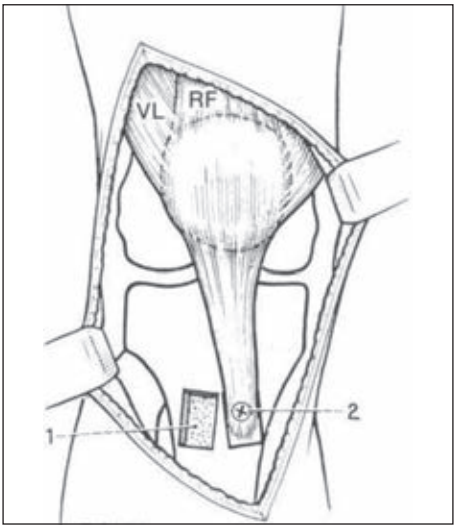


Fig. 1: Roux procedure

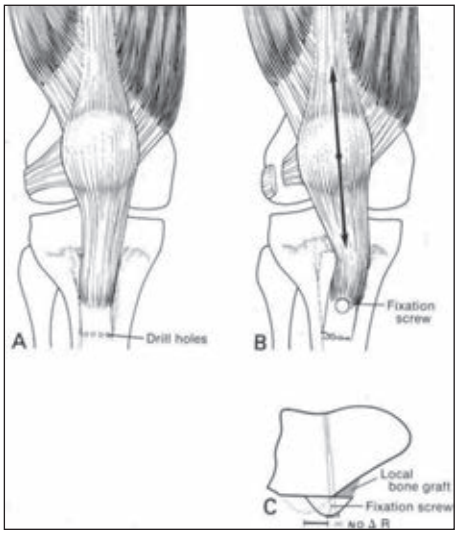
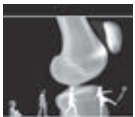


Fig. 2: Elmslie-Trillat procedure

It was not until 1938 when Hauser reported about 4 succesful cases of medial transfer the interest was renewed in distal procedures (ref.) (fig. 3).

Hauser his technique involved a transfer of the tibial tuberosity distal and medial. This caused as a consequence of the proximal tibia geometry also a transfer in dorsal direction. In addition to



the transfer a lateral release and medial plication was performed. Although the early results were encouraging later long term reports indicated and accelerated osteoarthritis caused by the increased contact pressures on the patello-femoral joint (fig. 4).

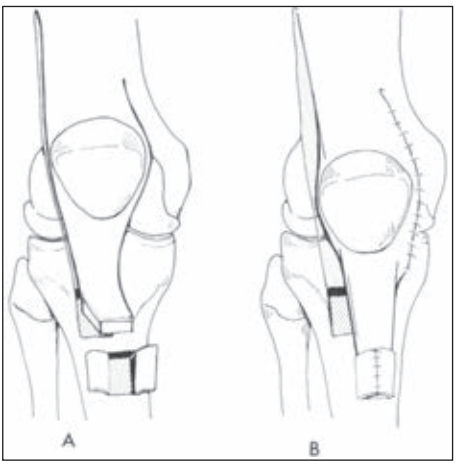


Fig. 3: Hauser procedure

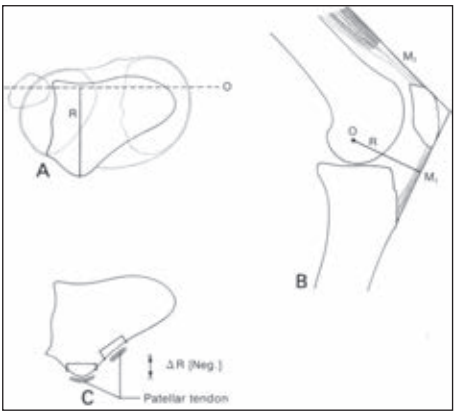


Fig. 4: Biomechanical effect Hauser procedure

After the publications of Trillat the Lyon school further developed the analysis of the pathophysiology of patellofemoral instability and pain. Subsequently scientific papers were reported in the English speaking arena. An important publication was the paper by Dejour in 1994 in KSSTA 1994 summarizing the complete concept of quadriceps dysplasia, patella alta, the trochlea dysplasia and the pathological lateralisation of the tuberosity as the four cornerstones that play a role in objective patellofemoral instability and potential patellofemoral instability (ref). The current medial soft tissue repair of the MPFL was not included in the concept but a reefing was suggested with more than 20 degrees tilt as measured on CT scans.

APPROACHES IN EUROPE AND US IN TIME

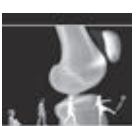
In Europe the Hauser procedure was frequently performed but subsequently abandoned due to the increased patellofemoral arthritis osteo-arthritis. This observation kept many surgeons from using the modified Trillat operation which does not increase the patellofemoral pressure as opposed to the Hauser technique.

Hampson *et al.* (ref) reported arthrosis caused by the posteriorisation after the Hauser procedure. Arnbjornsson et al reported in 1992 in the JBJS a high percentage of arthrosis in conservative and operatively treated patello-femoral instability. It also became clear that the tuberosity transfers done for undefined pain without a lateral tuberosity position had poor results due to overmedialisation and creating medial patellofemoral arthritis (Crelsamer).

Another problem was the limited successes of the soft tissue procedures available at that time such as the Galeazzi procedure (semitendinosus tenodesis) and the Krogius plasty (medial reefing with lateral release) and Green plasty and later the lateral release epidemic.

Despite the hesitations to operate on the patellofemoral joint due to lack of understanding of the pathology and the less favourable outcomes a number of papers reported on the Trillat operation. On average they found around 80% good results in terms of pain and stability after the procedure.

Trillat (1974) reported 78% good/excellent results and 92% improved. Morshuis and



Pavlov (1990) reported 84% good results declining to 70% after 30 months with an anteromedialisation of the tuberosity with a correlation with the cartilage status. Conti *et al.* (1992) found 87% good subjective results after the ET procedure. Aglietti *et al.* (1994) reported good results with tuberosity transfer with and without soft tissue correction. Koskinen found better results after an ET procedure compared to a Roux-Goldwaith (1998). Kruger *et al.* (1999) found 70% good results after the ET procedure.

Kumar *et al.* (2000) reported 61% good results with better outcome for instability patients. Diks *et al.* (2003) found 85% good results with CT guided tuberosity transfer, patients with an objective lateral tracking patella had better pain relief. Marcacci *et al.* (2004) reported about a variant with a medial slip of the patellar tendon tenodesed on the anteromedial side of the tibia inadjunct to the normal ET procedure in cases where a simple ET operation does not restore patella tracking. Kataraglis *et al.* (2006) found in the 73% good results in patients with anterior knee pain or instability with better results for the instability group. Koeter *et al.* (2007) found the good results in a prospective study with a self-aligning tuberosity technique in flexion of the knee for correct positioning of the tuberosity. The average pain reduction was 5 points on a 10 points pain score. Endres and Wilke (2011) round excellent results in 16 of 18 patients after 10 years an reported no progressive arthritic changes at follow-up interestingly.

The US approach around the tuberosity started with Goldtwaith (1904) who developed a technique with medialisation of a lateral slip of the patellar tendon sutured to the medial capsule but this technique became not very popular. Later a lot of enthusiasm for the Hauser operation came up but MacNab et al reported in 1954 arthritic changes after this procedure.

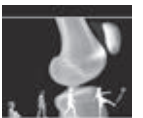
Hughston (1979) reported about his subtle tuberosity medialisation similar to the Elmslie-Trillat of around 5mm in combination with a medial soft tissue procedure and found over a 25 year period 70% good results. Brown in 1984 81% good results with an average of 10 mm medialisation at 42 months of follow-up, they found a correlation with the q-angle and undercorrection resulted in less good results. Fulkerson in 1983 reported a variant of the ET procedure with an anteromedialisation of the tuberosity with an oblique osteotomy simulating the effects of both the ET procedure and the Maquet operation with ventralisation of the patellar tendon.

Despite these developments the proximal realignment remained relatively more popular in the US. Later in 1994 Shelbourne reported the ET procedure being effective in treating patella instability and maltracking by reducing the abnormal patellar congruence angle.

Crosby and Insall in 1976 also reported arthritic changes after tuberosity transfer and found 20% recurrent instability and the arthritic changes were not seen after soft tissue procedures. Carney *et al.* (2005) reported a 26 year follow-up after ET procedures and found 7% recurrent dislocation rate and a decline from 73% good results after 3 years to 54% good results after 26 years. Barber *et al.* (2008) found a 91% succesrate in eliminating instability and a satisfaction rate of 8 out of 10 points after an average of 8 years follow-up.

Cosgarea *et al.* (1999) found in biomechanical analysis failure to fracture at a lower load in the Fulkerson osteotomy techniques and recommend protected weight bearing after surgery. Ramappa *et al.* (2006) found in lab tests similar effects of both techniques on correction of abnormal patella tracking and pressures but the medialisation was more effective in reducing lateral facet pressures.

In recent review paper from Amis (2007) the strong biomechanical effect of the ET procedure has well been described and the operation should be performed with care not to overmedialise since this can easily cause too high medial pressures. However the medial soft tissues also play in concert with the lateral side an important role in guiding the patella in the trochlea in combination with the muscles. Future development of a comprehensive approach to analyse all the pathology of the patellofemoral



joint and develop an evidence based treatment algorithm will further refine the treatment of patellofemoral instability and maltracking.

The Lyon Knee School has been of major importance in the development of diagnostics and treatment approaches for patellofemoral pathology. And it is without any doubt that the "good old" and biomechanical powerfull ET procedure as described by Albert Trillat will keep its central place in the tools for the surgeon to treat patellofemoral pathology.

LITERATURE

TRILLAT A, DEJOUR H, COUTETTE A. Diagnostic et traitement des subluxations de la rotule. *Chir Orthop 53; 331-42, 1967.*

ROUX C. Luxation habituelle de la rotule. Traitement opératoire. *Rev Chir Paris 1888;8; 682-9.*

HAUSER EW. Total tendon transplant for slipping patella: a new operation for recurrent dislocation of the patella. *Surg Gyn Obste 1941;66: 199-214.*

DEJOUR H, WALCH G, NOVE-JOSSERAND L, GUIER C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol. Arthrosc (1994) 2:19-26.*

ARNBJÖRNSSON A, EGUND N, RYDLING O, STOCKERUP R, RYD L. The natural history of recurrent dislocation of the patella. Long-term results of conservative and operative treatment. *J Bone Joint Surg Br (1992) 74: 140-2.*

GRELSAMER R. Current concepts review: patellar malalignment. *J Bone Joint Surg Am (2000) 82: 1639-47.*

HAMPSON W, HILL P. Late results of transfer of the tibial tubercle for recurrent dislocation of the patella. *J Bone Joint Surg Br (1975). 57: 209-13.*

FULKERSON J, BECKER G, MEANEY J. Anteromedial tibial tubercle transfer without bonegraft. *Am J Sports Med (1990) 18: 490-6.*

MORSHUIS WJ, PAVLOV PW, DE ROOY KP. Anteromedialisation of the tibia tuberosity in the treatment of patellofemoral pain and malalignment. *Clin Orthop 1990; 205; 242-50.*

AGLIETTI P, BUZZO R, DE BIASE P *et al.* Surgical treatment of recurrent dislocation of the patella. *Clin Orthop 1994; 308: 8-17.*

KOSKINEN SK, RANTANEN JP, NELIMARKKA OL *et al.* Effect of Elmslie-trillat en Roux-Golwthwaith procedures on patellofemoral relationships and symptoms in patients with patellar dislocations. *Am J Knee Surg 1998; 11,3: 167-73.*

ENDRES S, WILKE A. A 10 year follow-up study after Roux-Elmslie-Trillat treatment for cases of patellar instability. *BMC Musculoskeletal Disorders 2011, 12; 48: 1-6.*

KARATAGLIS D, GREEN MA, LEARMONTH DJA. Functional outcome following modified Elmslie-trillat procedure. *Knee 2006; 13: 464-8*.

KRUGER T, BIRKE A, DECKER T *et al.* Ergebnisse der operation nach Elmslie-trillat bei Patellaluxation und subluxation. *Unfall Chirurg 1999, 102: 700-7.*

DIKS M, WYMENGA AB, ANDERSON PG. Patients with lateral tracking patella have better pain relief following CT guided tuberosity transfer than patients with an unstable patella. *KSSTA 2003; 11: 384-8.*

KOETER S, DIKS M, ANDERSON P *et al.* A modified tibial tubercle osteotomy for patellar maltracking. *J Bone Joint Surg (Br) 2007;89-B; 180-5.*

CONTI C, BERRUTO M, BIANCHI M. The Elmslie-Trillat procedure for recurrent subluxation of the patella. One to five year follow-up. *Ital J Orthop Traumat 1992;8: 341-9.*

KUMAR A, JONES S, BICKERSTAFF DR *et al.* Functional evaluation of the modified Elmslie-Trillat procedure for patellofemoral dysfunction. *The Knee 2001:8: 287-92.*

MARCACCI *et al.* Treatment of chronic patellar dislocation with a modified Elmslie-Trillat procedure. *Arch Trauma Surg 2004, 124; 250-7.*

MACNAB I. *J Bone and Joint Surg 1952; 34A: 957.*

CROSBY EB, INSALL J. Recurrent dislocation of the patella. *J bone and Joint Surg 58A 9-13, 1976.*

GOLDTWAITH Slipping of recurrent dislocation of the patella. *Bost Med Surg J 1904: 150; 169-74.*

HUGHSTON JC, WALSH WM. Proximal and distal reconstruction of the extensor mechanism for patellar subluxation. *Clin Orthp 1979;144: 36-42.*

BROWN, ALEXANDER, LICHTMAN. The Elsmlie-Trillat procedure: evaluation in patellar dislocation and subluxation. *Am J Sports Med 1984, 12,2: 104-9.*

FULKERSON JP. Anteromedialisation of the tibial tuberosity for patellofemoral malalignment. *Clin Orthop 1983;153: 176.*

COX J. Evaluation of the Roux-Elmslie-Trillat procedure for knee extensor realignment. *Am J Sports Med 1982;10;5: 303-10.*

SHELBOURNE KD, PORTER DA, ROZZI W. Use of a modified Elmslie-Trillat procedure to improve abnormal patellar congruence angles. *Am J Sports Med 1994;22; 3: 318-23*

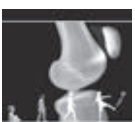
COSGAREA AJ, SCHATZKE MD, STH AK *et al.* Biomechanical analysis of flat and oblique tibial tubercle osteotomy for recurrent patellar instability. *Am J Sport Med 1999; 27;4; 507-12.*

RAMAPPA AJ, APRELEVA M, HARROLD FR *et al.* The effects of medialisation and anteromedialization of the tibia tubercle on patellofemoral mechanics and kinematics. *Am J Sport Med 2006,34;5: 749-56.*

BARBER A, MCGARRY JE. Elmslie-Trillat procedure for the treatment of recurrent patellar instability. *Am J Sports Med 2008; 24; 77-81.*

CARNEY JR, MOLOGNE TS, MULDOON M *et al.* Long term evaluation of the Roux-Elmslie-Trillat Procedure for patellar instability. *Am J Sport Med 2005; 33, 8,1220-23.*

AMIS Current concepts on anatomy and biomechanics of patellar stability. *Sports Med Arthrosc rev 2007;15: 48-56.*



PATELLAR TENDON TENODESIS FOR THE TREATMENT OF PATELLA ALTA

R.A. MAGNUSSEN, C. MAYER, E. SERVIEN, G. DEMEY, M. JACOBI, S. LUSTIG, P. NEYRET

INTRODUCTION

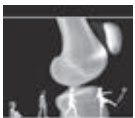
Numerous anatomic factors have been shown to contribute to episodic patellar dislocation (EPD), including injury to the medial patello-femoral ligament (MPFL) [8] trochlear dysplasia [4, 5] increased tibial tubercle-trochlear groove (TT-TG) distance [7], abnormal patellar tilt, and patella alta [6, 16, 19, 25]. Traditionally an under-appreciated contributor to patellar instability, patella alta has been noted to be present in 24% of patients with EPD but in only 3% of normal controls [5]. Further, patella alta has been shown to be a risk factor for recurrent instability in patients with EPD following conservative treatment [13] or isolated MPFL reconstruction [23].

The reason for the association between patella alta and EPD is not entirely clear and is likely multifactorial. Patella alta has been shown to cause decreased contact between the patella and trochlea [14, 21, 24] contributing to decreased resistance to lateral translation of the patella in these patients [20, 25]. It has also been suggested that increased patellar tendon length itself is the culprit, with increased length allowing a pathologic increase in coronal plane motion of the patella [16].

One study identified increased patellar tendon length (greater than 52mm) rather than a "too proximal" position of the tibial tubercle in patients with patella alta and EPD [16]. It may thus be desirable to address the length of the patellar tendon itself rather than simply altering its insertion site through tibial tubercle distalization. One potential method for shortening an abnormally long patellar tendon is to distalize the tibial tubercle and tenodese the distal portion of the patellar tendon into the original location of the tibial tubercle. We have described good results of this technique in a recently published case series [15]. The goal of this presentation is to explore indications and technique for patellar tendon tenodesis and review available outcomes data.

INDICATIONS FOR PATELLAR TENDON TENODESIS

It must be stressed that patellar tendon tenodesis associated with a distalization of the tibial tubercle is indicated in only a small fraction of patients treated for EPD. Patients undergoing evaluation for EPD should undergo a careful assessment of their anatomy and all predisposing factors for patellar dislocation as has been described in detail by H. Dejour *et al.* [5]. Those with significant patella alta (defined by a Caton-Deschamps index [1] greater than 1.2) should be considered for tibial tubercle distalization.



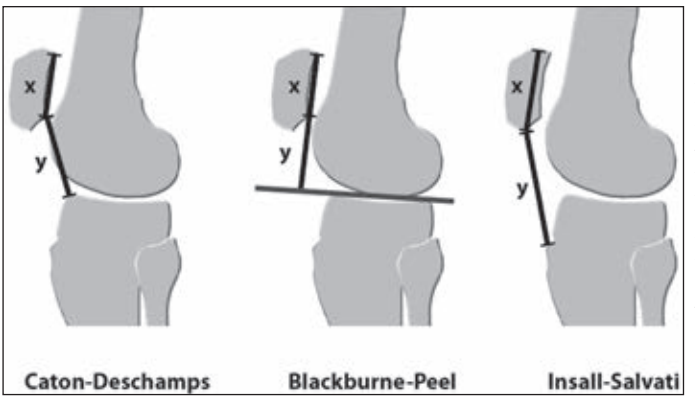


Fig. 1: Schematic drawing of the method of calculation of the Caton-Deschamps index (A), the Blackburne-Peel index (B), and the Insall-Salvati ratio (C). With each method, the value is calculated by dividing the distance marked as "y" on the figure by the distance marked as "x".

Among the population of patients with patella alta, one encounters a wide variety of patellar tendon lengths. Previous work has defined an abnormally long patellar tendon as greater than 52mm in length. We consider a patellar tendon longer than 52mm in association with patellar alta in a patient with EPD to be an indication for combined tibial tubercle distalization and patellar tendon tenodesis.

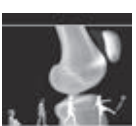
It should be noted that while the Caton-Deschamps index1 is an effective measure of patellar height, the Insall-Salvati ratio [11] is a more direct measure of the relative length of the patellar tendon (fig. 1). Further work may result in criteria for the performance of combined tibial tubercle distalization and patellar tendon tenodesis based on information obtained from both of these indices, but such data are currently unavailable.

CASE SERIES OF PATIENTS TREATED FOR PATELLA ALTA AND ELONGATED PATELLAR TENDON

Patients

Between 1996 to 2004, 134 patients underwent surgery for EPD at our center. Twenty-five of these patients were treated with distalization of the tibial tubercle in association with tenodesis of the patellar tendon into the proximal tibia by an original technique developed by Philippe Neyret and described at the *Journées Lyonnaises de Chirurgie du Genou* in 2002 [22]. The patients included 9 males and 16 females (6 of whom underwent bilateral treatment). Complete radiographic assessment and clinical follow-up of at least five years were available for 27 of 31 knees (87%). The average age at surgery was 20.4 years (range 14 to 30 years). All patients were skeletally mature at the time of surgery and none had undergone previous surgery on the index knee. All had a history of at least one patellar dislocation, symptoms of recurrent instability (either recurrent dislocation or subjective feelings of instability limiting function), and a positive patellar apprehension test. No patients in the series had severe trochlear dysplasia or excessive patellar tilt.

Radiographic evaluation included weight-bearing anteroposterior and lateral views of the knee and an axial view of the patella with the knee in 30 degrees of flexion. The height of the patella was quantified by measurement of both the Caton-Deschamps index1 and the Insall-Salvati ratio [11]. Additionally, all patients underwent a CT scan for measurement of the tibial tubercle-trochlear groove (TT-TG) distance. A patellar tendon tenodesis was performed in association with a tibial tubercle distalization in all patients with patellar alta and a tendon length greater than 52mm as described above. In addition to the distalization, the tibial tuberosity was also medialized in patients with a TT-TG greater than 20mm [5]. No other associated procedures were performed on patients in this series.



Operative technique and Rehabilitation

Surgery was performed in the supine position with a tourniquet on the proximal thigh. The procedure included a distalization of the tibial tubercle followed by tenodesis of the patellar tendon (fig. 2) [22]. A longitudinal incision was created from the inferomedial patellar border extending 6cm below the tibial tubercle, allowing visualization of the medial and lateral borders of the tendon and the tibial tubercle. After creation of two 3.2mm drill holes for fixation, the tibial tubercle was osteotomized as a 6cm bone block approximately 8mm in thickness. The bone block was then distalized an average of 9mm (range 6-16mm) depending on the degree of patellar alta that was present with a goal of achieving a Caton-Deschamps index of 1.0. Medialization was also performed in 22 knees (81.5%) (mean medialization 7mm, range: 2 to 11mm).

Prior to fixation of the bone block, two suture anchors were placed near the top of the original location of the tibial tubercle, approximately 3cm below the joint line (fig. 2A). The bone block was then fixed in the distalized position with two 4.5mm bicortical screws (fig. 2B). The sutures from each anchor were then passed through the tendon and tied, tenodesing the patellar tendon into the proximal tibia (fig. 2C). An advancement of the vastus medialis obliquus (VMO) muscle was then performed [9, 10] and the incision was then closed over a drain.

Weightbearing was allowed on the first postoperative day with the use of an extension brace. A supervised rehabilitation protocol was initiated focusing on gradual restoration of knee range of motion. Flexion was limited to 90 degrees for the first month following surgery, after which unrestricted motion was allowed. Strengthening began one month post-operatively with a focus on the quadriceps and VMO in particular. Biking was allowed from two months post-operative and a return to unrestricted activities including running was expected 4 to 6 months following surgery.

Outcome Assessment

Clinical examination at last follow-up included a physical examination with assessment of patellar apprehension. Any subsequent operations on the index knee or recurrent patellar dislocations were documented. Knee function was assessed with the IKDC subjective knee evaluation [12]. Patient satisfaction with the procedure was also assessed. Radiological examination included assessment of patella height by the Caton-Deschamps index and the Insall-Salvati method. The length of the patellar tendon was determined by measuring the distance from the inferior tip of the patella to the site of the patellar tendon tenodesis (metal suture anchors) (fig. 3).

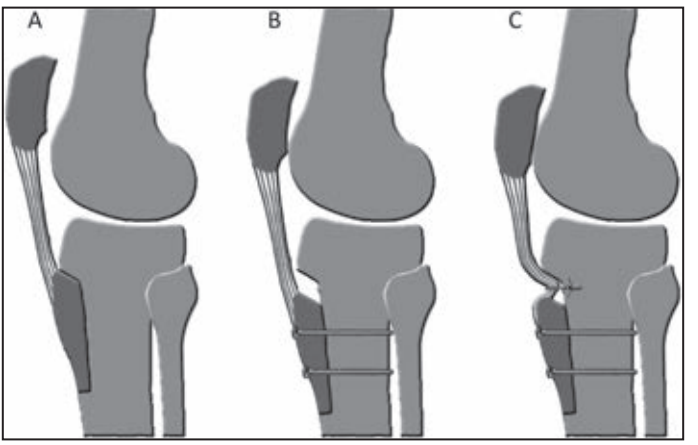
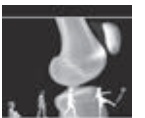


Fig. 2: Schematic drawing of a patient with patella alta and a long patellar tendon (A) treated with distalization of the tibial tubercle (B) and patellar tendon tenodesis (C).



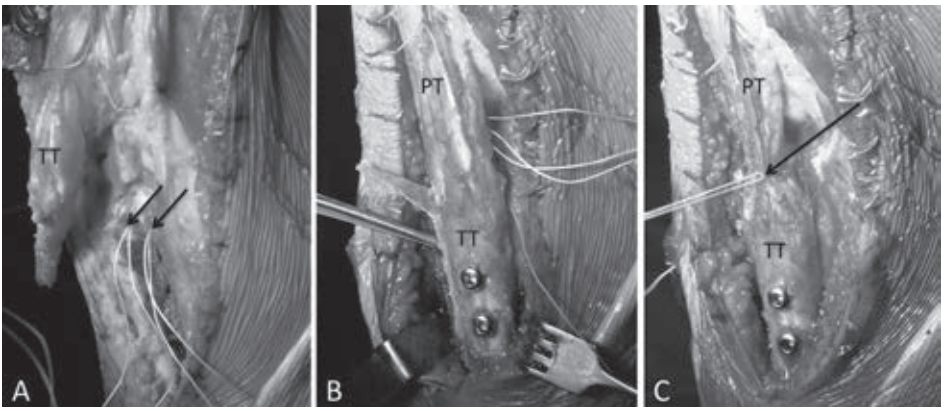


Fig. 3: Intra-operative photographs of treatment of patella alta and elongation of the patellar tendon with tibial tubercle distalization and patellar tendon tenodesis. A: Prior to fixation of the tibial tubercle bone block (TT), two suture anchors (arrows) are placed near the top of the original position of the tibial tubercle. B: The tibial tubercle (TT) is the fixed in a distalized position. The sutures are visible behind the patellar tendon (PT). C: The sutures are then passed through the tendon and tied (arrows), tenodesing the patellar tendon (PT) proximal to the tibial tubercle (TT).

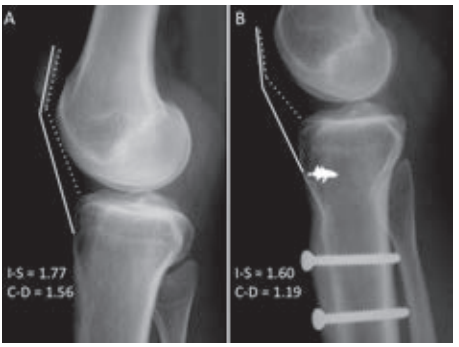


Fig. 4: Pre-operative (A) and post-operative (B) lateral radiographs demonstrating distalization of the tibial tubercle and tenodesis of the patellar tendon. The distances used to calculate the Insall-Salvati ratio (solid lines) and Caton-Deschamps index (dashed lines) are shown on both films.

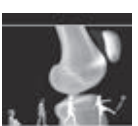
RESULTS

Mean follow-up was 9.6 years (range, 6 to 14 years). The mean length of the patellar tendon changed from 56.3 ± 2.7mm (range, 52.2 to 61.7mm) pre-operatively to 44.3 ± 8.6mm (range, 32.2 to 60.9mm) post-operatively ($p<0.0001$). Significant changes in patellar height were also noted between the pre- and post-operative radiographs (Table 1). No cases of patellar dislocation occurred post-operatively. No radiographic evidence of degenerative change was noted in the patello-femoral or tibiofemoral compartments at follow-up.

The mean post-operative subjective IKDC score was 75.6±9.5 (range 55.2 to 98.9).

Table 1: Changes in Patellar Tendon Length and Patellar Height with Surgery

	Pre-operative	Post-operative	Significance
Patellar Tendon Length (mm)	56.3 ± 2.7	44.3 ± 8.6	p < 0.0001
Caton-Deschamps Index1	1.22 ± 0.17	0.95 ± 0.22	p < 0.0001
Insall-Salvati Ratio11	1.42 ± 0.17	0.91 ± 0.18	p < 0.0001



Patients reported they were very satisfied with their knee in fifteen cases (55.5%), satisfied with their knee in ten cases (37.0%), and disappointed with their knee in two cases (7.4%) at final follow-up. The patellar apprehension test remained positive in nine knees (33.3%).

OUTCOMES DATA

The case series above demonstrates that technique of tibial tubercle distalization and patellar tendon tenodesis outlined above results in normalization of patellar tendon length and a stable patellofemoral joint in patients with patella alta and EPD. Long-term follow-up revealed high patient satisfaction and knee function according to the subjective IKDC score.

Numerous other authors have hypothesized that addressing patella alta in patients with EPD through distalization of the tibial tubercle would stabilize the patella [2, 5]. A recent study by Caton *et al.* described achieving perfect patellar stability in 77% of patients treated with distalization/medialization of the tibial tubercle, but do not clearly define the criteria used for this assessment [3]. Simmons *et al.* reported no recurrent dislocations in a group of 15 patients treated with tibial tubercle distalization [19]. Pritsch et al reported 6-year results of their series of 63 knees treated for EPD with tibial tubercle medialization in all cases and distalization in 90% of cases [18]. They reported good or very good Lysholm scores in 73% of patients, recurrent dislocation in one patient, and recurrent patellar subluxation in 5 patients. The patellar apprehension test remained positive in 15% of knees. Palmer *et al.* reported 5.6-year outcomes of 59 knees with patellar instability treated with tibial tubercle medialization and distalization [17]. They noted two cases of recurrent dislocation and 79% had good or excellent results. We are aware of no other studies in which episodic patellar instability was addressed through a tenodesis of the patellar tendon.

CONCLUSION

Patellar tendon tenodesis results in normalization of patellar tendon length and a stable patellofemoral joint and good long-term knee function when performed in association with tibial tubercle distalization in appropriately selected patients with EPD associated with patella alta and an elongated patellar tendon.

LITERATURE

[1] CATON J, DESCHAMPS G, CHAMBAT P, LERAT JL, DEJOUR H. Patella infera. Apropos of 128 cases. *Rev Chir Orthop Reparatrice Appar Mot. 1982; 68(5):317-325. PMID: 6216535.*

[2] CATON J, MIRONNEAU A, WALCH G, LEVIGNE C, MICHEL CR. Idiopathic high patella in adolescents. Apropos of 61 surgical cases. *Rev Chir Orthop Reparatrice Appar Mot. 1990; 76(4): 253-60. PMID: 2148403.*

[3] CATON JH, DEJOUR D. Tibial tubercle osteotomy in patello-femoral instability and in patellar height abnormality. *Int Orthop. Feb 2010; 34(2): 305-9. PMID: 20066411.*

[4] DEJOUR H, WALCH G, NEYRET P, ADELEINE P. Dysplasia of the femoral trochlea. *Rev Chir Orthop Reparatrice Appar Mot. 1990;76(1): 45-54. PMID: 2140459.*

[5] DEJOUR H, WALCH G, NOVE-JOSSERAND L, GUIER C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc. 1994; 2(1):19-26. PMID: 7584171.*

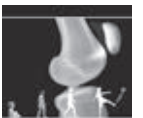
[6] GEENEN E, MOLENAERS G, MARTENS M. Patella alta in patellofemoral instability. *Acta Orthop Belg. 1989; 55(3): 387-93. PMID: 2603680.*

[7] GOUTALLIER D, BERNAGEAU J, LECUDONNEC B. The measurement of the tibial tuberosity. Patella groove distanced technique and results (author's transl). *Rev Chir Orthop Reparatrice Appar Mot. Jul-Aug 1978; 64(5):423-428. PMID: 152950.*

[8] GUERRERO P, LI X, PATEL K, BROWN M, BUSCONI B. Medial patellofemoral ligament injury patterns and associated pathology in lateral patella dislocation: an MRI study. *Sports Med Arthrosc Rehabil Ther Technol. 2009;1(1):17. PMID: 19643022.*

[9] HENRY JH, CRAVEN PR Jr Surgical treatment of patellar instability: indications and results. *Am J Sports Med. Mar-Apr 1981;9(2):82-85. PMID: 7223925.*

[10] INSALL J, BULLOUGH PG, BURSTEIN AH. Proximal "tube" realignment of the patella for chondromalacia



patellae. *Clin Orthop Relat Res. Oct 1979(144):63-69. PMID: 535252.*

[11] INSALL J, SALVATI E. Patella position in the normal knee joint. *Radiology Oct 1971;101(1): 101-104. PMID: 5111961.*

[12] IRRGANG JJ, ANDERSON AF, BOLAND AL, *et al.* Development and validation of the international knee documentation committee subjective knee form. *Am J Sports Med. Sep-Oct 2001; 29(5): 600-13. PMID: 11573919.*

[13] LARSEN E, LAURIDSEN F. Conservative treatment of patellar dislocations. Influence of evident factors on the tendency to redislocation and the therapeutic result. *Clin Orthop Relat Res. Nov-Dec 1982(171): 131-36. PMID: 7140059.*

[14] LUYCKX T, DIDDEN K, VANDENNEUCKER H, LABEY L, INNOCENTI B, BELLEMANS J. Is there a biomechanical explanation for anterior knee pain in patients with patella alta?: influence of patellar height on patellofemoral contact force, contact area and contact pressure. *J Bone Joint Surg Br. Mar 2009; 91(3): 344-50. PMID: 19258610.*

[15] MAYER C, MAGNUSSEN RA, SERVIEN E, *et al.* Patellar tendon tenodesis in association with tibial tubercle distalization for the treatment of episodic patellar dislocation with patella alta. *Am J Sports Med. Feb 2012; 40(2): 346-51. PMID: 22109545.*

[16] NEYRET P, ROBINSON AH, LE COULTRE B, LAPRA C, CHAMBAT P. Patellar tendon length--the factor in patellar instability? *Knee Feb 2002;9(1): 3-6. PMID: 11830373.*

[17] PALMER SH, SERVANT CT, MAGUIRE J, MACHAN S, PARISH EN, CROSS MJ. Surgical reconstruction of severe patellofemoral maltracking. *Clin Orthop Relat Res Feb 2004(419): 144-148. PMID: 15021145.*

[18] PRITSCH T, HAIM A, ARBEL R, SNIR N, SHASHA N, DEKEL S. Tailored tibial tubercle transfer for patellofemoral malalignment: analysis of clinical outcomes. *Knee Surg Sports Traumatol Arthrosc. Aug 2007; 15(8):994-1002. PMID: 17429609.*

[19] SIMMONS E Jr, CAMERON JC. Patella alta and recurrent dislocation of the patella. *Clin Orthop Relat Res. Jan 1992(274): 265-9. PMID: 1729011.*

[20] SINGERMAN R, DAVY DT, GOLDBERG VM. Effects of patella alta and patella infera on patellofemoral contact forces. *J Biomech. Aug 1994;27(8): 1059-65. PMID: 8089160.*

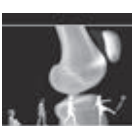
[21] STEFANIK JJ, ZHU Y, ZUMWALT AC, *et al.* Association between patella alta and the prevalence and worsening of structural features of patellofemoral joint osteoarthritis: the multicenter osteoarthritis study. *Arthritis Care Res (Hoboken). Sep 2010;62(9): 1258-65. PMID: 20506169.*

[22] TAD L, SERVIEN E, AIT SI SELMI T, NEYRET P. La "tenodese" du tendon rotulien. In: Chambat P, Neyret P, Bonnin M, et al., eds. *Le Genou du Sportif. Lyon: ALRM; 2002: 49-53.*

[23] THAUNAT M, ERASMUS PJ. Recurrent patellar dislocation after medial patellofemoral ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc. Jan 2008; 16(1): 40-43. PMID: 17973099.*

[24] WARD SR, POWERS CM. The influence of patella alta on patellofemoral joint stress during normal and fast walking. *Clin Biomech (Bristol, Avon). Dec 2004; 19(10): 1040-47. PMID: 15531054.*

[25] WARD SR, TERK MR, POWERS CM. Patella alta: association with patellofemoral alignment and changes in contact area during weight-bearing. *J Bone Joint Surg Am. Aug 2007; 89(8):1749-55. PMID: 17671014.*



THE CLINICAL EXAMINATION IN PATELLAR INSTABILITY

D.C. FITHIAN

INTRODUCTION

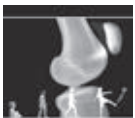
The patellofemoral joint (PFJ) is a unique articulation with a complex architecture. Its motion differs from that of most other joints in that it is mostly comprised of sliding rather than rolling. This places unique challenges on the articular cartilage. The most common disorders of the PFJ are thought to occur as a result of alterations in the mechanics of this joint, leading to cartilage overload or gross instability.

Diagnosis of patellofemoral instability can be difficult because patellar instability, patellofemoral pain, meniscal and cruciate ligament insufficiencies can produce a similar presentation of nonspecific pain and knee instability. Thus, careful evaluation including history, examination and imaging are helpful in making the correct diagnosis. This chapter focuses on the physical examination. A general approach is taken for completeness of the examination; but it must be emphasized that only direct assessment of patellar motion limits can document MPFL insufficiency, which is necessary for a correct diagnosis of patellar instability.

CLINICAL EVALUATION

Pertinent history

The knee extensor mechanism is a complicated and delicate system, the treatment of which is only further complicated by surgery if the surgeon has not clearly defined the pathological basis of the presenting complaint. The chief complaint should guide the workup and the discussion of treatment options; this will help the physician to focus on the patient's needs and expectations. For example, the patient who seeks help for relief of daily pain has a very different problem than one complaining of occasional sharp pain and giving-way. Although pain itself can result in knee instability (a symptom), it is important to differentiate painful knee giving-way from episodic pain due to excessive patellofemoral laxity. The latter is due specifically to insufficient constraint of mediolateral patellar motion. Pain and catching in early flexion points to an articular lesion at the inferior patella or proximal trochlea; pain throughout the range indicates a more diffuse, perhaps extra-articular process.



As the clinician develops an understanding of the symptoms and complaints, he or she can begin to develop one or more hypotheses, which can be tested in the physical examination and with subsequent imaging studies. The isolated complaint of pain, with no objective findings to suggest a specific source (pathology) representing an indication for surgery, should be treated nonoperatively.

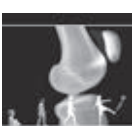
Physical examination

Due to the complex and delicate interactions between the knee extensor system and lower limb function, clinical evaluation of patellofemoral complaints can be challenging. After other disorders have been ruled out, specific testing for disorders of the patellofemoral joint can be performed. The patient should be evaluated standing, walking, and stepping up and down from a small step, squatting, sitting, supine, running, and jumping. Observation of the patient during a few functional activities yields a great deal of information about patellofemoral loading and neuromuscular conditioning (fig. 1). Any hindfoot valgus, forefoot pronation, and/or heel cord tightness should be noted as they can affect tibial rotation and patellofemoral alignment [1].

Fig. 1: The step-down test is a simple test that can be done in the clinic to evaluate core and hip control. This patient demonstrates pelvic weakness with hip adduction and medial collapse of the knee. It is apparent that her weakness results in a variety of functional alignment abnormalities, including: (1) contralateral pelvic drop, (2) right femoral internal rotation, (3) right knee valgus, (4) right tibia internal rotation, and (5) right foot pronation.

The prone position allows estimation of several important variables, including quadriceps tightness (important if correction of patella alta is being considered), femoral internal and external rotation limits, and foot-thigh angle (FTA) and/or trans-maleolar axis (TMA) (fig. 2). Femoral and tibial rotation can be estimated by examining the patient prone with the hips extended, the knees flexed 90°, and the feet and ankles in a neutral, comfortable position with the soles of the feet parallel to the floor after the method of Staheli *et al.* [2]. Kozic et [3] showed that on physical examination, femoral anteversion should be suspected if prone hip internal rotation exceeds external rotation by at least 45 degrees. With respect to estimating FTA and TMA, Staheli reported a wide range of normal values, with mean values of 10° for FTA and 20° for TMA [2]. Souza and Powers also confirmed the reliability of the Staheli method for estimating femoral anteversion, though axial imaging was more precise [4]. Our preferred approach is to use the prone physical examination to screen for torsion of the tibia and femur, and to obtain CT scan to assess rotational alignment if hip IR exceeds ER by at least 20 degrees or if the prone foot-thigh axis or TMA is greater than 20 degrees.

The lines of action of the quadriceps and the patellar tendon are not collinear. The angular difference between the two is the quadriceps angle, or "Q-angle". Because of this angle, the force generated by the quadriceps serves to both extend the knee and to drive the patella laterally, compressing the femoral trochlea in order to convert tension in the quadriceps into extension torque at the knee. The relative magnitude of the laterally directed force is related to the Q-angle. External rotation of the tibia, internal rotation of the femur, and increasing knee valgus all cause an increase in the Q-angle and thus an increase in the laterally directed force within the PFJ (fig. 3) [1].



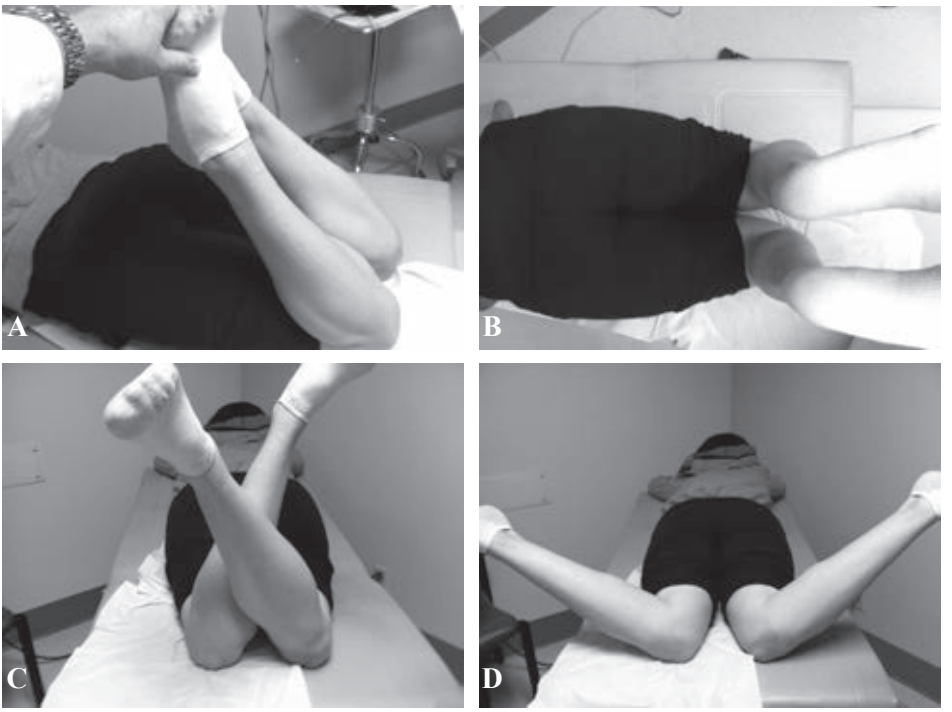


Fig. 2: The prone examination allows assessment of femoral and tibia torsion as well as quadriceps tightness. (A) evaluation of knee flexion with the hip in extended, which is a measure of quadriceps flexibility; (B) assessment of tibial torsion; (C) hip external rotation and (D) hip internal rotation.

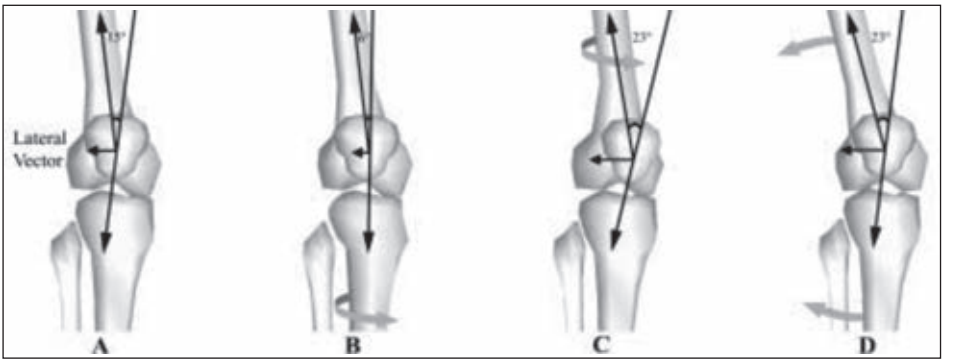
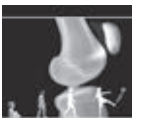


Fig. 3: (A) The Q angle is measured as the angle formed by the intersection of the line drawn from the anterior superior iliac spine to the midpoint of the patella and a proximal extension of the line drawn from the tibial tubercle to the midpoint of the patella. Normal alignment of the tibia and femur results in an offset in the resultant quadriceps force vector (proximal) and the patellar tendon force vector (distal), creating a lateral vector acting on the patella; (B) tibia internal rotation decreases the Q angle and the magnitude of the lateral vector acting on the patella; (C) femoral internal rotation increases the Q angle and the lateral force acting on the patella; (D) knee valgus increases the Q angle and the lateral force acting on the patella. JOSPT grant to use Figure 1 from Powers CM. The influence of altered lower-extremity kinematics on patellofemoral joint dysfunction: a theoretical perspective. J Orthop Sports Phys Ther 2003;33(11):639-646.



However, use of the Q-angle alone grossly underestimates the complexity of patellofemoral alignment and often leads to errors of diagnosis and treatment. Furthermore, the Q-angle is highly variable and unreliable as a measurement [5, 6]. These considerations have led the International Patellofemoral Study Group (IPSG) to recommend abandonment of clinical measurement of Q angle, preferring imaging studies to estimate the lateralizing vector at the PFJ. The preferred method is to use axial imaging (CT or MRI) to assess this vector [7].

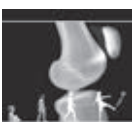
Careful palpation of both medial and lateral retinaculum is helpful to localize tenderness. Studies have shown that 90% of patients with patellofemoral pain syndrome (PFPS) had pain in some portion of the lateral retinaculum [8]. Palpation of the specific site of pain can help guide further investigation of what mechanical overload, if any, has occurred. The patella should be displaced to the side undergoing examination so that while fibers are being palpated, they are also brought away from underlying structures in order to avoid confusion about the site of tenderness.

The term "patellar tracking" refers to the change in position of the patella relative to the femur during active knee flexion and extension. While it is obviously important, no clinically useful tracking measurement systems exist. The J sign is a useful but non-specific sign of patellofemoral pathology. It represents a patella that does not seat immediately as the knee is flexed, as well as the rotational torque between the extensor hood (tendons, retinaculum and patella) and the femur. But many factors can contribute to abnormal tracking, such as trochlear dysplasia, patella alta, and medial retinacular laxity. Thus, the clinical usefulness of abnormal tracking in the assessment of alignment remains unclear because its relationship to the loading characteristics of the joint is not a simple one.

Normal tracking of the patella within the trochlear groove has been described by translation and tilt, both of which change with knee flexion angle [9]. As the normal knee begins to flex, the patella becomes engaged in the trochlea, causing it to translate medially approximately 4mm by 20 degrees of knee flexion. With progressive flexion it then follows the trochlear groove approximately 7mm laterally by 90 degrees of knee flexion. While it is translating laterally, it also tilts medially in a progressive linear fashion about 7 degrees at 90 of flexion. Deep in flexion, it is more medially tilted with the odd (far medial) facet articulating with the medial trochlea. The patella flexes with the knee at a rate of about 0.7 degrees per degree of knee flexion [9].

Abnormal patellar tracking may be caused by muscle weakness, soft tissues deficiencies, abnormal joint geometry, or limb malalignment. Early in flexion the medial retinaculum (specifically the medial patellofemoral ligament) provides much of the restraint to lateral displacement of the patella. Its contribution to patellar restraint decreases with flexion from 50% at 0 degrees of flexion to 30% at 20 degrees knee flexion, as the patella begins to engage the femoral trochlea. The lowest force required to displace the patella laterally occurs at 30 degrees of flexion. With further flexion, the patella engages the trochlear groove and trochlear geometry becomes the primary constraint to mediolateral patellar motion. In cadaver studies where the trochea has been modified (flattened) to simulate a dysplastic trochlea, the constraint of the patella is reduced by 70% [10].

The diagnosis of episodic lateral patellar dislocation and other conditions associated with insufficient patellar constraint (e.g. medial patellar instability following excessive lateral release) requires documentation of excessive laxity of the retinacular constraints. Comparison to the opposite knee can be helpful, provided that the complaint is unilateral [11]. Patellar mobility is best assessed both at 0º and at 30º of flexion (fig. 4). The checkrein often is easier to recognize at 0º because in this position the trochlea does not constrain the patella, so it is easier to feel an "endpoint" as you displace the patella laterally [12]. At 30º of flexion the patella is seated in the trochlear groove and it is easier to quantify the amount of mobility in each direction [12]. Normal translation should



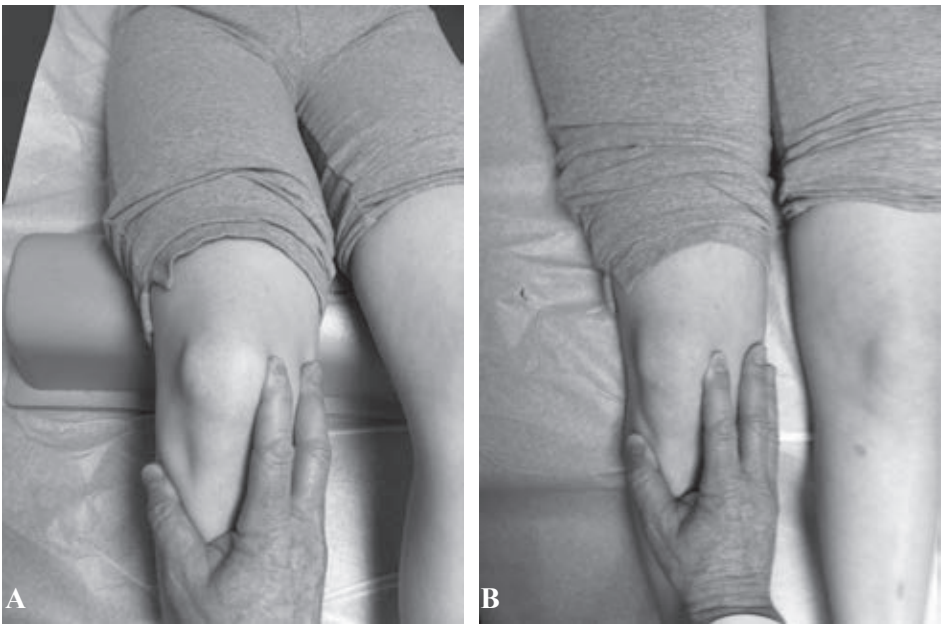
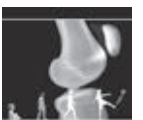


Fig. 4: (A) At 30º of flexion the patella is seated in the trochlear groove and it is easier to quantify the amount of mobility in each direction (medially and laterally) [11]. Normal translation should be symmetric in each direction and not exceed 7-10mm with a 5-lb load [11, 13]. (B) The checkrein, or endpoint of MPFL laxity usually is easier to recognize at 0º because in this position the trochlea does not constrain the patella, so it is easier to feel an "endpoint" as you displace the patella laterally [12]

be symmetric in each direction and not exceed 7-10mm with a 5-lb load [11, 13]. Alternatively, the patella can be divided into 4 quadrants and the displacement can be recorded in quadrants. Particularly in heavy patients, stress xrays may be more useful for assessing patellar mobility [14]. If patients are apprehensive as the patella is moved, an exam or stress X-rays under anesthesia can be very helpful to confirm pathological laxity prior to proceeding with surgical stabilization. Stabilization is never indicated unless excess laxity has been documented either in the clinic or under anesthesia.

SUMMARY AND CONCLUSION

The history and physical examination are key to understanding a patient's presenting complaint, and for determining further workup and treatment. This chapter focused on the physical features of patients presenting with complaints of knee pain and instability. It is important to realize that meniscus tears, cartilage flap tears and ligament insufficiencies other than MPFL laxity sometimes result in a patient being referred for treatment of patellar instability. It is the responsibility of the consultant to identify MPFL laxity prior to embarking on any surgical treatment for supposed patellar instability. At this time, patellar instability can be documented only by clinical examination or by stress radiography. While several ancillary points of physical examination are raised in this chapter, these should not distract our focus from the primary point of the chapter. Excessive laxity of patellar constraints, principally of the MPFL, is the sine qua non of patellar instability. No patient should undergo patellar stabilization unless the clinical workup has demonstrated deficient constraint of medial or lateral patellar motion with respect to the trochlear groove.



LITERATURE

[1] POWERS CM. The influence of altered lower-extremity kinematics on patellofemoral joint dysfunction: a theoretical perspective. *J Orthop Sports Phys Ther, 2003. 33(11): p. 639-46.*

[2] STAHELI LT *et al.* Lower-extremity rotational problems in children. Normal values to guide management. *J Bone Joint Surg Am, 1985. 67(1): p. 39-47.*

[3] KOZIC S *et al.* Femoral anteversion related to side differences in hip rotation. Passive rotation in 1,140 children aged 8-9 years. *Acta orthopaedica Scandinavica, 1997. 68(6): p. 533-6.*

[4] SOUZA RB, CM POWERS. Concurrent criterion-related validity and reliability of a clinical test to measure femoral anteversion. *J Orthop Sports Phys Ther, 2009. 39(8): p. 586-92.*

[5] GREENE CC *et al.* Reliability of the quadriceps angle measurement. *The American journal of knee surgery, 2001. 14(2): p. 97-103.*

[6] TOMSICH DA *et al.* Patellofemoral alignment: reliability. *The Journal of orthopaedic and sports physical therapy, 1996. 23(3): p. 200-8.*

[7] FITHIAN D, P NEYRET, E SERVIEN. Patellar Instability: The Lyon Experience. *Techniques in Knee Surgery, 2007. 6(2): p. 112-23.*

[8] FULKERSON JP. The etiology of patellofemoral pain in young, active patients: a prospective study. *Clin Orthop, 1983(179): p. 129-33.*

[9] AMIS AA, W SENAVONGSE, AM BULL. Patellofemoral kinematics during knee flexion-extension: an in vitro study. *J Orthop Res, 2006. 24(12): p. 2201-11.*

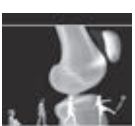
[10] SENAVONGSE W, AA AMIS. The effects of articular, retinacular, or muscular deficiencies on patellofemoral joint stability. *J Bone Joint Surg Br, 2005. 87(4): p. 577-82.*

[11] FITHIAN DC *et al.* Instrumented measurement of patellar mobility. *Am J Sports Med, 1995. 23(5): p. 607-15.*

[12] SENAVONGSE W *et al.* Quantitative measurement of patellofemoral joint stability: force-displacement behavior of the human patella in vitro. *J Orthop Res, 2003. 21(5): p. 780-6.*

[13] HAUTAMAA PV *et al.* Medial soft tissue restraints in lateral patellar instability and repair. *Clin Orthop, 1998. 349: p. 174-82.*

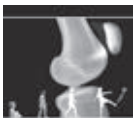
[14] TEITGE RA *et al.* Stress radiographs of the patello-femoral joint. *J Bone Joint Surg Am, 1996. 78(2): p. 193-203.*



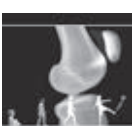
CLINICAL EXAMINATION FOR PATELLA INSTABILITY

C. FINK, P.J. ERASMUS
(Video Presentation)

Patient Standing	**Alignment** • Neutral • Varus • Valgus
Patient Walking	**Gait** • Kneecap position (squinting patella, etc) • Tubercle position • Foot progression angle - Out toeing (>15°) - Neutral - In toeing **Patella height** - Normal - High - Low **Patella movement**



Patient sitting on the side of the bed	**Active knee extension and flexion** Lateral movement (Tanaka and Thawait) • Proximal - Neutral - Lateral maltracking - J-sign - Diagonal movement • Distal - Subluxation in flexion Medial movement (iatrogenic secondary to lateral release) **Extension against resistance from 90 degree flexion to full extension.** • Crepitation positive or negative - Negative - Positive *(Note at what flexion angle it appears and disappears)* **Patella stability at 30° knee flexion** **Tibial torsion** **Ankle movement**
Patient lying flat	**Knee range of movement** **Patella** • Tenderness - Epicondyle - Medial patella border • Stability test at 30° knee flexion • Movement - Side to side - Tilt **Hip Rotation** • Hip in extension • Hip in flexion
Patient on side	**Patella and knee stability** • Patella Stability test at 30° knee flexion • Possible antero lateral laxity (ACL)
Squatting from standing to heel sitting	Squating • Pain • Crepitations *(Note at what flexion angle it appears and disappears)*



CLINICAL EXAMINATION OF THE PATELLOFEMORAL JOINT

S. DONELL

INTRODUCTION

In the management of patients with patellar instability the most important points are obtained from the history. This will establish the functional problems the patient is experiencing, and the therapeutic measures already tried. The patient's attitude to an operation should have been explored, as well as key risk factors for recurrence, notably a positive family history [1]. There are also associated important problems, such as hypermobility syndrome, which influence the outcome. The association of anterior knee pain with a poor operative result is crucial to understand. The ideal patient is post-adolescent with a normal body mass index, no hypermobility, no previous operations, a normal lower limb rotational profile, plays regular sports, and only has intermittent dislocations where, in between, they have a normal functioning knee. They should ideally live locally.

The purpose of the examination is to confirm the diagnosis patellar instability, and to exclude any important alternative diagnoses such as anterior cruciate ligament rupture, medial collateral ligament rupture, and inherited collagen diseases such as Marfan's and Ehlers-Danlos syndromes.

EXAMINATION TESTS OF THE PATELLOFEMORAL JOINT

There are 17 reported tests for patellofemoral joint [2]; many are well known.

Hypermobility

Hypermobility is measured using the Beighton score, a nine-item score. In the United Kingdom a self-help group, the Hypermobility Syndrome Association (HMSA) has a useful website [http://www.hypermobility.org/].

Gait pattern

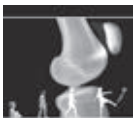
Observing a patient walking may reveal abnormal biomechanics, notably in the foot with pronation and persistent heel valgus during step-off. A leg length discrepancy may also be seen.

Lower limb alignment evaluation

With the patient standing the overall profile of the lower limb is noted looking for femoral anteversion, varus or valgus alignment, and tibial torsion.

Q-angle

With the patient supine a line is drawn from the anterior superior iliac spine to the centre of the



patella. A second line is drawn from the centre of the patella to the tibial tubercle. The resultant angle is the quadriceps (Q-) angle. Normal is valgus 10º to 15º degrees in men and 15º to 20º for women.

Quadriceps definition

The technique is not described but reduced definition of the quadriceps may indicate atrophy.

VMO capability

The patient sits on the edge of the examination couch. The leg is actively extended and held at 45º flexion. A concavity on the medial aspect of the distal thigh indicates vastus medialis obliquus (VMO) atrophy.

Patellar glide test

The patient lies supine with the knee relaxed at 30º flexion. The patella is pushed medially and laterally. It is divided into four quadrants and displacement equal to three or more quadrants indicates reduced patellar restraint.

Patellar apprehension test

With the patient supine and the knee relaxed at 30º flexion, the patella is pushed laterally. If the patient exhibits anxiety and/or an involuntary quadriceps contraction then this is a positive apprehension test.

Modified apprehension test

With the patient supine and the knee relaxed at 30º flexion and in neutral rotation, the patella is pushed in a distal and lateral direction at 45º (towards the fibular head). This is said to isolate the MPFL by relaxing the medial patellotibial ligament. It is also said to reduce the effect of the lateral trochlear flare by the distal displacement.

Bassett's sign

This is demonstrated when there is tenderness on palpation of the adductor tubercle and medial epicondyle. It indicates damage at the origin of the MPFL.

Palpation medial retinaculum

Palpation of the medial retinaculum and medial patellar border reveals a defect or tenderness at the site of injury i.e. along the middle portion of the MPFL to its insertion into the patella.

Gravity subluxation test

With the patient lying in lateral decubitus and the affected leg uppermost, the patient relaxes as the examiner abducts the leg. In patients with medial subluxation the patella displaces medially. If when the patient isometrically contracts the quadriceps and the patella remains medially displaced this indicates complete disruption of the lateral retinaculum. If it relocates, the lateral retinaculum is intact at the level of the vastus lateralis muscle.

Patellar positioning

The patient initially lies supine and is re-tested sitting. The knee is relaxed in full extension and the tilt, height, and mediolateral displacement of the patella observed whilst the knee actively flexes to full flexion. Abnormal positions of the patella are noted, including patella alta and infera and excessive lateral tilt.

Patellar tilt test

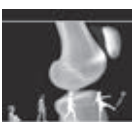
With the patient supine and the knee relaxed at 20º flexion, the patella is held between thumb and index finger and the medial side is pushed down to elevate the lateral edge. Normal results in an elevation of neutral to 20º, less than this is associated with lateral retinacular tightness.

Quadriceps pull test

With the patient supine and the knee relaxed in full extension the centre point of the patella is marked and a line drawn to the tibial tubercle. The patient performs an isometric quadriceps contraction and its horizontal displacement from the reference line is measured. Greater than 15mm is abnormal and suggests imbalance of the muscle forces.

Tibial tubercle to Trochlear Groove (TTTG) Assessment

The patient lies semi-recumbent and the midpoint between the pubic symphysis and the



anterior superior iliac spine. With the knee at 90º callipers are placed across the epicondyles and a piece of string passed from the proximal reference point over the knee at the midpoint of the callipers and continues in a straight line down the tibia. The transverse distance between the centre of the tibial tubercle and the string is the TTTG.

J-sign

The patient sits on the edge of the couch with the knee in full extension. They then actively flex the knee fully. The examiner observes the tracking of the patella. A positive J-sign occurs when the patella moves from lateral to medial in the first 20º of knee flexion.

Smith *et al's* [2] review suggested that patellar instability can be confirmed with all of these tests. However the sensitivity-specificity and the reliability-validity of the test were open to doubt. Surprisingly, despite the extent of the literature on the Q-angle, and patellar medio-lateral orientation and position, little of this pertained to patellar instability populations. It was concluded that, despite examination being a cornerstone in diagnosis, the evidence for the utility of these tests of patellar instability was methodologically flawed and there was insufficient evidence to support their use. As a result a study was set up to address this.

INTRA- AND INTER-OBSERVER RELIABILITY OF PATELLAR INSTABILITY TESTS [3]

Under the auspices of the International Patellofemoral Study Group (IPSG), five patients with known patellar instability were recruited at the Norfolk & Norwich University Hospital. They were all female with an average age of 27 years (18 to 38 years) with a mean duration of symptoms of 11 years (2 to 26 years) and a mean body mass index of 22 (19 to 28). Two had had previous patellar stabilisation procedures, and all had bilateral symptoms.

Five experienced clinicians from the IPSG were invited to take part in the study, which was ethically approved. The clinicians were instructed only to perform the tests that they routinely used in their practices when evaluating a patient with patellar instability. Each patient was randomly allocated to a clinician who then performed and recorded the results of the tests, screened from the other examiners.

After a break, the clinicians then repeated the tests with further randomisation to the patient order.

Assessment of inter- and intra-observer reliability was made using Kappa analysis by an independent statistician. These were interpreted as showing no agreement when <0.00, slight agreement at 0.00 to 0.20, fair at 0.21 to 0.40, moderate at 0.41 to 0.60, substantial at 0.61 to 0.80, and almost perfect at 0.81 to 1.00.

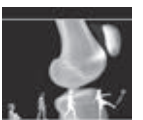
Results

The inter-observer agreement was very poor for most of the tests. Fair-to-moderate agreement was found for the J-sign, patellofemoral crepitus, and assessment of foot arch position. The inter-observer reliability was greatest for more generic lower limb examinations (foot arch position, patellofemoral crepitus, and pain on palpation of the medial retinaculum) rather than more specialist tests (Q-angle, gravity subluxation test, and tubercle-sulcus angle).

Intra-observer reliability showed moderate-to-substantial agreement, particularly for alignment, and observational & palpation tests (tibial torsion, Bassett's sign, Q-angle performed at 30 degrees flexion).

Discussion

Inter-observer reliability was best for more general examination tests and qualitative assessments better than quantitative i.e. tibial torsion "abnormal" as against "n degrees".



Abnormal was therefore more useful than the actual number of degrees. In fact quantitative information can be gained from images, where this is needed. Experienced clinicians showed moderate consistency in their examination findings despite the poor agreement between them.

This means that examination tests cannot be relied upon to give consistent and accurate numerical data, and therefore cannot be used as decision tools for surgical management of the patient. Standardisation of the physical examination, both the test chosen, and how it is performed and recorded, is needed for research studies and clinical management.

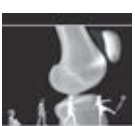
PERSONAL EXAMINATION TESTS

My personal protocol when examining a patient with a history of patellar instability does not include the Q-angle as it does not add anything [4]. The only measure that is likely to be reliable, although we have not achieved consensus on this is the mediolateral glide [5]. My personal examination protocol is:

1. Assess the Beighton score for hypermobility before asking the patient to lie on the couch.
2. Assess the rotational profile of the lower limb with the patient supine and the knees extended. The feet are raised noting the relative leg lengths, and knee alignment (e.g. recurvatum or valgus), then internally and externally rotate the ankles to check femoral version. Tibial torsion is noted by pointing the patellae vertically and noting the foot alignment with the ankle plantegrade.
3. Perform a stroke test to find a slight knee effusion.
4. Ask the patient to push their knees backwards and palpate the VMOs to check their presence and power.
5. Palpate the medial border of the patella, the medial retinaculum and adductor tubercle and test for apprehension in extension. My personal grading system for apprehension is 0 = none, + = apprehension after lateral patellar displacement, ++ = apprehension at the start of lateral displacement and +++ = apprehension on moving the hand towards the knee. Then assess the mediolateral glide in extension, noting if there is any crepitus.
6. Note the active range of knee flexion. With the knee in full flexion palpate the anterior surface of the distal femur and note if there is a normal groove or dysplasia.
7. Check the integrity of the ACL.
8. Sit the patient over the side of the bed and note patella tracking with active extension from 90º flexion. The range of abnormalities is much greater than just the J-sign. A slight J is normal, especially in the presence of recurvatum. In some the maltracking may be almost L-shaped, or with a visible clunk at 20º to 30º flexion. There may be bayonet tracking, dislocation in flexion, or permanent lateral dislocation. The more severe the more likely the patient has a significant trochlear dysplasia.
9. Check core and gluteal muscle control. The former is demonstrated by the patient lying supine and flexing both knees to 90º. They are then asked to lift their pelvis so that their chest pelvis and knees are in a straight line. They then fold their arms across their chest. They are then asked to straighten each knee keeping their pelvis lifted (normal side first). With weak lumbar muscles the patient is unable to sustain the elevation of the pelvis. Gluteal muscle control is demonstrated by being balanced on each leg, and then performing a unilateral squat. With a weak gluteus maximus muscle the femur internally rotates on squatting, often precipitating the symptoms.

CONCLUSION

Examination of the patellofemoral joint in patellar instability is undertaken to confirm the diagnosis. The evidence base shows that there is no reliable test that can be used to decide on surgical management. The more severe the grade of apprehension, the more likely an



operation may benefit the patient. In the majority I perform a medial patellofemoral ligament reconstruction, the question to answer is do they need a trochleoplasty? On examination the dysplasia may be palpated (if the patient is not obese) and the severity is associated with the type of maltracking noted. Ultimately surgical management is decided by quantitative measurements on images, and not on the examination.

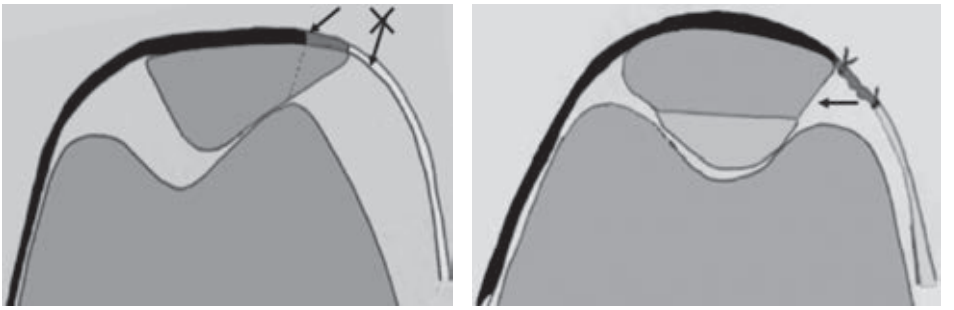


Fig. 1

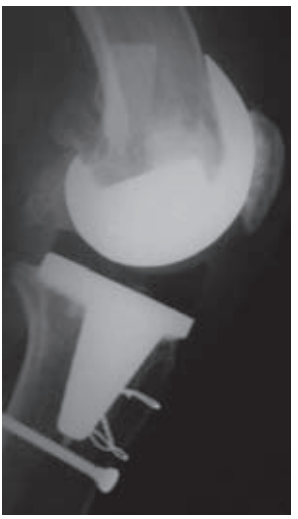


Fig. 2

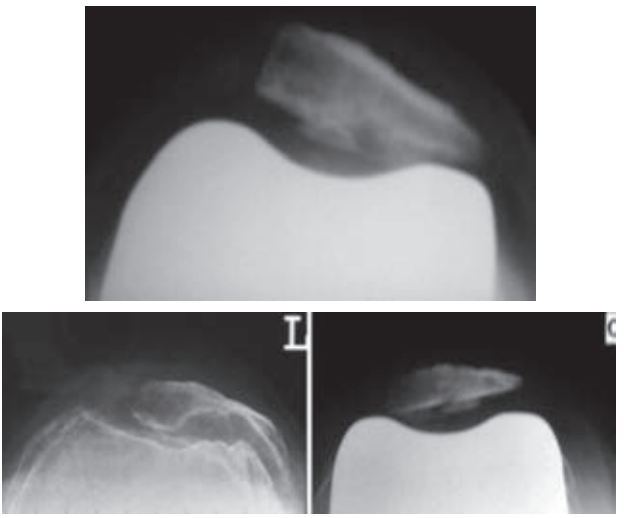


Fig. 3

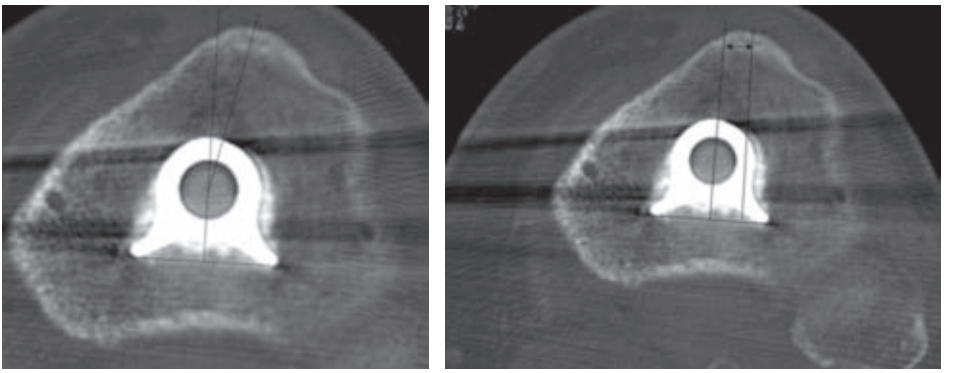


Fig. 4

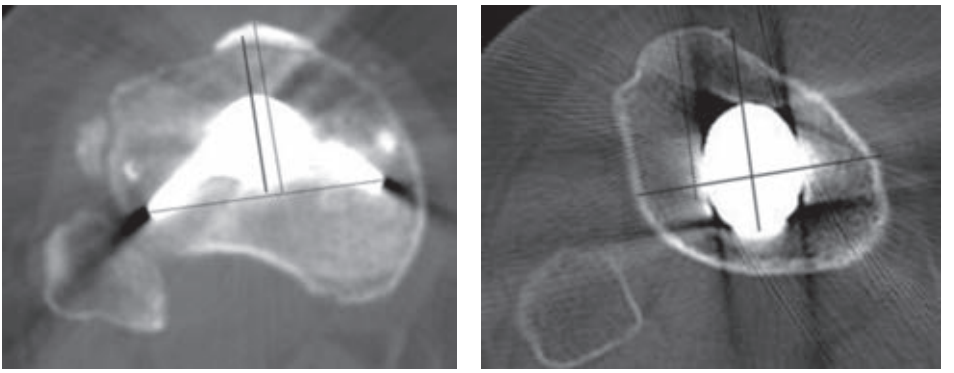


Fig. 5

LITERATURE

[1] PALMU S, KALLIO PE, DONELL ST, *et al.* Acute Patellar Dislocation in Children and Adolescents: A Randomized Clinical Trial. *J Bone Joint Surg [Am] 2008; 90A: 463-70.*

[2] SMITH TO, DAVIES L, O'DRISCOLL ML, *et al.* An evaluation of the clinical tests and outcome measures used to assess patellar instability. *The Knee 2008; 15: 255-62.*

[3] SMITH TO, CLARK A, NEDA S, *et al.* The intra- and inter-observer reliability of the physical examination methods used to assess patients with patellofemoral joint instability. *The Knee 2012: 19: 404-10.*

[4] SMITH TO, HUNT N, DONELL ST. The reliability and validity of the Q-angle: A systematic review. *Knee Surgery, Sports Traumatology, Arthroscopy 2008; 16: 1068-79.*

[5] SMITH TO, DAVIES L, DONELL ST. The reliability and validity of assessing medio-lateral patellar position: A systematic review. *Manual Therapy 2009; 14: 355-62.*

LUXATION DE ROTULE EXAMEN ARTHROSCOPIQUE ÉVALUATION AVANT ET APRÈS STABILISATION

J.M. FAYARD, M. THAUNAT,
B. SONNERY-COTTET, P. CHAMBAT

INTRODUCTION

L'arthroscopie, au cours de la chirurgie de stabilisation rotulienne, présente un intérêt indéniable : elle permet d'inspecter l'articulation fémoro-patellaire, d'évaluer le statut cartilagineux, d'analyser la position de la rotule (médio-latérale, proximo-distale). Par ailleurs, l'arthroscopie permet une analyse dynamique de la cinétique rotulienne avant et après la réalisation d'un geste de stabilisation.

INSTALLATION

L'intervention s'effectue classiquement en décubitus dorsal, le garrot étant placé le plus haut possible sur la racine de la cuisse afin de gêner le moins possible la cinétique rotulienne.

Delaunay [8] souligne le fait que l'anesthésie générale ou locorégionale parasite l'analyse de la cinétique fémoro-patellaire. Les travaux de Johnson [9] semblent confirmer cette notion. En effet, sur 11 genoux sains examinés arthroscopiquement sous anesthésie locale, lors de la flexion active, le recentrage se fait autour de 20° de flexion. En revanche, lors de la flexion passive, celui-ci s'effectue autour de 45°.

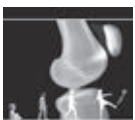
Par ailleurs, le remplissage articulaire modifie la cinématique rotulienne. Selon Delaunay [7], l'angle de flexion permettant d'obtenir une congruence articulaire parfaite ou angle de flexion de recentrage (AFR) est de 31° pour un genou sans irrigation articulaire. Cet angle est de 38° après inflation articulaire.

VOIES D'ABORD

Les voies de choix pour l'analyse de l'articulation fémoro-patellaire sont les voies arthroscopiques hautes et en particulier la voie supéro-latérale. Elle permet une analyse de l'ensemble des surfaces articulaires. En raison du volume du cul-de-sac sous-quadricipital, la vision est aisée.

Les voies classiques antéro-médiale et antéro-latérale gardent tout de même une place. Néanmoins, la vision peut s'avérer difficile dès que l'on veut analyser la cinétique en flexion en raison du ligament adipeux infra-patellaire. En effet, l'engagement rotulien plaque la rotule contre la trochlée et ferme l'espace antérieur.

Nous positionnons le patient sur une table classique un contre appui et une cale sous le pied



permettant de maintenir le genou fléchi à 90°. Pour Delaunay [7], seule le positionnement genou fléchi sur un arthrostress permet d'analyser de façon précise le degré de flexion du genou.

ANALYSE STATIQUE

L'analyse de la morphologie rotulienne est aisée par voie antéro-latérale. Elle s'effectue jambe en extension, l'arthroscope est positionné horizontalement et orienté vers le haut. Néanmoins, le ligament adipeux peut parfois gêner sa visualisation et l'utilisation du shaver peut s'avérer nécessaire pour étudier la portion distale.

L'arthroscopie permet d'évaluer l'aspect général de la rotule, le caractère dysplasique ou non, la taille de la facette médiale par rapport à la facette latérale.

On appréciera également la tendance spontanée à la translation latérale de la rotule comme le souligne Kita [10]. On évaluera l'importance de cette translation en pourcentage de la facette latérale.

Par ailleurs, la hauteur de la rotule peut être également appréciée par rapport à la trochlée. En effet, en cas de patella alta, celle-ci siège à la partie haute de la trochlée.

Cette partie de la trochlée, siège des dysplasies, est mieux appréciée par voie supéro-latérale. En revanche, l'analyse de la gorge trochléenne est plus facile par voie antéro-latérale. Le scope est dirigé progressivement vers le bas en glissant le long de la berge externe afin d'explorer la trochlée dans sa totalité.

Le caractère dysplasique ou non de la trochlée est appréciée : hypoplasie de la berge médiale, obliquité de la berge latérale, saillie de la trochlée.

Le statut cartilagineux rotulien et trochléen est également analysé (fig. 1). Ces lésions cartilagineuses sont fréquentes sur la rotule. Nomura [12] fait état de 96 % de lésions cartilagineuses après luxation de rotule. L'auteur décrit deux types de lésions : les fissurations (fig. 2) siégeant principalement sur le dôme et les érosions siégeant principalement sur la facette médiale. Kita [10] rapporte des observations similaires. Par ailleurs, l'opérateur s'attachera à rechercher d'éventuels corps étrangers articulaires, fréquents dans le cadre des luxations de rotule.

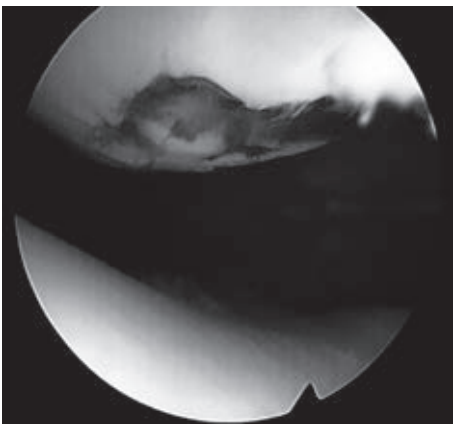


Fig. 1 : Fracture ostéochondrale de la berge médiale de la rotule s'étendant à la crête médiane

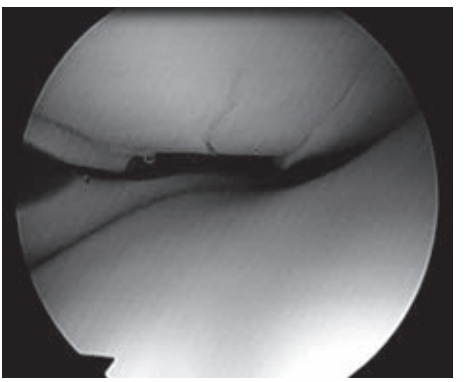
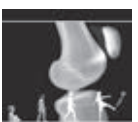


Fig. 2 : Fissures du dôme patellaire



ANALYSE DE LA DYNAMIQUE ROTULIENNE

Analyse avant stabilisation

En extension, seule la facette latérale s'articule avec le fémur. Au cours de la flexion, la crête médiane puis la facette médiale s'engagent dans la gorge trochléenne [5]. Ainsi selon Cascells [4] et Metcalf [13], dans des conditions normales, la crête médiane doit être engagée au cours des 45° premiers degrés de flexion. La facette externe s'engage vers 20°, la facette interne vers 50°. Chez les patients présentant une instabilité rotulienne objective, la crête médiane s'engage à 50° de flexion et la facette interne à 85°.

La cinétique rotulienne dans le plan frontal peut également être appréciée. On notera le caractère luxable ou subluxable ainsi que l'importance de la subluxation (fig. 3). Ceci peut être mesuré en pourcentage de la surface rotulienne ou en quadrants. On évaluera la réductibilité de la translation externe spontanée. Si cela n'est pas le cas, cela signe une rétractation des structures externes.

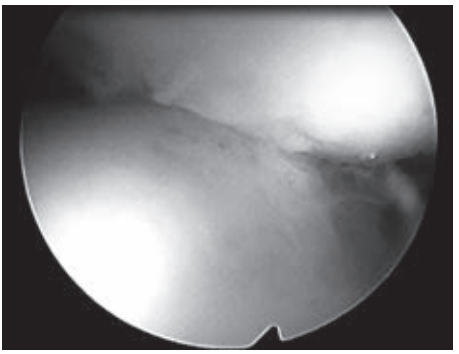


Fig. 3 : Rotule luxable

Analyse des gestes de stabilisation

Après gestes de transferts distaux

L'effet des gestes de transposition de la tubérosité tibiale antérieure demeure difficile à quantifier sous arthroscopie.

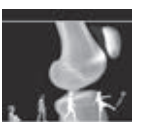
Néanmoins en cas de patella alta, il est possible d'évaluer le positionnement de la rotule par rapport à la limite supérieure de la trochlée genou en extension et l'angle de flexion permettant un contact complet de l'ensemble de facettes articulaires rotuliennes (Angle de flexion de recentrage ou AFR). Après abaissement de la tubérosité tibiale antérieure, le positionnement rotulien genou en extension ainsi que l'AFR sont notés.

Un tel geste doit permettre le recentrage plus précoce de la rotule sur la trochlée tant lors de l'analyse statique que dynamique.

L'efficacité d'un geste de médialisation reste plus difficile à évaluer. Celui-ci agit sur le recentrage rotulien tant en flexion qu'en extension en diminuant la TAGT [8]. Nous avons constaté chez un patient ayant bénéficié d'une médialisation de la tubérosité tibiale antérieure, plusieurs années auparavant, un engagement rotulien "inversé" : la facette médiale entre en contact en premier avec la trochlée puis la crête médiane et enfin la facette latérale. Kuroda [11] et Benvenutti [3] soulignent que la médialisation de la tubérosité tibiale antérieure augmente les pressions de contact sur le compartiment femoro-patellaire médial.

Après plastie du ligament patellofémoral médial (MPFL)

Le MPFL joue le rôle de frein primaire au déplacement latéral de la rotule. Il est systématiquement atteint en cas luxation de rotule. Cette atteinte reste difficile à analyser actuellement sous arthroscopie (fig. 4).



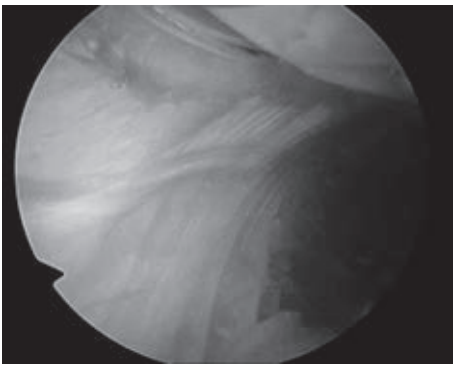


Fig. 4 : MPFL natif

En revanche, après stabilisation, il est possible de vérifier le positionnement purement extra-articulaire de la greffe et de l'absence de brèche articulaire lors du passage de celle-ci. Celle-ci est visualisable comme une bandelette venant s'imprimer en avant de la capsule articulaire médiale (fig. 5). En revanche, la qualité du positionnement des insertions fémorale et patellaire ainsi que la tension adéquate de la greffe ne nous semble pas être évaluable avec précision.

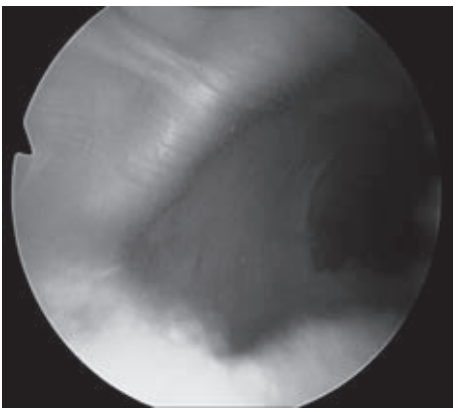


Fig. 5 : Plastie du MPFL

Kita [10] rapporte l'analyse arthroscopique de 25 genoux opérés d'un MPFL pour luxation récidivante de rotule. La cinétique rotulienne a été analysée avant stabilisation et immédiatement après. Avant stabilisation, l'auteur rapporte une tendance à la translation latérale de la rotule quel que soit le degré de flexion du genou. Immédiatement après plastie du MPFL, une cinétique rotulienne normale était restaurée entre 0 et 60° de flexion. De même, dans une étude cadavérique, Sandmeier [16] note que la reconstruction du MPFL améliore la cinétique rotulienne. Amis [1] souligne le fait que le MPFL est le frein primaire à la luxation lors des 20 premiers degrés de flexion c'est-à-dire lors de l'engagement dans la trochlée [2] puis l'action du MPFL diminue lors de la flexion du genou [15] au fur et à mesure que la congruence osseuse augmente.

Après stabilisation, il faut à tout prix éviter que la rotule soit hyper-réduite et fixée dans le plan frontal afin de limiter le risque d'hyperpression médiale. Sandmeier [16] et Ostermeier [14] ont montré que la plastie MPFL a tendance à hyperpermédialiser la rotule et de ce fait surcharger le compartiment fémoropatellaire médial.

Ainsi, l'opérateur s'attachera à évaluer la restauration d'une cinétique rotulienne normale lors de l'engagement. Afin de limiter le risque d'hypercorrection, il est important que la rotule garde un degré de liberté dans le plan frontal. Il est actuellement impossible de fixer des critères de correction idéale sous arthroscopie. Nous avons fixé arbitrairement une translation latérale sur environ la moitié de la facette latérale, genou en extension, comme étant un critère de mobilité résiduelle satisfaisante.

CONCLUSION

L'arthroscopie est un outil de choix pour analyser l'articulation fémoro-patellaire grâce aux voies d'abord antérieures classiques mais éga-

lement grâce aux voies supérieures. Outre l'analyse morphologique statique, elle permet d'étudier la cinétique rotulienne normale et pathologique. De ce fait, elle présente un intérêt certain avant et après chirurgie de stabilisation rotulienne. Néanmoins, il est nécessaire d'établir des critères fiables et reproductibles de mesure de la cinétique rotulienne normale et pathologique afin de pouvoir évaluer la qualité des gestes de stabilisation.

RÉFÉRENCES

[1] AMIS AA, FIRER P, MOUNTNEY J, SENAVONGSE W, THOMAS NP Anatomy and biomechanics of the medial patellofemoral ligament. *Knee. 2003 ;10: 215-20.*

[2] ARENDT EA, FITHIAN DC, COHEN E Current concepts of lateral patellar dislocation. *Clin Sports Med 2002; 499-519.*

[3] BENVENUTTI JF, RAKOTOMANANA L, LEYVRAZ PF, PIOLETTI DP, HEEGAARD JH, GENTON MG Displacement of the tibial tuberosity. *Clin Orthop 1997 (343): 224-34.*

[4] CASCELLS SW The arthroscope in the diagnosis of disorders of the patellofemoral joint. *Clin Orthop 1979; 144: 45-50.*

[5] CUSHNER FD, SCOTT NW Arthroscopic examination and treatment of the patellofemoral joint. *In Scuderi GR : The Patella 1995, Springer-Verlag : 201-21*

[6] DELAUNAY C Evaluation arthroscopique du recentrage femoro-patellaire et corrélation clinique. *Rev Chir Orthop Reparatrice Mot 2000; 86: 482-90.*

[7] DELAUNAY C, KAPANDJI T Effet du remplissage liquidien arthroscopique sur l'engagement fémoropatellaire. *Rev Chir Orthop Réparatrice Mot 1998 ; 84 : 346-9.*

[8] GOUTALLIER D, BERNAGEAU J, LECUDONNEC B. The measurement of the tibial tuberosity. Patella groove distanced technique and results (traduction de l'auteur). *Rev Chir Orthop Reparatrice Mot 1978 ; 64 : 423-8.*

[9] JOHNSON LL Arthroscopic évaluation of the patellofemoral articulation. In JM. Fox & W. Del Pizzo: *The Patellofemoral Joint, 1993, MC Graw-Hill; 335-50.*

[10] KITA K, HORIBE S, TORITSUKA Y, NAKAMURA N, TANAKA Y, YONETANI Y, MAE T, NAKATA K, YOSHIKAWA H, SHINO K Effects of médial patellofemoral ligament reconstruction on patellar tracking. *Knee Surg Sports Traumatol Arthrosc 2012; 20: 829-37.*

[11] KURODA R, KAMBIC H, VALDEVIT A, ANDRISH JT Articular contact pressure after tibial tubercle transfer. A cadaveric study. *Am J Sports Med 2001 (29): 403-9.*

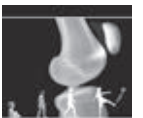
[12] NOMURA E, INOUE M Cartilage lesions of the patella in recurrent patellar dislocation. *Am J Sports Med. 2004; 32: 498-502.*

[13] METCALF RW An arthroscopic method for latéral release of the subluxating or dislocating patella. *Clin Orthop 1982; 167: 9.*

[14] OSTERMEIER S, HOLST M, BOHNSACK M, HURSCHLER C, STUKENBORG-COLSMAN C, WIRTH CJ In vitro measurement of patellar kinematics following reconstruction of the medial patellofemoral ligament. *Knee Surg Sports Traumatol Arthrosc 2007; 15: 276-85.*

[15] PHILLIPOT R, BOYER B, TESTA R, FARIZON F, MOYEN B The rôle of the medial ligamentous structures on patellar tracking during knee flexion. *Knee surg Sports Traumatol Arthrosc 2012; 20: 331-6.*

[16] SANDMEIER RH, BURKS RT, BACHUS KN, BILLINGS A The effect of reconstruction of the medial patellofemoral ligament on patellar tracking. *Am J Sports Med 2000; 28: 345-9.*



BIOMÉCANIQUE DU LIGAMENT PATELLO-FÉMORAL MÉDIAL
MPFL BIOMECHANICS

J. CHOUTEAU

INTRODUCTION

Le ligament patello-fémoral médial (MPLF) est situé sur la face médiale du genou et unit le bord médial de la patella à la métaphyse fémorale.

La face médiale du genou présente plusieurs plans tissulaires. Le plan superficiel est constitué par le rétinaculum médial. Le deuxième plan, ou plan intermédiaire, est celui du ligament patello fémoral médial (MPLF) et du ligament collatéral médial (MCL). Enfin, le plan profond est composé de 2 structures. Il s'agit tout d'abord du ligament patello méniscal médial (MPML) dont la longueur est de 39,4 ± 3,2 mm et la largeur de 9,6 ± 1,2 mm. Enfin, le ligament patello tibial médial (MPTL), plus inconstant, a une longueur de 54,6 ± 8,4 mm et une largeur de 21,8 ± 4,4 mm. La présence simultanée du MPFL et du MPTL est retrouvée dans 47 % des cas.

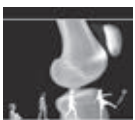
ANATOMIE DU MPFL

Philippot [1], sur une étude anatomique de 23 genoux cadavériques, a constamment retrouvé la présence du MPFL. Sa longueur était de 57.7 ± 5.8 mm et sa largeur de 12.2 ± 2.6 mm à son insertion fémorale et de 24.4 ± 4.8 mm à son insertion patellaire. Une jonction entre le MPFL et le vaste interne (VMO) a constamment été retrouvée. Il s'agissait d'une zone de réflexion avec un entrecroisement de fibres provenant du MPFL et du VMO d'une longueur de 25.7 ± 6 mm.

Un rapport étroit entre les fibres du faisceau superficiel du MCL et le MPFL sous la forme d'une arche ligamentaire a été mise en évidence sur 40 % des genoux étudiés par plusieurs études [2, 3].

L'insertion fémorale du MPFL a été étudiée précisément par Philippot [1] grâce à la réalisation d'un repère orthonormé centré sur l'insertion fémorale du MPFL. Celle-ci se situait postérieurement et proximalement par rapport à l'épicondyle médial de 10.7 ± 3.3 mm et en avant et distalement de 11.2 + 5.9 mm par rapport au tubercule de l'adducteur.

L'insertion patellaire a été constamment retrouvée sur la moitié supérieure de la berge médiale de la patella. Une extension du MPFL peut aussi s'insérer sur l'insertion patellaire du tendon quadricipital. La présence combinée d'une extension d'insertion sur le tendon quadricipital et sur le vaste interne a été retrouvée par certaines études [2-4].



BIOMÉCANIQUE DU MPFL

Données générales

Amis [5] a retrouvé, sur une étude de 10 genoux cadavériques (âge moyen 70 ans), une résistance moyenne du MPFL à la rupture de 208N.

Les contraintes transmises au MPFL durant la flexion du genou ont été étudiées notamment par Elias [6]. Elles atteignent un maximum de 20N à 30° de flexion et sont proches de 0N à 90° de flexion.

Le MPFL participe au contrôle de la translation latérale de la patella pour une proportion de 53 % pour Conlan [3], de 60 % (41 à 80 %) pour Desio [7] et de 50 % pour Hautamaa [8].

Cinématique de la patella sur genou sain et après section du MPFL

Philippot [9] a mis au point un protocole opto-électronique d'évaluation de la cinématique du MPFL. Pour cela le fémur, le tibia et la rotule ont été marqués à l'aide de marqueurs tripodes. De cette manière, les mouvements des 3 segments pouvaient être enregistrés les uns par rapport aux autres pour les 6 degrées de liberté.

Cinématique de la patella sur genou sain

La bascule de la patella (patellar tilt) était quasi nulle durant les premiers 45° de flexion du genou. A partir de 45° de flexion, la patella montrait une bascule externe qui s'accentuait aux alentours de 90° de flexion, atteignant un maximum de -3.7 ± 8.9 mm par rapport à sa position de référence initiale.

La translation de la patella (patellar shift) était progressive et latérale pour atteindre, à 90° de flexion, une translation maximale de 3.18 ± 9.67 mm par rapport à sa position de référence.

Enfin, durant les premiers 30° de flexion du genou, la patella présentait une rotation médiale (patellar rotation) atteignant une amplitude maximale de 0.8 ± 4.6 à 15° de flexion. Ensuite, se produisait une inversion vers la rotation latérale atteignant un maximum de 1.45 ± 6.5 à 90° de flexion.

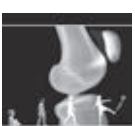
Cinématique de la patella après section du MPFL

Après section du MPFL, la translation externe de la patella augmentait de manière statistiquement significative (maximale en extension et diminuant avec la flexion du genou). La translation externe augmentait ainsi de 72 % en extension complète et de 52 % à 90° de flexion.

La bascule externe de la patella augmentait aussi de manière significative et était maximale en extension. L'augmentation était ainsi de 76 % en extension complète et de 28 % à 90° de flexion. Enfin, après section du MPFL, la patella montrait une rotation externe, de l'extension à 35° de flexion produisant une augmentation de la rotation externe de 62 % en extension maximale et de 0 % à 35° de flexion.

CONCLUSION

Le ligament patello-fémoral médial est une structure constamment retrouvée au sein du second plan tissulaire de la face médial du genou. Il est le stabilisateur principal de la patella entre l'extension complète du genou et la flexion à 30°.



RÉFÉRENCES

[1] PHILIPPOT R, CHOUTEAU J, WEGRZYN J, TESTA R, FESSY MH, MOYEN B. Medial patellofemoral ligament anatomy: implications for its surgical reconstruction. *Knee Surg Sports Traumatol Arthrosc. 2009 May; 17(5): 475-9.*

[2] SMIRK C, MORRIS H. The anatomy and reconstruction of the medial patellofemoral ligament. *Knee. 2003 Sep; 10(3): 221-7.*

[3] CONLAN T, GARTH WP Jr, LEMONS JE. Evaluation of the medial soft-tissue restraints of the extensor mechanism of the knee. *J Bone Joint Surg Am. 1993 May; 75(5): 682-93.*

[4] TUXOE JI, TEIR M, WINGE S, NIELSEN PL. The medial patellofemoral ligament: a dissection study. *Knee Surg Sports Traumatol Arthrosc. 2002 May; 10(3): 138-40.*

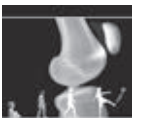
[5] AMIS AA, FIRER P, MOUNTNEY J, SENAVONGSE W, THOMAS NP. Anatomy and biomechanics of the medial patellofemoral ligament. *Knee. 2003 Sep; 10(3): 215-20.*

[6] ELIAS JJ, COSGAREA AJ. Technical errors during medial patellofemoral ligament reconstruction could overload medial patellofemoral cartilage: a computational analysis. *Am J Sports Med. 2006 Sep; 34(9): 1478-85.*

[7] DESIO SM, BURKS RT, BACHUS KN. Soft tissue restraints to lateral patellar translation in the human knee. *Am J Sports Med. 1998 Jan-Feb; 26(1): 59-65.*

[8] HAUTAMAA PV, FITHIAN DC, KAUFMAN KR, DANIEL DM, POHLMEYER AM. Medial soft tissue restraints in lateral patellar instability and repair. *Clin Orthop Relat Res. 1998 Apr(349): 174-82.*

[9] PHILIPPOT R, CHOUTEAU J, TESTA R, MOYEN B. In vitro analysis of patellar kinematics: validation of an opto-electronic cinematic analysis protocol. *Knee Surg Sports Traumatol Arthrosc. 2010 Feb; 18(2): 161-6.*



INLAY OR ONLAY PFA

P.B. SCHOETTLE

Patellofemoral arthritis is a pathology which can be caused by several factors. Depending on the interplay of the structures involved in the patellofemoral joint there is a difference between the direct and indirect development of the PFA. The group of direct causes includes direct blunt trauma, fracture of the patella, traumatic patellar dislocation, chronic overloading by profession, sports or overweight and rarely osteochondritis dissecans. Malalignment, a poor tracking and positioning of the patella, is the cause for the indirect development of PFA. It is mainly induced by trochlear dysplasia, valgus deformity and femoral anteversion. Regarding successful therapy of PFA the precise clinical and radiological diagnostics of the underlying causes is a necessary precondition.

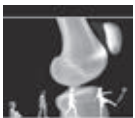
Since biological treatment options in the PFJ are limited in their success, and anterior transfer of the tuberosity can only be carried out by accepting a medial or lateral increase of the PF loading, which may be not sufficient in some cases, especially with an underlying trochlear dysplasia, the indication for an isolated patellofemoral replacement may be given.

For a long time, reputation of these partial arthroplasties have been not very promising (in comparison with the UKR i.e.), since the outcome was no as satisfying due to a symmetrical, non-physiological V-shape of the systems.

However, actual products respect the given anatomy and can be separated in traditional onlay and recent inlay systems.

The main difference in between those two systems is the surgical technique and the postoperative joint volume: in onlay systems, the arthroplasty is cemented onto a bony plain, flattened by an anterior femoral osteotomie, where the degenerative cartilage and the underlying bone has been removed (fig. 1). While some rotational malalignments and even valgus mismatches can be influenced positively by an anterior osteotomie, the only technique comprises the risk of a postoperative bone-volume augmentation: in cases of small knees, where the anterior osteotomie is limited by given anatomical facts to avoid a weak spot of the distal femur and the risk of a postoperative fracture, an anteriorisation of the femoral joint line and an therefore an augmentation of the patellofemoral joint pressure with ongoing pain can occur.

Other then that, in inlay systems, the defect area is reamed only down to the demanded depth of the trochlea, respecting the supero-inferior curvature of the given joint. Therewith it is possible to recreate the given trochlea shape or to change a before dysplastic into a physiological trochlea, respecting the surrounding bone shape and curvature. In these



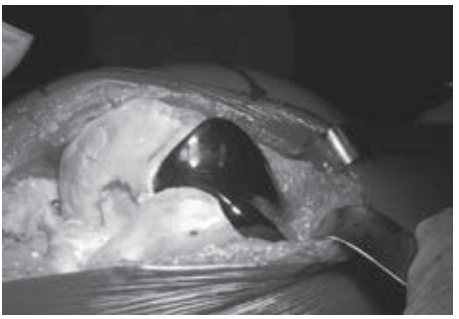


Fig. 1

techniques, the new surface rather works like liquid metal without the risk of an overstuffing (fig. 2). However, any bone malalignment, especially a pathological internal rotation can not be treated with these methods.

As a conclusion: nowadays, an inlay system seems to be the preferred thechnique in patellofemoral arthritis, since a more anatomical result can be achieved. In cases with an additional (causing) malalignment, either an onlay technique with a correcting anterior femur cutting osteotomie or a combinative treatment of an osteotomie and an inlay technique has to be performed.

Additional resurfacing of the patella seems to be indicated only in cases with a concave patella without any physiological gliding properties or for improving kinematics and stability, while the focal degeneration of the patellar cartilage has no negative influence on the outcome if not treated.

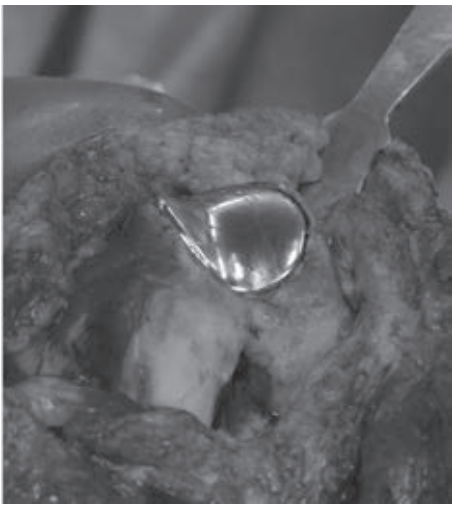


Fig. 2a

Fig. 2b

LATERAL-SIDED SURGERY WITH MPFL RECONSTRUCTION: WHEN IS THIS NEEDED?

E.A. ARENDT

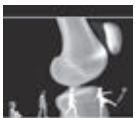
This chapter discusses when lateral-sided surgery is necessary to perform concomitant with a Medial Patellofemoral ligament (MPFL) reconstruction. Specifically, when does one need a lateral retinacular release/lengthening as part of the surgical stabilization for the patella to prevent recurrent lateral dislocations? In order to answer this one needs to review "lateral patella tilt".

If one looks at the historic roots of patella instability, the majority of early surgical procedures favored increasing the directional pull of medial-sided structures while eliminating or diminishing the pull of (abnormal) lateral-sided forces (i.e.) a lateral retinacular release [1, 2, 3]. *Campbell's Operative Orthopaedics* (1980), in reviewing surgical procedures for patella instability, reported that procedures used to surgically correct lateral patella dislocations that have been the most widely used and the most successful included "release of tight and contracted soft tissues, including the vastus lateralis tendon, to remove deforming forces" [4].

Patella position in the sagittal view has been analyzed and measured since late 1930's when Blumensaat recognized patella position and its relationship to patella dislocation [5].

Patella tilt, however, began to be analyzed and measured with the introduction of radiographic axial imaging. The addition of axial slicing afforded by CT scans and later MR imaging added to the recognition and evaluation of this anatomic feature. The position of the patella in relation to the trochlear groove was a primary component of "patella malalignment."

H. Dejour and G. Walch, leading a combined effort with the entire "Lyonnaise" team, analysed over 1800 radiographic cases of patellofemoral (PF) patients and controls. This analysis utilized precise radiographic review of the trochlea on lateral view radiographs with a strict superposition of both posterior femoral condyles, and a standardised CT protocol. Within this body of work the Lyonnais team defined principle imaging factors associated with lateral patella dislocations (objective instability). The authors defined lateral patella tilt by overlapping CT images comparing the long axis of the patella to the posterior femoral condylar line. A lateral patella tilt >20° was the threshold value associated with objective patella instability [6]. Excessive lateral patella tilt was included as a correctable risk factor in the "le menu à la carte". An early hypothesis of the etiology of lateral patella tilt was one of muscle weakness, specifically VMO dysplasia.



The Lyonnais team further described a surgical algorithm to treat patella instability by correcting each anatomical (radiographic imaging) abnormality. Excessive lateral patella tilt was treated with a lateral retinacular release, and a "VMO plasty".

Several seminal articles describe lateral patella tilt as an injury variable in need of surgical correction, describing lateral patella tilt as an imaging feature without a physical exam association [7, 8].

The reader is left with the assumption that the imaging sign of excessive lateral patella tilt is associated with tight lateral structures that presumably needed to be lengthened or released. Although patella tilt is difficult to define objectively by physical exam, it is recognized that lateral patella tilt is associated with a decrease in medial patella translation and negative medial patella tilt test (the inability to bring the lateral board of the patella past the level of the horizon) (fig. 1). When you associate excessive lateral patellae tilt on X-ray with a physical exam sign of lateral tightness, this represents patella tilt that may need to be surgically corrected.

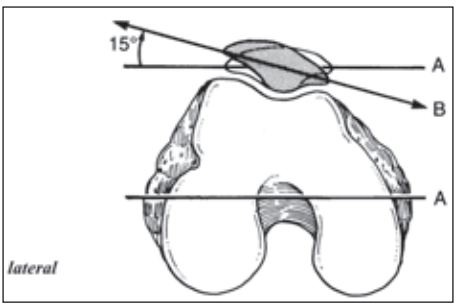


Fig. 1: Medial Patella Tilt Test

A critical aspect of patella tilt analysis is that excessive lateral patella tilt may represent lateral tightness, but it always represents some degree of medial retinacular laxity. This is particularly important when viewing an acute image; one nearly always sees excessive lateral patella tilt and a large hemarthrosis due to the associated trauma of an acute patella dislocation. Therefore, measurement of lateral patella tilt can be grossly over-estimated on MR, CT, or axial radiograph when it is associated with a large degree of knee swelling as is typical in the acute injury phase.

Lateral patella tilt can also be mis-leading when it is associated with loss of cartilage in the lateral patellofemoral joint. Laurin *et al.* [9], describe lateral patella tilt as being due to cartilage loss from the lateral trochlea and lateral patella facet, not tightness of the lateral retinaculum. Indeed, we often see a "pseudo tilt" associated with patellofemoral arthrosis, where the lateral tilt of the patella is due to loss of cartilage in the lateral patellofemoral joint space [10] (fig. 2), rather than lateral patella tilt being obligated by one's own innate morphologic anatomy.

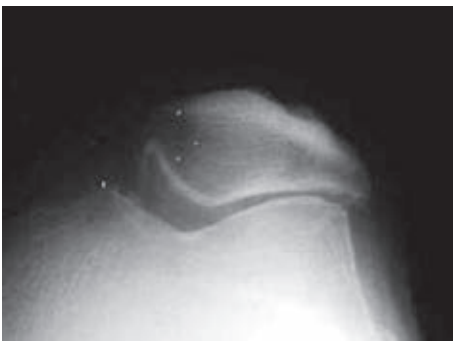
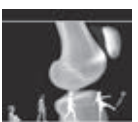


Fig. 2 : Patella tilt due to loss of cartilage in the lateral PF compartment (Iwano Type 3 classification of PF Arthritis).

Noting the concerns of mis-interpreting patella tilt *as always associated with "too tight" lateral soft tissues structures*, it is noted that the degree of lateral patella tilt has been shown to have a direct relationship to the degree of trochlear dysplasia (i.e.), high grade trochlear dysplasia is associated with greater patella tilt [11]. For students of patellofemoral morphology, the lateral soft tissue structures offer the most challenging and diverse soft tissue dilemmas in dysplastic patellofemoral joints.



When to release or lengthen the lateral soft tissue structures:

With the above discussion as a prologue, one can attempt to answer the question "When are lateral sided structures a deforming force restricting a balanced patella?".

The most correct statement would be: one should relax the lateral structures by lengthening and/or releasing the lateral structures when they offer resistance to normalization of patella position in early flexion. The harder question is: how does one determine this?

The discussion of when to release/lengthen lateral sided retinacular structures will be restricted to those cases that one assumes the major feature preventing normalization of the patella position is a soft tissue structure, not the trochlea. Certainly, the morphology of the trochlea in early flexion can also be a reason to have excessive lateral patella tilt [11].

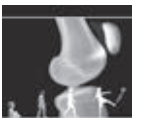
However, when one is talking about high-grade trochlear dysplasia associated with large degrees of patella tilt, one nearly always needs to lengthen or release the lateral structures; that is not what is under discussion in this article. This article attempts to address those more subtle cases of "isolated MPFL reconstructions" that may or may not need a lateral retinacular lengthening/release.

An ideal candidate for an isolated MPFL reconstruction (without bony work (eg) tibial tubercle osteotomies/trochleoplasties) might have the following profile of risk factors:
- Trochlear dysplasia, type A, or normal trochlea.
- A tubercle sulcus angle of 0 to 5° valgus/or TT-TG less than 20mm. (no significant mal-alignment of the patellofemoral joint).
- Patella alta less than 1.3 (Insall/Salvati or Caton/Deschamps index) and/or "reasonable" overlap of the patella and trochlea surfaces on sagittal MR [12] (functional patella engagement with the trochlea).

In regards to lateral lengthening, the author reviews the following pre-operative factors to aide in the decision of how to manage the lateral soft tissue structures.

- Lateral patella tilt less than 20° utilizing axial image with posterior femoral condyles as a reference, measured on an image without notable knee effusion (in a non-acute injury setting) usually does not need lateral lengthening.
- If the non-acute axial image in full extension shows increased lateral patella tilt but the tilt corrects in early flexion (20° Laurin's view or a 30° Merchant's view), the patella rarely needs lateral structures lengthened.
- Axial radiographs taken in early flexion reveal excessive lateral tilt on both sides, with no injury to the opposite (non-injured) knee; this is a strong sign that lateral sided deforming forces are present.
- Patella tilt that has no lateral tightness on physical exam after the patella is relocated does not need lateral side lengthening. (This may be necessary to evaluate intra-operatively).

The final decision for how to manage lateral soft tissue structures is made intra-operatively. One way the author has evaluated lateral sided tightness intra-operatively when doing MPFL reconstructions is the following: a K wire is passed medial to lateral across the patella at the most superior aspect of the patella's insertion of the MPFL, exiting at the lateral border of the patella. This gives the surgeon a true representation of the medial to lateral axis of the patella. One can then test the lateral tightness by seeing if the K wire remains level (parallel to the horizon) at full extension, and then again at 20°, or when the patella is initially engaged in the trochlear groove. If the K-wire remains tilted, the author lengthens the lateral sided structures (fig. 3). This can be referred to as an "intra-operative patella tilt test", with a very visible structure (the K-wire) representing the long axis of the patella. In the author's MPFL technique, this K-wire is used to



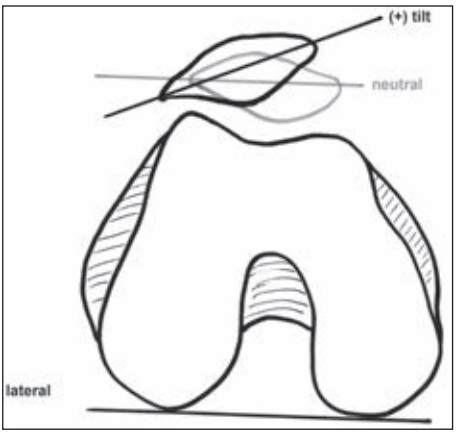


Fig. 3 : K-wire placed thru the M-L axis of the patella intra operatively.
Black: patella M-L axis cannot be brought to a horizontal level.
Grey: post-lateral retinacular lengthening/release.

establish a short docking station for the MPFL by over-drilling the K-wire with a 4mm cannulated reamer for a length of 10mm. Even if one's preferred surgical technique involves a different kind of patella fixation, this technique can be used by inserting a K-wire as described above and later removing it.

The author's preference is to lengthen, not release, the lateral sided soft tissue structures of the patella (fig. 4). In support of lateral sided lengthening: it is a more precise balance of the patellofemoral forces and it reduces the potential for excessive medial patellofemoral translation. Conversely, lateral retinacular lengthening does require either a larger incision or two incisions. It slightly increases the operating room time. At times, the gap between the lateral structures is too great to be lengthened and a release is necessary; however in the surgical profile detailed above ("isolated" MPFL reconstruction), the gap is rarely >20mm. When one performs a lengthening of the lateral tissue, one often has 15-22mm of tissue to lengthen.

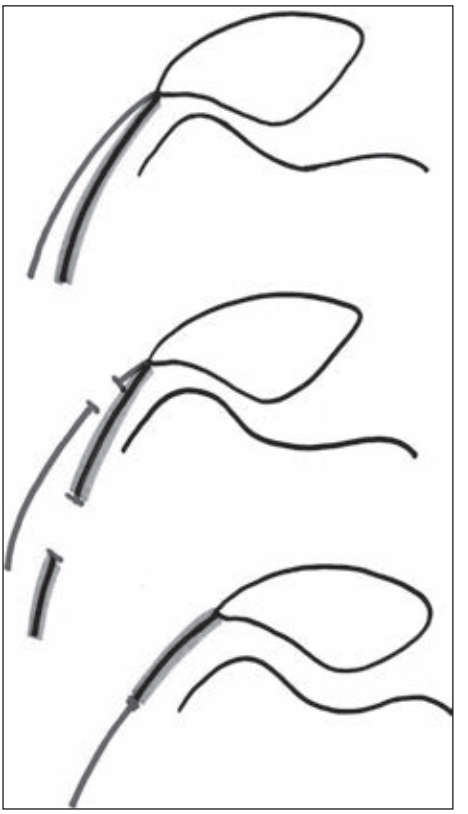


Fig. 4 : Schematic drawing of lateral retinacular lengthening : ITB = Iliotibial band, LPFL= lateral patellofemoral ligament
Dark-grey: ITB
Light-grey: LPFL

LITERATURE

[1] ROUX C. Luxation habituelle de la rotule: Traitement operatoire. *Rev Chir Orthop 1888. 8, 682-9.*

[2] GOLDTHWAIT JE. Dislocation of the patella. *Am Orthop Assn 1895. 8, 237-8.*

[3] TRILLAT A, DEJOUR H, COUETTE A. 1964. [Diagnosis and Treatment of Recurrent Dislocations of the Patella]. *Rev Chir Orthop Reparatrice Appar Mot 50, 813-24.*

[4] CAMPBELL WC, EDMONSON JH, CRENSHAW JR. In: Campbell, W.C., Edmonson, J.H., Crenshaw, J.R. (Eds.), *Campbell's Operative Orthopaedics, 6th Edition. Elsevier (Mosby), Waltham, p. 464, 1980.*

[5] BLUMENSAAT C. Die lageabweichungen and verrenkungen der kniescheibe. *Ergeb Chir Orthop 1938. 31, 149-223.*

[6] DEJOUR H, WALCH G, NOVE-JOSSERAND L, GUIER C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc 1994. 2, 19-26.*

[7] DEJOUR H, WALCH G, NOVE-JOSSERAND L, GUIER C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc 1994. 2, 19-26.*

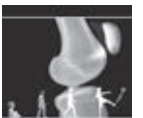
[8] SCHUTZER SF, RAMSBY GA, FULKERSON JP. The Evaluation of Patellofemoral Pain Using Computerized Tomography: A Preliminary Study. *Clin Ortho 1986: 204: 286-93.*

[9] LAURIN *et al.* (Laurin CA, Levesque HP, Dussault R, Labelle H, Peides JP "The abnormal patellofemoral angle: ADiagnostic Roentgenographic sign of recurrent patellar dislocation". *JBJS, 60A55-60 (1978).*

[10] IWANO T, KUROSAWA H, TOKUYAMA H, HOSHIKAWA Y. Roentgenographic and clinical findings of patellofemoral osteoarthrosis. With special reference to its relationship to femorotibial osteoarthrosis and etiologic factors. *Clin Orthop Rel Res. 1990; 252: 190-7.*

[11] PR SAGGIN, D DEJOUR, X MEYER, T TAVERNIER. CT and arthro CT scan in patellofemoral disorders. In Zaffagnini S, Dejour D, Arendt EA (Eds.), Patellofemoral Pain, Iinstability and Arthritis. *Springer-Verlag, Berlin Heidelberg, pp.73-78, 2010.*

[12] BIEDERT RM, ALBRECHT S. The patellotrochlear index: a new index for assessing patellar height. *Knee Surg Sports Traumatol Arthrosc 14, 707-12, 2006.*



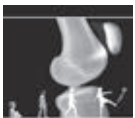
ANATOMICAL DOUBLE BUNDLE MPFL RECONSTRUCTION

P.B. SCHOETTLE

INTRODUCTION

The medial patellofemoral complex, consisting of the medial patellofemoral ligament (MPFL) and the medial patellotibial ligament, is the main passive stabilizer of the patellofemoral joint. Since it has been shown that rupture of the MPFL is the main pathological consequence of patellar dislocation [1] and biomechanical studies have demonstrated that the MPFL is the main restraint against lateral patellar displacement, reconstruction of the MPFL has become a widespread technique for restoration of patellofemoral stablity. An additional reason that MPFL reconstruction became as popular is the fact that distal realignment procedures such as transfer of the tibial tuberosity or release at the lateral patellar retinaculum/capsule have provided inadequate restoration of patellofemoral stability in every patient, frequently leading to increased mediolateral instability, increased patellofemoral pressure or arthritic degeneration.

Therefore, numerous techniques for reconstruction of the medial patellofemoral complex have been described with promising clinical results [2]. However, since it is known that a non-anatomical reconstruction of the MPFL can lead to non-physiologic patellofemoral loads and kinematics [3], the goal of a surgical intervention must be an anatomical reconstruction. Since the femoral insertion of the MPFL has been evaluated anatomically [3], biomechanically [4], and radiologically [5], the complications of increased patellofemoral pressure in flexion associated with non-anatomic femoral graft fixation that is too anterior/proximal [3] can be avoided. Upon careful observation of the anatomical shape of the original MPFL, it is apparent that the patellar insertion is much wider than the femoral one. Additionally, Amis *et al.* have proven double bundle structure provides a more stable proximal and distal ligamentous structure [3]. Respecting this anatomic condition, a double bundle reconstruction at the patellar side is reasonable to restore native ligamentous morphologic and biomechanical properties; moreover, this method lessens the patellar rotation during flexion-extension movement that may occur during single bundle reconstruction. Under these conditions, the double bundle reconstruction, described earlier [6] shows very satisfying clinical results. As we know from ACL reconstruction, direct anatomical/aperture fixation, [7] provides the highest time-zero fixation by avoiding elongation of the graft or "bungee" effect, resulting in the possibility of early rehabilitation with full range of motion. In a similar manner, these concepts may be applied to MPFL reconstruction.



Although most of the actual surgical techniques utilize a free tendon graft to reconstruct the MPFL as the only method for anatomic double bundle graft fixation, an all aperture fixation has not yet been described.

Recent studies have described an anatomical double bundle reconstruction, using an aperture fixation at the femoral insertion [6, 8], while the patellar fixation remains relatively indirect resulting in the eventual risk of post-operative micromotion and subsequent loosening. Patellar graft fixation has been described with either an anchor system, attaching the graft into a bony rim, [6] or by tying the attached graft sutures to each other at the lateral patellar edge [8]; however, this method may potentially result in graft slippage by degloving [7].

Until today, only one technique described anatomical patellar fixation by looping graft through bone tunnels without any additional fixation device [9]. This technique appears to produce stable fixation at the patella. However, in soft bone, a widening of the tunnel could occur in the long-term; moreover, in patients with a short gracilis graft, the tendon length may not be long enough to reach the anatomical femoral insertion.

The double bundle technique described here offers an aperture fixation at the patella and the femur, providing a high initial stability on both insertions, resulting in improved bony in growth, and consequently, an earlier return tofull range of motion.

SURGICAL TECHNIQUE

Harvesting and preparing of the gracilis tendon

After completion of the arthroscopy, a 2cm long oblique incision is performed at the pes anserinus. After incising the sartorius aponeurosis, the gracilis tendon is harvested and used as an autograft. The load to failure force of the Gracilis graft – even as a single bundle-exceeds the failure to load of the MPFL (208N). [3] The usable part of the tendon should be at least 18cm long. After harvesting the tendon with the stripper and removing the muscle tissue, the doubled tendon diameter is determined and both ends are whipstitched with an absorbable braided suture over a length of 15mm.

Preparing the soft tissue layer

A 2cm skin incision is performed from the superomedial corner to the end of the medial margin of the patella, where the patellar MPFL insertion is located [4, 10] (fig. 1). As the MPFL is situated central to the Vastus medialis obliquus (VMO) in the second layer of the medial patellofemoral complex [10], the central part of the VMO is identified and a scissor is brought along to the medial femoral epicondyle in between the VMO and the joint capsule, cautiously avoiding any injury to the joint. After the opened scissors are removed, a right angle clamp is brought into the separated layer and the tip is directed towards the skin in the area of the adductor tubercle, where the femoral MPFL insertion is located. Then a small longitudinal skin incision is performed over the tip in 30° knee flexion, the position wherethe graft will be finally fixed. Finally, in preparation for passing the final graft, a suture loop is inserted in between the second and the third layer using the right angle clamp.

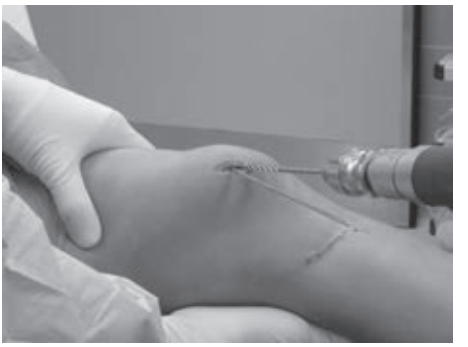
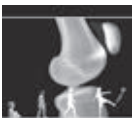


Fig. 1: After the anatomical landmarks of the patella are marked, a 2cm incision from the supermedial corner of the patella to the medial margin is performed and two 4.0mm holes are drilled for the MPFL insertion.



Preparing the femoral insertion site

To avoid non-physiological patellofemoral forces, the femoral MPFL insertion has to be very accurate. Therefore, a guide wire with an eyelet is placed slightly posterior to the midpoint of the medial epicondyle and the adductor tubercle and the entering point into the bone is marked with a clamp [10]. Then the guide wire placement is controlled by a picture intensifier on a straight lateral view to obtain the correct anatomical femoral insertion; if the graft is placed too anterior or proximal, abnormal graft tensioning will lead to increased patellofemoral pressures during flexion [3]. Therefore, we use the radiographic landmark of the anatomical MPFL insertion which has been shown to be located slightly anterior to an elongation of the posterior femoral cortex in between the proximal origin of the medial condyle and the most posterior point of Blumensaat's line [6]. If necessary, the guide wire entry point is corrected before overdrilling to the contralateral cortex with a drill diameter 1mm larger than that of the graft loop.

Preparing the patellar insertion site

To achieve aperture fixation at the patellar side, the free graft ends have to be fixated directly to the patella. Therefore, the medial patellar margin is prepared and two guide wires are drilled tangentially into the patella at the proximal and distal end of the medial edge. The guide wires are subsequently overdrilled with a cannulated 4mm drill to a depth of 20mm.

Graft fixation

The two free sutured graft ends are fixed into the patellar holes one after each other, using a 4.75x15mm Swivel Lock (Fa. Arthrex), achieving a direct anatomical graft fixation. To accomplish this, the graft sutures are pulled through the PEEK eyelet of the Swivel Lock, and pushed into the drill holes. Keeping the suture under tension, the graft ends are fixed with the 4.75x15mm Swivel Lock screw (fig. 2). In this way, a double bundle aperture fixation at the patellar side is achieved, leaving the graft loop free (fig. 3).

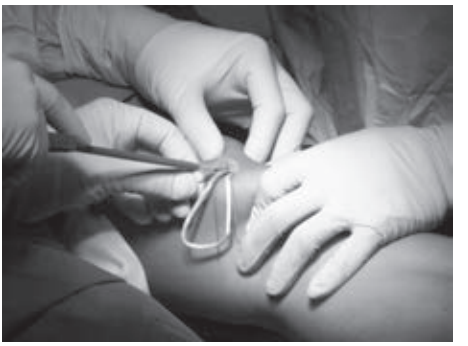


Fig. 2: Using two Swivellock anchors, the armed graft ends are inserted and fixated into the patella.

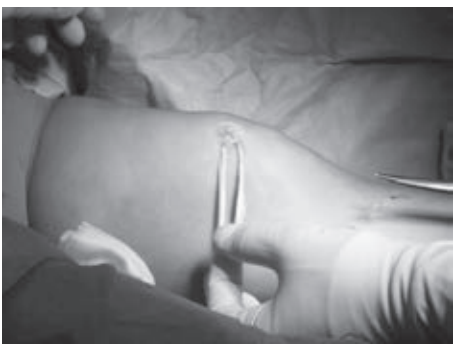
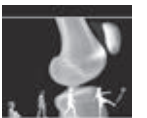


Fig. 3: Graft ends are fixed using a 4.75x15mm Swivel Lock screw while tensioning the graft.

The suture loop is then used to pull the graft in between layer 2 and 3 to the femoral insertion. Next, a Nitinol wire is inserted into the femoral drill hole and the suture loop of the graft is pulled laterally using the guide wire. Finally, while maintaining equal tension on both bundles, the graft is pulled into the femoral socket. Since biomechanical studies have shown that the MPFL has its maximal length and restraint against patella lateralisation in 30° of flexion (fig. 4) [3], femoral fixation is performed in 30° of flexion with the lateral patellar edge positioned in line with the lateral trochlear border using a bioresorbable interference screw. An anatomical femoral insertion avoids an overcorrection, since an



overtension of the graft can only occur if the femoral tunnel is placed too far anterior or proximal. In this case, the insertion point would move towards posterior in flexion, leading to a lengthening of the distance between patellar and femoral insertion, increasing the load onto the graft, and consequently, onto the patello-femoral joint.

If adequate medial restraint has been restored, lateral patellar dislocation should no longer be possible and routine skin closure is performed after reattaching the aponeurosis of the VMO back to the medial edge of the patella with resorbable sutures.

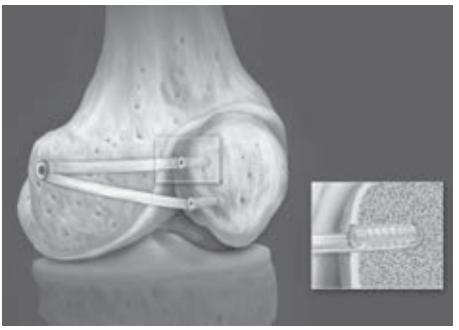


Fig. 4: The double bundle aperture fixation is achieved at the patellar side as well as at the femoral insertion.

POSTOPERATIVE TREATMENT

Compared to other techniques, this aperture fixation with a biotenodesis screw at the patellar insertion provides an immediate stable tendon to bone fixation with an ultimate load to failure force at the patellar side higher than the 208N needed to rupture an intact MPFL [3]. Weight bearing is allowed, however, no more than 20kg until wound healing, while leg raising and quadriceps setting exercises can be started immediately with a free range of motion as tolerated.

Low impact activities such as running or cycling are allowed at 6 weeks post-op; full activity is permitted at 3 months.

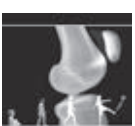
DISCUSSION

The most important finding and improvement in using the above described technique was the possibility of an immediate full range of motion due to the aperture fixation at both sides. The benefit of anatomic graft positioning in ligament reconstruction has been known for a long time and has been clearly demonstrated in ACL reconstruction. Anatomical reconstruction of the MPFL is particularly important as biomechanical studies have demonstrated that the length change pattern of a MPFL reconstruction depends critically on the site of the femoral attachment; moreover kinematics change significantly when the patellar or the femoral insertion has been off by only 5mm [4]. Aside from tunnel placement, graft fixation is the other determining factor in ligament reconstruction [7]. Non-aperture fixation at either the femoral or patellar insertion can increase the risk of a delayed or insufficient tendon to bone healing, which may result in early loosening or slackening of the graft. To avoid this, a restricted range of motion is recommended by some surgeons; however, this may lead to arthrofibrosis, potentially necessitating an additional arthroscopic arthrolysis, carrying the additional risk of damage to the graft.

However, until today, only one technique describes a double bundle aperture fixation at both sides where the graft is looped through a bone tunnel in the patella [9]. In this technique, the graft is shuttled through the patella and fixated press fit without any fixation devices, providing a high intial fixation strength.

The aim of this manuscript was therefore to describe a procedure for an anatomical double bundle reconstruction of the MPFL, respecting not only the ligament shape and both the anatomical patellar and femoral insertion areas, but also an aperture fixation.

In recent studies, a tendon transfer is described either to the patella or to the femur for reconstructing the MPFL [11, 12]. However, in these techniques, not only the is transferred muscle weakened in its original motion, but



neither the patella nor the femoral insertion can be reconstructed at its anatomical insertion, and the graft used is always a single bundle graft despite the fact that the MPFL consists of a proximal and a distal bundle [3]. This also includes a single point fixation at the patellar side, increasing the rotational moment of the patella in flexion extension movement. In terms of the fixation itself, some techniques fix the graft to the surrounding soft tissue [13] and not to the patellar and/or femoral bone even though a ligament is a structure in between bones, and recent studies have proven the high resistance to failure of tendon-bone interfaces.

Since tendon to bone healing showed excellent results in ACL reconstruction with hamstring tendon grafts, the same tendon-to-bone fixation is used at both, the femoral and the patellar side in our technique.

While tendon-to-bone tunnel healing is created on the femoral side using a biodegradable screw for fixing the graft loop, the free graft ends are now fixed directly and anatomically into the patella using a biotenodesis device. This technique seems to provide a higher load to failure strength than an earlier described technique, where a laminar attachment of the graft was performed at the patellar side using two suture anchors [6], which may result in loosening of the knot during full range of motion testing; or, the patellar bone may be too soft for a secure anchoring. Knotting the free graft ends to each other at the patellar edge [8] are comparable to a non-aperture indirect fixation in ACL surgery. With the suture anchor technique, a secure graft to bone healing can not be provided in every case and loosening of the graft could occur.

Another technique, looping the graft through the patella provides a very high initial fixation strength and preliminary results are promising [9]. However, if micromotion in the patellar tunnels develops, it may lead to a slackening of the graft at later follow up. However, if the graft is very short, the femoral insertion can not be reached and an overly anterior fixation has to be accepted, leading eventually to an increased patellofemoral pressure or loss of fexion.

The use of a free autograft in the above described technique allows us to place the graft at the anatomical insertion with a sufficient length and to recreate the double bundle structure of the MPFL as it was described in an anatomical study by Amis [3]. This provides a higher stability, as the proximal bundle seems to stabilise in extension, while the distal bundle stabilises in flexion. Furthermore, the double bundle reconstruction decreases patellar rotation in contrast to techniques where only a single point fixation is performed or the middle part of the quadriceps tendon is flipped medially [4]. Reproducing the anatomy of the native MPFL enables the reconstructed ligament to have an isometric function, and therefore avoids increased patellofemoral pressure in higher degrees of knee flexion [6]. We also estimate that in long-term follow up, slackening of the graft will not occur due to an improved tendon to bone healing by using direct fixation at the femoral as well as at the patellar insertion.

REFERENCES

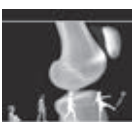
[1] SM DESIO, RT BURKS, KN BACHUS Soft tissue restraints to lateral patellar translation in the human knee. *Am J Sports Med 1 (1998) (26):59-65.*

[2] E NOMURA, M INOUE Surgical technique and rationale for medial patellofemoral ligament reconstruction for recurrent patellar dislocation. *Arthroscopy 5 (2003) (19): E47.*

[3] AA AMIS, P FIRER, J MOUNTNEY, W SENAVONGSE, NP THOMAS Anatomy and biomechanics of the medial patellofemoral ligament. *Knee 3 (2003) (10): 215-20.*

[4] RN STEENSEN, RM DOPIRAK, WG MCDONALD, 3rd The anatomy and isometry of the medial patellofemoral ligament: implications for reconstruction. *Am J Sports Med 6 (2004) (32): 1509-13.*

[5] PB SCHOTTLE, A SCHMELING, N ROSENSTIEL, A WEILER Radiographic landmarks for femoral tunnel placement in medial patellofemoral ligament reconstruction. *Am J Sports Med 5 (2007) (35): 801-4.*



[6] PB SCHOTTLE, J ROMERO, A SCHMELING, A WEILER Technical note: anatomical reconstruction of the medial patellofemoral ligament using a free gracilis autograft. *Arch Orthop Trauma Surg 5 (2008) (128): 479-84.*

[7] J BRAND Jr, A WEILER, DN CABORN, CH BROWN Jr, DL JOHNSON Graft fixation in cruciate ligament reconstruction. *Am J Sports Med 5 (2000) (28): 761-74.*

[8] M THAUNAT, PJ ERASMUS The favourable anisometry: an original concept for medial patellofemoral ligament reconstruction. *Knee 6 (2007) (14): 424-8.*

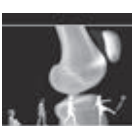
[9] SE CHRISTIANSEN, B JACOBSEN, B LUND, M LIND Reconstruction of the medial patellofemoral ligament with gracilis tendon autograft in transverse patellar drill holes. *Arthroscopy 1 (2008) (24):82-7.*

[10] E NOMURA, M INOUE, N OSADA Anatomical analysis of the medial patellofemoral ligament of the knee, especially the femoral attachment. *Knee Surg Sports Traumatol Arthrosc (2005) 7 (13): 510-15.*

[11] S OSTERMEIER, C STUKENBORG-COLSMAN, CJ WIRTH, M BOHNSACK Reconstruction of the medial patellofemoral ligament by tunnel transfer of the semitendinosus tendon. *Oper Orthop Traumatol 5-6 (2007) (19): 489-501.*

[12] RN STEENSEN, RM DOPIRAK, PB MAURUS A simple technique for reconstruction of the medial patellofemoral ligament using a quadriceps tendon graft. *Arthroscopy 3 (2005) (21): 365-70.*

[13] JL ELLERA-GOMES, LR STIGLER-MARCZYK, P CESAR DE CESAR, CF JUNGBLUT Medial patellofemoral ligament reconstruction with semitendinosus autograft for chronic patellar instability: a follow-up study. *Arthroscopy 2 (2004) (20): 147-51*



TECHNIQUE DU MPFL AVEC FIXATION SUR LES PARTIES MOLLES

V. CHASSAING

Cette technique de reconstruction du MPFL [1, 2], utilisant le tendon du gracilis, parfois celui du semi-tendineux, avec fixation sur les parties molles, est utilisée depuis 1993 [3]. Elle a été systématiquement associée à :
- une section du rétinaculum latéral, sous arthroscopie ou à ciel ouvert,
- et une plicature du rétinaculum médial.

Une transposition de la tubérosité tibiale antérieure associée a été rarement utilisée pour une médialisation, mais systématiquement pour un abaissement chaque fois qu'il existait une patella alta, suivant l'index de Caton-Deschamps [4].

FIXATION FÉMORALE

La fixation fémorale s'effectue sur le rétinaculum médial, à sa partie postérieure, près de son insertion fémorale. Le repère utilisé pour cette fixation est l'épicondyle médial, toujours bien palpable. Comme il s'agit d'une fixation sur des parties molles, il faut tenir compte de la souplesse du rétinaculum lors de la mise en tension de la greffe et placer cet amarrage, non pas en regard de l'épicondyle, mais un centimètre en arrière.

Après l'incision cutanée, on aborde les parties molles para-patellaires médiales. Une incision longitudinale du rétinaculum médial est effectuée le long du bord médial patellaire. Vers le haut, elle se prolonge dans le tendon quadricipital à un demi-centimètre des fibres du vaste médial. Il faut veiller à ne pas ouvrir la synoviale et à cliver progressivement vers l'arrière l'espace situé entre synoviale et rétinaculum médial. C'est dans ce plan que la greffe est introduite (fig. 1), avec une petite pince courbe qui a chargé le fil de laçage du tendon. Son extrémité progresse vers l'arrière jusqu'au niveau de l'épicondyle médial. La pince perfore alors le rétinaculum médial, près de son insertion fé-

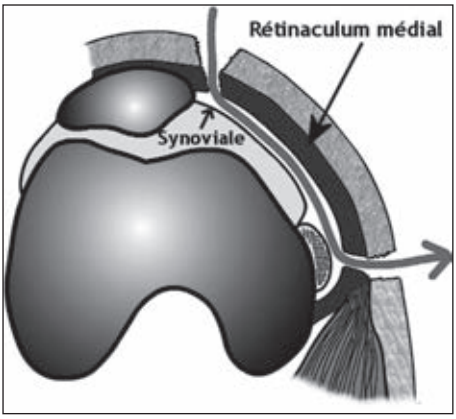
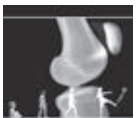


Fig. 1 : Premier passage du tendon. Introduction de la greffe après ouverture para-patellaire du rétinaculum médial. En arrière elle traverse l'épaisseur du rétinaculum puis s'extériorise par ouverture cutanée punctiforme.



morale. Sa pointe s'extériorise au niveau d'une ponction cutanée postéro-médiale, permettant ainsi un premier passage du tendon du gracilis à travers le rétinaculum médial. Un deuxième passage de la pince, dans le même plan, permet d'effectuer une deuxième perforation du rétinaculum médial à distance (un à deux centimètres) de la précédente. Après ce passage, la pince charge le fil de traction de la greffe, et l'attire dans un trajet inverse du précédent, obtenant ainsi un aller-retour du tendon, avec amarrage en "U" sur le rétinaculum médial en regard de l'épicondyle fémoral médial (fig. 2). On teste la solidité de cette fixation : on doit pouvoir soulever le genou par traction sur le tendon.

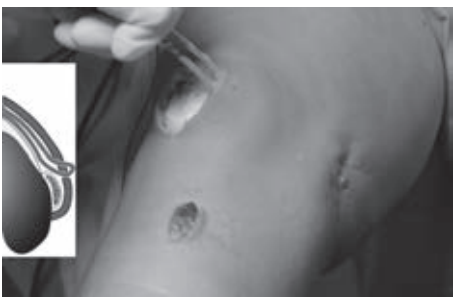


Fig. 2 : Fixation fémorale
La greffe est amarrée au rétinaculum médial par un aller-retour en U. La solidité de cette fixation est contrôlée en tirant sur les deux brins tendineux. L'invagination au niveau de la ponction cutanée postéro-médiale sera supprimée par libération d'adhérences sous-cutanées.

FIXATION PATELLAIRE

C'est une fixation sous-périostée, permettant un amarrage au niveau du bord médial de la patella, à sa partie moyenne. Une petite rugine effectue un trajet sous périosté, depuis le milieu de la face antérieure de la patella jusqu'à son bord médial (fig. 3). Pour assurer une solidité suffisante à cette fixation, il est important de soulever la totalité du périoste en créant un passage ostéo-périosté étroit, adapté au diamètre de la greffe, avec une rugine étroite et légèrement courbée (fig. 4) de façon à rester au contact de la face antérieure, convexe, de la patella. Dans le même temps, grâce à une petite perforation de son extrémité, la rugine a pu faire passer dans ce tunnel ostéo-périosté un fil de traction. Ce dernier va permettre d'y attirer un brin du gracilis, dont l'extrémité a été solidement et finement lacée de façon à faciliter ce passage.

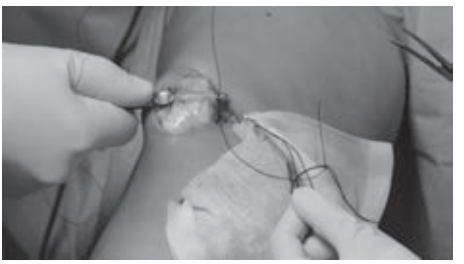
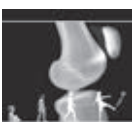


Fig. 3 : Préparation de la fixation patellaire pour le passage sous périosté.

Fig. 4 : Rugine courbe perforée.

A sa sortie, le tendon est retourné sur lui-même, après avoir croisé la face antérieure de la patella.

C'est au niveau de cette fixation patellaire que l'on règle la tension de la greffe. Sa mise en tension se fait sur un genou fléchi à 45°, de façon à éviter une hypercorrection médiale. En maintenant la tension par traction sur les deux brins tendineux, on contrôle la stabilité patellaire par des mouvements d'extension et de flexion. Il faut vraiment tendre suffisamment la greffe, à la limite de la subluxation médiale de la patella. Tout en maintenant la tension ainsi choisie, le tendon est suturé au périoste prépatellaire, puis à lui-même par de nombreux points (fig. 5).



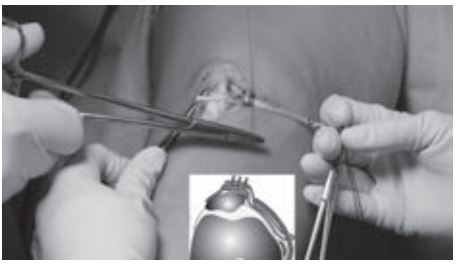


Fig. 5 : Suture du tendon au périoste et à lui-même. Les deux pinces sur les deux extrémités tendineuses maintiennent la tension choisie.

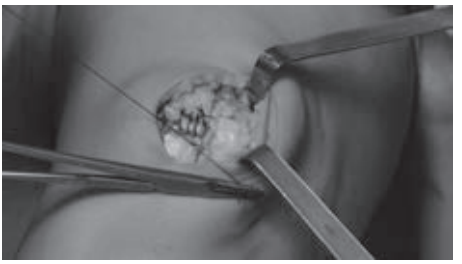


Fig. 6 : Suture en paletot du rétinaculum médial, qui vient recouvrir en partie la patella et la suture de la greffe.

SUTURE EN PALETOT DU RÉTINACULUM MÉDIAL [5]

Cette suture à points séparés (fig. 6) vient compléter et renforcer la mise en tension du plan interne. Comme précédemment, elle est effectuée sur un genou à 45° de flexion pour éviter une hypercorrection. Les points supérieurs permettent de réaliser une plastie du vaste médial.

SUITES OPÉRATOIRES

Il n'y a pas d'immobilisation. L'appui immédiat est autorisé sur un genou en extension sous couvert de deux cannes béquilles pendant 15 jours. La rééducation, entreprise au bout de deux semaines, est prévue pendant un mois et demi à deux mois.

CONCLUSION

Cette fixation sur les parties molles lors de la reconstruction du MPFL, utilisée depuis 20 ans, présente beaucoup d'avantages :

- elle est suffisamment solide pour éviter toute immobilisation et permettre une rééducation précoce. Elle n'est cependant pas rigide, garde une certaine souplesse qui diminue le risque d'hypercorrection,
- elle peut être pratiquée par des petites incisions cutanées, ce qui est appréciable chez ces patients souvent jeunes,
- elle est peu coûteuse puisqu'elle ne nécessite aucun implant (vis d'interférence, ancres…),
- elle n'a pas été à l'origine de séquelles douloureuses, en particulier au niveau de la fixation fémorale.

LITTÉRATURE

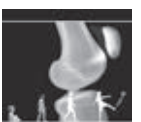
[1] CHASSAING V, TREMOULET J. Luxation de la rotule : techniques chirurgicales et indications. *J Traumatol Sport, 2000, 17, 74-82.*

[2] CHASSAING V, TREMOULET J. Plastie du ligament fémoro-patellaire médial avec le tendon du gracile pour stabilisation de la patella. *Revue de chirurgie orthopédique 2005, 91, 335-40.*

[3] CHASSAING V, PERRAUDIN JE Stabilisation arthroscopique et percutanée de la rotule. *Annales Orthopédiques de l'Ouest, volume 27, pages 37-40, 1995.*

[4] CATON J. Méthode de mesure de la hauteur de la rotule. *Acta Orthop. Belg, 1989, 55, N° 3, 385-6.*

[5] INSALL J, FALVO KA, WISE DW. Chondromalacia patellae. A prospective study. *J Bone Joint Surg., 58 A, 1, 1976, 1-8.s*



ALL SURGICAL PROCEDURES (video) Patellar tendon procedure

G.L. CAMANHO

INDICATIONS

The indications are:
- Acute dislocation of the patella when magnetic resonance imaging (MRI) demonstrates an extensive lesion of the MPFL (fig. 1).

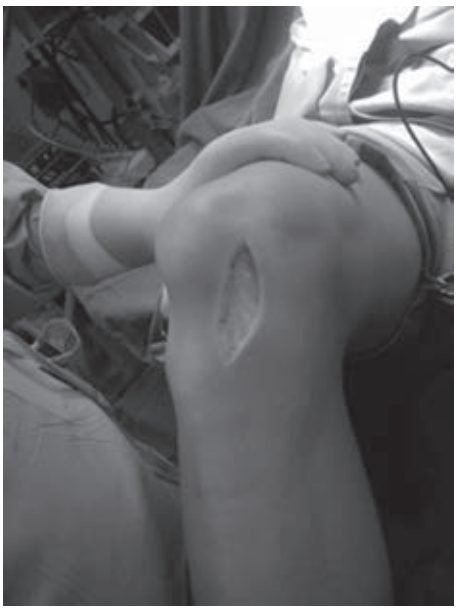


Fig. 1 : Skin incision

PREOPERATIVE PLANNING

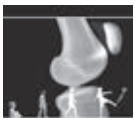
In acute cases, MRI is fundamental for analyzing the type of MPFL lesion. In cases of extensive lesions, reconstruction is indicated. In the other hand, in cases of isolated lesions with defined borders in the patellar insertion, repair should be the best approach.

SURGICAL TECHNIQUE

Our preference is to use subarachnoid anesthesia. We then perform an examination under anesthesia to confirm the presence of patellofemoral instability. A pneumatic tourniquet is routinely used.

We start the procedure with arthroscopy to treat possible cartilage lesions. We do not perform lateral release.

The surgical incision is planned as follows: an incision is started proximally at the level of the upper margin of the patella, centrally, between the medial margin of the patella and the medial epicondyle (fig. 1). We make an incision going down towards the superomedial margin of the ATT. This incision will be associated with the incision for the inferomedial portal for arthroscopy.



We perform layer-by-layer dissection to reach the peritendon of the patellar ligament, as done when taking a graft from the patellar tendon to reconstruct the anterior cruciate ligament. The peritendon will be incised vertically but medially, since only the medial portion of its structure will be used. We use the medial third of this tendon.

By means of a subperiosteal incision, we release the distal extremity of the tendon from the ATT and flip this strip, medially and superiorly. Before this, however, with the purpose to reproducing the anatomy of the MPFL at its patellar insertion, we perform subperiosteal release on the patella, as far as the junction of the proximal third with the medial third (fig. 2). We then perform reinforcement using resistant thread, suturing the graft to the extensor apparatus, which is inserted into the patella.

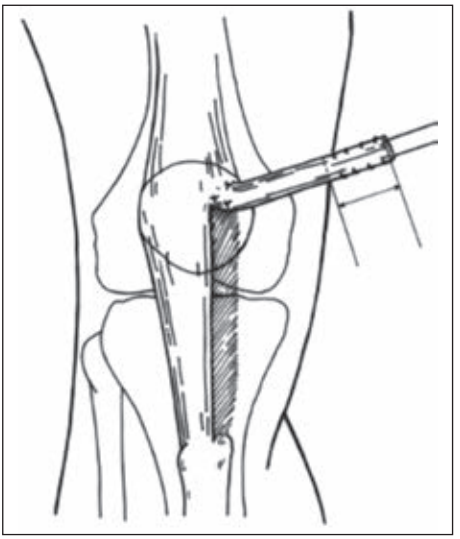


Fig. 2 : Diagram showing removal of the medial third of the patellar tendon

The femoral insertion covers a more diffuse area and is posterior and proximal to the medial epicondyle. At this point, we need to measure the distance to the patellar insertion and the graft fixation point on the femur. We perfom Krackow suturing on the free end of the tendon and position the graft at the femoral insertion site, allowing the entrance of the stitches into femoral tunnel to further fixation with interference screws. Next, we make the tunnel using a drill bit of the same size as the graft is ad at the end using an absorbable interference screw (fig. 3). If the graft is too short, we have the possibility of not making the tunnel and fixing using absorbable or non-absorbable anchors. The fixation should be done at 60 degrees of flexion, the position at which we apply tension to the ligament.

It is of prime importance at this point to be concerned about testing the total flexion and stability of the fixation method.

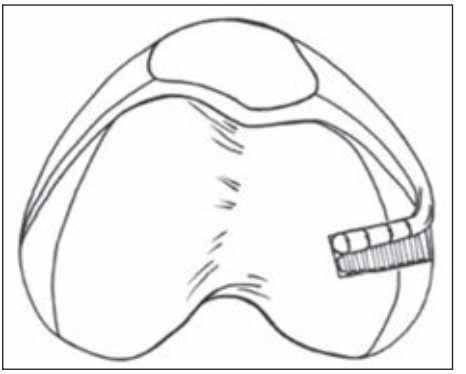
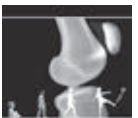


Fig. 3 : Diagram showing fixation in the femoral insertion using interference screw

We complement this technique by developing a dynamic system between the reconstructed ligament and the lower border of the vastus medialis muscle, by means of stitches to suture the vastus medialis to the MPFL (fig. 4).

After washing the wound, we close the peritendon using Vicryl® 3.0 and then close the wound using intradermal stitches of colorless Monocryl®. We do not routinely use a drain and we prefer removal using the tourniquet and perform hemostasis.

We perform infiltration using local anesthetics in the skin incisions and we finish by bandaging and immobilizing using an inguinal-malleolar brace.



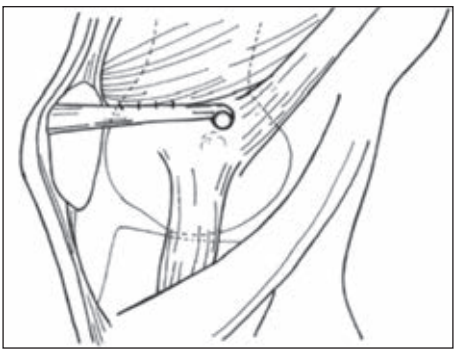


Fig. 4 : Suture of the vastus medialis muscle to the ligament graft

POSTOPERATIVE TREATMENT

Rehabilitation is started at the time of the first outpatient return visit. The individual keeps the brace on for three weeks, during which isometric exercises are started and analgesia (cryotherapy) and electrostimulation are administered. We initially advise patients to use a pair of crutches and walk without loadbearing on the knee, and to perform cryotherapy at home. During the first week, the load will gradually be applied, up to the patient's pain limit. Starting in the first to third week, we add in larger gains in range of motion, after the third week, the immobilizer is keep off and bicycle are use with loading and initial proprioception exercises. From the sixth week onwards, we start closed kinetic chain exercises and gradually start to open kinetic chain exercises. Our aim is that patients should be able to return to their preoperative sports activities after approximately 10 to 12 weeks.

FUTURE POSSIBILITIES FOR THE TECHNIQUE

We are seeking to adapt the skin incision, so that the result is more cosmetic.

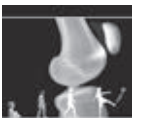
ABSTRACT

In 2001 we presented a proposal for a surgical technique to reconstruct the Medial Patello-femoral Ligament – MPFL using the patellar tendon and in 2007 we published the technique [1].

The technique consists of dissection to reach the peritendon of the patellar ligament, taking a graft from the medial third of the patellar tendon to reconstruct the Medial Patellofemoral Ligament. By means of a subperiosteal incision, we release the distal extremity of the tendon from the anterior tuberosity of the tibia and flip this strip, medially and superiorly. Initially, we perform a subperiosteal release on the patella, as far as the junction of proximal third, with the medial third as a graft positioned at femoral insertion site and fixed using an interference screw within femoral tunnel. If the graft is too short, there is a possibility of not making the tunnel and fixing it with absorbable or non-absorbable anchors.

LITERATURE

[1] CAMANHO GL, BITAR AC, HERNANDEZ AJ, OLIVI R. Medial patellofemoral ligament reconstruction: a novel technique using the patellar ligament. *Arthroscopy 2007, 23(1): 108.e1-4.*



MINIMAL INVASIVE RECONSTRUCTION OF THE MPFL USING QUADRICEPS TENDON

C. FINK, C. HOSER, M. VESELKO

INTRODUCTION

Reconstruction of the medial patellofemoral ligament (MPFL) for the treatment of patella instability has achieved increased attention over the last few years.

Several surgical techniques have been described which most of them using hamstring tendons as the graft of choice [2-5, 8, 9].

All of these techniques utilize bone tunnels and/or anchors for graft fixation on the patella. Besides implant costs, complications such as implant breakage or patella fractures through bone tunnel have been experienced [1, 2, 7].

There are few reports on MPFL reconstruction using a strip of quadriceps tendon without anchors or bone tunnels in the patella [6, 10]. Despite very good clinical results, the cosmetic appearance of longitudinal scars over the thigh as well as technical difficulties harvesting a uniform 2-3mm strip of the quadricepstendon (fig. 1) have not lead to a widespread use of this techniques.

Therefor we developed a new harvesting technique for the quadriceps tendon which allows not only a constant graft harvest with respect to width and thickness but also is cosmetically better accepted (only a 2-3cm transverse skin incision) (fig. 2).

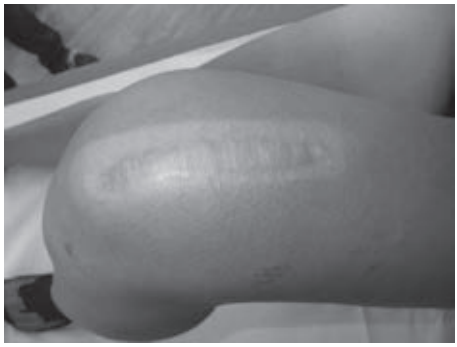


Fig. 1

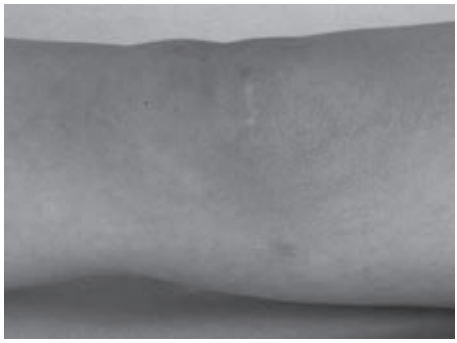
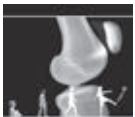


Fig. 2



SURGICAL TECHNIQUE

Positioning

Patient positioning has to allow free knee motion between 0° and 120°.

The intraoperative access for the fluoroscope is important to be kept in mind and ideally checked prior to draping.

We prefer fixation of the operative leg in an electric leg holder (fig. 3).

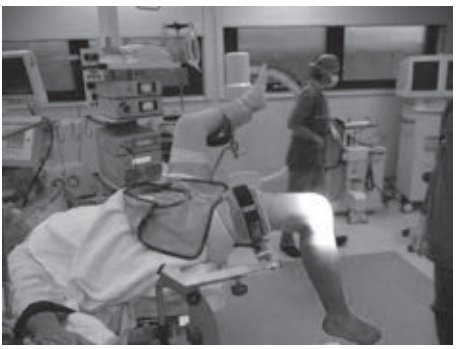


Fig. 3

Surgical steps

In 90° of knee flexion a 2.5-3cm transverse skin incision is placed over the superomedial pole of the patella. The prepatellar bursa is incised longitudinally and the quadriceps tendon is then carefully exposed.

A long Langenbeck retractor is then introduced and the quadriceps tendon subcutaneously exposed proximal to the patella.

The double knife (Karl Storz) in 10 or 12mm width is then introduced starting over the middle of the superior patella boarder and pushed up to a minimum of 8cm (mark on the instrument) (fig. 4).

The thickness of the graft is then determined with 2 or 3mm by a second special knife (Karl Storz). The knife is pushed proximal to the same mark (minimum 8cm) (fig. 5).

Finally the tendon strip is cut subcutaneously by a special tendon cutter (Karl Storz) (fig. 6).

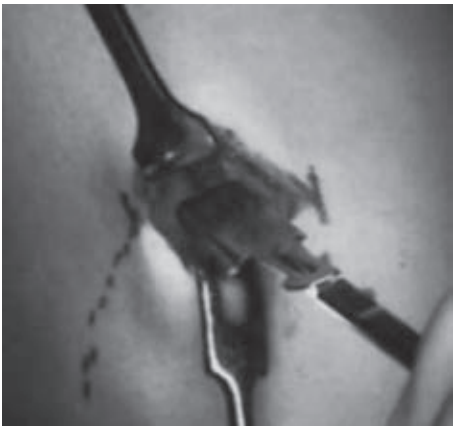


Fig. 4

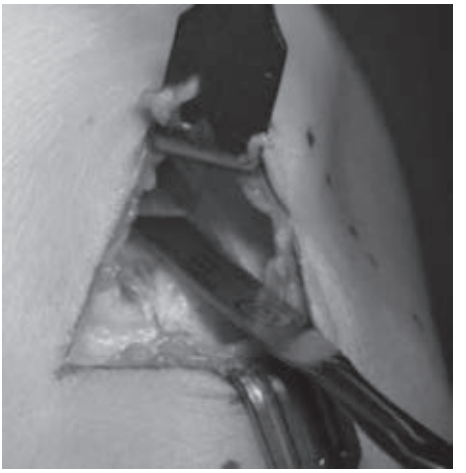


Fig. 5

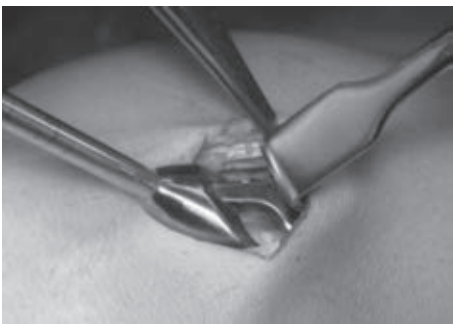
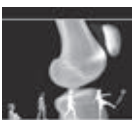


Fig. 6



The graft is left attached distally and the free proximal end is armed with resorbable sutures in web stitch technique.

Distally the longitudinal cuts are continued with a surgical knife towards the patella and over the patellar surface in the chosen width (10 or 12mm) (Lateral: 2cm, medial: 1cm on the surface of the patella). The quadriceps tendon strip is than subperiostally elevated from the surface of the patella.

The proximal 1.5cm of the medial patellar border is then exposed. From the medial patella border the prepatellar tissue is elevated towards laterally creating a tunnel reaching the medial edge of the graft. This is performed best with a periostial elevator (fig. 7)), so that anterior cortex of the patella in the tunnel is gently abraded. A surgical clamp is introduced into the tunnel from medial to lateral and the graft is passed through the tunnel (fig. 8). The graft pulled through the tunnel should lie flat on the abraded cortex of the patellar surface, so that fast and broad graft-to-bone healing can be expected. The graft is then secured to the retinaculum tissue on the medial patellar edge by resorbable sutures (fig. 9).

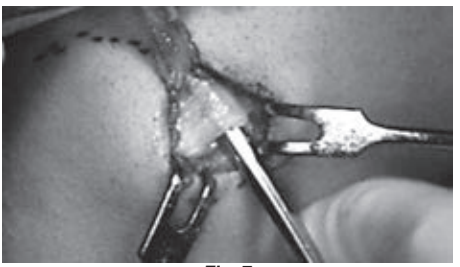


Fig. 7

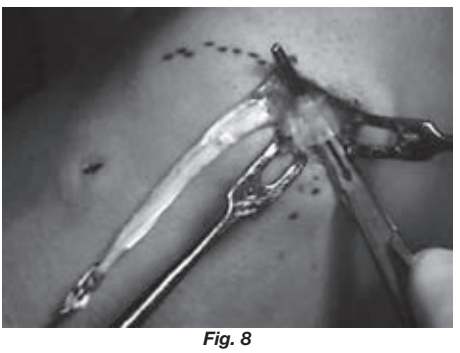


Fig. 8

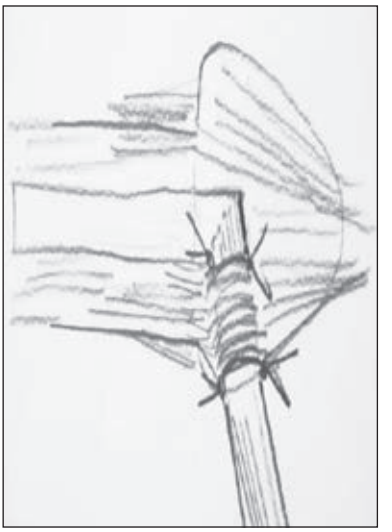


Fig. 9

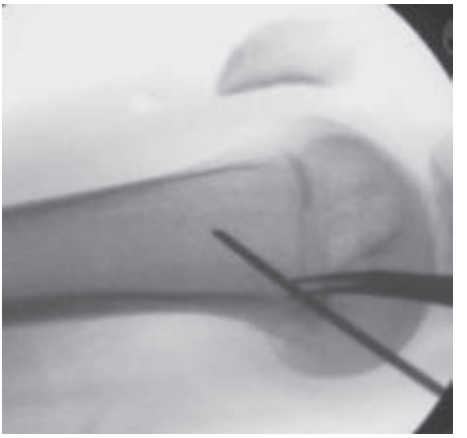
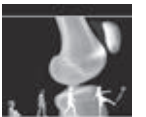


Fig. 10

A 1.5cm skin incision is then made over the adductor tubercle. Starting at the patella a curved clamp is used to create a tunnel in the space between the vastus medialis and the capsule. A suture loop is then pulled through the tunnel. This loop is used later to pull the graft towards the femoral insertion.

Under fluoroscopic guidance a 2.4mm guide pin is drilled into the insertion of the MPFL [8]. It is directed antero-laterally to exit the femur



on the lateral cortex well proximal to the lateral epicondyle. If found accurate by fluoroscopy the guide pin is overreamed with an 8mm cannulated reamer to e depth of 30mm.

The graft is then pulled into the tunnel. The knee is cycled 5 times with moderate tension on the graft. Fixation is performed with an 8x28mm resorbable interference screw at 20 degrees of knee flexion.

Remark: In children with open physes the graft can be fixed with a bone anchor or sutures alone [10].

Postoperative Treatment

A knee brace with ROM 0-90° is used. The patient is mobilized with 20kg partial weightbearing for 3 weeks. Full weightbearing as tolerated is started thereafter. Passive ROM exercises to a maximum of 90degrees is started thereafter immediately postoperatively. Stationary cycling is started 6 weeks postop. Full return to pivoting sports is between 4 and 5 months after the operation.

CONCLUSION

MPFL reconstruction using a strip of quadricepstendon harvested subcutaneously in the described technique has been found to result in excellent clinical and cosmetical results. This technique can also be recommended for MPFL revision surgery as well as for treatment of patella instability in children with open growth plates.

LITERATURE

[1] BOLLIER M, J FULKERSON *et al.* "Technical failure of medial patellofemoral ligament reconstruction". *Arthroscopy (2011). 27(8): 1153-9.*

[2] CHRISTIANSEN SE, BW JACOBSEN *et al.* Reconstruction of the medial patellofemoral ligament with gracilis tendon autograft in transverse patellar drill holes. *Arthroscopy (2008). 24(1): 82-7.*

[3] FISHER B, J NYLAND *et al.* Medial patellofemoral ligament reconstruction for recurrent patellar dislocation: a systematic review including rehabilitation and return-to-sports efficacy. *Arthroscopy (2010). 26(10): 1384-94.*

[4] MAENO S, D HASHIMOTO *et al.* Medial patellofemoral ligament reconstruction with hanger lifting procedure. *Knee Surg Sports Traumatol Arthrosc (2010). 18(2): 157-60.*

[5] MATTHEWS JJ, P SCHRANZ. Reconstruction of the medial patellofemoral ligament using a longitudinal patellar tunnel technique. *Int Orthop (2010). 34(8): 1321-5.*

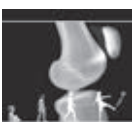
[6] NOYES FR, JC ALBRIGHT. Reconstruction of the medial patellofemoral ligament with autologous quadriceps tendon. *Arthroscopy (2006). 22(8): 904 e901-7.*

[7] LEGRAND AB, GREIS PE, DOBBS RE, BURKS RT MPFL reconstruction. *Sports Med Arthrosc (2007) 2: 72-7.*

[8] SCHÖTTLE P, A SCHMELING *et al.* Anatomical reconstruction of the medial patellofemoral ligament using a free gracilis autograft. *Arch Orthop Trauma Surg (2009). 129(3): 305-9.*

[9] SCHÖTTLE PB, D HENSLER *et al.* Anatomical double-bundle MPFL reconstruction with an aperture fixation. *Knee Surg Sports Traumatol Arthrosc (2010). 18(2): 147-51.*

[10] STEENSEN RN, RM DOPIRAK *et al.* A simple technique for reconstruction of the medial patellofemoral ligament using a quadriceps tendon graft. *Arthroscopy (2005). 21(3): 365-70.*



MPFL RECONSTRUCTION: NAVIGATION AND ANGLE OF FIXATION

S. ZAFFAGNINI, P.G. NTAGIOPOULOS, D. DEJOUR, B. SHARMA, S. BIGNOZZI, N. LOPOMO, Ing. F. COLLE, M. MARCACCI

INTRODUCTION

Medial patellofemoral ligament (MPFL) is an established primary static stabilizer of lateral patellar dislocation and its lesion labeled as essential for acute and chronic cases of patellar dislocation (fig. 1) [1-4]. Over the last decade, its anatomic reconstruction in clinical patellar instability has been studied. Combination of MPFL reconstruction with other procedures like tibial tubercle osteotomies and trochleoplasties has also generated interest [5-9]. There is no clear consensus on a single surgical technique, graft options, fixation methods and angle of flexion at fixation [5, 6, 10, 11]. A single, blind femoral tunnel, between the adductor tubercle and medial epicondyle, with two patellar tunnels using one hamstring tendon is currently accepted widely [12-15].

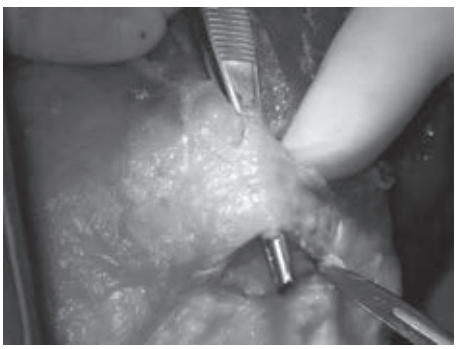


Fig. 1: Cadaveric Knee showing the isolated, native Medial Patello-femoral Ligament.

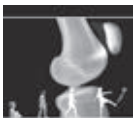
In this background, it is suitable to study MPFL reconstruction technique using the navigation system to record patellar kinematics on a cadaveric model.

MATERIALS AND METHODS

The study was carried out on six normal cadaveric knee specimens, aged between 41-60 years, two males and four females. After hip disarticulation and subcutaneous dissection 20cm proximally, each femur was clamped and quadriceps tendon was loaded with 60N axial load (fig. 2). The tibia was moved manually through 0-90 degrees of flexion with native and then, reconstructed MPFL to record the patellar kinematics via an imageless navigation system (BLUIGS, Orthokey LLC, Lewes, Delaware). The femoral insertion of the native and graft MPFL was acquired after resection of the native MPFL, along with the medial epicondyle.

Surgical technique for MPFL reconstruction

A 22-25cm length of gracillis graft was prepared using a standard harvesting technique



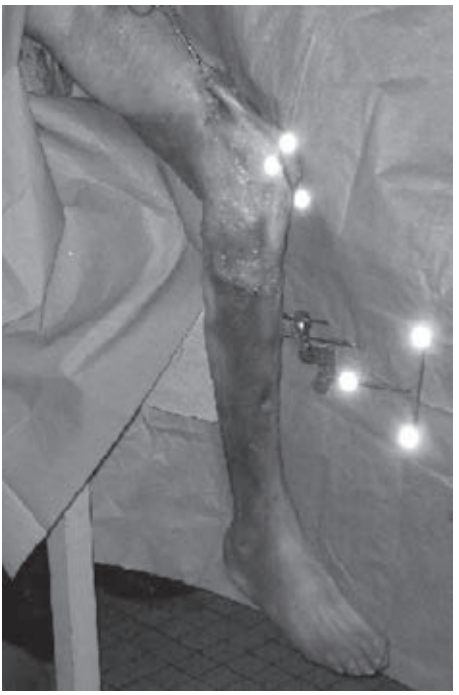


Fig. 2: Experimental Setup showing frames for navigation and axial quadriceps loading.

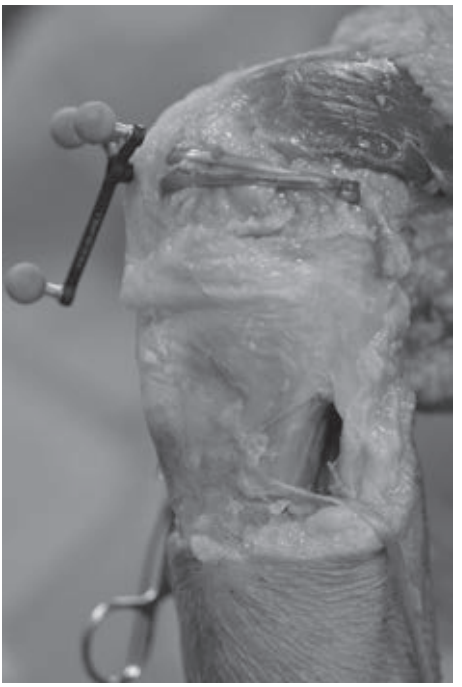


Fig. 3: Picture demonstrating a MPFL graft in position, extending from a single blind femoral tunnel on the right to a U-tunnel on medial patella on the left.

and 2-0 braided sutures (Vicryl) (fig. 3). The superior 2/3rd of the medial border of the patella was exposed and freshened via sub-periosteal dissection, without damaging the synovial membrane. Two antero-posterior tunnels, 3.5mm wide each, were drilled short of the patellar articular surface, 10mm below superior patellar pole and 15mm lateral to medial patellar pole, separated with 20mm bone in between. These tunnels were connected in a U-shaped tunnel, while a pulley was fashioned in the medial retinaculum, 1cm medial to the patella (fig. 4). After dissection of the MPFL, the graft tunnel was positioned in the antero-superior part of the native insertion, using a 7mm drill, over a k-wire, without violating the lateral femoral cortex (fig. 5). The graft was passed with suture passers, through the U-shaped patellar tunnel, under the pulley and into the blind femoral tunnel, when the knee was cycled 10 times. The tension in the MPFL was set to allow one quadrant lateral translation of the medial facet with a firm end-point, without a medial tilt. The tension was maintained, while the graft was fixed with interference screws, in 70 degrees of knee flexion to ensure central position in the trochlea.

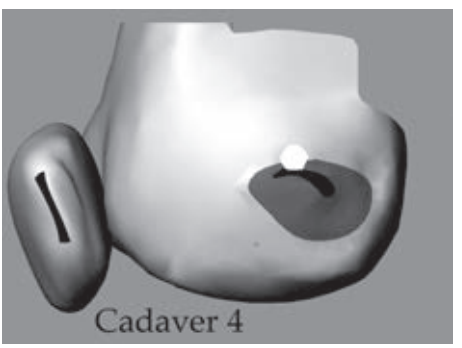
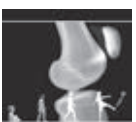


Fig. 4: Schematic comparison of Native (black area) and Graft MPFL Femoral Insertion (white area). The graft was proximal and anterior to the MPFL insertion.



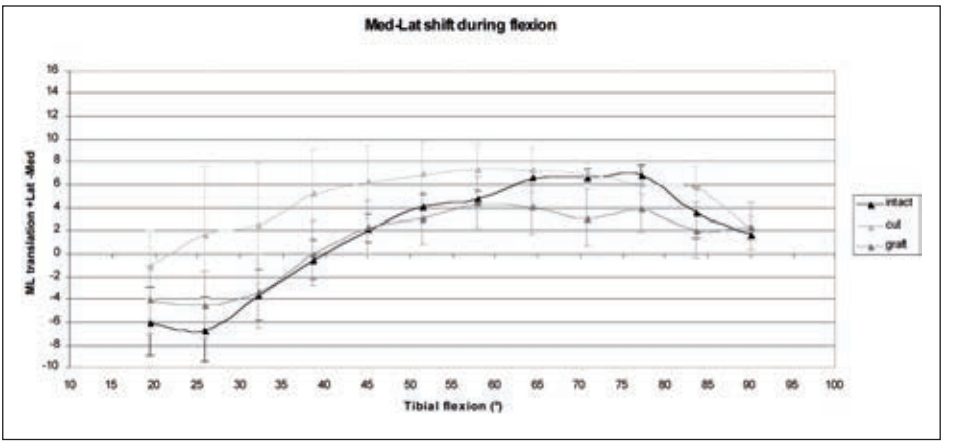


Fig. 5: Medial to lateral patellar translation (shift) in 3 different states of MPFL (intact/cut/reconstructed) in different degrees of flexion. Medial translation (in mm) expressed in negative (-) values and lateral translation expressed in positive (+) values.

Statistical Analysis

The mean ± standard deviation of the femoral insertion of the native MPFL insertion was compared with that of the graft MPFL. The native and graft MPFL were compared using the non-parametric Kruskal-Wallis test in terms of patellar tracking ($p<0.05$).

RESULTS

The graft femoral insertion was found to be proximal and anterior to the geometric center of the femoral insertion of the native MPFL (fig. 4). While the difference was statistically significant in proximal direction, it was not so anteriorly. The navigation system recorded a maximum medial shift in early flexion and

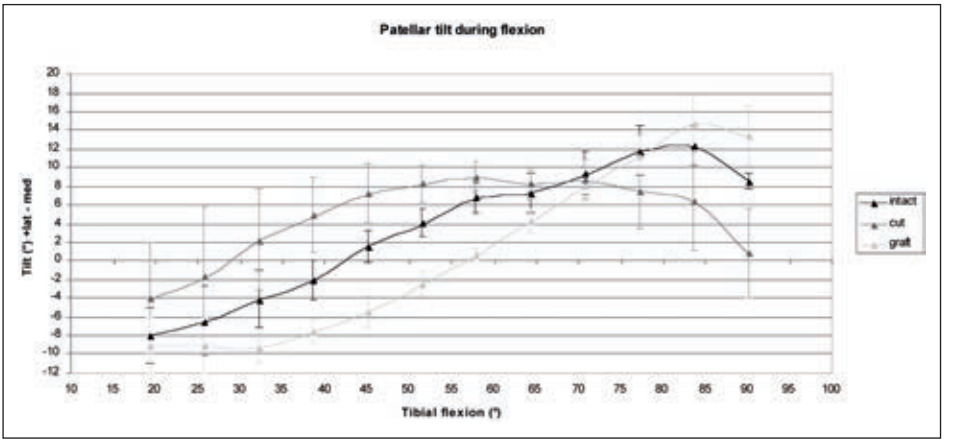
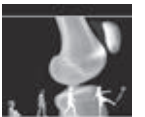


Fig. 6: Changes of patellar tilt in 3 different states of MPFL (intact/cut/reconstructed) in different degrees of flexion. Medial tilt expressed in negative (-) values and lateral tilt expressed in positive (+) values.



thereafter a lateral shift (fig. 5). The variability of patellar shift reduced between 60-90 degrees, While there was no medial tilt in early flexion, the lateral tilt reduced after 85 degrees of knee flexion. After MPFL reconstruction, the patella medial shift was restored, along with normal patellar tilt (fig. 6). No statistically significant difference was noted between the two states; native MPFL and MPFL reconstruction.

DISCUSSION

Over the last decade, MPFL and its reconstruction has gained significance in patellar instability [16]. While its anatomy [17-19], role in patellar instability [3, 18] and lesion in lateral patellar dislocation [1, 2, 20, 21] has been well established, the exact technique of its reconstruction is still debated. The site of femoral insertion, pre-cycling, initial tension and angle of fixation are all debated. It has been suggested that the effect of these variations should be benchmarked on the basis of patellar kinematics. The current study demonstrates how navigation could help define not only a standard technique of MPFL reconstruction, but also be used intra-operatively to customize the surgery for every patient.

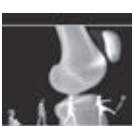
One of the challenges in MPFL reconstruction is the discrepancy between the femoral insertion of the native MPFL and the graft. The area of the native MPFL is much larger than the 7-8mm cylindrical hamstring graft. Secondly, the biomechanical properties of the graft and the native MPFL are different. Added to these are the controversies whether the MPFL is an isometric structure or not, and its resting length tension.

There are also concerns of excessive medial tilt after MPFL reconstruction, given the posteromedial orientation of the graft, increasing the medial contact pressure in the patello-femoral joint4. Excessive medial tilt may compromise long term results by exacerbating existing cartilage damage in patellar instability [9, 18, 22-25]. Furthermore, the femoral insertion of the original MPFL is fan-shaped and of larger diameter than the patellar insertion. In comparison, the hamstring graft used is a cylindrical 7mm graft with very different biomechanical properties from the native MPFL. Incorrect femoral placement can diversely affect the affect range of motion and constraint of the graft in flexion and extension.

Therefore, the ideal MPFL reconstruction would place the graft in an appropriate femoral and patellar insertion, with adequate tension, aim at an ideal patellar position that would result in unconstrained patellar tracking throughout the range of knee motion.

In the present study on normal cadavers, kinematic based navigation demonstrated that a statistically similar patellar tracking could be reproduced. Patellar shift and tilt after MPFL reconstruction was comparable to that with native MPFL. The chosen femoral insertion was in a significantly proximal and slightly anterior end of the native MPFL insertion. The U-shaped patellar tunnels reduce the risk of fractures and the medial retinaculum pulley provides a proper orientation to the graft. The tension of graft was set in extension in such a way to allow passive lateralization of one quadrant of the patella in extension with a firm endpoint, while the femoral insertion was fixed at 70 degrees of flexion to centralize the patella in the trochlea and to provide adequate range of motion. The navigation system also demonstrated the absence of an excessive medial tilt, thus avoiding excessive contact pressure in the medial facet of the patella.

The femoral insertion of the MPFL is more controversial compared to the patellar insertion, which is relatively well defined and identified. Often a fluoroscopy is used to locate an anatomically referenced MPFL insertion. However, the current study illustrates the difficulty in isolating the ideal graft femoral insertion even within the wide native femoral insertion. The insertion ultimately is one of the most important factors to not just stabilize the patella, but also maintain range of motion of the knee and normal excursion of the patella during the same [26, 27].



There has been various principles proposed in literature as related to the degree of knee flexion and the tension in which to fix the graft MPFL. Tensioning the graft at 20-30 degrees of flexion is a widely used option, given that the patellar instability occurs in this range [2], but others chose to tension the reconstructed ligament in greater degrees of flexion, when the patella is more fully captured by the trochlea [2, 26]. The authors do not recommend the traditional graft tensioning between 20° to 30° of flexion. The exact knee position during fixation is less important if knee cycling and graft pre-tensioning precede the final fixation. Testing the lateral patellar translation in extension (in order not to exceed 1/3 of patella width), graft pre-tensioning, and making the femoral fixation last in order were the key steps of the reconstruction.

In the presence of dysplasia, a trochlea that would not serve as a fulcrum for patella stability in late knee flexion, is a question to be further studied in all patients with patellar instability that undergo MPFL reconstruction. The findings of the previously-published manuscripts are based on normal knees with no trochlear dysplasia and with no concern of abnormal patella height. The presence of a normal trochlear groove is a prerequisite for the function of MPFL beyond certain degrees of flexion. All these are key factors for the reconstruction of MPFL, which would involve a construct that serves its native fashion [1, 2, 26]. But in order for this to succeed, a normal trochlear anatomy is of paramount importance, and therefore in cases of trochlear dysplasia (which account for 96% of the objective patellar instability population [28]), the lack of trochlear depth and patella containment must be taken into account. In these cases there is a trend towards overtensioning the graft to avoid lateral patellar translation [22].

CONCLUSIONS

The material presented demonstrates the potential use of navigation systems to align patellar tracking after MPFL reconstruction to the native state. It also identifies some key principles that could be used for graft fixation in the procedure. As intra-operative patellar tracking becomes common place, with the resolution of controversies surrounding patellar kinematics, a customization of the proposed MPFL reconstruction technique may be possible in each patient.

LITERATURE

[1] AMIS AA. Current concepts on anatomy and biomechanics of patellar stability. *Sports Med Arthrosc. 2007 Giu; 15(2): 48-56.*

[2] BICOS J, FULKERSON JP, AMIS A. Current concepts review: the medial patellofemoral ligament. *Am J Sports Med. 2007 Mar; 35(3): 484-92.*

[3] CONLAN T, GARTH WP Jr, LEMONS JE. Evaluation of the medial soft-tissue restraints of the extensor mechanism of the knee. *J Bone Joint Surg Am. 1993 Mag; 75(5): 682-93.*

[4] NOMURA E. Classification of lesions of the medial patello-femoral ligament in patellar dislocation. *Int Orthop. 1999; 23(5): 260-3.*

[5] CHRISTIANSEN SE, JAKOBSEN BW, LUND B, LIND M. Isolated repair of the medial patellofemoral ligament in primary dislocation of the patella: a prospective randomized study. *Arthroscopy. 2008 Ago; 24(8): 881-7.*

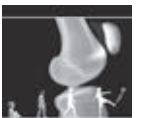
[6] DAINER RD, BARRACK RL, BUCKLEY SL, ALEXANDER AH. Arthroscopic treatment of acute patellar dislocations. *Arthroscopy 1988; 4(4): 267-71.*

[7] FUKUSHIMA K, HORAGUCHI T, OKANO T, YOSHIMATSU T, SAITO A, RYU J. Patellar dislocation: arthroscopic patellar stabilization with anchor sutures. *Arthroscopy 2004 Set; 20(7): 761-4.*

[8] HASPL M, CICAK N, KLOBUCAR H, PECINA M. Fully arthroscopic stabilization of the patella. Art*hroscopy 2002 Gen; 18(1): E2.*

[9] SANDMEIER RH, BURKS RT, BACHUS KN, BILLINGS A. The effect of reconstruction of the medial patellofemoral ligament on patellar tracking. *Am J Sports Med. 2000 Giu; 28(3): 345-9.*

[10] HALBRECHT JL. Arthroscopic patella realignment: An all-inside technique. *Arthroscopy 2001 Dic; 17(9): 940-5.*



[11] REDFERN J, KAMATH G, BURKS R. Anatomical confirmation of the use of radiographic landmarks in medial patellofemoral ligament reconstruction. *Am J Sports Med. 2010 Feb; 38(2): 293-7.*

[12] GIORDANO M, FALCIGLIA F, AULISA AG, GUZZANTI V. Patellar dislocation in skeletally immature patients: semitendinosous and gracilis augmentation for combined medial patellofemoral and medial patellotibial ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc [Internet]. 2011 Nov 25 [citato 2012 Lug 13]; Available da: http://www.ncbi.nlm.nih.gov/pubmed/22116266*

[13] JACOBI M, REISCHL N, BERGMANN M, BOUAICHA S, DJONOV V, MAGNUSSEN RA. Reconstruction of the medial patellofemoral ligament using the adductor magnus tendon: an anatomic study. *Arthroscopy 2012 Gen; 28(1): 105-9.*

[14] KITA K, HORIBE S, TORITSUKA Y, NAKAMURA N, TANAKA Y, YONETANI Y, *et al.* Effects of medial patellofemoral ligament reconstruction on patellar tracking. *Knee Surg Sports Traumatol Arthrosc. 2012 Mag; 20(5): 829-37.*

[15] SILLANPÄÄ PJ, MÄENPÄÄ HM, MATTILA VM, VISURI T, PIHLAJAMÄKI H. A mini-invasive adductor magnus tendon transfer technique for medial patellofemoral ligament reconstruction: a technical note. *Knee Surg Sports Traumatol Arthrosc. 2009 Mag; 17(5): 508-12.*

[16] REIDER B, MARSHALL JL, WARREN RF. Persistent vertical septum in the human knee joint. *J Bone Joint Surg Am. 1981 Set; 63(7): 1185-7.*

[17] ARENDT EA, FITHIAN DC, COHEN E. Current concepts of lateral patella dislocation. *Clin Sports Med. 2002 Lug; 21(3): 499-519.*

[18] HAUTAMAA PV, FITHIAN DC, KAUFMAN KR, DANIEL DM, POHLMEYER AM. Medial soft tissue restraints in lateral patellar instability and repair. *Clin Orthop Relat Res 1998 Apr;(349): 174-82.*

[19] NOMURA E, HORIUCHI Y, INOUE M. Correlation of MR imaging findings and open exploration of medial patellofemoral ligament injuries in acute patellar dislocations. *Knee 2002 Mag; 9(2): 139-43.*

[20] BURKS RT, DESIO SM, BACHUS KN, TYSON L, SPRINGER K. Biomechanical evaluation of lateral patellar dislocations. *Am J Knee Surg. 1998; 11(1): 24-31.*

[21] NOMURA, HORIUCHI, KIHARA. Medial patellofemoral ligament restraint in lateral patellar translation and reconstruction. *Knee 2000 Apr 1;7(2): 121-7.*

[22] BECK P, BROWN NAT, GREIS PE, BURKS RT. Patellofemoral contact pressures and lateral patellar translation after medial patellofemoral ligament reconstruction. *Am J Sports Med. 2007 Set; 35(9): 1557-63.*

[23] CONLAN T, GARTH WP Jr, LEMONS JE. Evaluation of the medial soft-tissue restraints of the extensor mechanism of the knee. *J Bone Joint Surg Am. 1993 Mag; 75(5): 682-93.*

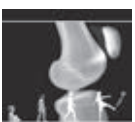
[24] OSTERMEIER S, HOLST M, BOHNSACK M, HURSCHLER C, STUKENBORG-COLSMAN C, WIRTH CJ. Dynamic measurement of patellofemoral contact pressure following reconstruction of the medial patellofemoral ligament: an in vitro study. *Clin Biomech (Bristol, Avon). 2007 Mar; 22(3): 327-35.*

[25] OSTERMEIER S, HOLST M, BOHNSACK M, HURSCHLER C, STUKENBORG-COLSMAN C, WIRTH CJ. In vitro measurement of patellar kinematics following reconstruction of the medial patellofemoral ligament. *Knee Surg Sports Traumatol Arthrosc. 2007 Mar; 15(3): 276-85.*

[26] AMIS AA, FIRER P, MOUNTNEY J, SENAVONGSE W, THOMAS NP. Anatomy and biomechanics of the medial patellofemoral ligament. *Knee 2003 Set; 10(3): 215-20.*

[27] ELIAS JJ, COSGAREA AJ. Technical errors during medial patellofemoral ligament reconstruction could overload medial patellofemoral cartilage: a computational analysis. *Am J Sports Med 2006 Set; 34(9): 1478-85.*

[28] DEJOUR H, WALCH G, NOVE-JOSSERAND L, GUIER C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc 1994; 2(1): 19-26.*



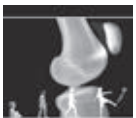
A PHILOSOPHY AND TECHNIQUE FOR RECONSTRUCTION OF THE MEDIAL PATELLOFEMORAL LIGAMENT

P.J. ERASMUS, M. THAUNAT

PHILOSOPHY

The patella is a sesamoid bone in a soft-tissue sleeve that originates on the anterior iliac spine and proximal femur and inserts distally on the tibial tubercle. The patella aligns itself in this soft-tissue sleeve and not with the femur as such [1]. Until the end of gestation, the form of the patella and trochlea probably has a genetic basis. After birth, the knee goes into full extension and a bipedal stance develops that results in a femoral obliquity and secondary valgus of the extensor mechanism soft-tissue sleeve. These epigenetic factors now determine the position of the patella in relation to the trochlea and probably play a major role in the eventual shape of the patella and trochlea, both of which develop congruent articulating surfaces [2, 3]. There is a difference between the bony and cartilage morphology of the patellofemoral joint [4, 5]. This means that congruent cartilaginous articulation may coexist with an underlying bony incongruence. In the last 30° of extension, the patella lies outside the bony constraints of the trochlea and is now dependent on soft-tissue constraints [6]. The MPFL has been shown to be the primary stabiliser against lateral dislocation [7]. The lateral retinaculum also has a restraining effect against lateral dislocation of the patella [8]. Beyond 30° of flexion, patellar stability is provided by the trochlea and the soft tissues become less important. The exact origin of the MPFL on the medial epicondyle is still undecided. Steensen [9] suggests that it attaches anterior to the epicondyle, while Smirk [10] postulates a posterior implantation, although some of his specimens reveal an anterior origin. In reconstructions, we prefer an anterior position on the epicondyle as this prevents a windscreen-wiper effect as well as an abnormal and sensitive prominence. In a study presented in 1997, we were able to demonstrate that the MPFL is non-isometric and becomes tight in extension and lax in flexion [11] (see illustrations 1, 2). This position has subsequently been confirmed by others [9, 10]. In recent unpublished cadaver studies, we could demonstrate that patella alta increases the non-isometry of the MPFL.

Placing the reconstruction more proximally, on the medial epicondyle, will result in a reconstructed MPFL that is lax in extension and tight in flexion, which may cause loss of knee flexion and excessive pressure on the medial patellar facet [12] (fig. 1). Conversely, placing the reconstruction too distally, on the medial epicondyle, will result in a too tight MPFL in extension and a lax ligament in flexion. A reconstruction that is too tight in extension may result in an extensor lag as the tension in the reconstructed ligament may be more than in the patellar tendon when the quadriceps muscles are maximally contracted (fig. 2). In reconstructing the MPFL, the aim



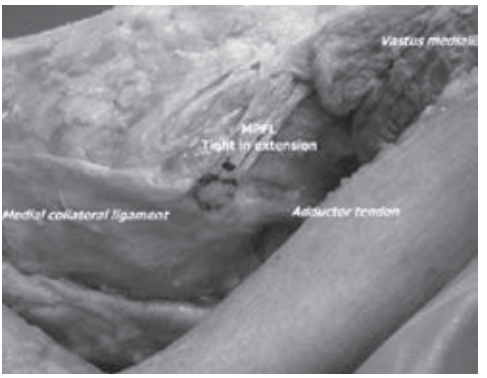


Illustration 1 : MPFL tight in extension

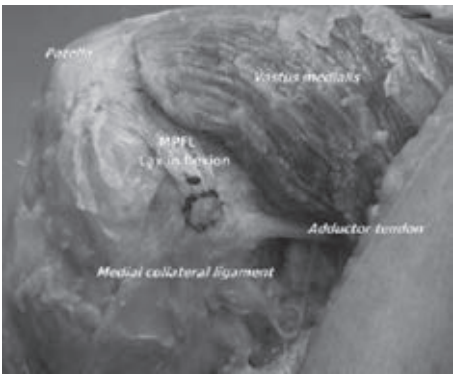


Illustration 2 : MPFL lax in flexion

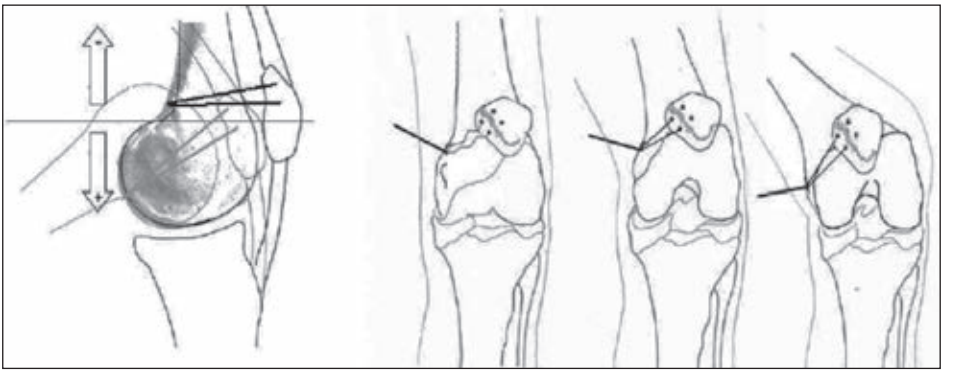


Fig. 1: Femoral position more proximal, MPFL is now more lax in extension and tighter in flexion.

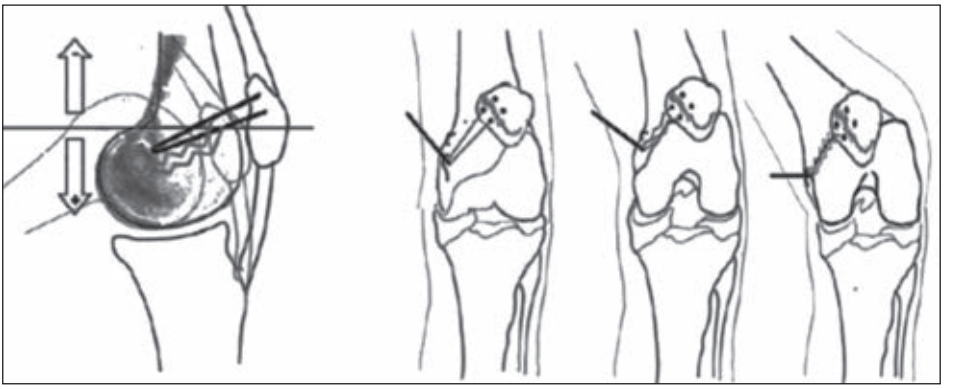
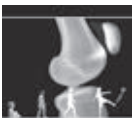


Fig. 2: Femoral position more distal, MPFL tighter in extension and more lax in flexion.



should be to create a "favourable anisometry" in the reconstructed ligament.13 In cases of severe patella alta, it may be impossible to achieve a "favourable anisometry" as non-isometry increases progressively with the height of the patella. In these cases, a distalisation of the patella might be necessary to improve the isometry.

In contrast to the MPFL, the lateral retinaculum is lax in extension and tightens in flexion [14, 15]. In nearly all patella dislocations there is damage to the MPFL. In our own series, 70% had damage at the patellar insertion while the remaining 30% were damaged at the femoral origin. In all cases, however, there was some interstitial damage to the whole ligament. These findings correspond with that of Garth [17] but differ from the MRI findings of Sallay [16].

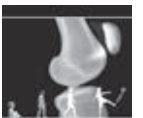
MANAGEMENT

In the majority of patients who present with patellar dislocation, there is underlying pathology such as ligamentous hyperlaxity, trochlear dysplasia and patella alta [18]. This underlying pathology predisposes the patient to an acute overload of the soft-tissue stabilisers and rupture of the MPFL with patella dislocation. Primary repair has a high failure rate: in our own series, 31% of the cases suffered redislocations in a four-year follow-up period. This corresponds with the results published by Nikku [19]. Most cases of primary dislocations are now treated non-surgically with a brace that allows full flexion but restricts the last 30% of extension. By restricting full extension, the MPFL is relaxed and may heal in a more favourable length. In exceptional cases, a primary reconstruction or direct repair of the MPFL and medial retinaculum would be considered. The principle of our repair philosophy is to reconstruct the MPFL with stronger tissue than before to compensate for the underlying predisposing pathology and without changing the original position of the patella and its original conformity with its underlying trochlea. The normal MPFL fails at 208N with an elasticity of 8N/mm [20]. A double gracilis fails at 1550N with an elasticity of 336N/mm [21]. At present, we prefer a double gracilis graft that, although stronger than the MPFL, is not as strong and stiff as a double semitendinosus tendon. Pre-operative evaluation consists of a proper clinical examination with specific attention to dynamic patella tracking, patella height and possible P-F chondral damage. The contra-lateral patella is also properly evaluated, as the principle is to restore the injured knee to the predislocation situation. Standard X-rays of the knee are done including a true lateral with the quads maximally contracted. This lateral X-ray is used to evaluate patellar height according to the Bernageau technique [22]. On MRI images, the ratio described by Biedert [23] can be used. The only surgery to be considered in addition to a MPFL reconstruction is a distal tibial tubercle transfer in cases of severe patella alta.

SURGICAL TECHNIQUE OF MPFL RECONSTRUCTION

Three 3cm long incisions are made over the gracilis tendon, over the medial edge of the patella and over the medial femoral epicondyle (fig. 3). The gracilis tendon is harvested with a routine technique. At the incision over the medial edge of the patella, an incision is made through the second fascial layer. From here a dissecting scissors is used to tunnel between the second and third fascial layers towards the medial epicondyle. At the medial epicondyle, the second fascial layer is again incised over the tip of the scissors (fig. 4).

A guide wire is inserted slightly proximally on the anterior slope of the epicondyle. In the proximal third of the medial edge of the patella, two 3mm drill holes are made approximately 10 to 12mm apart. These drill holes should be on the edge of the patella. Larger drill holes and holes that go into the centre of the patella might act as stress raisers, which can lead to a stress fracture of the patella and should therefore be avoided. A tape is now placed around the guide wire at the medial epicondyle, then between the second and third fascial layers and through the drill holes at the medial edge of



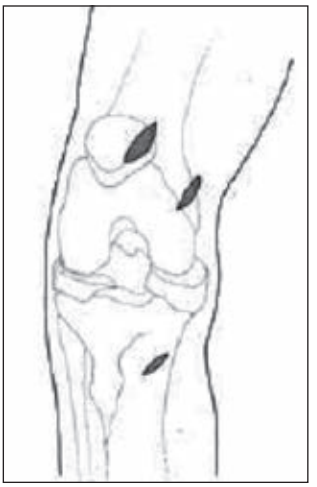


Fig. 3 : Skin incisions over the gracilis, the medial patella and the medial epicondyle.

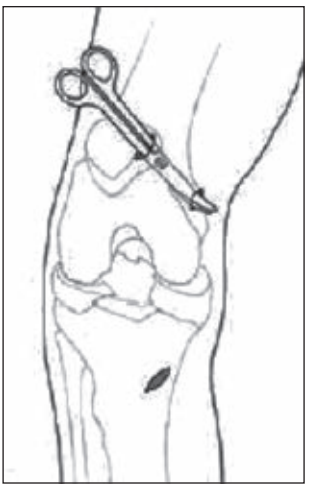


Fig. 4 : Dissecting with scissors between the second and third layer from the patella to the epicondyle.

the patella. With the knee in full extension, a bone hook is inserted at the distal pole of the patella. While pulling proximally on the bone hook, in the direction of the femoral shaft, the tape is temporary tied in the drill holes on the patella (fig. 5). The stability of the patella is compared with that of the opposite knee and the length changes in the tape are observed as the knee is flexed and extended. If the femoral fixation point is correct the patella will be stable in full extension. The tape should be maximally tight at full extension and become progressively more lax with flexion. If this tension pattern is not seen the position of the guide pin on the femur needs to be adjusted. Moving the guide pin more proximally will decrease the tension in extension and increase the tension in flexion (fig. 1). Conversely, moving the guide pin more distally will increase the tension in extension and decrease tension in flexion (fig. 2). The ideal position is where the tape is at its tightest in extension and becomes lax with flexion while stability of the patella is maintained. Care should be taken to ensure that there is more tension in the patellar tendon than in the reconstructed MPFL. This is best

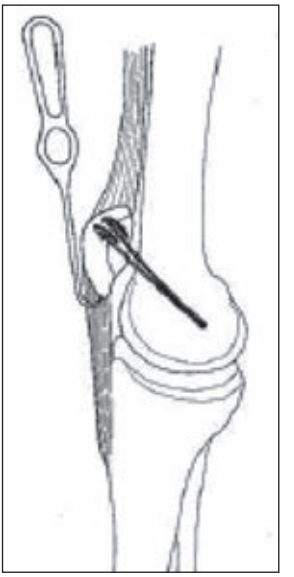


Fig. 5 : Pull proximally with a bone hook on the patella and the knee in full extension. Tension in the patellar tendon should be more than in the reconstructed MPFL.

achieved by pulling the patella proximally with the bone hook when tying the temporary tape. When a satisfactory tension pattern, in both the tape and patellar tendon is achieved, the guide wire in the epicondyle is overdrilled with a 4.5mm cannulated drill. A 5mm bone anchor is placed in the depth of the drill hole on the femur. The loop of the double gracilis tendon is tied into the femoral bone tunnel with the anchor. The two free ends of the looped tendon are now brought between the second and third fascial layers to the exposed medial edge of the patella and through the two 3mm drill holes on the medial edge. The free ends of the gracilis tendon are then folded back on themselves (fig. 6). The reconstructed ligament is tensed in the same manner as described above with the testing tape. Tensing is done with the knee in full extension while simultaneously pulling with a bone hook on the patella, in the direction of the femoral shaft. This manoeuvre prevents over-tensing of the reconstructed MPFL. Excessive tension in the reconstructed ligament can lead to an extensor lag. This happens when the tension in this reconstructed ligament is more than in the patellar tendon with the knee locked in full extension by maximum quadriceps contraction.

After tensing, the medial and lateral movement of the operated patella should be similar to that of the contralateral patella, the idea being to restore stability to the pre-dislocation situation. We suggested draping both knees to allow intra-operative comparison of the patellar movement. Once the tensing is satisfactory, the free end of the folded back tendon is sutured to itself and the surrounding soft tissue with non-absorbable material (fig. 7). Postoperatively, immediate full passive motion is encouraged. Active flexion and light isometric quadriceps exercises are done. For the first 4 weeks postoperatively the patient is mobilised partially weight-bearing, using two crutches. After 4 weeks, the crutches are discarded and intensive quadriceps rehabilitation starts. Quadriceps rehabilitation is often prolonged and can take up to 6 months or even longer. Normal sports activities can be resumed as soon as full quadriceps rehabilitation is achieved.

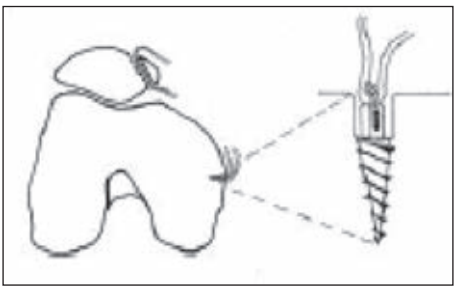


Fig. 6 : 5mm bone anchor anterior to the medial epicondyle. Two 3mm drill holes through the medial patellar rim.

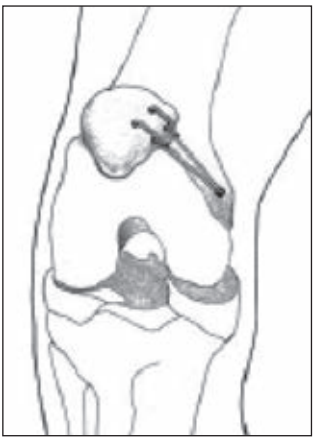
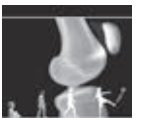


Fig. 7 : Reconstructed MPFL from the medial epicondyle to the medial patella.



LITERATURE

[1] ECKHOFF DG, BROWN AW, KILCOYNE RF, STAMM ER. Torsion of the knee associated with anterior knee pain. *Corr 1997; 339: 152-5.*

[2] TARDIEU C, DUPONT JY. Origine des dysplasies de la trochlea fémorale. *Revue Dechirurgie Orthopedique 2001; 87: 373-83.*

[3] FUCENTESE SF, VON ROLL A, KOCH PP, EPARI DR, FUCHS B, SCHOTTLE S. The patella morphology in trochlea dysplasia – a comparative study. *Knee 2006; 13(2): 145-50.*

[4] STAEUBLI HU, BOSSHARD C, PORCELLINI P, RAUSCHNING W. Magnetic resonance imaging for articular cartilage: cartilage-bone mismatch. *Clin Sports Med 2002; 21: 417-33.*

[5] SHIH YF, BULL AM, AMIS AA. The cartilaginous and osseous geometry of the femoral trochlea groove. *Knee Surg Sports Traumatol. Arthrosc 2004; 12: 300-6.*

[6] HEEGARD J, LEYVRAZ PF, VAN KAMPEN A, RAKOTOMANANA L, RUBIN PJ, BLANKEVOORT L. Influence of soft tissue structure on patella three-dimensional tracking. *Corr 1994; 299: 235-43.*

[7] HAUTMAA PV, FITHIAN DC, KAUFMAN KR, DANIEL DM. Medial soft tissue restraints in lateral patellar instability and repair. *Clinical Orthopedics and Related Research 1998; 349: 174-82.*

[8] DESIO SM, BURKS RT, BACHUS KN. Soft tissue restraints to lateral patellar translation in the human knee. *The American Journal of Sports Medicine 1998; 26(1): 59-65.*

[9] STEENSEN RN, DOPIRAK RM, McDONALD WG 3rd. The anatomy and isometry of the medial patellofemoral ligament. *The American Journal of Sport Medicine 2004; 32(6): 1509-13.*

[10] SMIRK C, MORRIS H. The anatomy and reconstruction of the medial patellofemoral ligament. *Knee 2003;10: 221-7.*

[11] ERASMUS PJ. Reconstruction of the medial patellofemoral ligament in recurrent dislocation of the patella. ISAKOS Buenos Aires May 1997. *Arthroscopy Suppl 1998; 14:42.*

[12] ELIAS JJ, COSGAREA AJ. Technical errors during medial patellofemoral ligament reconstruction could overload the medial patellofemoral cartilage: a computational analysis. *AJSM 2006; 34(9): 1478-85.*

[13] THAUNAT M, ERASMUS PJ. Favourable anisometry: An original concept for MPFL reconstruction. *Knee 2007; 14: 424-8.*

[14] LUO ZP, SAKAI N, RAND JA, AN KN. Tensile stress of the lateral patellofemoral ligament during knee motion. The American. *Journal of Knee Surgery 1997; 10(3): 139-44.*

[15] ISHIBASHI Y, OKAMURA Y, OTSUKA H, TSUDA E, TOH S. Lateral patellar retinaculum tension in patella instability. *Clinical Orthopedics and related research 2002; 397: 362-9.*

[16] SALLAY PI, POGGI J, SPEER KP, GARRETT WE. Acute dislocation of the patella: Acorrelative patho-anatomic study. *The American Journal of Sports Medicine 1996; 24: 52-60.*

[17] GARTH WP. Delayed proximal repair of the medial patellofemoral ligament in patients with recurrent patellar instability. *Clin Orth 2000; 377: 132-44.*

[18] NEYRET P. Patella tendon length: the factor in patellar instability? *Knee 2002; 9(1): 3-6.*

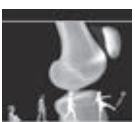
[19] NIKKU R. Operative versus closed treatment of primary dislocation of the patella. *Acta Orthopedica Scandinavia 1997; 68(5): 419- 23.*

[20] MOUNTNEY J, SENAVONGSE W, AMIS AA, THOMAS NP. Tensile strength of the medial patellofemoral ligament before and after repair or reconstruction. *JBJS 2005; 87B(1): 36-40.*

[21] HAMNER DL, BROWN CH, STEINER ME, HECKER AT, HAYES WC. Hamstring tendon grafts for reconstruction of the anterior cruciate ligament: Biomechanical evaluation of the use of multiple strands and tensioning techniques. *JBJS 1999; 81(A): 549-57.*

[22] BERNAGEAU J, GOUTALLIER D. Examin radiologique de l'articulation fémoro-patellaire: Actualités rheumatologiques. *Expansion scientifiique francaise 1984: 105-10.*

[23] BIEDERT RM, ALBRECHT S. The patello trochlear index: a new index for assessing patellar height. *Knee Surg Sports Traumatol. Arthroscopy 2006; 14: 707-12.*



LA BALLONISATION DU TUNNEL FÉMORAL APRÈS RECONSTRUCTION DU LIGAMENT FÉMORO-PATELLAIRE MÉDIAL

J.B. BERARD, S. ÖZCAN, G. DEMEY, S. LUSTIG, P. NEYRET, E. SERVIEN

INTRODUCTION

En cas de luxation récidivante de la rotule, l'instabilité est le principal symptôme nécessitant une intervention chirurgicale. Différentes procédures chirurgicales peuvent être envisagées : les interventions osseuses [1, 2, 3] (trochléoplastie, ostéotomie de la tubérosité tibiale antérieure) et/ou les interventions sur les tissus mous [4, 5] (section de l'aileron externe, plastie du vaste médial, reconstruction du ligament médio-patellaire fémoral [MPFL]). Depuis quelques années, la reconstruction du MPFL est devenue le geste chirurgical de première intention, isolé ou non. Il a été clairement démontré que le MPFL est la structure anatomique la plus souvent touchée après une luxation rotulienne [6, 7] et son rôle stabilisateur dans les premiers degrés de flexion a été largement étudié. Son rôle est primordial dans les premiers degrés de flexion puisqu'il va diriger la patella médialement à l'entrée de la trochlée. Il se détend alors, vers 20° de flexion, dans une trochlée normale. La stabilisation patellaire est alors assurée par le vastus médialis et la congruence trochléenne. Deux théories prédominent : la recherche d'une isométrie la plus parfaite possible [8] et la théorie de l'anisométrie favorable où le ligament est tendu en position d'extension et se détend avec la flexion [9]. Dans les deux cas, la tension appliquée au ligament ne doit pas augmenter en flexion ce qui laisse libre le travail de récupération précoce des amplitudes articulaires.

Plusieurs techniques de reconstruction du MPFL ont été décrites avec des résultats cliniques encourageants. De nombreuses techniques utilisent un tunnel fémoral pour la fixation du greffon [7, 10-16].

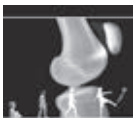
Grâce à notre expérience [17], nous avons noté qu'il existait parfois, au cours du suivi radiologique postopératoire, un élargissement du tunnel fémoral, appelé "ballonisation".

De la même façon qu'il a été décrit un élargissement du tunnel osseux dans la reconstruction du LCA [18-20], nous pensons qu'il existe pour le MPFL, des causes mécaniques et biologiques [21] pouvant expliquer cette ballonisation.

Le but de ce travail était de déterminer l'incidence de la ballonisation dans notre série prospective de reconstruction du MPFL, et de rechercher les causes mécaniques influençant l'isométrie du MPFL.

MATÉRIEL ET MÉTHODES

Entre 2005 et 2010, 66 patients ont été opérés pour luxation récidivante de la rotule de première intention. Les critères d'exclusion étaient



les chirurgies itératives du genou, les gestes associés autres que la transposition de la tubérosité tibiale antérieure (TTA) et les causes neurologiques.

La série prospective comportait 51 patients, soit un total de 55 genoux opérés (26 genoux gauches et 29 genoux droits).

Il y avait 37 femmes et 14 hommes (sex ratio : 2,6/1).

39 interventions ont consisté en une reconstruction isolée du MPFL et 16 en une reconstruction du MPFL + transposition de TTA.

Analyse préopératoire

Tous les patients avaient en préopératoire des radiographies de face en charge et de profil strict à 15° de flexion, ainsi que des vues axiales de rotules et un scanner du genou en extension selon un protocole identique.

Tous les patients remplissaient le score *International Knee Documentation Committee* (IKDC) subjectif [22, 23].

Les items analysés en préopératoire étaient :
- *Pour la clinique :* l'âge, l'indice de masse corporel (BMI)
- *Pour le score fonctionnel :* l'IKDC subjectif
- *Pour l'imagerie :* la TA-GT sur le scanner, le stade de dysplasie de trochlée et l'index de Caton-Deschamps [24] sur la radio de profil.

La dysplasie trochléenne était classée en 4 grades selon la classification de D. Dejour [25].

Technique chirurgicale de reconstruction du MPFL

La technique chirurgicale consistait en une arthroscopie première à la recherche de lésions associées (cartilage) et pour l'évaluation de la course rotulienne.

Le prélèvement du tendon du gracilis était effectué à l'aide d'un stripper, puis replié sur lui-même autour d'un fil tracteur et suturé sur 2 cm.

Deux tunnels rotuliens étaient réalisés à la mèche 3,2 mm puis 4,5 mm dans le tiers proximal de la rotule par un point d'entrée médial et un orifice de sortie à la face antérieure distant de 8 à 10 mm.

Le tunnel fémoral borgne était réalisé avec une mèche d'un diamètre de 7 mm et mesurait 25 à 30 mm.

Le greffon était alors tracté dans le tunnel grâce au fil passé dans le chas de la broche et fixé par une vis d'interférence résorbable de diamètre 7 mm.

Les extrémités du greffon étaient glissées dans le plan de clivage sus-capsulaire et récupérées par l'incision rotulienne où ils passaient dans les tunnels et étaient suturés sur eux-mêmes : la rotule devait être centrée lorsque la suture était réalisée, de manière à obtenir une bonne tension du greffon. La rotule devait rester mobilisable mais non luxable. Cette suture était réalisée à 30° de flexion du genou.

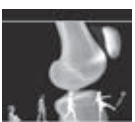
Dans le cas où l'index de Caton-Deschamps était supérieur à 1,2, un abaissement de TTA était réalisé dans le même temps opératoire que le MPFL.

Analyse postopératoire

Les patients étaient revus à 45 jours, 6 mois et 1 an avec radio de face et de profil strict puis tous les deux ans.

Les patients remplissaient un score IKDC subjectif à 1 an.

Sur les radios de profil à 1 an, le tunnel fémoral était analysé. Deux mesures étaient rapportées : la mesure verticale du rayon du tunnel qui est effectuée dans l'axe de la diaphyse fémorale et la mesure horizontale du rayon du tunnel, perpendiculaire à l'axe de la diaphyse (fig. 1).



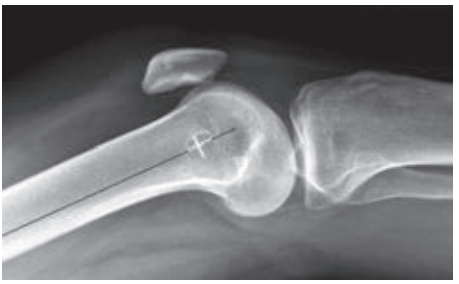


Fig. 1 : Mesure du rayon vertical et horizontal du tunnel fémoral

Le positionnement des tunnels était analysé selon la méthode de Schöttle [26], modifié par Servien [17] : le tunnel était considéré comme mal positionné s'il était situé à plus de 7 mm (fig. 2) de n'importe quelle partie du point de Schöttle (centre anatomique du MPFL natif).

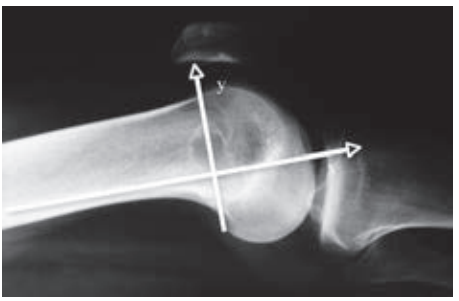


Fig. 2 : Exemple de malposition du tunnel fémoral

Les mesures ont été effectuées par deux observateurs indépendants et comparées.

L'aire du tunnel a été calculée pour chaque radiographie selon la formule $\pi*r^2$ afin de déterminer le groupe des tunnels "normaux" et le groupe des tunnels "ballonisés".

L'aire théorique du tunnel a été calculée en fonction du diamètre de la mèche et de la vis d'interférence : $\pi*r*r = 38,5\ mm^2$.

L'analyse de l'aire du tunnel a permis de différencier deux groupes : celui des tunnels normaux où l'aire du tunnel mesurée est inférieure à 2 X l'aire théorique et celui des tunnels ballonisés où l'aire est supérieure à 2 X l'aire théorique.

Analyse statistique

Les items ont été comparés entre les deux groupes (moyenne, valeurs minimales et maximales et écart type selon Pearson) et un test de Student à distribution unilatérale sur les deux populations hétéroscédastiques a été réalisé à l'aide du logiciel Excel. La différence était statistiquement significative pour $P \leq 0,05$.

RÉSULTATS

Série générale

L'âge moyen à l'intervention était de 24,2 ans (13 - 60,5 ans).

Le BMI moyen des patients à l'intervention était de 22,5 kg/m^2 (17 à 33,8 ; Ecart type à 3,2).

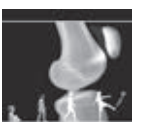
La répartition de dysplasie de trochlée selon D. Dejour était de : 14 au stade A, 22 au stade B, 13 au stade C et 3 au stade D. Trois trochlées n'étaient pas dysplasiques.

L'index de Caton-Deschamps moyen était de 1,1 (0,6 à 1,5 ; Ecart type à 0,17).

L'IKDC subjectif préopératoire moyen était de 56 (23 à 80 ; Ecart type à 13) et postopératoire de 76 (28 à 98 ; Ecart type à 14).

La moyenne de suivi était de 25,4 mois (12 - 64 mois).

Il y avait 32 genoux avec tunnel non ballonisé et 23 genoux avec tunnel ballonisé.



Groupe non ballonisé

La hauteur rotulienne était normale dans 72 % des cas, soit 23 genoux et il y avait 28 % de rotules hautes, soit 9 genoux.

La moyenne de l'IKDC postopératoire était de 76 (28 à 97 ; Ecart type à 15,4).

Il y avait 66 % de tunnels bien positionnés, soit 21 genoux et 34 % de tunnels mal positionnés, soit 11 genoux.

Groupe ballonisé

La hauteur rotulienne était normale dans 48 % des cas, soit 11 genoux et il y avait 52 % de rotules hautes, soit 12 genoux.

La moyenne de l'IKDC postopératoire était de 76 (50 à 98 ; Ecart type à 13).

Il y avait 56 % de tunnels bien positionnés, soit 13 genoux et 44 % de tunnels mal positionnés, soit 10 genoux.

Comparaison

Nous avons retrouvé plus de rotules hautes dans le groupe ballonisé (52 % ; n=12) que dans le groupe non-ballonisé (28 % ; n=9) de façon statistiquement significative (p=0,03).

Le score IKDC postopératoire était identique dans les deux groupes : 76 pour les non ballonisés et pour les ballonisés (p=0,43).

Dans le groupe ballonisé, nous avons retrouvé plus de tunnels mal positionnés (44 % ; n=10) que dans le groupe non ballonisé (34 % ; n=11) de façon significative (p=0,05).

Il n'y avait aucune corrélation liée au poids (p=0,17) ou à l'âge (p=0,28) entre les patients des deux groupes. Il n'y avait pas de corrélation sur la répartition des dysplasies de trochlée entre le groupe ballonisé et non ballonisé (p=0,11).

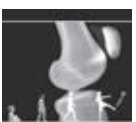
DISCUSSION

Série générale

Sur le plan thérapeutique, la reconstruction du MPFL est une intervention efficace (aucune luxation n'est survenue en postopératoire au recul actuel) et intéressante sur le plan fonctionnel (IKDC passant de 56 de moyenne en préopératoire à 76 en postopératoire).

Tableau 1 : Récapitulatif de la comparaison entre les deux groupes

Moyenne	Groupe non ballonisé	Groupe ballonisé	P value
Dysplasie de trochlée	Stade 0 : 6,25 % Stade A : 34,3 % Stade B :37,5 % Stade C : 21,8 % Stade D : 0 %	Stade 0 : 4 % Stade A : 13 % Stade B : 43 % Stade C : 26 % Stade D : 13 %	p=0,11
Index Caton-Deschamps	<1,2 : 72 % ≥1,2 : 28 %	<1,2 : 48 % ≥1,2 :52 %	p=0,03
Score IKDC post-op	76	76	p=0,43
Positionnement tunnel	Bon : 66 % Mauvais : 34 %	Bon : 56 % Mauvais : 44 %	p=0,05
BMI (Kg/m²)	21,8	23,2	p=0,17
Age (ans)	25	24	p=0,28



La ballonisation

Il a été retrouvé significativement plus de ballonisation du tunnel fémoral chez les patients avec une rotule haute et une malposition du tunnel, ce qui semble être en faveur d'une cause mécanique. Les situations où le transplant est trop tendu (rotule haute et/ou tunnel trop distal ou trop postérieur) peuvent être à l'origine d'une anisométrie franche [9].

Données de la littérature

L'élargissement des tunnels osseux dans le cadre de la reconstruction du ligament croisé antérieur (LCA) a été bien étudié sur le plan biomécanique [15-17] et biologique [27, 28] et il paraîtrait dangereux d'extrapoler ces concepts à l'identique pour la reconstruction du MPFL sans les avoir scientifiquement validés. Cependant, ces précédents travaux nous donnent une idée des études à réaliser pour expliquer ce phénomène.

Il s'agit, à notre connaissance, de la seule étude portant sur la ballonisation du tunnel fémoral dans la reconstruction du MPFL, sur une série prospective de patients.

L'étude était unicentrique avec une technique chirurgicale bien conduite et superposable d'un opérateur à l'autre.

CONCLUSION

La ballonisation du tunnel fémoral dans la reconstruction du MPFL est un phénomène fréquent qui se voit plus souvent en cas de rotule haute et/ou de malposition du tunnel fémoral. Cependant, cela n'affecte pas le score IKDC et ne provoque pas de luxation itérative de rotule, au recul minimum de 1 an.

LITERATURE

[1] SERVIEN E, VERDONK PC, NEYRET P. Tibial tuberosity transfer for episodic patellar dislocation. *Sports Med Arthrosc. 2007 juin;15(2):61-7.*

[2] UTTING MR, MULFORD JS, ELDRIDGE JDJ. A prospective evaluation of trochleoplasty for the treatment of patellofemoral dislocation and instability. *J Bone Joint Surg Br. 2008 févr; 90(2): 180-5.*

[3] FARR J, SCHEPSIS A, COLE B, *and al.* Antero-medialization: review and technique. *J Knee Surg. 2007 avr; 20(2): 120-8.*

[4] DAVIS DK, FITHIAN DC. Techniques of medial retinacular repair and reconstruction. *Clin Orthop Relat Res 2002 sept;(402): 38-52.*

[5] MYERS P, WILLIAMS A, DODDS R, *and al.* The three-in-one proximal and distal soft tissue patellar realignment procedure. Results, and its place in the management of patellofemoral instability. *Am J Sports Med. 1999 oct;27(5): 575-9.*

[6] AMIS AA, FIRER P, MOUNTNEY J, *and al.* Anatomy and biomechanics of the medial patellofemoral ligament. *Knee 2003 sept; 10(3): 215-20.*

[7] FARR J, SCHEPSIS AA. Reconstruction of the medial patellofemoral ligament for recurrent patellar instability. *J Knee Surg. 2006 oct; 19(4): 307-16.*

[8] STEENSEN RN, DOPIRAK RM, MAURUS PB. A simple technique for reconstruction of the medial patellofemoral ligament using a quadriceps tendon graft. *Arthroscopy 2005 mars; 21(3): 365-70.*

[9] THAUNAT M, ERASMUS PJ. The favourable anisometry: an original concept for medial patellofemoral ligament reconstruction. *Knee 2007 déc; 14(6): 424-8.*

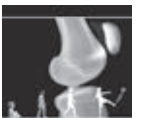
[10] DEIE M, OCHI M, SUMEN Y, *and al.* A long-term follow-up study after medial patellofemoral ligament reconstruction using the transferred semitendinosus tendon for patellar dislocation. *Knee Surg Sports Traumatol Arthrosc. 2005 oct;13(7): 522-8.*

[11] ELIAS JJ, COSGAREA AJ. Technical errors during medial patellofemoral ligament reconstruction could overload medial patellofemoral cartilage: a computational analysis. *Am J Sports Med. 2006 sept;34(9): 1478-85.*

[12] GOMES JLE. Medial patellofemoral ligament reconstruction with half width (hemi tendon) semitendinosus graft. *Orthopedics 2008 avr; 31(4): 322-6.*

[13] LEGRAND AB, GREIS PE, DOBBS RE, and al. MPFL reconstruction. *Sports Med Arthrosc. 2007 juin; 15(2): 72-7.*

[14] NOMURA E, INOUE M. Hybrid medial patellofemoral ligament reconstruction using the semitendinous tendon for recurrent patellar dislocation: minimum 3 years' follow-up. *Arthroscopy. 2006 juill; 22(7): 787-93.*



[15] NOYES FR, ALBRIGHT JC. Reconstruction of the medial patellofemoral ligament with autologous quadriceps tendon. *Arthroscopy. 2006 août; 22(8): 904.e1-7.*

[16] SCHÖTTLE P, SCHMELING A, ROMERO J, *and al.* Anatomical reconstruction of the medial patellofemoral ligament using a free gracilis autograft. *Arch Orthop Trauma Surg 2009 mars; 129(3): 305-9.*

[17] SERVIEN E, FRITSCH B, LUSTIG S, *and al.* In vivo positioning analysis of medial patellofemoral ligament reconstruction. *Am J Sports Med 2011 janv; 39(1): 134-9.*

[18] IORIO R, VADALÀ A, ARGENTO G, *and al.* Bone tunnel enlargement after ACL reconstruction using autologous hamstring tendons: a CT study. *Int Orthop. 2007 fëvr; 31(1): 49-55.*

[19] NEBELUNG W, BECKER R, MERKEL M, *and al.* Bone tunnel enlargement after anterior cruciate ligament reconstruction with semitendinosus tendon using Endobutton fixation on the femoral side. *Arthroscopy. 1998 déc; 14(8): 810-5.*

[20] WEBSTER KE, FELLER JA, HAMEISTER KA. Bone tunnel enlargement following anterior cruciate ligament reconstruction: a randomised comparison of hamstring and patellar tendon grafts with 2-year follow-up. *Knee Surg Sports Traumatol Arthrosc. 2001;9(2): 86-91.*

[21] WILSON TC, KANTARAS A, ATAY A, *and al.* Tunnel enlargement after anterior cruciate ligament surgery. *Am J Sports Med. 2004 mars; 32(2): 543-9.*

[22] HEFTI F, MÜLLER W. Current state of evaluation of knee ligament lesions. The new IKDC knee evaluation form. *Orthopade 1993 nov; 22(6): 351-62.*

[23] IRRGANG JJ, ANDERSON AF, BOLAND AL, *and al.* Development and validation of the international knee documentation committee subjective knee form. *Am J Sports Med. 2001 oct; 29(5): 600-13.*

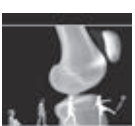
[24] CATON J. Method of measuring the height of the patella. *Acta Orthop Belg. 1989; 55(3): 385-6.*

[25] DEJOUR D, REYNAUD P, LECOULTRE B. Douleur et instabilité rotulienne : essai de classification. *Med Hyg.1998 ;5:1466-71.*

[26] SCHÖTTLE PB, SCHMELING A, ROSENSTIEL N, *and al.* Radiographic landmarks for femoral tunnel placement in medial patellofemoral ligament reconstruction. *Am J Sports Med. 2007 mai;35(5):801-4.*

[27] HUNT P, REHM O, WEILER A. Soft tissue graft interference fit fixation: observations on graft insertion site healing and tunnel remodeling 2 years after ACL reconstruction in sheep. *Knee Surg Sports Traumatol Arthrosc. 2006 déc;14(12):1245-51.*

[28] WEILER A, PEINE R, PASHMINEH-AZAR A, *and al.* Tendon healing in a bone tunnel. Part I: Biomechanical results after biodegradable interference fit fixation in a model of anterior cruciate ligament reconstruction in sheep. *Arthroscopy. 2002 fëvr; 18(2): 113-23.*



COMPLICATIONS AFTER MPFL RECONSTRUCTION

P.J. ERASMUS, M. THAUNAT

INTRODUCTION

Similar to other ligamentous reconstructions, around the knee, MPFL reconstructions can lead to complications. These complications relates to a lack understanding of the biomechanics of the MPFL ligament and technical errors made during the reconstruction.

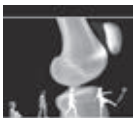
BIOMECHANICS OF THE MPFL

The MPFL should be seen as a check rein in preventing abnormal lateral movement of the patella, at and near full extension; it is not suppose to pull the patella medially and is of no importance once the patella has engaged the trochlea [15].

In the literature, on reconstruction of the MPFL, most authors suggest that the reconstructed ligament should be isometric. So called most isometric points [25, 27] for the reconstruction have been suggested.

However in measurements of the normal length changes in the MPFL it has repeatedly been shown to be a non isometric ligament [25, 29]. According to Steensen there is a 5.4mm length change from 0° to 90° of flexion and an average of 7.2mm length change from 0° to 120°. It is important to realize that the MPFL is a non isometric ligament that is at it's tightest at full extension and becomes more lax with flexion, as the patella engages into the trochlea [15]. Victor [33] confirmed the non isometry of the MPFL and has suggested that there is a difference in the non isometry of the proximal and distal fibres of the MPFL; the proximal fibres are at their tightest at full extension and the distal at 30° of flexion. In cadaveric studies it was shown that an anatomic MPFL reconstruction will restore patella kinematics better than an isometric reconstruction [22]. The position of the reconstructed ligament on the patella has very little effect on the isometry of the ligament. In contrast the position on the femur has a major effect on the on the isometry of the ligament. A more distal position increases tightness in extension and laxity in flexion; conversely a more proximal position results in a graft that is lax in extension and tight in flexion (fig. 1).

In nearly all patella dislocations there is underlying causes like patella alta, trochlear dysplasia, ligamentous hyper laxity, etc. that predisposes the patient to a patella dislocation. Patella alta seems to be the most constant predisposing factor in patella dislocation [16] Patella height has an influence on the isometry of the MPFL, the higher the patella the bigger



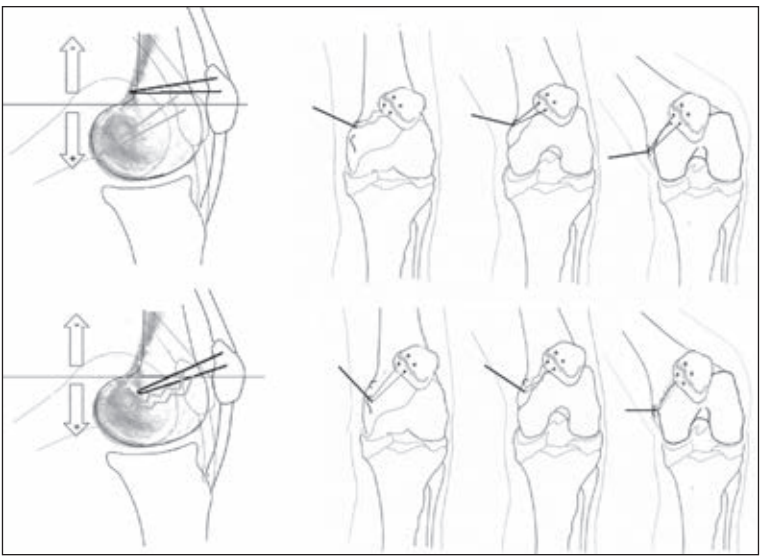


Fig. 1 : A proximal position on the femur will result in a graft that is loose in extension and tight in flexion, conversely a distal femoral position will result in a graft that is tight in extension and loose in flexion.

the non isometry of the ligament. In unpublished cadaveric experiments we found that that the average length changes in the MPFL from 0°-90° was 4mm. If the tibial tubercle was moved 10mm proximally the average change was 6mm.

When the tubercle was moved 10mm distally the average length change was only 3mm [9]. Considering this increase in non isometry with patella alta the distance, at full extension, from the origin of the MPFL on the medial femoral condyle to its insertion on the patella, with the quads fully contracted, is important in planning surgery. At present there is no specific way to measure this distance. The most commonly used measurements for the patella height like Caton – Deschamps, Blackburne – Peel and Insall – Salvati measures patella height in relation to the tibia. What is however more important is the height of the patella in relation to the superior border of the trochlea as suggested by Bernageau [3] on X-rays and Biedert on MRI's [4, 1]. Patella alta is associated with a long patella tendon and patella tendon length is more sensitive than Caton-Deschamps index for patella instability [21]?

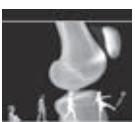
In reconstructing the ligament the aim should be to use a ligament that is stronger than the original to compensate for the underlying predisposing factors. The reconstructed ligament should duplicate the non isometry.

The aim of the reconstruction should be to create a "favourable anisometry" [29] that duplicates that of the original ligament before injury. Failure to create favourable non isometry can lead to redislocation, extensor lag and loss of flexion. Loss of flexion will also lead to overload in the patella femoral joint especially in the medial facet with flexion.

COMPLICATIONS

Loss of motion

In the long term follow up in our series of more than 200 MPFL reconstructions, done from 1995 till 2008, extensor lag, with full passive extension and no loss of flexion was the most common complication. There was no long term loss of flexion. Not with standing loss of motion



these patients still had an average Kujala score of 92.7 (72-100) indicating that this score is not sensitive to a minor extensor lag. Smith *et al.* [26] did a comprehensive literature research on the clinical and radiological results of MPFL reconstructions. They could only find eight papers, involving 186 MPFL reconstructions, which met the criteria of their scoring system. In only two of these eight papers did the authors report on the post operative range of motion and in both there were loss of flexion compared to the non operated leg. None reported on loss of active knee extension or extensor lag. Only one paper, in this review, reported on quadriceps atrophy with an incidence of 60% not withstanding a mean Kujala score of 88.6 [7]. Loss of motion after an MPFL reconstruction is directly related to the tension in the reconstructed ligament. If it is too tight in extension (femoral insertion too distal) there will be an extensor lag although passive full extension will not be affected. If it is too tight in flexion (femoral insertion too proximal) there will be loss of flexion both passive and active; in this situation the patella might still be unstable in extension.

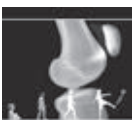
Both the gracilis and semitendinosis tendons, generally used, for reconstructing the MPFL is stronger and stiffer than the original MPFL [20]. The strength is a positive factor considering the underlying predisposing factors leading to the first dislocation. The stiffness on the other hand can theoretically lead to overload in the P-F joint especially in cases where the reconstructed MPFL is not in the optimal position.

In our technique of MPFL reconstruction we try to recreate the normal non isometry of the ligament so called "favourable anisometry" [29].

This creates a ligament that is tight in extension and lax in flexion. There is however the danger that the ligament can be too tight in extension resulting in an extensor lag. Post operative quadriceps inhibition is very common and should be distinguished from a permanent extensor lag as a result of an over tight MPFL reconstruction. At 3 months post operative follow-up there was on average a 4° (5°-15°) extensor lag, probably as a result of quads inhibition, in 45% of our patients. This extensor lag was however temporary and over the long term only 4 out of the more than 200 cases had permanent loss of active full extension which was caused by an over tight reconstruction in extension [27].

Elias [8] has shown experimentally that a too proximal placed femoral position for the MPLF graft will lead to increased patello femoral load with potential overload of the articular surface in the P-F joint. Loss of both active and passive flexion will also be associated with this. In techniques where the aim is to have an isometric reconstructed MPFL, the danger of a having a ligament that is too tight in flexion is increased. Femoral insertions near or at the adductor tubercle, although advocated by some authors, should be avoided as this will lead to reconstruction that is too tight in flexion and too loose in extension [25, 27, 23].

In prevention of motion loss complications special attention should be given to the technique of determining the tension in the reconstructed ligament. Beck showed [2] that overtensioning can be avoided by applying low loads to medial patellofemoral ligament reconstructions, which reestablished normal translation and patellofemoral contact pressures. The aim of the MPFL reconstruction should be to restore the tension in the MPFL to the same tension that it had before being torn with a graft that is stronger than the original ligament. If the patella of the opposite knee is stable the amount of transverse patella movement in the reconstructed patella should be similar to that of the uninjured knee. This can be achieved intra operatively by draping both knees and comparing the amount of transverse movement. Fithian [13] advises adjusting the graft tension in such a way that a 5 lb displacing force result in 7-9mm of lateral displacement of the patella We recommend that the isometry of the ligament should be tested till the "favourable anisometric point" is found by, using a guide pin in the proposed femoral implantation site. The "favourable anisometric point" would be a point where the reconstructed ligament will be



tight in extension end lax in flexion. It is further recommended that the ligament should be tightened in full extension, pulling proximally on the patella with a bone hook in the direction of the anterior superior iliac spine in order to ensure that, with maximum quads contraction, there will be more tension in the patellar tendon than in the reconstructed MPFL (fig. 2). In cases of severe patella alta a distal tibial tubercle transfer should be considered as this will decrease the non isometry of the reconstruction allowing easier and more precise tensioning of the reconstructed ligament [9] (fig. 3). We will consider a distal tubercle transfer where the Bernageau measurement is more than 8mm or the patella tendon length more is than 60mm, especially if this combined with a clinically marked positive J-Sign. A distal transfer of as little as 6mm is usually adequate in these cases. Other authors have recommended different techniques for tensioning the reconstructed MPFL. The most popular being to tension the ligament between 30° to 60° of flexion when the patella is already centred in the trochlea [5, 20, 6]. The major length changes in the MPFL happens after 30° of flexion [25, 29] and considering this tensioning the ligament in early flexion should prevent over tensioning provided that, on the femur, the correct non isometric point have been selected.

Post operative quadriceps inhibition, although temporary, can result in an increased rehabilitation period and a late return to sporting activities.

Drez [7] reported quadriceps atrophy in more than 50% of his patients at an average follow up of 31.5 months (24-43).

In an effort to combat this we start the patients on isometric quads contraction program preoperatively. Post operatively no braces are used and immediate active and passive full range of motion is encouraged.

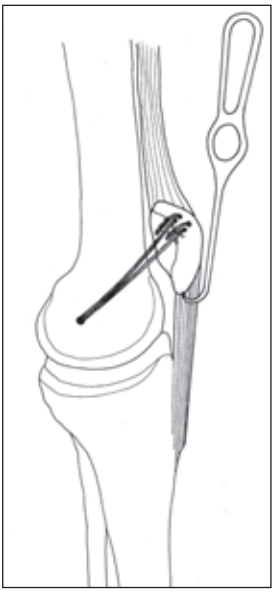


Fig. 2 : Tensioning the MPFL graft in full extension ensuring that the tension in the reconstruction is less than in the patellar tendon.

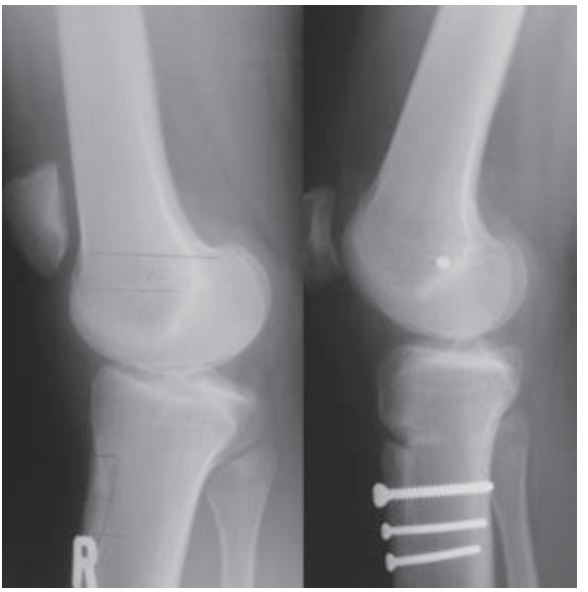


Fig. 3 : Distalization of the tibial tubercle combined with a MPFL reconstruction.

Isometric quadriceps exercises are continued. Full weight bearing, with the support of one or two crutches, as necessary, is allowed). In our follow up of 22 patients at average 29 months (8-65) the difference in the side to side upper leg circumference, 15cm above the knee, was only 0.19cm (0-1.5cm) [9].

Should loss of motion persist for longer than 9 months, after an MPFL reconstruction, we would recommend a percutaneus sequential fish scale type of tenotomy, near the implantation of the ligament on the patella, till full range of motion is restored [31].

Fractures

In our series we only had had three redislocations, all associated with fractures of the medial rim of the patella [30]. In all these patients the fracture occurred with a definitive injury; in two this happened in contact sport, one in soccer and the other in rugby football.

The third patient sustained a redislocation when she fell off a chair trying to replace a fused light bulb. The fractures occurred respectively 2, 5 and 10 years after the initial surgery. In our reconstructing technique two 3 -3.5mm drill holes, 10mm apart, are made on the medial edge of the patella exiting the anterior cortex approximately 10mm from the medial edge. These fractures were similar to that seen in acute primary patelladislocations [32]. A gracilis outograft was used for reconstructing the ligament.

This reconstructed ligament is stronger than the original MPFL and as the underlying predisposing factors have not been addressed there will, at times, be high strain on the ligament. This can result in a patella fracture through the drill holes which acts as stress raisers. In all three cases the fracture involved not more than the 1cm of the medial patella (fig. 4).

The fractures were reduced and fixed with screws all resulting in a stable patella with no longstanding sequel from the fractures.

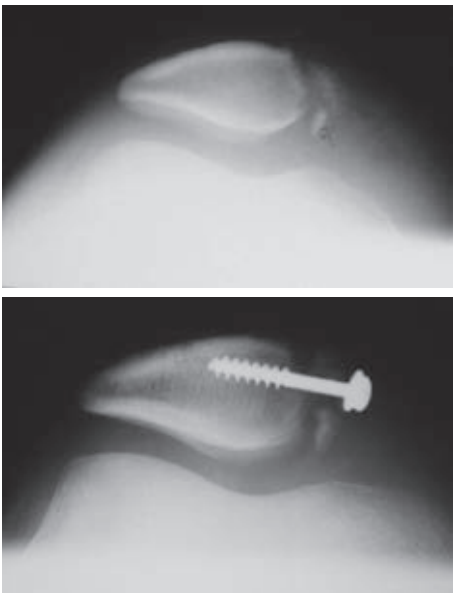
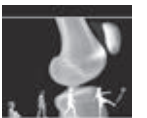


Fig. 4 : Redislocation after MPFL reconstruction with a fracture of the medial rim reattached with a screw and washer.

Mikashima [17] reported two fractures in 12 knees. A single transverse drill hole, of 4.5mm was done from medial to lateral through the patella.

These fractures all occurred within 6 weeks from the surgery resulting in a nearly 16% incidence of fractures. Both Christiansen [5] using two 4.5mm and Gomes using a single 7mm [14] transverse drill holes, reported non traumatic patella fractures. It is possible that too large drill holes increase the possibility of a fracture especially when they transverse the patella. Fractures of the medial rim of the patella usually do not involve the articular surface of the patella and is relatively easy to treat in contrast transverse fractures which always involve the articular surface of the patella and in most cases there is a disruption of the cortex which makes it more serious and an anatomical reduction difficult (fig. 5).



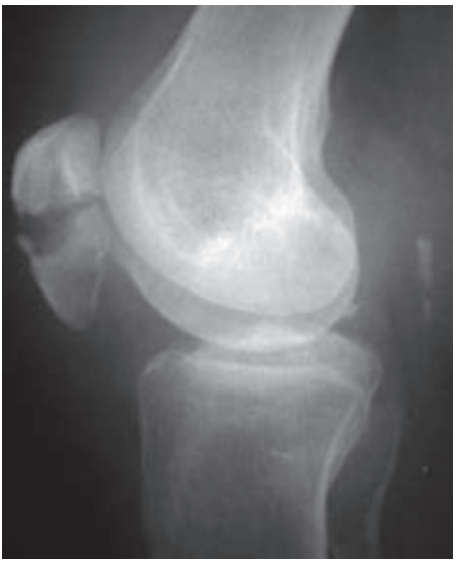


Fig. 5 : Tranverse patella fracture secondary to transverse drill hole for MPFL reconstruction.

It has to be expected that drill holes in the patella will act as stress raisers and might therefore predispose to fractures. Keeping this in mind the size and position of the hole should be carefully considered. Holes larger than 3.5mm should probably be avoided. Transverse drill holes through the patella will result in more serious fractures then drill holes through the medial rim.

Redislocations

Redislocations seems to be rare, in the literature it varies between 0-4% [5, 7, 24]. In our series there were only three redislocations all three associated with a fracture of the medial rim of the patella. In Smith's [26] review article there was only two post reconstruction patella dislocations or subluxations in 186 knees; in five patients there were a positive apprehension sign.

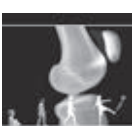
Localized tenderness

Localized tenderness in the region of the medial epicondyle related to either the graft or the internal fixation used can be an irritating complication. Nomura [20] reported an incidence of 40% as a result of using a staple for fixation just distal to the adductor tubercle. Christiansen [5] reported 50% tenderness over the medial epicondyle Steiner [28] had to remove irritating screws in 10% of his patientsWe [10] use a deep seated bone anchor on the medial femur and had a 6% incidence of mild localized tenderness at the exit of the graft; no surgical intervention was necessary for this.

P-F degeneration

In a average 7 (4.4-9.3) year follow-up of our first 29 patients [10] the Tegner (5.8) Lysholm (88.5) and IKDC (81) scores were statistically unchanged at 3,5 and 7 years follow-up. P-F cartilage damage at the time of the MPFL reconstruction had a negative effect on the Lysholm score but no effect on the Tegner and IKDC scores. In a 29 month follow-up in a consecutive group of isolated MPFL reconstructions in 22 of our patients [9] there was no statistical correlation between the Kujala score; trochlea dysplasia and Caton Deschamps index. There was a weak correlation between a lower score; patella tendon length and Bernageau measurement of patella height. There was however a statistical significant correlation between P-F degeneration at the time of the reconstruction and a low Kujala score.

It does seem that development or even progression of P-F degeneration might be prevented or at least stalled by an isolated MPFL reconstruction. Nomura, in a 12 year follow up, came to the same conclusion [20].



CONCLUSIONS

MPFL reconstructions seem to give good result with few complications not withstanding the varied techniques described in the literature. There is however certain principles that should be adhered to prevent complications. The reconstructed ligament should be tight in extension and lax in flexion. In cases of severe patella alta a distalization osteotomy of the tibial tubercle should be considered to improve the non isometry of the MPFL. The ligament should be tensioned in such a way that with maximum quadriceps contraction the tension in the patellar tendon should be more than the tension in the reconstructed ligament to prevent a permanent extensor lag.

Drill holes through the patella should be as small as possible preferably not exceeding 3.5mm. These drill holes should be made through the medial rim of the patella. Transverse drill holes through the patella should be avoided to prevent the possibility of transverse fractures through the patella.

Prominence of the reconstructed graft or fixation material over the medial condyle will lead to localized tenderness over this area which can be annoying to the patient and is easily avoided by using non prominent fixation devices.

There seems to be no progression in P-F degeneration in follow up periods of 7 to 12 years. However, patella degeneration at the time of the MPFL reconstruction will have a negative effect on the functional result.

LITERATURE

[1] BARNETT A, PRENTICE M, MANDALIA V. The Patellotrochlear Index: A More Clinically Relevant Measurent of Patella Height? *JBJS (2009) (B) 91-B 413.*

[2] BECK P, BROWN NA, GREIS PE. *et al*. Patellofemoral Contact Pressures and Lateral Patellar Translation After Medial Patellofemoral Ligament Reconstruction. *American Journal of Sports Medicine (2007) 35(9) 1557-63.*

[3] BERNAGEAU J, GOUTALLIER D. Exam radiologique de l'articulation fémorale-patellaire. L'actualité rhumatologique. *Paris Expansion Scientifique Francaise (1984).*

[4] BIEDERT R, ALBRECHT S. The Patellotrochlear Index: a New Index for Assessing Patellar Height. *Knee Surg Sports Traumatol Arthrosc (2006) 14: 707-12.*

[5] CHRISTIANSEN SE, JACOBSEN BW, LUND B. *et al.* Reconstruction of the medial patellofemoral ligament with gracilis tendon outograft in transverse patellar drill holes. *Arthroscopy (2008) 24 (1) 82-7.*

[6] DEIE M, OCHI Y, SUMEN M. *et al.* Reconstruction of the medial patellofemoral ligament for the treatment of habitual or recurrent dislocation of the patella in children. *JBJS 85B (2003) (6)887-90.*

[7] DREZ D, EDWARDS TB, WILLIAMS CS. Results of medial patellofemoral ligament reconstruction in the treatment of patella dislocation. *Arthroscopy (2001) 17: 298-306.*

[8] ELIAS JJ, COSGAREA AJ. Technical errors during MPFL reconstruction could overload the medial Patello Femoral Cartilage. *Am J Sports Med 34(9) (2006) 1478-85.*

[9] ERASMUS PJ. Influence of patella height on the results of MPFL reconstruction. *ISAKOS Florence Italy (2007).*

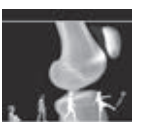
[10] ERASMUS PJ. Long term follow-up of MPFL reconstruction. *American Orthopedic Society for Sport Medicine (AOSS) Washington (2005).*

[12] ERASMUS PJ. Reconstruction of the medial patellofemoral ligament in recurrent dislocation of the patella. (ISAKOS Buenos Aires May 1997). *Arthroscopy (1998) 14:S42 (suppl, abstr).*

[13] FITHIAN DC, GUPTA N. Patellar instability: principles of soft tissue repair and reconstruction. *Tech Knee Surg (2006) 5:19-26.*

[14] GOMES EJ, MARCZYK LS, DE CESAR PC. *et al.* Medial patellofemoral ligament reconstructionwith semitendinosus autograft for chronic patellar instability: follow-upstudy. *Arthroscopy (2004) 20: 147-51.*

[15] HEEGAARD J, LEYVRAZ PF, VAN KAMPEN A. *et al.* Influence of soft tissue structure on patella three dimensional tracking. *Clinical orthopedics and related research (1996) 299, 235-43.*



[16] HVID I, ANDERSEN L, SCHMIDT H. Patellar Height and Femoral Trochlea Development. *Acta Orthop. Scand (1983) 54, 91-3.*

[17] MIKASHIMA Y, KIMURA M, KOMAYASHI Y. *et al.* Clinical results of isolated reconstruction of the medial patellofemoral ligament for recurrent dislocation and subluxation of the patella. *Acta Orthop Belg., (2006) 72, 65-71.*

[18] MOUNTNEY J, SENAVONGSE W, AMIS AA. *et al.* Tensile strength of the medial patellofemoral ligament before and after repair or reconstruction. *JBJS (B) 87(1): (2005) 36-40.*

[19] NOMURA E, INOUE M. Hybrid medial patellofemoral ligament reconstruction using semitendinosis tendon for recurrent patellar dislocation: minimum 3 years follow up. *Arthroscopy (2006) 22: 787-93.*

[20] NOMURA E, MOTOYASU I, KOBAYASHI S. Long-term Follow-up and Knee Osteoarthritis Change After Medial Patellofemoral Ligament Reconstruction for Recurrent Patellar Dislocation. *Am J Sports Med (2007) 35 (11)1851-8.*

[21] NEYRET P, ROBINSON AH, LE COULTRE B. *et al.* Patella tendon length – the factor in patella instability? *Knee 9 (2002) (1) 3-611.*

[22] PARKER DA, ALEXANDER JW, CONDITT MA. *et al.* Comparison of isometric and anatomic reconstruction of the medial patellofemoral ligament: a cadaveric study. *Orthopedics (2008) 31(4) 339-43.*

[23] SILLANPÄÄ PJ, MÄEMPÄÄ H, MATILLA W. *et al.* A mini-invasive adductor magnus tendon transfer technique for medial patellofemoral ligament reconstruction:a technical note. *Knee Surg Sports Traumatol Arthrosc (2009) 17: 508-12.*

[24] SCHÖTTLE PB, FUCENTESE SF, ROMERO J. Clinical and radiological outcome of medial paytellofemoral ligament reconstruction with a semitendinosis outograft for patella instability. *Knee Surg Sports Traumatol Artrosc (2005) 13, 516-21.*

[25] SMIRK C, MORRIS H. The anatomy and reconstruction of the medial patellofemoral ligament. *Knee (2003) 10, 221-7.*

[26] SMITH TO, WALKER J, RUSSEL N. Outcomes of medial patellofemoral ligament reconstruction for patellar instability: a systemic review. *Knee Surg Traumatol Arthrosc (2007) 15: 1301-14.*

[27] STEENSEN RN, DOPIRAK RM, MCDONALD WG. The anatomy and Isometry of the Medial Patello Femoral Joint. *Am J Sport Med (2004) 32(6) 1509-13.*

[28] STEINER TM, TORGA-SPAK R, TEITGE RA. Medial patellofemoral ligament reconstruction in patients with lateral patella instability and trochlear dysplasia. *Am J Sport Med 34, (2006), 1254-61.*

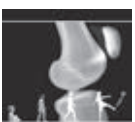
[29] THAUNAT M, ERASMUS P. The favourable anisometry: An original concept for medial patellofemoral ligament reconstruction. *Knee 9 (2007) (1) 3-6.*

[30] THAUNAT M, ERASMUS PJ. Recurrent patella dislocation after medial patellofemoral recomstruction. *Knee Surgery, Sport Traumatology Arthroscopy (2008) 16(1) 40-3.*

[31] THAUNAT M, ERASMUS PJ. Management of overtight medial patellofemoral ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc (2009) 17: 480-3.*

[32] TORITSUKA Y, HORIBE S, HIRO-OKA A. *et al.* Medial marginal fracture of the patella following patellar dislocation. *Knee. (2007) 14(6): 429-33.*

[33] VICTOR J, WONG P, WITVROUW E, SLOTEN JV. *et al.* How isometric are the medial patellofemoral, superficial medial collateral and lateral collateral ligaments of the knee? *Am J Sports Med 37 N° 10, (2009) 2028-36.*



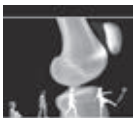
THE HISTORY OF THE TROCHLEAR DYSPLASIA IN PATELLA DISLOCATION

D. DEJOUR, P.G. NTAGIOPOULOS

Patellofemoral disorders are a transversal link in knee pathologies. They present with a wide range of symptoms from pain and feeling of instability to documented episodes of true patellar dislocation. This special pathology gives symptoms from childhood and adolescence with a high-demand in sports activities to the degenerative arthritic knee. One of the least understood pathologies in an otherwise thoroughly studied knee joint is patellar dislocation. Patients with recurrent patellar dislocation experience a disabling everyday life, are predisposed to future patellofemoral arthritis and the causes of dislocation and arthritis seem to overlap [1-3]. A huge volume of surgical and bibliographic interest on the treatment of this disease has enriched the orthopedic literature. In the last decades the surgeons' arsenal gained new techniques on both soft-tissue and bony surgeries. But even after many different operations on these patients, there is still a small subgroup of this population with near-to-normal knee kinematics or even unsatisfactory post-operative results [3]. This is probably attributed to the multifactorial cause of patellar dislocation and the inability to provide to all of these patients a definitive treatment with the present techniques [1, 3].

The causes of patellar dislocation include bony abnormalities, soft-tissue trauma or even anatomical abnormalities and impaired function of the surrounding musculature [1, 3, 4]. Although patellar dislocation leads to deficiency of the medial patellofemoral ligament (MPFL), the medial patellomeniscal ligament (MPML) and the medial patellotibial ligament (MPTL) [5], whose chief role in patellar stability is clear [3, 6, 7], the principal causes of dislocation are osseous anomalies [4]. Their importance lies on the fact that patellar dislocation has been associated with threshold values of their normal anatomy, and therefore the amount for their surgical correction has been identified [4, 8]. MPFL rupture or elongation is the consequence of patellar dislocation, while the basic causes of patellar dislocation are anatomic anomalies such as trochlea dysplasia, patella alta and extensor mechanism malalignment [3, 4, 8-11]. All of them have to be well identified in order to correct the primitive anatomic anomalies and to repair the *consequence* of patellar dislocation.

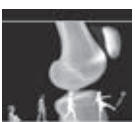
Regardless of the age of onset or the pattern (permanent, habitual or recurrent) of patellar dislocation, four main anatomic anomalies with statistical thresholds have been identified in this population since 1987 [12]: trochlear dysplasia, patella alta, excessive trochlear groove-tibial tuberosity distance (TT-TG) and excessive lateral patellar tilt are the underlying key factors predisposing to instability [4].



Trochlear dysplasia (roots from the Greek words *"dys-"*: mal- and *"plasis":* creation [13]) is a developmental condition in which the femoral trochlea lacks of its normal and sophisticated concave anatomy, that is absolutely necessary to engage the patella, and instead, it becomes shallow, flat or even convex. According to A. Amis [14], who published the first biomechanical results on patellar instability in trochlear dysplasia and the efficacy of trochleoplasty procedures, it is recognized that the mediolateral flattening of the anterior surface of the trochlear facets results predominantly from an *excess of bone centrally in the groove*. In its major pattern it forms a supratrochlear prominence or "bump" anterior to the shaft of the femur, which the patella has to override when the knee starts to flex in order to engage in the groove for the remaining degrees of flexion. Amis *et al.* showed that simulated trochlear dysplasia led to significant reduction in lateral stability and that by re-creating a deep trochlear groove with a deepening trochleoplasty procedure, lateral stability increased significantly similarly to the intact knee [14].

The genetic and primitive origin of trochlear dysplasia and its familiar occurrence have been shown by C. Tardieu and J.L. Jouve. There is evidence that the asymmetrical trochlear shape in adults exists in the foetus since the third trimester of pregnancy, something that could prove the genetic roots of trochlear dysplasia [15, 16]. The shape of the articular trochlea is variable in mammals depending on their type of locomotion: unguligrade, digitigrade or plantigrade [17]. The asymmetrical ingression of the patella into the normal trochlea is a characteristic of the modern man. Christine Tardieu's extensive anthropomorphometric studies on this field have showed that the femoral valgus angle, the femoral bicondylar angle and the morphology of the normal trochlea and its articulation with the patella are not present in prime mammals or non-walking children and are the result of human erect stance and bipedalism [17-19]. These anatomic characteristics of the trochlea could have been integrated into the genome during the course of evolution [17, 20]. According to Tardieu, the oblique angle of the femur is the major feature, which initiated the later modifications of the patellofemoral joint that over 3 million years before, were never inscribed in the human genome. The elevated lateral femoral facet and the deep trochlear groove are features that *"were first acquired, then once selected, genetically assimilated, and now appear on the foetal cartilaginous epiphysis"* [17].

It is surprising that the origin of the study on the abnormal trochlear shape in patellar dislocation starts more than 200 years ago in Europe. Although the term 'dysplasia' was not originally used, the earliest reference on this condition can be attributed to Richerand in 1802. According to Isermeyer [21], Richerand made the very first description of the abnormal morphology of the trochlear groove and the lateral femoral facet in paediatric patients with patellar dislocation. The interest on the abnormal trochlear shape in (mainly) congenital patellar dislocation is also documented in the works of B. Pollard in 1891 [22] and D. Drew in 1908 [23], who focused on the effects of a *"possibly congenital"* reduced lateral facet height on improper patellar engagement and lateral patellar dislocation, and even attempted to surgically create a new groove for the patella. According to S. Donell and C. Hing [24], in 1914, J. Murphy also considered the shallow trochlear groove as a cause for patellar dislocation, and he performed a similar surgical correction by burring the groove and adding fat tissue between the patella and the femur to reduce scarring and adhesions by the exposed cancellous bone [25]. The next year, in 1915, Fred H. Albee from the *"New York Postgraduate Medical School Clinic"*, was the first who tried to surgically correct trochlear dysplasia properly with his pioneer procedure: the lateral facet elevating trochleoplasty (fig. 1) [26]. This was a completely opposite concept and it included adding a bone graft under the lateral facet in order to augment the required mechanical block for a pathological lateral patellar translation. This procedure gained, for a period of time, overseas attraction mostly in the U.K. and less in France.



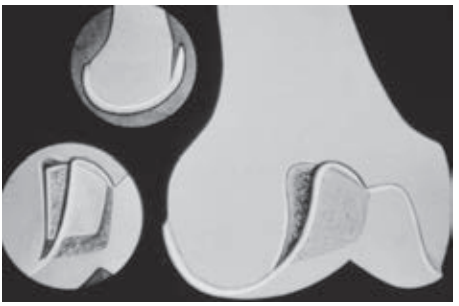


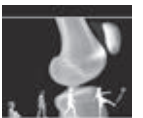
Fig. 1: The lateral facet elevating trochleoplasty proposed by Albee.

It took almost 50 years, for a more meticulous study on the anatomic characteristics of the patients with instability by H. Brattström, a paediatric orthopaedist from Österliden in Sweden with a strong interest in rheumatologic conditions, who focused on the reduced height of the lateral trochlear facet in children. He was the first to publish, in 1964, a study on trochlear dysplasia on axial knee X-rays at 30° of flexion and described it as a flattening of the trochlear groove [27]. This was the first large-scale study on the anatomy of the trochlea, the depth of the groove and the height of the lateral facet was described in patients with patellar dislocation, but without notion of pathologic threshold value. He discovered three types of trochlear dysplasia: ***1)*** hypoplasia of the medial femoral condyle which is the most frequent type, ***2)*** aplasia of the medial condyle and ***3)*** total dysplasia of both condyles with a flat or convex distal femoral trochlea. At that time, there was a growing interest in many European centers, where surgeons believed that the flattened trochlear groove was a major factor in patellar dislocation [10]. Then, a more drastic concept of surgically correcting the dysplasia was proposed by a french surgeon from Paris Y. Masse in 1978, who focused on a technique to remodel the trochlea without injuring its cartilage [28] in which the goal was to remove the posterior part of the trochlea and then push it posteriorly with an impactor, but with no consideration on the medial or lateral facet.

It wasn't until 1985, that the Belgians radiologists B. Maldague and J. Malghem focused on the study of trochlea *"and its dysplasias"* in lateral X-rays [29, 30]. They described an anomaly that could be either generalized or focal to the proximal trochlea and they correlated an increased sulcus angle of more than 145° with trochlear dysplasia. They measured the proximal trochlear depth 1cm below the upper limit of the trochlear groove in lateral X-rays and found that it was on average 2.7 mm in patients with patellar dislocation, in contrast to 5.9 mm in the asymptomatic group.

Until that time, trochlear dysplasia was a pathologic radiographic finding in patients with patellar dislocation. Although how strongly trochlear dysplasia was correlated in the clinical setting with patellar dislocation not only to the paediatric population was not yet documented, the interest on trochlear dysplasia was emerging in the university hospital of Lyon during the 1980's with a series of publications by Henri Dejour and Gilles Walch, who presented some of the most essential and classic data on patellar instability (fig. 2). They were the first to link the occurrence of trochlear dysplasia in patients with patellar dislocation and in asymptomatic controls. They used their 1984 classification of

Fig. 2: Henri Dejour (left) and Gilles Walch pioneers in the field of patellofemoral disorders during the 1980's.



permanent, habitual, and recurrent patellar dislocation, in which they started performing X-ray measurement of the trochlea and its position related to the anterior femoral cortex. It is worth mentioning that at the time, surgeons in Lyon were already using true lateral X-rays for a different method developed for measuring anterior tibial translation in ACL ruptures. They introduced the "crossing sign", which became the pathognomonic sign for trochlear dysplasia on lateral X-rays (fig. 3).

This fundamental work on the definition of trochlear dysplasia and on its consistent presence in patients with patellar dislocation was presented and published in 1987 during the 6th edition of the meeting *"Les Journées Lyonnaises de Chirurgie du Genou"* [12] and was later issued in 1990 [31]. It was the result of a huge effort from the whole "Lyon's School" of surgeons who studied the radiographic findings in more than 1,800 patients and presented their standardized approach on patellofemoral instability that would be quoted and followed widely in the future. H. Dejour and G. Walch introduced the definition of dysplasia as a shallow, flat or even convex trochlear groove by using some of the radiographic methods of Raguet [32], Maldague and Malghem [29, 30] and by examining true lateral X-rays where the posterior femoral condyles were *superimposed* [8]. They recorded that trochlear dysplasia was present in 96% of patients with documented patellar dislocation and only in 3% of a control population, thus presenting dysplasia as the *most consistent anatomic abnormality underlying in patients with patellofemoral instability*. Yet, the most important attribution of this classic study was the introduction of the first *quantitative and qualitative* criteria of trochlear dysplasia. H. Dejour and G. Walch described the crossing sign as a simple yet fundamental qualitative factor of trochlear dysplasia. It represented the point where the line of the trochlear floor intersects the anterior contour of the lateral femoral condyle and the level where the trochlea is flat. The crossing sign could have three different appearances in reference to the level of the intersection (proximally or distally) and the symmetry or not of the medial and lateral femoral condyles (fig. 4). With these data, the first classification of trochlear dysplasia was introduced, which was focused on how proximally or distally (on the flexion-extension arc of the knee) the trochlea becomes dysplastic: *Type I* corresponds to minor dysplasia where the trochlea is flat at only one point in its superior (proximal) portion and the condyles are symmetrical, *Type II* has separate

Fig. 3: (Original slide) The introduction of the 'crossing sign' as pathognomonic sign and qualitative factor for trochlear dysplasia.

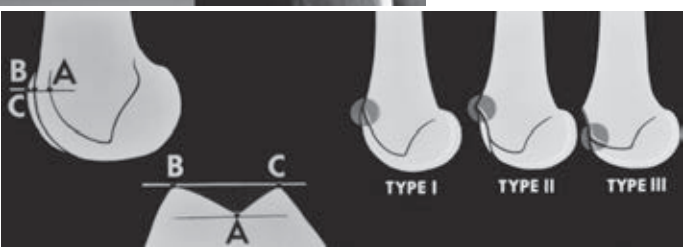
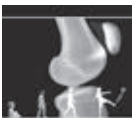


Fig. 4: The crossing sign had 3 different types in reference to the level of the intersection (proximally or distally) and the symmetry or not of the medial and lateral femoral condyles.



crossings of the two condyles with the floor of the trochlea and the condyles are asymmetrical, *Type III* is a severe form of trochlear dysplasia where the condyles are symmetrical and the crossing is situated low (distally) in the groove (fig. 4).

H. Dejour and G. Walch also introduced two quantitative criteria for trochlear dysplasia that they were not integrated in this first classification. The "trochlear bump" or prominence represents the elevated trochlear floor from the anterior distal femoral cortex proximally to the crossing sign, and the "trochlear depth" that measures the depth of the trochlear floor from the condyles distally to the crossing sign (fig. 5). A trochlear depth of 4mm or less was found to be pathological [8]. The importance of this radiographic analysis was that for the first time trochlear dysplasia was related to the reduced height of the lateral facet (trochlear depth), the elevated trochlear floor (prominence) or both. The impact of the work of this group of surgeons was so great that for many years, French surgeons were treating patellofemoral instability based on these results and consider patellar dislocation as a multifactorial entity of imaging findings surrounding trochlear dysplasia [10], a concept that initially attracted a lot of interest and was sometimes a field for overseas criticism. The conclusions from these new findings about trochlear dysplasia and the trochlear prominence led Henri Dejour to propose a new surgery for the treatment of high-grade trochlear dysplasia. He described a deepening trochleoplasty where the bump was removed and the groove was recreated by doing a osteotomy in the native groove and then by fixing it with two screws medially and laterally leading to compression of both facets [8, 31, 33] (fig. 6) [28].

In 1994, the Swiss surgeon H. Bereiter recommended a third operation for the treatment of trochlear dysplasia [34]. This was known as the "Bereiter" procedure [35]. In 2002, D. Goutallier performed the fourth type of trochleoplasty procedure [36]. The first results of the "recession-wedge" trochleoplasty were published, and this technique was embraced by P. Beaufils in Paris [37]. In 2010, L. Blønd from Copenhagen presented his innovative technique of Bereiter's arthroscopic trochleoplasty [38].

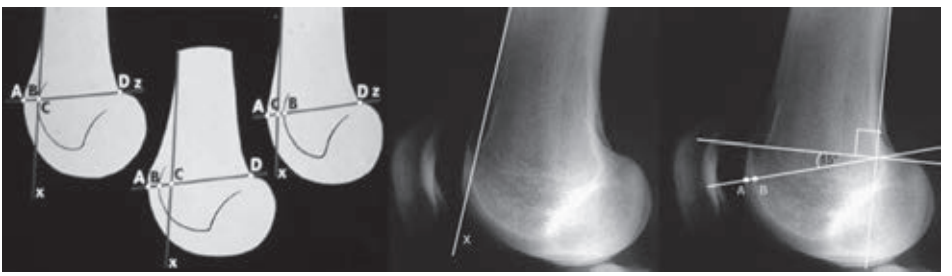


Fig. 5: (Original slide) The 'trochlear bump' or 'prominence' represents the elevated trochlear floor from the anterior distal femoral cortex (distance A-B, left) proximally to the crossing sign, and the 'trochlear depth' (distance B-C, left) represents the depth of the trochlear floor from the condyles distally to the crossing sign.

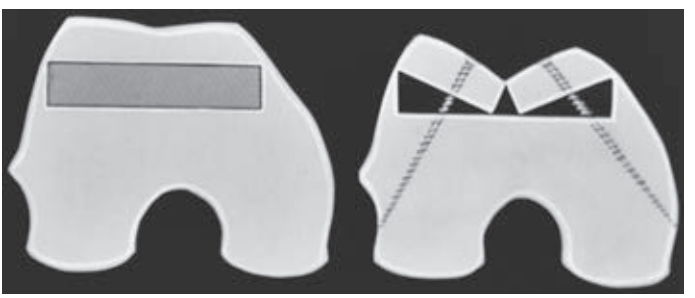
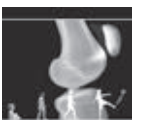


Fig. 6: (Original slide) The deepening trochleoplasty proposed by H. Dejour.



In 1998, Franck Remy and François Gougeon published a study showing that the first 1987 classification of H. Dejour and G. Walch had poor inter- and intra- observer reliability especially for the type II. It was probably due to the fact that the prominence or bump and the shape of the facet and their asymmetry were not taken into account in the three-type classification [39, 40]. At that time they observed the presence of the trochlear bump and measured the trochlear depth in the available X-rays, but did not correlate them to slice imaging (CT scan) (fig. 7). It was a three-dimensional anomaly with a two-dimensional imaging based only on X-rays. In 1998, David Dejour and Bertrand Lecoultre did a study on 177 objective patellar dislocations comparing the true sagittal view, the axial view and the CT scan (fig. 8) [41]. Two more signs were added to the definition of trochlear dysplasia: the "supratrochlear spur" and the 'double contour sign'. The supratrochlear spur is the prominence of the whole trochlea. It starts often proximal to the cartilaginous part of the trochlea on the lateral side (fig. 9). The double contour sign is the osteochondral projection on the sagittal view of the *hypoplastic medial facet* (fig. 8). Using this three signs, they were able to publish in 1998 a four grade classification which will become in the future the most widely used classification of trochlear dysplasia until now [4, 41-43] (fig. 10): ***Type A*** involves mild dysplasia where the trochlea *is shallower than normal* with the characteristic crossing sign.

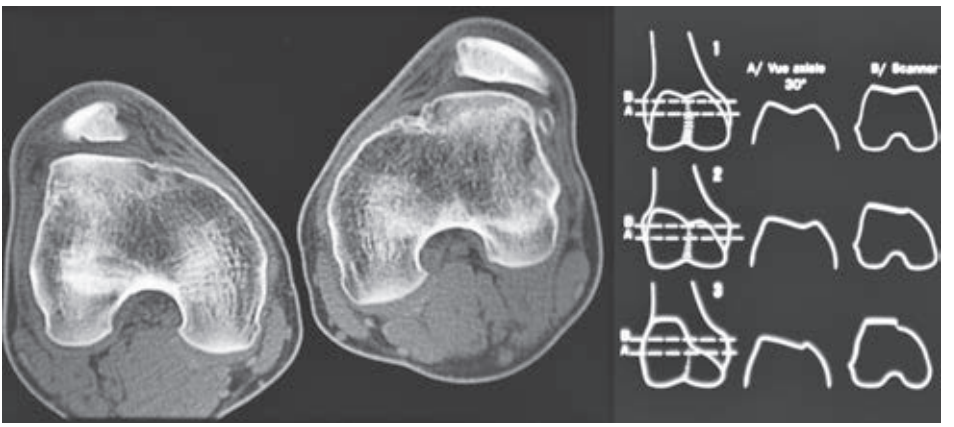


Fig. 7: (Original slide) The identification of trochlear bump and the measurement the trochlear depth in X-rays that was not correlated to slice imaging at that time (left).

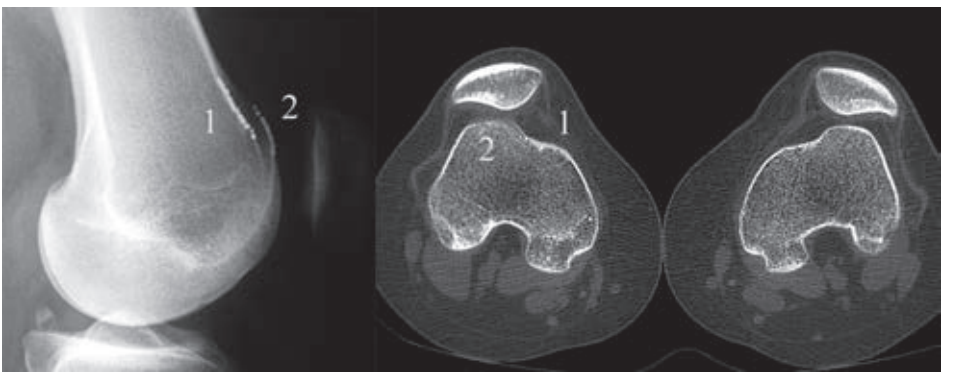
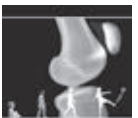


Fig. 8: The correlation of true lateral X-rays and slice imaging by D. Dejour and B. Lecoultre revealed the presence of (1) the medial hypoplastic facet and the 'double contour sign', and (2) the supratrochlear spur.



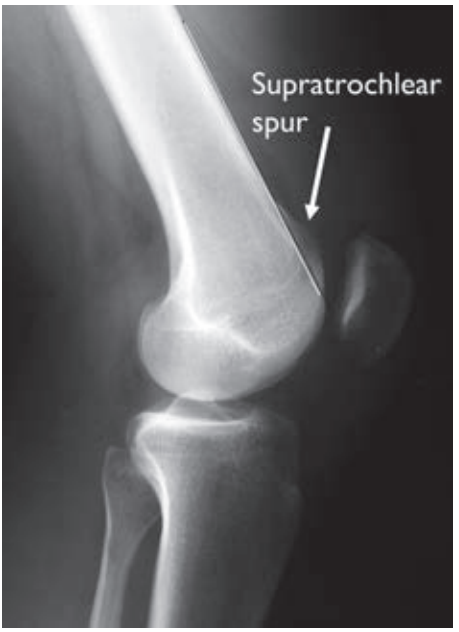


Fig. 9: The supratrochlear spur on a true lateral X-ray represents the prominence of the whole trochlea.

Type B is pathologic for a flat trochlea with a prominence and the additional *supratrochlear spur*. In ***Type C***, the trochlea is convex and has the "crossing sign" and the "double contour sign" on sagittal X-rays. In the most severe ***Type D***, the trochlear groove is elevated above the anterior femoral cortex; it has a hypoplastic medial facet and a "cliff-pattern" appearance with all three previous signs on the lateral X-rays. This new classification scheme was more reproducible and had a high observer agreement [44, 45].

The interest on studying the morphology of trochlear dysplasia on axial views was also strong in a study by R. Biedert, who analyzed the decreased trochlear depth caused by either an elevated trochlea floor or a flattened lateral/medial condylar height on MRI [46]. He compared the height of the medial, central and lateral third of the trochlea according to the width of the lateral condyle and he discovered that a reduced height of the lateral condyle more than 77% was pathologic and that in more

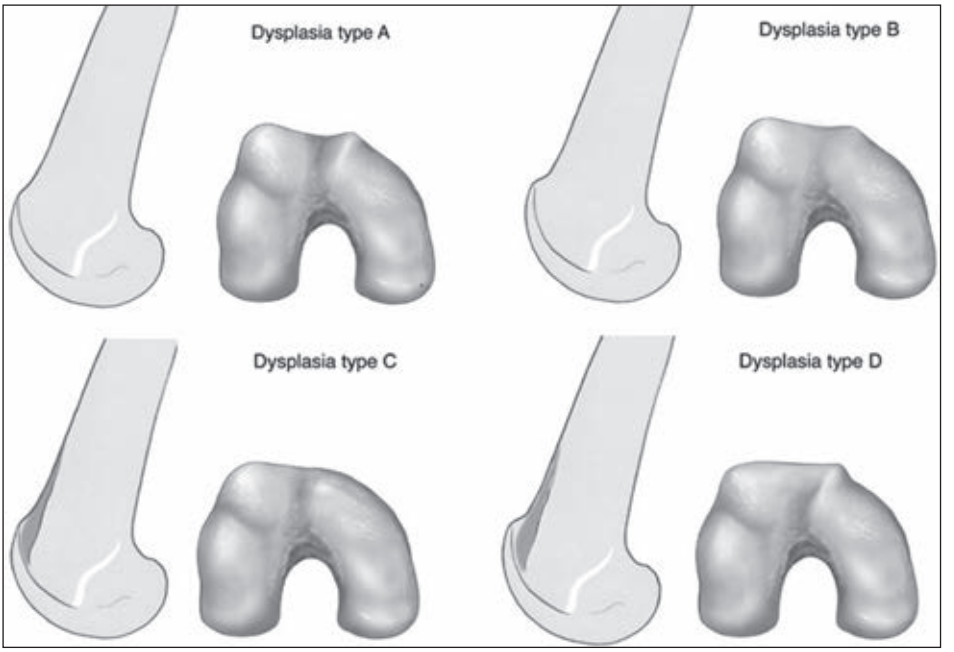
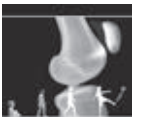


Fig. 10: The four-type classification for trochlear dysplasia proposed by D. Dejour.



than 80% of patients with patellar dislocation the pathology is located in the middle (elevated groove) and medial (hypoplastic facet) third [46]. These patients would benefit from a deepening trochleoplasty procedure [46]. Biedert *et al.* also studied trochlear dysplasia with the use of convetional X-rays, axial CT and MRI and presented another type of trochlear dysplasia that could not be included in the Dejour classification and where none of the previously described radiological findings existed: the "too short lateral articular trochlea" [47]. They described the lateral condyle index for assessing the length of the lateral trochlea and defining the patients suffering from a short trochlea who could be good candidate for elevating trochleoplasty.

Finally, in 2010, D. Dejour presented the modification of the "Lyon's School" sulcus-deepening trochleoplasty with the combination of soft-tissue procedures for the treatment of recurrent patellar dislocation in patients with underlying high-grade trochlear dysplasia (Type B and D) [48]. The rationale of this surgical procedure is to restore the normal anatomy and to re-shape the trochlea by undermining the cancellous bone and deepening the groove, to decrease the sulcus angle and additionally, to perform a 'proximal' re-alignement procedure by lateralizing the trochlear groove (fig. 11) [48].

Although the origins of trochlear dysplasia are scarce and reported up to two centuries ago, its future study seems very promising. Currently, there is ample interest on the diagnosis and treatment of trochlear dysplasia. There is also observer agreement on the classification of trochlear dysplasia [44, 45]. Its contribution to future degenerative joint disease is another reason for the introduction of various techniques to surgically correct it [2, 49]. Trochleoplasty procedures are more and more widely performed as a primary or revision option in selected patients with recurrent patellar dislocation and underlying trochlear dysplasia. The recognition of the importance of trochlear dysplasia in the aetiology of patellar dislocation is growing and has been embraced by surgeons in Europe but also, in the United States [2, 9, 10], in the U.K. [1, 24] and in Japan [50, 51]. There are convincing data that there is a subgroup of patients with recurrent patellar dislocation and underlying high-grade trochlear dysplasia, in which the *"benign neglect"* of the latter and the application of traditional surgery is ill-fated [9, 35-37, 52, 53]. The sound biomechanical evidence from the surgical treatment of trochlear dysplasia [14] and the satisfactory clinical results published by Von Knoch [35], Verdonk [54], Donell [24], Blønd [38] and Schöttle [55], Goutallier [36], Fucentese [56], Thaunat [52], Beaufils [37], Dejour [57] and others, confirm that trochlear dysplasia is a distinctive pathology in the aetiology of patellar dislocation, which must not be ignored or under-diagnosed, and that its treatment should be in the armamentarium of knee surgeons.

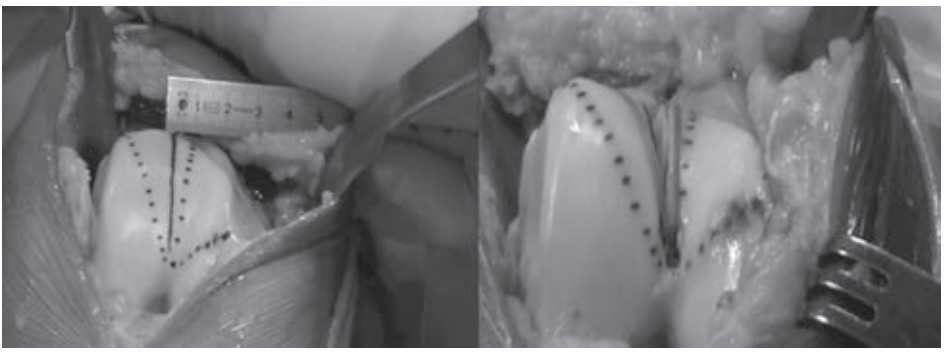


Fig. 11: The sulcus-deepening trochleoplasty removes cancellous bone and deepens the groove, decreases the sulcus angle and performs a 'proximal' re-alignement procedure by lateralizing the trochlear groove.

LITERATURE

[1] MULFORD JS, WAKELEY CJ, ELDRIDGE JD. Assessment and management of chronic patellofemoral instability. *J Bone Joint Surg Br 2007; 89: 709-16.*

[2] ARENDT E. Anatomy and malalignment of the patellofemoral joint: its relation to patellofemoral arthrosis. *Clin Orthop Relat Res 2005: 71-5.*

[3] ARENDT EA, FITHIAN DC, COHEN E. Current concepts of lateral patella dislocation. *Clin Sports Med 2002; 21: 499-519.*

[4] DEJOUR D, LE COULTRE B. Osteotomies in patello-femoral instabilities. *Sports Med Arthrosc 2007; 15: 39-46.*

[5] PHILIPPOT R, BOYER B, TESTA R, FARIZON F, MOYEN B. The role of the medial ligamentous structures on patellar tracking during knee flexion. *Knee Surg Sports Traumatol Arthrosc 2012; 20: 331-6.*

[6] AMIS AA, FIRER P, MOUNTNEY J, SENAVONGSE W, THOMAS NP. Anatomy and biomechanics of the medial patellofemoral ligament. *Knee 2003; 10: 215-20.*

[7] BICOS J, FULKERSON JP, AMIS A. Current concepts review: the medial patellofemoral ligament. *Am J Sports Med 2007; 35: 484-92.*

[8] DEJOUR H, WALCH G, NOVE-JOSSERAND L, GUIER C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc 1994; 2: 19-26.*

[9] BOLLIER M, FULKERSON JP. The role of trochlear dysplasia in patellofemoral instability. *J Am Acad Orthop Surg 2011; 19: 8-16.*

[10] FITHIAN DC, NEYRET P, SERVIEN E. Patellar instability: the Lyon experience. *Current Orthopaedic Practice 2008; 19: 328-38.*

[11] FITHIAN DC, PAXTON EW, COHEN AB. Indications in the treatment of patellar instability. *J Knee Surg 2004; 17: 47-56.*

[12] DEJOUR H, WALCH G. Morphologic factors in patellar instability: clinical, radiologic, and tomographic data. *In: "Journées Lyonaise de Chirurgie du Genou", Lyon, 1987. pp 25-35.*

[13] "Dysplasia" Dorland's Medical Dictionary. *Elsevier (2012).*

[14] AMIS AA, OGUZ C, BULL AM, SENAVONGSE W, DEJOUR D. The effect of trochleoplasty on patellar stability and kinematics: a biomechanical study *in vitro*. *J Bone Joint Surg Br 2008; 90: 864-9.*

[15] GLARD Y, JOUVE JL, GARRON E, ADALIAN P, TARDIEU C, BOLLINI G. Anatomic study of femoral patellar groove in fetus. *J Pediatr Orthop 2005; 25: 305-8.*

[16] GARRON E, JOUVE JL, TARDIEU C, PANUEL M, DUTOUR O, BOLLINI G. Anatomic study of the anterior patellar groove in the fetal period. *Rev Chir Orthop Reparatrice Appar Mot 2003; 89: 407-12.*

[17] TARDIEU C, DUPONT JY. The origin of femoral trochlear dysplasia: comparative anatomy, evolution, and growth of the patellofemoral joint. *Rev Chir Orthop Reparatrice Appar Mot 2001; 87: 373-83.*

[18] SHEFELBINE SJ, TARDIEU C, CARTER DR. Development of the femoral bicondylar angle in hominid bipedalism. *Bone 2002; 30: 765-70.*

[19] TARDIEU C. Morphogenesis of the femoral diaphysis in humans: significance of function and evolution. *Folia Primatol (Basel) 1994;63: 53-8.*

[20] TARDIEU C, GLARD Y, GARRON E, BOULAY C, JOUVE JL, DUTOUR O *et al.* Relationship between formation of the femoral bicondylar angle and trochlear shape: independence of diaphyseal and epiphyseal growth. *Am J Phys Anthropol 2006;130: 491-500.*

[21] ISERMEYER H. Über die pathologische Luxation der Patella. *Arch Klin Chir 1967: 1-23.*

[22] POLLARD B. Old dislocation of patella reduced by intra articular operation. *Lancet 1891: 988.*

[23] DREW D. Dislocation of the Patella (? Congenital); Operation; Cure. *Proc R Soc Med 1908;1: 11-3.*

[24] DONELL ST, JOSEPH G, HING CB, MARSHALL TJ. Modified Dejour trochleoplasty for severe dysplasia: operative technique and early clinical results. *Knee 2006; 13: 266-73.*

[25] MURPHY JB Congenital luxation of the patella. In: Clinics of John B. Murphy, vol. 1. *WB Saunders, pp 817-38, (1914).*

[26] ALBEE FH. Bone graft wedge in the treatment of habitual dislocation of the patella. *Med Record 1915; 88: 257.*

[27] BRATTSTROEM H. Shape of the Intercondylar Groove Normally and in Recurrent Dislocation of Patella. A Clinical and X-Ray-Anatomical Investigation. *Acta Orthop Scand Suppl 1964; 68: Suppl/ 68:1-148.*

[28] MASSE Y. Trochleoplasty. Restoration of the inter-condylar groove in subluxations and dislocations of the patella. *Rev Chir Orthop Reparatrice Appar Mot 1978; 64: 3-17.*

[29] MALGHEM J, MALDAGUE B. Depth insufficiency of the proximal trochlear groove on lateral radiographs of the knee: relation to patellar dislocation. *Radiology 1989; 170: 507-10.*

[30] MALDAGUE B, MALGHEM J. Significance of the radiograph of the knee profile in the detection of patellar instability. Preliminary report. *Rev Chir Orthop Reparatrice Appar Mot 1985;71 Suppl 2: 5-13.*

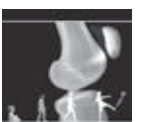
[31] DEJOUR H, WALCH G, NEYRET P, ADELEINE P. Dysplasia of the femoral trochlea. *Rev Chir Orthop Reparatrice Appar Mot 1990; 76: 45-54.*

[32] RAGUET M. Mesure radiolgique de la hauteur trochléenne. *J Traumatol Sport 1989: 210-3.*

[33] WALCH G, DEJOUR H. Radiology in femoro-patellar pathology. *Acta Orthop Belg 1989;55: 371-80.*

[34] BEREITER H, GAUTIER E. Die trochleaplastik als chirurgische Therapie der rezidivierenden Patellaluxation bei Trochleadysplasie des Femurs. *Arthroskopie 1994; 7.*

[35] VON KNOCH F, BOHM T, BURGI ML, VON KNOCH M, BEREITER H. Trochleaplasty for recurrent patellar dislocation in association with trochlear dysplasia. A 4- to



14-year follow-up study. *J Bone Joint Surg Br 2006; 88: 1331-5.*

[36] GOUTALLIER D, RAOU D, VAN DRIESSCHE S. Retro-trochlear wedge reduction trochleoplasty for the treatment of painful patella syndrome with protruding trochleae. Technical note and early results. *Rev Chir Orthop Reparatrice Appar Mot 2002; 88: 678-85.*

[37] BEAUFILS P, THAUNAT M, PUJOL N, SCHEFFLER S, ROSSI R, CARMONT M. Trochleoplasty in major trochlear dysplasia: current concepts. *Sports Med Arthrosc Rehabil Ther Technol 2012; 4:7.*

[38] BLØND L, SCHÖTTLE PB. The arthroscopic deepening trochleoplasty. *Knee Surg Sports Traumatol Arthrosc 2010;18: 480-5.*

[39] REMY F, BESSON A, MIGAUD H, COTTEN A, GOUGEON F, DUQUENNOY A. Reproducibility of the radiographic analysis of dysplasia of the femoral trochlea. Intra- and interobserver analysis of 68 knees. *Rev Chir Orthop Reparatrice Appar Mot 1998; 84: 728-33.*

[40] REMY F, CHANTELOT C, FONTAINE C, DEMONDION X, MIGAUD H, GOUGEON F. Inter- and intraobserver reproducibility in radiographic diagnosis and classification of femoral trochlear dysplasia. *Surg Radiol Anat 1998; 20: 285-9.*

[41] DEJOUR D, REYNAUD P, LECOULTRE B. Douleurs et Instabilite Rotulienne. Essai de Classification. *Med Hyg 1998; 56: 1466-71.*

[42] TAVERNIER T, DEJOUR D. Knee imaging: what is the best modality. *J Radiol 2001; 82: 387-405; 7-8.*

[43] TECKLENBURG K, DEJOUR D, HOSER C, FINK C. Bony and cartilaginous anatomy of the patellofemoral joint. *Knee Surg Sports Traumatol Arthrosc 2006; 14: 235-40.*

[44] LIPPACHER S, DEJOUR D, ELSHARKAWI M, DORNACHER D, RING C, DREYHAUPT J *et al.* Observer agreement on the dejour trochlear dysplasia classification: a comparison of true lateral radiographs and axial magnetic resonance images. *Am J Sports Med 2012; 40: 837-43.*

[45] RÉMY F, GOUGEON F, ALA EDDINE T, MIGAUD H, FONTAINE C, DUQUENNOY A. Reproducibility of the new classification scheme of femoral trochlear dysplasia by Dejour: predictive value for severity of femoropatellar instability in 47 knees. *J Bone Joint Surg Br 2002; 84-B: 43.*

[46] BIEDERT RM, BACHMANN M. Anterior-posterior trochlear measurements of normal and dysplastic trochlea by axial magnetic resonance imaging. *Knee Surg Sports Traumatol Arthrosc 2009; 17: 1225-30.*

[47] BIEDERT RM, NETZER P, GAL I, SIGG A, TSCHOLL PM. The lateral condyle index: a new index for assessing the length of the lateral articular trochlea as predisposing factor for patellar instability. *Int Orthop 2011; 35: 1327-31.*

[48] DEJOUR D, SAGGIN P. The sulcus deepening trochleoplasty-the Lyon's procedure. *Int Orthop 2010; 34: 311-6.*

[49] MAENPAA H, LEHTO MU. Patellofemoral osteoarthritis after patellar dislocation. *Clin Orthop Relat Res 1997: 156-62.*

[50] KOBAYASHI A, OU A. Condyloplasty of lateral epicondyle of the femur for patellar instability. Seikei-saigaigeka. *Orthop Surg Traumatol 1994: 49-57.*

[51] HORIKAWA A, KODAMA H, MIYAKOSHI N, YAMADA S, MIYAMOTO S. Recurrent dislocation of the patella accompanying hypotrochlea of the femur and malalignment of the patella. *Ups J Med Sci 2011; 116: 285-8.*

[52] THAUNAT M, BESSIERE C, PUJOL N, BOISRENOULT P, BEAUFILS P. Recession wedge trochleoplasty as an additional procedure in the surgical treatment of patellar instability with major trochlear dysplasia: early results. *Orthop Traumatol Surg Res 2011; 97: 833-45.*

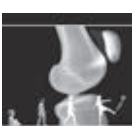
[53] UTTING MR, MULFORD JS, ELDRIDGE JD. A prospective evaluation of trochleoplasty for the treatment of patellofemoral dislocation and instability. *J Bone Joint Surg Br 2008; 90: 180-5.*

[54] VERDONK R, JANSEGERS E, STUYTS B. Trochleoplasty in dysplastic knee trochlea. *Knee Surg Sports Traumatol Arthrosc 2005; 13: 529-33.*

[55] SCHÖTTLE PB, SCHELL H, DUDA G, WEILER A. Cartilage viability after trochleoplasty. *Knee Surg Sports Traumatol Arthrosc 2007; 15: 161-7.*

[56] FUCENTESE SF, ZINGG PO, SCHMITT J, PFIRRMANN CW, MEYER DC, KOCH PP. Classification of trochlear dysplasia as predictor of clinical outcome after trochleoplasty. *Knee Surg Sports Traumatol Arthrosc 2011; 19: 1655-61.*

[57] DEJOUR D, BYN P, SAGGIN P Deepening Trochleoplasty for Patellar Instability. In: Zaffagnini S, Dejour D, Arendt E (eds) Patellofemoral Pain, Instability and Arhtritis, vol 1. *Springer, Berlin, Heidelberg, pp 225-32, (2010).*



INTRA- AND INTEROBSERVER AGREEMENT OF DEJOUR´S CLASSIFICATION OF TROCHLEAR DYSPLASIA

S. LIPPACHER, H. REICHEL, M. NELITZ

INTRODUCTION

Trochlear dysplasia is known to be an important cause for patellofemoral instability. Henri Dejour *et al.* [11] found that 96% of patients with a history of a true patellar dislocation had evidence of trochlear dysplasia.

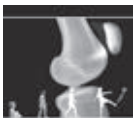
The lateral X-ray has always been a necessary part of the radiographic evaluation of the knee. Not only is it critical for assessing patellar height [2] but in 1989 Walch and H. Dejour [25] as well as 1992 Grelsamer and Tedder described the lateral trochlear sign [14] bringing to light its value in diagnosing trochlear dysplasia.

In addition to the lateral trochlear sign, which he later called crossing sign, H. Dejour described the supratrochlear bump or spur and the double contour as typical radiological signs of trochlear dysplasia [11].

H. Dejour *et al.* [10, 11] initially described three types of trochlear dysplasia on conventional lateral radiographs. Due to the lack of intra- and interobserver agreement, D. Dejour later proposed his classification of four types on two-dimensional radiographs and three-dimensional CT-scans (fig. 1) [8, 9].

Recently magnetic resonance imaging has become the diagnostic tool of choice to assess patellofemoral instability, because soft tissue injuries, potential flake fractures and the tibial tuberosity groove distance can also be evaluated [1, 7, 21, 22, 23]. On axial MRI trochlear dysplasia is diagnosed on the first craniocaudal image, where the complete cartilaginous trochlea can be seen.

Dejour's radiographic and axial CT classifications [11] are widely used in clinical practice and in the orthopaedic literature to assess the severity of trochlear dysplasia [6, 13, 19, 21]. The aim of this study was to assess the intra- and interobserver agreement of the radiographic and MRI based classification as described by D. Dejour.



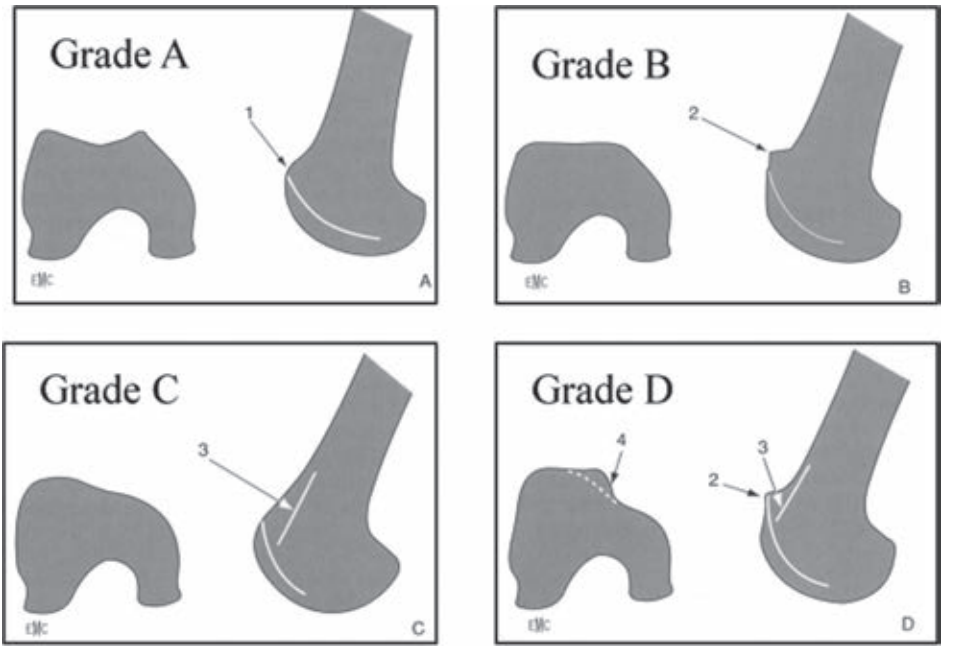


Fig. 1: Classification of trochlear dysplasia according to D. Dejour (reprinted with David Dejour's permission): 1 – crossing sign, 2 – supratrochlear bump or spur, 3 – double contour, 4 – vertical join or cliff pattern.

MATERIALS AND METHODS

Lateral radiographs and transverse MRI T2-weighted scans of fifty knees in fifty patients with patellar instability that were referred to our outpatient-clinic between 2007 and 2010 for subsequent surgery were analyzed. None of these patients had prior surgery of the knee. Patients with insufficient X-ray or MRI were excluded. In all included patients routine X-rays were taken to evaluate the patellofemoral joint. The lateral view was taken in the supine position and 0° knee flexion with superimposed condyles [17]. During clinical assessment a firm typing was made using the D. Dejour's classification.

This study compares the reliability and accuracy of two diagnostic studies in grading the severity of trochlear dysplasia.

The radiographic study (method 1) included a correct plain lateral view of the knee. According to D. Dejour [9] on lateral radiographs four different types of trochlear dysplasia were distinguished (fig. 1, 2).

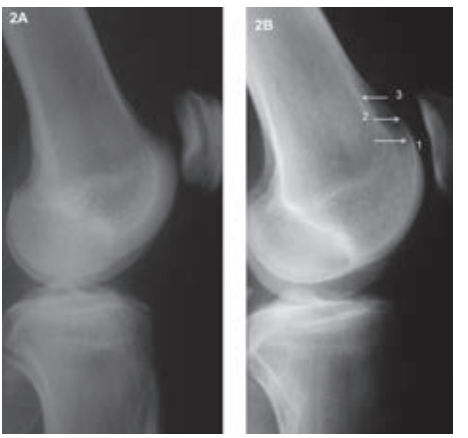


Fig. 2:
A: Normal patellofemoral joint.
B: trochlear dysplasia with crossing sign (1), small supratrochlear bump or spur (2), double contour

In method 2 the axial MRI of the proximal trochlea was used. Selected for this study was the most proximal image that included the entire width of the trochlea [9] (fig. 1).

The fifty lateral radiographs and the fifty MRI images were read by four different observers, two senior and two junior orthopaedic surgeons, who were blinded to the clinical history. The studies were read twice within a four week time span. For the second reading the studies were randomly arranged in a completely different order.

The results were first analyzed for interobserver agreement on the four classification types between the four readers. To see if there was better agreement if the dysplasia was divided into two groups instead of four, the interobserver agreement was then analysed when type A was compared to type B, C and D combined.

Statistical analysis

Contingency tables were used descriptively for intra- and interobserver agreement as well as for agreement of lateral radiographs and MRI-scans. For assessment of agreement, the proportion of observed agreement (po) was calculated incl. 95% confidence interval (CI).

Because of unsatisfactory results in the agreement of radiographs and MRI, an additional analysis was performed: Two mixed effects regression models [16] were fitted to investigate the influence of radiographs and MRI on the D. Dejour's four grades of radiological criteria of trochlear dysplasia (ordinal outcome) and on the two-graded distinguishing between low-grade and high-grade trochlear dysplasia (binary outcome). In both models the method (i.e. radiographs/MRI) was included as fixed effect and the rater was included as random effect.

RESULTS

The current study assesses how four blinded independent readers agree on typing trochlear dysplasia first using just the lateral view and then using just the MRI image. This was done twice with the studies being randomly shuffled for the second reading.

In the following, important results are summarized in terms of proportions of observed agreement (po).

1a) Intra-observer agreement, four-grade analysis

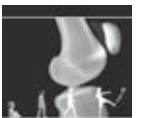
When observers classified trochlear dysplasia into D. Dejour's four grades the intra- observer agreement of the lateral radiographs between the first and the second reading was 30% to 78%. Intra-observer agreement for classification into four grades for the two readings of the axial MR images was 32% to 74%.

1b) Intra-observer agreement, two-grade analysis

When grade A was compared to type B, C and D combined to form low-grade and high-grade classifications the intra-observer agreement on the lateral radiograph between the first and the second reading was 56% to 88% and 70% to 90% for the axial MR images.

2a) Interobserver agreement, four-grade analysis (comparison of all possible pairs of raters)

Four-grade analysis of interobserver agreement of lateral X-rays showed at the first reading an agreement ranged from 36% to 52% and at the second reading an agreement ranged between 24% and 58%. Interobserver agreement of axial MR images was at the first reading 42% to 68% and at the second reading 28% to 40%.



2b) Interobserver agreement, two-grade analysis (comparison of all possible pairs of raters)

Two-grade analysis of lateral X-rays showed at the first reading an agreement between 60% and 76% and at the second reading an agreement between 52% and 76%. The interobserver agreement of axial MR images was at the first reading 86% to 96% and at the second reading 62% to 86%.

3a) Agreement of the radiographs and MRI, four-grade analysis

Using the four-grade classification according to D. Dejour the agreement of radiographs and MRI at the first reading was 36 to 56%. At the second reading an agreement of radiographs and MRI between 28 and 54% was achieved.

3b) Agreement of the radiographs and MRI, two-grade analysis

For the two-grade classification the agreement of radiographs and MRI at the first reading ranged from 66 to 82%. At the second reading an agreement of radiographs and MRI of 64 to 74% was measured.

Because of unsatisfactory results in the agreement of radiographs and MRI, an additional regression analysis was performed: Two mixed effects regression models were fitted to investigate the influence of radiographs and MRI.

In the first regression model, the influence of the method (radiographs vs. MRI) on the D. Dejour's classification was investigated. The result shows that the MRI has a higher odds for a higher level in D. Dejour's classification (odds ratio 1.29; 95% CI: 1.01; 1.66, p=0.04). In the second regression model, the influence of the method (radiographs vs. MRI) on the two-grade classification (low-grade and high-grade trochlear dysplasia) was investigated. The result of the second model shows that the MRI has a higher odds for high-grade trochlear dysplasia (odds ratio 2.24; 95% CI: 1.61; 3.13, p<0.01).

DISCUSSION

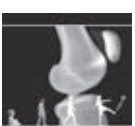
The results of the current study show that inter- and intraobserver agreement on D. Dejour's four grade classification of trochlear dysplasia on the lateral radiograph and the MRI is insufficient. In contrast the two-grade classification showed good to excellent agreement.

There are different possible reasons for these unsatisfactory results. The surface geometry of the articular cartilage cannot easily be divided in four groups as there is a wide variation of the anatomy of the trochlear geometry. For example we found it difficult to distinguish between type B and C dysplasia. One of the rater indicated a flat, but slightly descending trochlea as type B, whereas the other raters interpreted the slightly descending trochlea as hypoplastic and therefore classified it as type C. Type C dysplasia is difficult to see on lateral radiographs and often needs to be defined on coronal cuts on MRI-scans, as in different cases a small vertical join or cliff pattern on MRI-scans may lead one rater to classify it as type C and the other rater as type D (fig. 3, 4).

In clinical practice it can be furthermore difficult to achieve a true lateral radiograph with superimposed dorsal condyles. But true lateral radiographs are mandatory to confirm the diagnosis of trochlear dysplasia. Koëter et al. verified that minimal rotation aberrations cause radiographs misdiagnosis of trochlear dysplasia [15].

The results of the study confirm the conclusions of Remy *et al.* [20] saying that the crossing sign and the supratrochlear bump are the most reproducible signs whereas the double contour only showed agreements of 51%.

Because the lateral radiograph was inconsistent in differentiating D. Dejour's four grades of trochlear dysplasia, the ability to distinguish



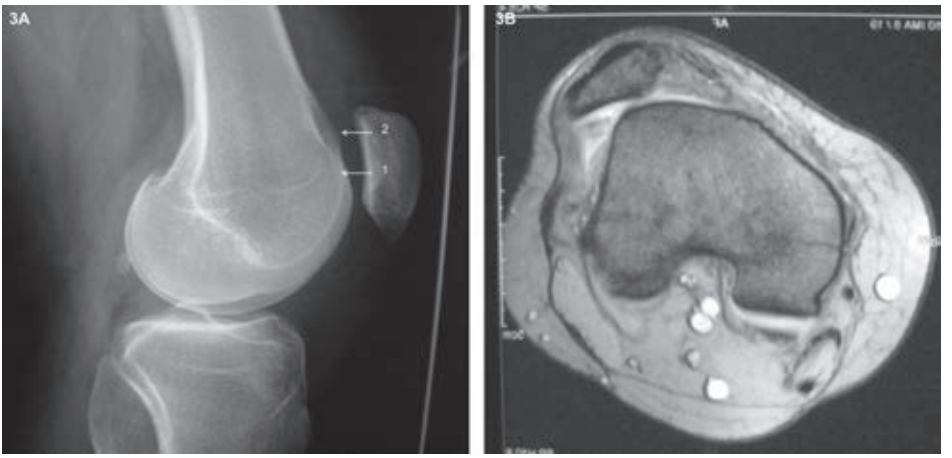


Fig. 3: Example for type B dysplasia.
A: Crossing sign (1), supratrochlear bump (2) on the lateral radiograph.
B: Flat trochlea on the MRI-scan.

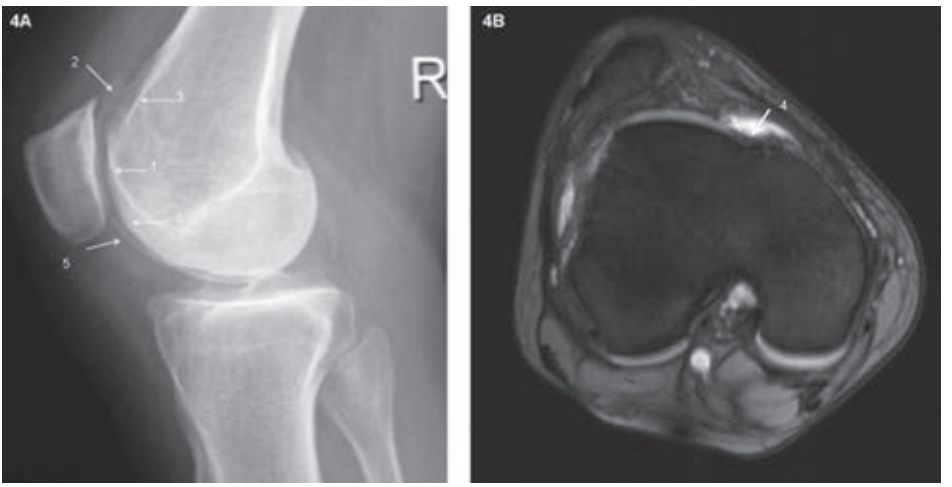
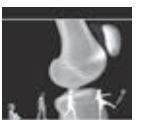


Fig. 4: Example for type D dysplasia:
A: Crossing sign (1), supratrochlear bump (2), double contour (3), lateral femoral condyle (5) and the medial femoral condyle (6) on the lateral radiograph.
B: Asymmetry of trochlear facets plus vertical join (4) on the MRI-scan.

between low-grade dysplasia, type A from high-grade dysplasia, type B, C and D was additionally studied.

Two-grade analysis showed much better results for intra- and interobserver agreement: The contingency tables exhibited better results for intraobserver agreement with proportions of observed agreement between 56 and 88% (lateral radiographs) and 70 and 90% (MRI) as well as for the interobserver agreement with proportions of observed agreement between 52 and 76% for lateral radiographs and 62 and 96% for MRI-scans.



The additional regression analysis showed a conspicuous effect of the method on D. Dejour's classification as well as two-grade classification which reinforces the poor agreement between radiographs and MRI. Less severe dysplasias were documented when analyzing lateral radiographs than analyzing MRI-scans.

Besides the D. Dejour classification, different measurements to assess the morphology of the femoral trochlea are described in the literature [3, 4, 5, 12, 18, 22, 24].

Although inter- and intra- observer agreements of the classification of Pfirrmann, Carrillon and Biedert [3, 4, 5, 18] seem to be higher, they are not as workable as D. Dejour's classification in clinical practice in our opinion.

So the authors still think that D. Dejour's classification is a good instrument for interpreting trochlear dysplasia. Although it is easy to apply using D. Dejour's classification several issues have to be considered:

1. The four-grade analysis shows fair intra- and interobserver agreement while the two-grade analysis show good to excellent agreement.
2. The best overall agreement was found for the two-grade analysis on MRI-scans.
3. The lateral radiograph tends to underestimate the severity of trochlear dysplasia compared to axial MR imaging.

In summary D. Dejour's classification is valid for typing trochlear dysplasia and is particularly useful in separating low-grade from high-grade cases. For clinical purposes the discrimination between low-grade and high-grade dysplasia is an important distinction because prognosis and treatment mainly depend on the severity of trochlear dysplasia.

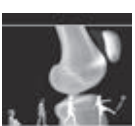
ABSTRACT

Trochlear dysplasia is known to be an important cause for patellofemoral instability. Dejour's radiographic and MRI classifications are widely used in clinical practice and in orthopaedic literature to assess the severity of trochlear dysplasia.

From fifty patients, fifty lateral radiographs as well as fifty MRI-scans were read twice independently within four weeks by four surgeons (two senior and two junior examiners). Analysis was made four-graded according to D. Dejour's four grades of radiological criteria of trochlear dysplasia as well as two-graded differentiating between low-grade (type A) and high-grade trochlear dysplasia (type B-D).

The four-grade analysis shows fair intra- and interobserver agreement while the two-grade analysis shows good to excellent agreement. The best overall agreement was found for the two-grade analysis on MRI-scans. The lateral radiograph tends to underestimate the severity of trochlear dysplasia compared to axial MR imaging.

D. Dejour's classification is valid for typing trochlear dysplasia and is particularly useful in separating low-grade from high-grade dysplasia.



LITERATURE

[1] BALCAREK P, AMMON J, FROSCH S, WALDE TA, SCHÜTTRUMPF JP, FERLEMANN KG, LILL H, STÜRMER KM, FROSCH KH. Magnetic resonance imaging characteristics of the medial patellofemoral ligament lesion in acute lateral patellar dislocations considering trochlear dysplasia, patella alta, and tibial tuberosity-trochlear groove distance. *Arthroscopy. 2010; 26:926-35.*

[2] BARNETT AJ, PRENTICE M, MANDALIA V, WAKELEY CJ, ELDRIDGE JD. Patellar height measurement in trochlear dysplasia. *Knee Surg Sports Traumatol Arthrosc. 2009; 17: 1412-15.*

[3] BIEDERT RM, BACHMANN M. Anterior-posterior trochlear measurements of normal and dysplastic trochlea by axial magnetic resonance imaging. *Knee Surg Sports Traumatol Arthrosc. 2009;17: 1225-30.*

[4] BIEDERT RM. Osteotomien. *Orthopäde. 2008; 37: 872-83.*

[5] CARRILLON Y, ABIDI H, DEJOUR D, FANTINO O, MOYEN B, TRAN-MINH VA. Patellar instability: assessment on MR images by measuring the lateral trochlear inclination-initial experience. *Radiology. 2000; 216: 582-5.*

[6] COLVIN AC, WEST RV. Current concepts review: Patellar instability. *J Bone Joint Surg Am. 2008; 90: 2751-62.*

[7] CUSMANO F, ADRAVANTI P, PEDRAZZINI M, AMPOLLINI A, PAVONE P. Radiologic assessment of femoro-patellar instability. Personal experience and review of the literature. *Radiol Med. 2001; 101: 66-74.*

[8] DEJOUR D, LE COULTRE B. Osteotomies in patello-femoral instabilities. *Sports Med Arthrosc Rev. 2007;15: 39-46.*

[9] DEJOUR D, SAGGIN P. The sulcus deepening trochleoplasty – the Lyon's procedure. *International Orthopaedics. 2010; 34: 311-316.*

[10] DEJOUR H, WALCH G, NEYRET P, ADELEINE P. La dysplasia de la trochlée fémorale. *Revue de Chirurgie Orthopédique. 1990;76: 45-54.*

[11] DEJOUR H, WALCH G, NOVE-JOSSERAND L, GUIER C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc. 1994; 2:19-26.*

[12] DELGADO-MARTINEZ AD, RODRIGUEZ-MERCHÁN R. BALLESTEROS EC, LUNA JD. Reproducibility of patellofemoral CT scan measurements. *International Orthopeadics. 2000; 24: 5-8.*

[13] FUCENTESE SF, VON ROLL A, KOCH PP, DEVAKARA RE, FUCHS B, SCHOTTLE PB. The patella morphology in trochlear dysplasia – A comparative MRI study. *Knee. 2006;13: 145-50.*

[14] GRELSAMER RP, TEDDER JL. The lateral trochlear sign: femoral trochlear dysplasia as seen on a lateral view roentgenograph. *Clin Orthop Relat Res. 1992; 281: 159-62.*

[15] KOËTER S, BONGERS EM, DE ROOIJ J, VAN KAMPEN A. Minimal rotation aberrations cause radiographic misdiagnosis of trochlear dysplasia. *Knee Surg Sports Traumatol Arthrosc. 2006;14: 713-17.*

[16] MOLENBERGHS G, VERBEKE G. Models for Discrete Longitudinal Data *Springer, Berlin, 2006.*

[17] MURRAY TF, DUPONT JY, FULKERSON JP. Axial and lateral radiographs in evaluation patellofemoral malalignment. *Am J Sports Med. 1999; 27: 580-84.*

[18] PFIRRMANN CWA, ZANETTI M, ROMERO J, HODLER J. Femoral trochlear dysplasia: MR findings. *Radiology. 2000; 216: 858-64.*

[19] REMY F, BESSON A, MIGAUD H, COTTON A, GOUGEON F, DUQUENNOY A. Reproductibilité de l'analyse radiographique de la dysplasia de la trochlée fémorale. Analyse intra- et interobservateur sur 68 genoux. *Rev Chir Orthop Reparatrice Appar Mot. 1998;84: 728-33.*

[20] RÉMY F, GOUGEON F, ALA EDDINE T, MIGAUD H, FONTAINE C, DUQUENNOY A. Reproducibility of the new classification of femoral trochlea dysplasia proposed by Dejour: Predictive value for severity of femoropatellar instability in 47 knees. *J Bone Joint Surg Br Proceedings. 2002; 84:43.*

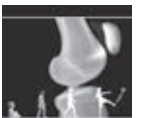
[21] SALZMANN GM, WEBER TS, SPANG JT, IMHOFF AB, SCHÖTTLE PB. Comparison of native axial radiographs with axial MR imaging for determination of the trochlear morphology in patients with trochlear dysplasia. *Arch Orthop Traum Surg. 2010; 130: 335-40.*

[22] SMITH TO, DAVIES L, TOMS AP, HING CB, DONELL ST. The reliability and validity of radiological assessment for patellar instability. A systematic review and meta-analysis. *Skeletal Radiol. 2010;40: 399-414.*

[23] TECKLENBURG K, FELLER JA, WHITEHEAD TS, WEBSTER KE, ELZARKA A. Outcome of surgery for recurrent patellar dislocation based on the distance of the tibial tuberosity to the trochlear groove. *J Bone Joint Surg Br. 2010;92: 1376-80.*

[24] TOMS AP, CAHIR J, SWIFT L, DONELL ST. Imaging the femoral sulcus with ultrasound, CT, and MRI: reliability and generalizability in patients with patellar instability. *Skeletal Radiol. 2009; 38: 329-38.*

[25] WALCH G, DEJOUR H. La radiologie dans la patologie fernoropatellaire. *Acta Orthop Belg. 1989; 55: 371-80.*



SULCUS DEEPENING TROCHLEOPLASTY FOR THE TREATMENT OF RECURRENT PATELLAR DISLOCATION WITH UNDERLYING TROCHLEAR DYSPLASIA

P.G. NTAGIOPOULOS, P. BYN, D. DEJOUR

INTRODUCTION

Since the documentation of trochlear dysplasia as the most consistent anatomic factor present in patients with recurrent patellar dislocation [1], a number of surgical procedures for its treatment has been introduced [2]. Trochleoplasty procedures are designed to reshape the abnormal trochlear groove and because they involve a certain amount of technical difficulty [3], they were initially accepted with skepticism or reserved as salvage options [4]. But recent literature contains several authors with encouraging short and mid-term results on the treatment of patients with patellar dislocation and underlying high-grade trochlear dysplasia leading to a great patellar instability and patellar dislocations [5-13].

Trochlear dysplasia is a developmental condition where the femoral trochlea loses its normal concave shape to an abnormal flat or even convex geometry [14]. Four basic trochleoplasty procedures have been proposed. Albee's pioneer procedure involved the elevation of the lateral facet in order to restore normal anatomy [15]. The second procedure is the "sulcus-deepening trochleoplasty", which was first proposed by Masse [16] and later standardized by Henri [17] and David Dejour [18] (fig. 1). The third procedure was introduced by Bereiter and Gautier in 1994 [19], was later followed by von Knoch [11] and is known as the "Bereiter procedure". An osteochondral flake with only 2mm of subchondral bone is elevated from the trochlea without osteotomy of the condyle, and the distal femoral subchondral bone is deepened and refashioned with osteotomes and a high-speed burr. The same procedure has been performed arthroscopically by Blønd and Schöttle [13]. The fourth procedure is the "recession wedge trochleoplasty" which was introduced by Goutallier [20] in 2002 and has been embraced by Beaufils [3]. In this technique, the shape of the trochlea is not changed but the trochlear bump is removed by a proximally-based wedge and a subsequent pressure on the trochlea.

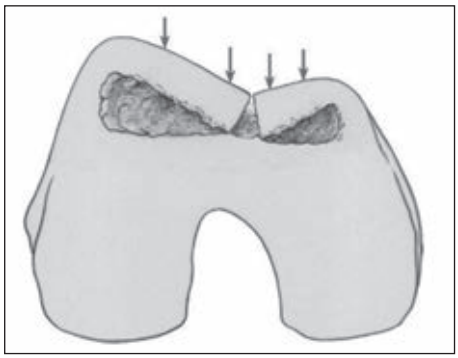
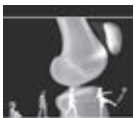


Fig. 1: The rationale of the sulcus-deepening trochleoplasty.



The purpose of the present study is to record the clinical, radiological and functional mid-term results from the application of "Lyon's sulcus-deepening trochleoplasty" [18] in patients with recurrent patellar dislocation and underlying trochlear high-grade dysplasia.

MATERIALS AND METHODS

This is a retrospective study that included patients treated for recurrent patellar dislocation between September 1993 to September 2006. Inclusion criteria for the study were the following: patients with recurrent or more than 3 documented episodes of patellar dislocation and underlying trochlear dysplasia and pathologic lateral patellar glide test using the quadrant test (patella can be shifted by 3 or more quadrants laterally by the examiner with patient's knee placed at full extension). Patients with open growth plates, patellofemoral arthritis and patellofemoral pain syndrome with no true dislocation were excluded from the study.

Pre-operative objective evaluation included apprehension test, lateral patellar glide test and patellar tracking. Subjective findings included the presence of patellofemoral pain and/or sense of instability. Radiological assessment included true lateral X-rays, axial view of the patella at 30° degrees of flexion and computed tomography. Trochlear dysplasia was graded and sulcus angle, lateral patellar tilt (without quadriceps contraction), tibial tuberosity – trochlear groove (TT-TG) distance and patellar height (according to Caton-Deschamps index [21]) were measured.

Patients were divided in two groups: Group A with recurrent dislocation after previous surgery and Group B with no surgical antecedent. Patients were treated surgically following an "à la carte surgery" rationale [17] that included the identification of abnormal anatomic factors contributing to instability and the correction of them one by one in the same stage [2, 14, 22] (fig. 2). In all patients, trochleoplasty was combined with medial soft-tissue surgery, such as vastus medialis obliquus (VMO) plasty or medial patellofemoral ligament (MPFL) reconstruction in all knees and, when required, with lateral soft-tissue surgery such as lateral release or lengthening. In patients where additional osseous etiologic factors were recognized, a re-alignment procedure was added.

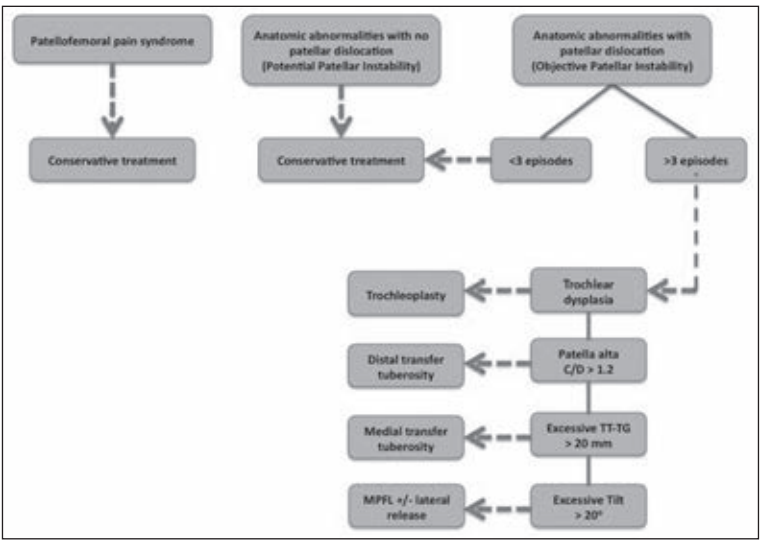


Fig. 2: The "à la carte surgery" algorithm that the authors followed for the therapeutic approach of patellofemoral instability.

Sulcus-deepening trochleoplasty was performed with the same surgical technique in all patients [18]. The rationale is to remove enough subchondral bone under the trochlea (fig. 1), so that the subsequent depression of the new trochlear groove will be flush with the anterior femoral cortex and the prominence will disappear (fig. 3). The trochlea is osteotomized with a scalpel carefully over the position of the desired new groove, so that a normal smaller sulcus angle is restored and the trochlear groove is positioned in a more lateral position to decrease the TT-TG distance (fig. 4) according to the TT-TG value. The new trochlea is fixed with 2 metallic staples. All patients followed the same rehabilitation protocol described in the relevant chapter.

Post-operative clinical evaluation was performed at 1, 3, 6, 12 months and the last follow-up was during May-August 2008. It included clinical and radiological examination, IKDC score [23] and level of activity at final follow-up. At final evaluation, patients were also asked on their overall satisfaction from the operation.

Statistical analysis: Differences between pre- and post-operative mean values were compared by the paired chi-square test. Level of statistical significance was <0.01.

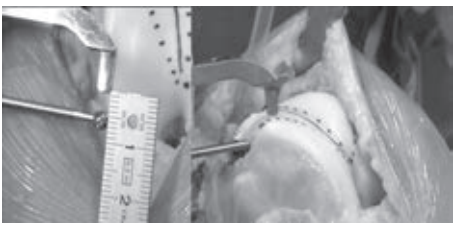


Fig. 3: Subchondral bone is removed under the trochlear without damaging the cartilage, in order to remove the prominence and re-shape the groove.

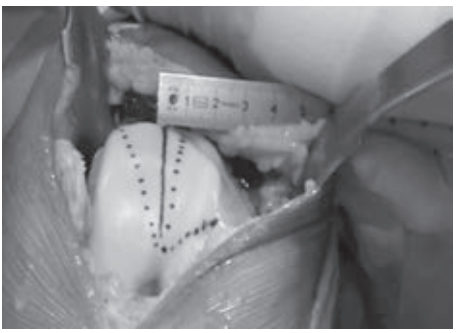


Fig. 4: According to pre-operative TT-TG measurement, the new trochlear groove (solid line) can be positioned in a more lateral position, serving as a "proximal re-alignment procedure".

RESULTS

Forty-nine patients were retrospectively included. Group A with previous patellofemoral surgery had 22 patients (24 knees) with prior unsuccessful surgery for patellar instability. Mean age was 23±5.5 years (14-33) at the time of surgery with a female to male ratio of 1.33. Thirty-three per cent had a positive familiar history (first degree relative) of recurrent patellar dislocation and 9% had bilateral surgery. Mean follow-up was 6 years (2-8) and no patients were lost to follow-up. Average number of previous surgery for patellofemoral instability was 2 per patient and included: medialization of the tibial tuberosity, distalization of the tibial tuberosity, soft-tissue surgery (medial structures augmentation, lateral structures release) and arthroscopy (Table 1). Patients' symptoms and clinical signs were lateral patellar dislocation (100%), patello-femoral pain (12.5%), positive apprehension sign (91.6%) and abnormal patellar tracking (81%). Trochlear cartilage lesions before trochleoplasty were: 62.4% had ICRS grade 0, 8.3% grade I, 20% grade II, 8.3% grade III, 0% grade IV, and for the patella 45.5% grade 0, 4% grade I, 21% grade II, 17% grade III, 12.5% grade IV. Sulcus-deepening trochleoplasty was combined with an additional operation in all cases: MPFL reconstruction in 45.8% of the cases (n=11), VMO plasty 41.6% (n=10), tibial tuberosity distalization 20.8% (n=5), tibial tuberosity medialization 29.1% (n=7), patellar tendon lengthening (for previous patella infera) 8.3% (n=2), lateral release 25% [6], patellar osteotomy 4.1% (n=1) (Table 1). Pre-operative and post-operative radiographic findings are

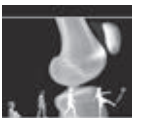


Table 1: Patient demographics, clinical signs, symptoms and associated procedures for the two groups.

	Group A	Group B
No. of knees	24	31
Age	23±5.5 years (14-33)	21±7.9 years (14-47)
Abnormal patellar tracking	81%	54.8%
Apprehension sign	91.6%	96.7%
Feeling of instability	100%	100%
Associated pain	12.5%	6.4%
Previous operations	Yes	No
Concomitant procedures		
1. VMO plasty	41.6%	83.8%
2. MPFL reconstruction	45.8%	16.1%
3. Distal transfer of tibial tuberosity	20.8%	51.6%
4. Medial transfer of tibial tuberosity	29.1%	67.7%
5. Lateral retinaculum release	25%	67.7%
6. Patellar osteotomy	4.1%	0%
7. Patellar tendon lengthening	8.3%	0%

shown in Table 3. Sulcus angle decreased significantly ($p<.01$) from 153°±14° pre-operatively to 141°±10° post-operatively, TT-TG distance decreased significantly ($p<.001$) from 16±6mm to 12±2mm and patellar tilt without quadriceps contraction decreased significantly ($p<.0001$) from 31°±14° to 11°±8° (Table 2). Pain decreased in 72% of the cases, remained unchanged or increased in 28% and there was no case of post-operative stiffness. Apprehension sign was negative post-operatively in 75%, patellar tracking was normal in all of the cases, lateral patellar tracking was <1 quadrant in 72% and <2 quadrants in 28% of the knees. Mean pre-operative IKDC score was 51.4±21.8 (23-75) and at last follow-up it increased to 76.7±13.0 (53-100), ($p<.001$). At the time of final follow-up, there was no case with patellofemoral arthritis according to Iwano criteria and 95.4% of the patients had returned to previous activities, including recreational sports. None of the patients had a patella dislocation recurrence and no post-operatively feeling of patellar instability. All of them responded that they were satisfied from surgery. There were no complications recorded in this group and there was no re-operation.

Group B included 27 patients with 31 knees that had more than 3 true episodes of patellar dislocation and no previous surgery for patellofemoral dislocation. Mean age was 21±7.9 years (14-47) at the time of surgery with a female to male ratio of 0.93. Mean follow-up was 7 years (2-9) and no patient was lost to follow-up. Forty-five per cent had a positive familiar history of recurrent patellar dislocation and 14.8% had bilateral surgery. Patients' symptoms and clinical signs were lateral patellar dislocation (100%), patello-femoral pain (6.4%), positive apprehension sign (96.7%) and abnormal patellar tracking (54.8%) (Table 1). Trochlear cartilage lesions before surgery were: 67.7% had ICRS grade 0, 6.4% grade I, 16.2% grade II, 3.2% grade III, 6.5% grade IV, and for the patella 25.8% grade 0, 0% grade I, 19.2% grade II, 22.5% grade III,

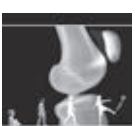


Table 2: Pre and post-operative imaging findings in patients that underwent trochleoplasty and had previous surgery for patellar dislocation.

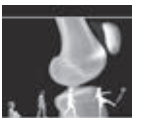
Group A: Knees with previous operation for patellofemoral instability		
	Pre-operative	**Post-operative**
Sulcus angle	153.0°±14.7° (130°-177°)	141±10.2° * NS (122°-163°)
Caton-Deschamps index	1.03±0.28 (0.5-1.48)	0.95±0.22 NS (0.69-1.29)
TT-TG distance	16.6±7.7mm (5-30mm)	12.6±4.2mm ** (5-20mm)
Patellar tilt	31.4o±14.3° (0°-58°)	11.8°±8.0° ** (1°-31°)
Trochlear dysplasia		
Type A	0%	33.3%
Type B	29.1%	0%
Type C	0%	0%
Type D	70.9%	0%
Normal or non-classifiable	0%	66.7%

*: p<.01, **: p<.001, NS: not significant, when compared to pre-operative values.

Table 3: Pre and post-operative imaging findings in patients that underwent trochleoplasty and had no previous surgery for patellar dislocation.

Group B: Knees with no previous operation for patellofemoral instability		
	Pre-operative	**Post-operative**
Sulcus angle	152°±16.2° (126°-178°)	141°±8.9° * NS (126°-155°)
Caton-Deschamps index	1.12±0.18 (0.78-1.4)	0.97±0.15 NS (0.64-1.25)
TT-TG distance	19±4.8mm (10-28mm)	12±5.5mm ** (6-24mm)
Patellar tilt	37°±7.8° (0°-58°)	15°±8.7° ** (1°-31°)
Trochlear dysplasia		
Type A	0%	45.1%
Type B	38.7%	0%
Type C	0%	0%
Type D	61.3%	0%
Normal or non-classifiable	0%	54.9%

*: p<.01, **: p<.001, NS: not significant, when compared to pre-operative values.



32.5% grade IV. Trochleoplasty was combined with an additional operation in all cases: MPFL reconstruction in 16.1% of the cases (n=5), VMO plasty 83.8% (n=26), tibial tuberosity distalization 51.6% (n=16), tibial tuberosity medialization 67.7% (n=21), lateral release 67.6% (n=21) (Table 1). Sulcus angle decreased significantly ($p<.01$) from 152°±16° pre-operatively to 141°±8° post-operatively, TT-TG distance decreased significantly ($p<.001$) from 19±4mm to 12±5mm and patellar tilt without quadriceps contraction decreased significantly ($p<.001$) from 37°±7° to 15°±8° (Table 3). Post-operative pain decreased in 77.4% of the cases, remained unchanged or increased at 22.6% and there was no case of stiffness. Apprehension sign remained positive in 19.3% of the cases, patellar tracking was normal in all cases, lateral patellar glide test was negative in 96.8% (<1 quadrant in 70%, <2 quadrants in 26.8%) and in 3.2% the quadrant test was 4/4 but with no patellar dislocation. Mean pre-operative IKDC score was 51.2±22.9 (25-80) and post-operative IKDC score was 82.5±17.9 (40-100), ($p<.001$). There was no radiographical evidence of patellofemoral arthritis according to Iwano criteria at the last follow-up. Eighty-seven per cent of the patients returned to their previous activities. There was no case with patella dislocation recurrence and no residual feeling of patellar instability and 93.6% replied they were satisfied from surgery. Three complicated cases were recorded: 2 cases of hardware (staples) breakage that had to be removed and one case of deep venous thrombosis that was treated accordingly.

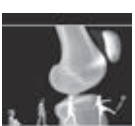
DISCUSSION

The mid-term clinical, radiological and functional results from the Lyon's sulcus-deepening trochleoplasty in the appropriate patient population for the treatment of recurrent patellar dislocation with underlying high-grade trochlear dysplasia are satisfactory without major complications and no sign of patello-femoral arthritis.

The authors' approach to the treatment of patients with recurrent patellar instability is based on the following algorithm for the identification of the major anatomic and etiologic factors (fig. 2) [2, 14, 22, 24, 25]: the presence of an excessive TT-TG distance more than 20mm is considered abnormal and should be treated with medialization osteotomy of the tuberosity. Patella alta is another significant factor contributing to patellar instability, and its surgical treatment involves distalization osteotomy of the tuberosity. An increased lateral patellar tilt over 20° is amenable to correction with lateral retinaculum release, VMO plasty or reconstruction of the MPFL. The presence of high-grade trochlear dysplasia requires a type of trochleoplasty for its correction [2, 18].

After the identification of any of the above anatomic parameters, the patient must be assessed for the presence and occurrence of patellar dislocation. ***1)*** Patients with no history of patella dislocation and pain as the only symptom, and ***2)*** patients with patellofemoral pain and with the presence of any of these anomalies but no history of patellar dislocation, are treated conservatively. ***3)*** Patients with any of these anatomic anomalies and recurrent patellar dislocation are candidates for surgery after at least 3 true episodes of patellar dislocation [14]. Surgical treatment of these patients involves the correction of one or more of the above-mentioned anatomic anomalies in one stage, which often requires the combination of more than one procedure. The presence of high-grade trochlear dysplasia (i.e. B or D) in these patients requires a deepening trochleo-plasty procedure for its correction. The goal of sulcus-deepening trochleoplasty is to reshape the trochlea, but patellar instability may be also caused by the presence of co-existent anatomic factors that must be addressed (e.g. tuberosity osteotomy for patella alta or increased TT-TG distance) and its treatment almost always requires a combined soft-tissue procedure like MPFL reconstruction or VMO plasty [14, 18].

The rationale of sulcus-deepening trochleo-plasty has three key elements: First, in cases of a flat trochlea, it reshapes the trochlea back to a more anatomic and concave geometry by deepening the proximal sulcus, so that it engages



the patella in late degrees of knee flexion [18]. Patellar stability during early flexion is mostly accomplished by MPFL which is tight in full knee extension and acts as a stabilizer during early flexion (15°-20°), brings the patella into the trochlear grove, and in greater degrees of flexion (>30°) is loose and the trochlea serves as a guide for normal patellar kinematics [1]. Second, in cases of a convex trochlea, trochleoplasty removes the sulcus prominence that the patella needs to override during flexion and that leads to patella dislocation off the lateral facet. Last, trochleoplasty creates a new trochlear groove in a more lateral position than the dysplastic one, thus decreasing the excessive TT-TG distance and serving as a "proximal realignment procedure" [18].

The biomechanical effects of the sulcus-deepening trochleoplasty have been studied by Amis *et al.* who reported that the mediolateral flattening of the anterior surface of the trochlear facets results predominantly from an excess of bone centrally in the groove. This forms a supratrochlear prominence or "bump" anterior to the shaft of the femur, which the patella has to override when the knee starts to flex in order to engage in the groove for the remaining degrees of flexion. The authors showed that "simulated" trochlear dysplasia led to significant reduction in lateral stability and by re-creating a deep trochlear groove with a trochleoplasty procedure, lateral stability increased significantly similarly to the intact knee [26].

Evaluation of the results from the application of trochleoplasty presents with certain difficulties. There is no agreement if satisfactory long-term results can by defined by the correction of imaging findings (fig. 5), the absence of pathological lateral patellar laxity, or the restoration of the pre-operative subjective patient's sense of instability, pain or the absence of apprehension [7, 8, 10]. Certainly, the latter along with post-operative satisfaction are the goals of any procedure for patellofemoral instability, but authors offer different functional scores with varying results after measuring post-operative satisfaction in the same population [7, 10]. Furthermore, selecting the right candidate for trochleoplasty has been controversial, since authors set different clinical (pain, instability, or both) or radiological inclusion criteria (type of dysplasia, height of prominence) for performing such procedure [3,

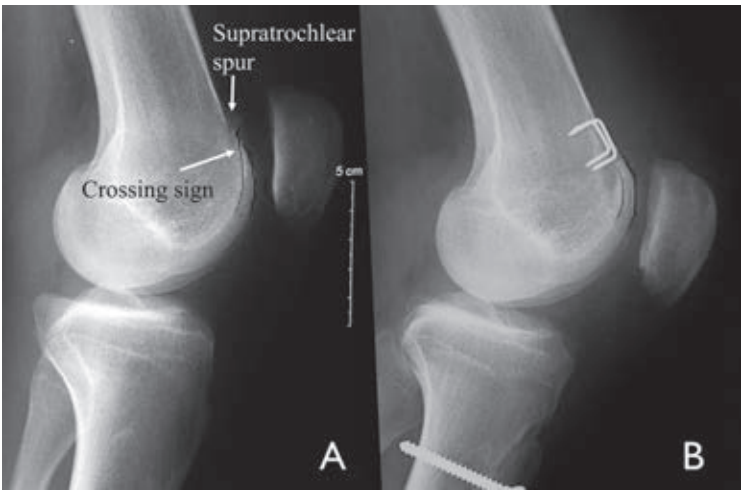
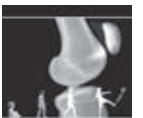


Fig. 5: Example of a (A) pre-operative dysplastic trochlea type B with the supratrochlear spur and the crossing sign, along with patella alta. (B) The supratrochlear spur and the crossing sign disappeared after trochleoplasty and the patella height was corrected with tuberosity osteotomy.



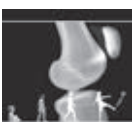
5-7, 10-12]. Trochleoplasty procedures are often performed with different techniques and, most important, with various rehabilitation protocols, which make the comparison of their results challenging. Last, evaluating the efficacy of trochleoplasty as a single procedure is very difficult to do in a patient population that usually presents with other concomitant anomalies that also need correction, and especially since trochleoplasty is almost always performed in combination with soft-tissue procedures, such as MPFL reconstruction [2, 3, 5-7, 11, 12, 14, 18, 20].

The mid-term clinical results from its application have been also reported. Fucentese *et al.* reported the results from its application in 44 cases after an average of 4 years [6]. Kujala score improved from 68 to 90 points ($p<0.001$), and instability ($p<0.001$) and pain ($p=0.027$) decreased significantly with no major complications, like chondrolysis or subchondral necrosis, except for deterioration of the cartilage on the lateral trochlear facet. Donell *et al.* reported their data after performing sulcus-deepening osteotomy in 17 cases after an average of 1 year [7]. Eleven cases had normal patellar tracking post-operatively, Kujala score improved from a mean of 48 to 75 ($p<0.05$) and only 8 patients required a re-operation (i.e. arthrolysis). Verdonk *et al.* also reported their results in 14 cases after a mean follow-up of 18 months [10]. In that study, patients were assessed using the Larsen-Lauridsen score on pain, stiffness, crepitus, and loss of function. Seven scored poorly, three fairly well and four well. On a subjective scoring system 77% of patients found the procedure satisfactory: six patients rated the result as very good, four as good, and one as satisfactory with only two patients rating the result as inadequate. All these authors conclude that sulcus-deepening trochleoplasty requires careful attention to detail [10]. It is a technically-demanding procedure that addresses a rare condition with satisfactory results and acceptable level of complications [7]. Sulcus-deepening trochleoplasty is more suitable for severe cases of type B and D of dysplasia, where the presence of trochlear prominence is amenable to correction with this technique [6].

In this study, the primary surgical treatment of patients with recurrent patellar instability and trochlear dysplasia included the sulcus-deepening trochleoplasty that was also combined with other procedures. The results of performing additional soft-tissue or bone surgery to trochleoplasty were satisfactory, IKDC score improved significantly and there was only one case of positive lateral glide test without patellar dislocation. Other authors report equal success rates from 90% to 100% [5, 7, 10-12, 20].

Except for the patellar height, all radiographic findings were significantly corrected post-operatively, with emphasis to the lateral patellar tilt. This can be attributed to both the trochleoplasty and the concomitant soft-tissue procedure (VMO plasty or MPFL reconstruction, respectively). Another interesting finding was the significant decrease of the TT-TG distance, caused by the position of the new groove in a more lateral position. This can be helpful in eliminating the need for an additional procedure (medial transfer of the tuberosity), since in cases of excessive TT-TG distance, re-positioning the trochlea, instead of the tuberosity, can reduce it.

Trochleoplasty procedures are a very appealing revision option in cases of previously operated patients with persistent patellar instability and an undiagnosed or underestimated underlying trochlear dysplasia [3, 5-7, 11-13, 20]. The satisfying results that are recorded in these patients after trochleoplasty, probably show the need for the early recognition of a group of patients with trochlear dysplasia in whom the benign neglect of the dysplasia and the application of conventional surgery for the treatment of patella dislocation (i.e. medial reefing, lateral release) is doomed to failure. In the present study, patients were also classified according to having previous surgery or not. All patients with previous patellofemoral surgery and persistent patellar dislocation had associated high-grade trochlear dysplasia (type B and D). In every case, trochleoplasty was combined with a soft-tissue procedure. Restoration of patellar stability, correction of lateral tilt, functional score and patient

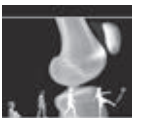


satisfaction were corrected significantly and were comparable to patients with no previous surgery that underwent trochleoplasty. Radiographic presence of patellofemoral arthritis was not recorded, although its appearance requires even longer follow-up periods. Subjective evaluation and overall satisfaction of these multiply-operated patient group was higher (p: NS) than patients with no previous surgery, with no need for re-operation or complications in this group.

On the other hand, trochleoplasty procedures are not void of surgery-specific complications. Early concerns on post-operative cartilage viability have been studied by Schöttle *et al.* who recorded normal subchondral bone, viable cartilage cells, bone flap healing and minimal risk for cartilage deterioration [9]. Over or undercorrection of normal trochlea geometry is another concern. Again this is difficult to verify since authors use different imaging parameters to compare post-operatively. Fucentese *et al.* showed significant decrease of TT-TG distance (17mm vs. 13mm), decrease of lateral patellar tilt (32° vs. 13°) and patellar translation (8mm vs. 0mm) after "Bereiter" trochleoplasty, in which the sulcus angle does not change [6, 8], but they recorded overcorrection of the trochlear groove in 2 out of 17 cases [8]. Von Knoch *et al.* showed significant correction the crossing sign (which remained positive only in 2 cases), increase of the trochlear depth from -0.1mm to 5.0mm, and decrease of the trochlear bump from 3.9mm to 0.4mm [11]. Donell *et al.* also recorded significant reduction to normal values of prominence height, TT-TG distance and patellar tilt without measuring the sulcus angle, which they considered inaccurate for estimating trochlear dysplasia [7]. Although they commented that the repetition of the imaging at 1 year, showed that correction of the patellar tilt angle was not achieved in a few cases, they followed the same "à la carte" rationale to reduce the risk of overcorrection [7]. In our study, there was a significant decrease of sulcus angle, TT-TG distance and lateral tilt. The clinical importance of such changes in radiographic findings is difficult to evaluate, because of the multifactorial nature of patellar instability. Yet, persistence of instability and need for re-operation after trochleoplasty procedures are rarely recorded [3, 5, 11], and when they are present, authors attribute it to other untreated pathology, i.e. MPFL deficiency [6]. Similarly, in this study, there was no case of post-operative patellar instability.

Post-operative stiffness and arthrofibrosis are potential complications after any knee operation, and the conflicting results of its occurrence by Verdonk (46%) [10], Donnell (33%) [7], Utting (2%) [5] and Thaunat (5%) [12], to its complete absence in the long-term study of von Knoch [11], prevent safe conclusions for the aetiology of trochleoplasty in post-operative arthrofibrosis. In this study there was no case of arthrofibrosis recorded. Similarly, reports of post-operative pain associated with trochleoplasty procedures vary among authors and it is difficult to compare, especially in the case of patients treated for patellar pain rather than dislocation [10, 20]. Even when the procedure is performed for the treatment of patellar dislocation, post-operative pain is a concern. Pain complicates trochleoplasty in previous studies from 15% [5] to 46% [10] and up to 100% [12]. In the mid and long-term evaluation, Von Knoch *et al.* [11] with an 8-year follow-up, recorded 49% of improvement of pain and 33% of increase, and Fucentese *et al.* [6] with 4 years follow-up, recorded increase of pain in 7% of their cases. In this series, patients were treated with trochleoplasty for recurrent patellar dislocation and not for patellofemoral pain alone. Post-operatively, 75% showed decrease in pain and 25% that their symptoms remained unchanged or increased. The unpredictability of post-operative pain [6] and its probably strongest association with severe cartilage lesions on both femoral and patellar side before surgery have been recorded [5-7].

The most concerning, though, potential complication of trochleoplasty procedures is the late development of osteoarthritis. Recurrent patellar dislocation can lead to degenerative changes if left untreated [27], and trochlear dysplasia can accelerate osteoarthritis as well [2, 11, 27], but the role of corrective surgeries



in the development of arthritis is poorly understood [28]. On one hand, trochlear dysplasia can predispose to patellofemoral arthritis and on the other hand, trochleoplasty can be an additional factor for arthritis as with any other corrective surgery for patellar dislocation [27, 29]. But the main goal of trochleoplasty remains to correct recurrent dislocation and not to prevent arthritic changes. An additional difficulty in estimating the risks of arthritis from trochleoplasty is the presence of severe cartilage lesions before surgery. The only available long-term data so far, show the presence of arthritic findings in 30% of patients after 8 years of trochleoplasty, but the authors recorded the presence of such degeneration in most of the cases at the time of surgery and attributed it to previous patellar dislocation [11]. In the present study, there were no cases of post-operative patellofemoral arthritis during the last follow-up, even for the cases with severe cartilage damage at the time of surgery.

In conclusion, sulcus-deepening trochleoplasty is a good option for the primary surgical treatment of carefully selected patients with recurrent patellar dislocation and high-grade trochlear dysplasia of type B or D. Concomitant etiologic factors must also be cautiously assessed. Trochleoplasty remains an important revision option in the case of previously operated patients with persistent patellar dislocation and undiagnosed or underestimated trochlear dysplasia, in whom neglect of dysplasia and conventional surgery is probably ill-fated. Combination of the procedure with soft-tissue surgery, such as MPFL reconstruction, is necessary to achieve normal patellar kinematics. The effects of such procedures are difficult to compare and there is need for agreement on the choice of the right candidate for trochleoplasty, the surgical technique, the rehabilitation protocol, and the post-operative parameters that must be corrected. Mid-term follow-up showed satisfactory restoration of patellar stability and functional knee scores, good to excellent patient satisfaction with no major complications of subsequent arthritis or deterioration of patellofemoral function.

LITERATURE

[1] DEJOUR H, WALCH G, NOVE-JOSSERAND L, GUIER C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc 1994; 2: 19-26.*

[2] MULFORD JS, WAKELEY CJ, ELDRIDGE JD. Assessment and management of chronic patellofemoral instability. *J Bone Joint Surg Br 2007; 89: 709-16.*

[3] BEAUFILS P, THAUNAT M, PUJOL N, SCHEFFLER S, ROSSI R, CARMONT M. Trochleoplasty in major trochlear dysplasia: current concepts. *Sports Med Arthrosc Rehabil Ther Technol 2012; 4:7.*

[4] BOLLIER M, FULKERSON JP. The role of trochlear dysplasia in patellofemoral instability. *J Am Acad Orthop Surg 2011; 19: 8-16.*

[5] Utting MR, MULFORD JS, ELDRIDGE JD. A prospective evaluation of trochleoplasty for the treatment of patellofemoral dislocation and instability. *J Bone Joint Surg Br 2008; 90: 180-5.*

[6] FUCENTESE SF, ZINGG PO, SCHMITT J, PFIRRMANN CW, MEYER DC, KOCH PP. Classification of trochlear dysplasia as predictor of clinical outcome after trochleoplasty. *Knee Surg Sports Traumatol Arthrosc 2011; 19: 1655-61.*

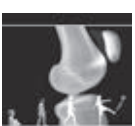
[7] DONELL ST, JOSEPH G, HING CB, MARSHALL TJ. Modified Dejour trochleoplasty for severe dysplasia: operative technique and early clinical results. *Knee 2006; 13: 266-73.*

[8] FUCENTESE SF, SCHÖTTLE PB, PFIRRMANN CW, ROMERO J. CT changes after trochleoplasty for symptomatic trochlear dysplasia. *Knee Surg Sports Traumatol Arthrosc 2007; 15: 168-74.*

[9] SCHÖTTLE PB, SCHELL H, DUDA G, WEILER A. Cartilage viability after trochleoplasty. *Knee Surg Sports Traumatol Arthrosc 2007; 15: 161-7.*

[10] VERDONK R, JANSEGERS E, STUYTS B. Trochleoplasty in dysplastic knee trochlea. *Knee Surg Sports Traumatol Arthrosc 2005; 13: 529-33.*

[11] VON KNOCH F, BOHM T, BURGI ML, VON KNOCH M, BEREITER H. Trochleaplasty for recurrent patellar dislocation in association with trochlear dysplasia. A 4- to 14-year follow-up study. *J Bone Joint Surg Br 2006; 88: 1331-5.*



[12] THAUNAT M, BESSIERE C, PUJOL N, BOISRENOULT P, BEAUFILS P. Recession wedge trochleoplasty as an additional procedure in the surgical treatment of patellar instability with major trochlear dysplasia: early results. *Orthop Traumatol Surg Res 2011;97: 833-45.*

[13] BLØND L, SCHÖTTLE PB. The arthroscopic deepening trochleoplasty. *Knee Surg Sports Traumatol Arthrosc 2010;18: 480-5.*

[14] DEJOUR D, LE COULTRE B. Osteotomies in patello-femoral instabilities. *Sports Med Arthrosc 2007; 15: 39-46.*

[15] ALBEE FH. Bone graft wedge in the treatment of habitual dislocation of the patella. *Med Record 1915; 88: 257.*

[16] MASSE Y. Trochleoplasty. Restoration of the intercondylar groove in subluxations and dislocations of the patella. *Rev Chir Orthop Reparatrice Appar Mot 1978; 64: 3-17.*

[17] DEJOUR H, WALCH G, NEYRET P, ADELEINE P. Dysplasia of the femoral trochlea. *Rev Chir Orthop Reparatrice Appar Mot 1990; 76: 45-54.*

[18] DEJOUR D, SAGGIN P. The sulcus deepening trochleoplasty-the Lyon's procedure. *Int Orthop 2010; 34: 311-6.*

[19] BEREITER H, GAUTIER E. Die trochleaplastik als chirurgische Therapie der rezidivierenden Patellaluxation bei Trochleadysplasie des Femurs. *Arthroskopie 1994; 7.*

[20] GOUTALLIER D, RAOU D, VAN DRIESSCHE S. Retro-trochlear wedge reduction trochleoplasty for the treatment of painful patella syndrome with protruding trochleae. Technical note and early results. *Rev Chir Orthop Reparatrice Appar Mot 2002; 88: 678-85.*

[21] CATON J, DESCHAMPS G, CHAMBAT P, LERAT JL, DEJOUR H. Patella infera. Apropos of 128 cases. *Rev Chir Orthop Reparatrice Appar Mot 1982; 68: 317-25.*

[22] COLVIN AC, WEST RV. Patellar instability. *J Bone Joint Surg Am 2008; 90: 2751-62.*

[23] IRRGANG JJ, ANDERSON AF, BOLAND AL, HARNER CD, KUROSAKA M, NEYRET P *et al.* Development and validation of the international knee documentation committee subjective knee form. *Am J Sports Med 2001; 29: 600-13.*

[24] DEJOUR D, SAGGIN P Disorders of the Patellofemoral Joint. In: Scott NW (ed) Insall & Scott Surgery of the Knee, vol 1. *Insall & Scott Surgery of the Knee, 5edn. Elsevier, Philadelhia (2012).*

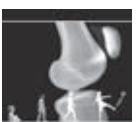
[25] SCHOETTLE PB, ZANETTI M, SEIFERT B, PFIRRMANN CW, FUCENTESE SF, ROMERO J. The tibial tuberosity-trochlear groove distance; a comparative study between CT and MRI scanning. *Knee 2006; 13: 26-31.*

[26] AMIS AA, OGUZ C, BULL AM, SENAVONGSE W, DEJOUR D. The effect of trochleoplasty on patellar stability and kinematics: a biomechanical study in vitro. *J Bone Joint Surg Br 2008; 90: 864-9.*

[27] MAENPAA H, LEHTO MU. Patellofemoral osteoarthritis after patellar dislocation. *Clin Orthop Relat Res 1997: 156-62.*

[28] SILLANPAA PJ, MATTILA VM, VISURI T, MAENPAA H, PIHLAJAMAKI H. Patellofemoral osteoarthritis in patients with operative treatment for patellar dislocation: a magnetic resonance-based analysis. *Knee Surg Sports Traumatol Arthrosc 2011; 19: 230-5.*

[29] ARNBJORNSSON A, EGUND N, RYDLING O, STOCKERUP R, RYD L. The natural history of recurrent dislocation of the patella. Long-term results of conservative and operative treatment. *J Bone Joint Surg Br 1992; 74: 140-2.*



RECESSION WEDGE TROCHLEOPLASTY FOR MAJOR TROCHLEAR DYSPLASIA

M. THAUNAT, C. BESSIERE, N. PUJOL, P. BOISRENOULT, P BEAUFILS

INTRODUCTION

The importance of dysplastic trochlea as a component of patellar instability has long been recognized. Elevation of the lateral trochlear facet was first described by Albee [1] in 1915. However, lateral trochlear elevation fell out of favor because of subsequent patellofemoral osteoarthritis. In 1966, operations to correct the abnormality by deepening the sulcus were introduced by Masse [2], who suggested removing subchondral bone and impacting the articular cartilage with a punch. This technique was later modified by Henri Dejour [3], who performed osteotomy of both femoral condyles to create a V-shaped trochlear groove. Von Knoch *et al.* [4] described another technique, known as "the Bereiter technique", in which an osteochondral flap is raised from the trochlea and a bony sulcus is fashioned using burrs; the flaps are then depressed, making a smooth groove, and fixed by vicryl tape; the technique was later described by Blønd and Schöttle [5] under arthroscopic control. A third type of trochleoplasty was described by Goutallier *et al.* [6], who performed "recession" trochleoplasty, whereby the prominent groove is recessed to the level of the anterior femoral cortex, without deepening the groove itself. This procedure is technically less demanding than a deepening trochleoplasty. Trochlear wedge recession decreases patellofemoral compression by increasing the angle between the quadriceps muscle force and the patellar tendon force (fig. 1). The aim is not to fashion a groove, but to reduce the bump without modifying patellofemoral congruence.

The purpose of the present short-term retrospective study was to determine whether recession wedge trochleoplasty according to the Goutallier technique was suitable for the surgical treatment of patellar instability with major dysplastic trochlea, defined as a domed, rather than flat or shallow, chondral surface of the proximal trochlea. It was hypothesized that

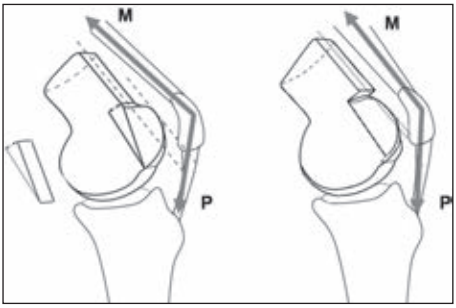
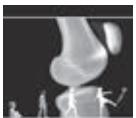


Fig. 1: Depression trochleoplasty according to the Goutallier technique [12]: the aim is not to fashion a groove but to reduce the bump without modifying patellofemoral congruence. The trochlear wedge recession decreases patellofemoral compression by increasing the angle between the quadriceps muscle force (M) and the patellar tendon force (P).



recession wedge trochleoplasty is a safe procedure for patellar instability with major dysplastic trochlea, and effective in preventing future patellar dislocation and in reducing anterior knee pain. Since this procedure may reduce patellofemoral joint reaction force without modifying patellofemoral congruence, it should also prevent late patellofemoral degeneration.

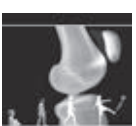
METHODS

Patients

Between April 2004 and March 2009, 20 depression trochleoplasties were performed by three different surgeons in our department on 18 patients (10 female, 8 male; 13 left knees, 7 right knees). One patient could not be contacted for reassessment and was considered lost to follow-up; 17 patients (19 knees) were assessed at last follow-up. Mean age at operation was 23 years (range, 18 to 45 yrs). Mean symptom duration before trochleoplasty was 7 years (range, 1 to 19 yrs); 7 knees had a total of 15 previous operations (Table 1). Indications for surgery were either patellofemoral instability (n=14) or pain (n=5). Patellofemoral instability was defined as recurrent subluxation (n=3) or recurrent dislocation (n=11) associated with dysplasia of the femoral trochlea. Dysplasia was defined by positive crossing sign and a trochlear bump, according to the Dejour and Walch classification (fig. 2) [7]. Persistent retropatellar pain and crepitus due to trochlear dysplasia was the indication in the remaining patients, who experienced either only 1 dislocation (n=4) or apprehension without dislocation (n=1). All patients had failed to respond to conservative treatment. Trochleoplasty was never isolated but associated to either realignment of the extensor apparatus or MPFL reconstruction, according to the "à la carte" surgery concept. Surgical treatment addressed each of 4 principal factors whenever present: trochlear dysplasia, tibial tubercle-trochlear groove offset (TT-TG), patella alta, and/or patella tilt. All patients were assessed clinically and had plain radiographs and CT scans. Plain radiographs in this study included weight-bearing AP view, non-weight-bearing lateral view and Merchant view. As long leg standing X-ray was not included in the preoperative planning, the mechanical axis of the leg could not be determined. Lateral views were used to measure patellar height, according to the Caton-Deschamps method [8] (normal range, 0.6 to 1.2), and trochlear dysplasia. Merchant views were used to measure patellar tilt angle (PTA), the angle subtended by a line between the edges of the patella and the horizontal; a line along the subchondral bone of the anterior patella can be substituted for the edge-to-edge line [9]. CT was used to measure

No	Gender	Age	Side	Length of symptoms (y)	Previous operations	Clinical findings				Length of follow-up (y)
						Apprehension	J-shaped	Dislocations	Pain	
1.	F	18	R	2	None	++	+	2	0	1
2.	F	30	L	3	Albee trochleoplasty	+	+	subluxation	+	3
3.	F	19	L	3	None	++	+	3	0	3
			R	4	None	++	+	1	I	2
4.	M	22	L	4	None	++	N/A	>10	0	4
5.	F	45	L	22	Lateral release+Elmslie Trillat	++	0	subluxation	+	6
6.	F	41	L	7	None	+	0	1	+	5
7.	M	29	L	14	arthroscopy, Emslie Trillat	+	+	0	+	2
8.	M	24	L	5	None	++	+	>10	0	7
9.	M	39	R	15	None	++	+	2	0	3
10.	F	20	R	9	None	++	+	6	+	1
11.	M	24	R	1	None	++	+	subluxation	0	5
12.	M	21	L	1	None	++	+	3	0	4
13.	M	32	L	5	Elmslie Trillat	++	+	>10	+	3
14.	F	19	L	7	Medial reefing	++	+	7	+	2
			R	7	Lateral release	++	+	5	+	1
15.	F	36	L	16	3 Emslie Trillat	++	+	>10	+	3
16.	F	21	L	7	None	++	+	1	+	1
17.	M	26	L	3	None	++	0	1	+	1

Table 1: Patient demographics. Patellar apprehension was graded from 0 to ++: 0, no apprehension; +, discomfort on extreme lateral translation of the patellar in extension, or true apprehension with voluntary quadriceps contraction on lateral translation in extension; and ++, when the patient stops the clinician touching the patella.



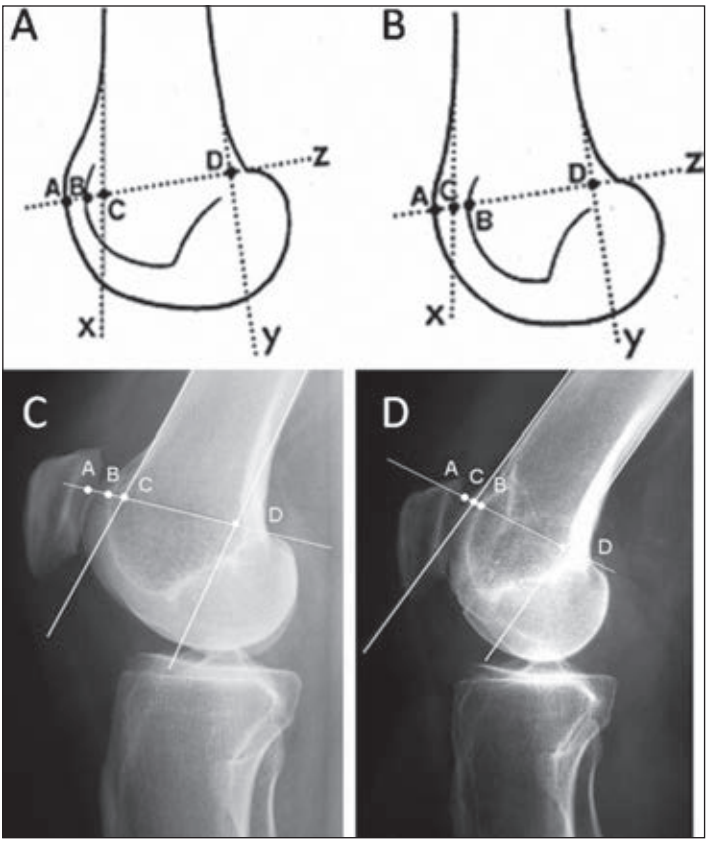
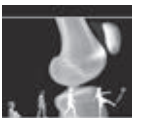


Fig. 2: Pre- and post-operative bump height measurement technique. 2A and 2B: Drawings showing the Dejour and Walch method [7] for calculating bump height. Point "D" is the junction between the posterior cortex and articular cartilage. Bump height is measured between points "B" and "C". 2C: Pre-operative lateral radiograph: the bump height is positive. 2D: Postoperative lateral radiograph: the bump height is now negative.

tibial tubercle offset from the trochlear groove (TT-TG distance) [10], to assess severity of trochlear dysplasia on the horizontal cuts according to the Dejour system [11] (*grade A:* crossing sign and shallow trochlea; *grade B:* crossing sign, supratrochlear spur, flat trochlea; *grade C:* crossing sign, double contour ("double shape"), asymmetry of trochlear facets; *grade D*: grade B + C, asymmetry of trochlear facets, and cliff pattern) and to measure PTA, subtended on CT scans by a line from one corner of the patella to the other and measured as the deviation of this line from the posterior femoral condyles.

Operative technique

The patient was positioned supine. The procedure was carried out under tourniquet. Approach was made just lateral to the patella, across the tibial tubercle (TT), and more distally along the anterior ridge of the tibia in order to perform TT transfer in the same step if required. The technique aimed to treat the underlying anatomical abnormality without compromising the articular surface. This operation was combined with proximal or distal realignment to allow improved patellar tracking and limit the risk of recurrence of patellar instability.



Thus, TT-TG distance and Caton-Deschamps index were addressed if outside the normal range; this included TT distalization for functional patella alta if the Caton-Deschamps index was greater than 1.2 and TT medialization in case of lateral patellar maltracking angle or when tuberosity offset from the trochlear groove was greater than 20 mm. MPFL reconstruction was also performed in case of increased lateral patellar mobility with positive apprehension test or in case of abnormal patellar tilt (greater than 20°) on CT. The lateral retinaculum was exposed, and resected with a 10 blade. The synovium was excised and all additional tethering tissue was released proximally and distally. The method for determining correction angle and wedge size included a pre-operative radiological plan and intra-operative measurement (fig. 3). Osteotomies were drawn on the bone with a dermographic pen according to the pre-operative plan. The anteroposterior cut was performed first, using a reciprocal saw. Then the posterior cut was made, strictly parallel to the frontal plane of the femur, from the lateral side, and directed medially; it was found safer

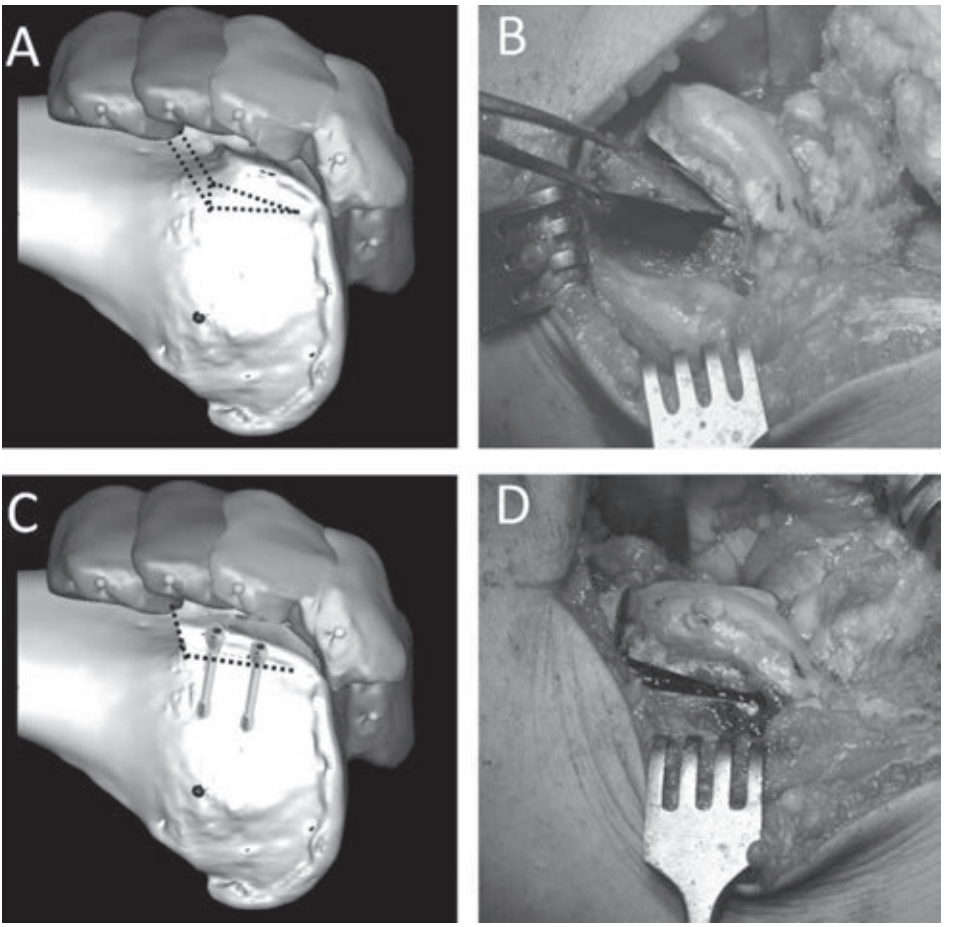


Fig. 3: Recession wedge trochleoplasty surgical technique. 3A and 3B: The base of the wedge which is removed should be the same in millimeters as the value of the trochlear bump, in order to allow the trochlea to settle into a deeper position, without modifying the trochlear groove. 3C and 3D: Correction is obtained after removal of the proximally based wedge by progressively applying pressure on the trochlea. Fixation uses two 3.5mm cancellous screws, positioned just laterally to the cartilage surface.

to begin the cut with a rigid osteotome and to complete it with the saw. The end of the cut ended approximately 5mm from the sulcus terminalis, in order to respect the distal osteochondral hinge and facilitate wedge closure. Then the anterior slanting cut was made so as to join the first two cuts. Osteotomy was completed and correction obtained after removal of the proximally based wedge by progressively applying digital pressure to the trochlea. The amount of bone removed was just enough to allow the trochlea to settle into a deeper position, without modifying the trochlear groove. Fixation used 3.5mm cancellous screws, positioned just laterally to the cartilage surface with heads which could be countersunk into the bone. Initially, 3 cancellous screws (2 lateral, 1 medial) were used; this was later changed to just 2 lateral screws, without any problem to date. Patients were managed in an extension brace for the first 6 weeks postoperatively. They were allowed to mobilize, bearing full weight on crutches. The protocol included restricting knee flexion to 0-60° for the first 3 postoperative weeks, and then slowly increasing the range of motion by daily increments up to 90° at the 6th week.

Postoperative assessment

Postoperative assessment included clinical examination, assessment of symptoms (pain) and functional scores. Patients were followed up with plain radiographs (standing AP, lateral and Merchant views). A physical exam was performed by an independent observer; KOOS [12], IKDC and Kujala [13] scores were used. Different subgroups of patients were compared to identify predictive factors for success of trochleoplasty: with or without prior surgery; patellofemoral chondropathy observed during surgery; degenerative change on preoperative radiographs. Patellofemoral cartilage damage was assessed using the ICRS classification. Patellofemoral chondropathy at the time of the index operation was defined as an abnormal (grade 2: lesions extending down to <50% of cartilage depth) or severely abnormal aspect of the cartilage (grade 3: lesions extending down to >50% of cartilage depth or to calcified layer or blisters; or grade 4: osteochondral injury, lesions extending just through the subchondral bone-plate, or deeper defects down into trabecular bone) [14].

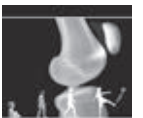
Patellar height and trochlear dysplasia were assessed on the lateral view. Measurements were performed by the same independent observer on a digitized version of the lateral X-rays, according to the technique described by Dejour [7] (fig. 2). PTA was assessed on the Merchant view [8].

RESULTS

Seven of the 19 knees had had previous surgery for instability (Table 1). Symptom duration prior to the index operation ranged from 1 to 22 years (mean, 11 years). Trochlear dysplasia was classified as grade A in 1 case, grade B in 7, grade C in 5 and grade D in 6. The patellofemoral articular surface was considered macroscopically normal at the time of operation in 14 knees and abnormal in 5 on the ICRS classification; lesions were grade 3 on the patella (n=3), grade 2 on the patella and lateral trochlea (n=1), and grade 2 on the patella (n=1). At the time of operation, all knees required additional procedures to correct instability factors (Table 2). Thirteen knees underwent TT medialization and distalization, 1 distalization only, 2 medialization only and 2 underwent TT lateralization to correct negative TT-TG caused by a previous surgery. Eight knees underwent MPFL reconstruction according to the technique described by Fithian *et al.* [15]. Minimum follow-up was 12 months, with a mean of 34 months (range, 12 to 71).

Complications

There were no intra- or peri-operative adverse events such as nerve injury, vascular problems or deep venous thrombosis. No patients showed non-union of the osteochondral block. One patient required arthrolysis for knee stiffness 1 year after the index operation: knee flexion before the second operation was 100°, and 140° after arthroscopic arthrolysis associated to



No.	Radiological findings										CT findings		intra-operative procedures				
	PH		BC (mm)		AC (mm)		PTA (°)		SA (°)		TT-TG (mm)	PTA (°)					
	Pre-op	Post-op	Pre-op	Post-op	Pre-op	Post-op	Pre-op	Post-op	Pre-op	Post-op	Pre-op	Pre-op	TD(mm)	LR	MPFL	MTT(mm)	DTT(mm)
1.	1.09	0.73	7	3	8	6	20	13	150	148	15	22	N/A	Y	N	10	10
2.	1.04	0.80	7	0	9	4	8	-1	150	148	8	17	N/A	Y	Y	0	0
3.	1.28	1.29	7	2	13	6	13	3	154	149	12	25	N/A	Y	Y	10	10
	1.80	1.25	6	1	10	7	6	5	148	147	6	15	7	Y	Y	10	12
4.	1.20	0.93	6	-1	10	3	10	2	170	154	17	20	10	Y	Y	10	8
5.	1.03	0.58	2	-5	6	1	15	9	154	150	1	2	N/A	Y	N	0	8
6.	1.03	0.76	5	-5	13	0	15	6	132	130	5	15	10	Y	N	10	5
7.	N/A	0.80	N/A	6	N/A	11	N/A	4	N/A	148	25	N/A	5	Y	N	10	0
8.	1.20	1.09	6	0	6	3	26	24	150	160	23	50	10	Y	N	10	10
9.	1.08	0.97	1	-8	8	-3	22	5	149	149	17	20	N/A	Y	Y	10	0
10.	1.06	0.91	0	-6	5	-1	18	10	145	147	22	45	N/A	Y	Y	10	7
11.	1.18	0.97	8	6	10	7	17	5	154	150	14	18	10	Y	N	10	10
12.	1.13	0.86	2	1	9	6	9	19	155	154	8	8	10	Y	N	10	10
13.	1.15	0.95	6	4	8	5	14	1	153	153	-8	6	10	Y	N	-10	0
14.	1.15	1.03	4	-3	13	5	8	2	138	149	21	4	8	Y	N	10	5
	1.22	1.13	4	0	12	7	8	2	136	136	23	2	N/A	Y	N	10	8
15.	1.00	0.54	5	-3	8	-1	9	2	140	150	-7	-6	10	Y	Y	-10	0
16.	1.20	1.00	7	-8	10	-4	20	N/A	N/A	N/A	20	N/A	10	Y	N	10	10
17.	1.30	1.00	8	0	9	2	14	2	150	157	22	30	9	Y	Y	10	7

Table 2: Pre- and post-operative radiological results and operative details. PH=patellar height, BC=Boss height, AC=Ridge height, TTTG=tibial tubercle–trochlear groove distance, PTA=patellar tilt angle, SA=sulcus angle, TD=trochlear depression, LR=lateral release, MPFL=medial patellofemoral ligament, MTT=medialization of tibial tubercle, DTT=distalization of tibial tubercle, Y=yes, N=no. Patellar apprehension was graded from 0 to ++: 0, no apprehension; +, discomfort on extreme lateral translation of the patellar in extension, or true apprehension with voluntary quadriceps contraction on lateral translation in extension; and ++, when the patient stops the clinician touching the patella.

hardware removal; the patient maintained full range of motion, and was satisfied at last follow-up. One patient required arthroscopic supratrochlear exostosectomy for a persistent ridge causing pain without symptoms of patellar instability; he too was satisfied, and had no complaint at last follow-up. A further 8 operations were needed to remove anterior tibial tubercle screws, due to pain over the screw site; screws were also systematically removed from the trochlea during the same revision operation, although well tolerated. One patient required 2 revisions for tibial tubercle non-union, which healed with a satisfactory result.

Clinical outcome

All patients except 1 were satisfied or very satisfied by the operation. Functional outcome on the various scores is summarized in Table 3. Mean objective knee score at last follow-up was 80 (±17) for the Kujala score, 70 (±18) for the KOOS and 67 (±17) for the IKDC. Patients with history of surgery, patellofemoral chondropathy discovered during surgery (ICRS grade ≥2) or degenerative change on pre-operative radiographs tended to show lower Kujala scores at last follow-up, but the difference was not significant (fig. 4).

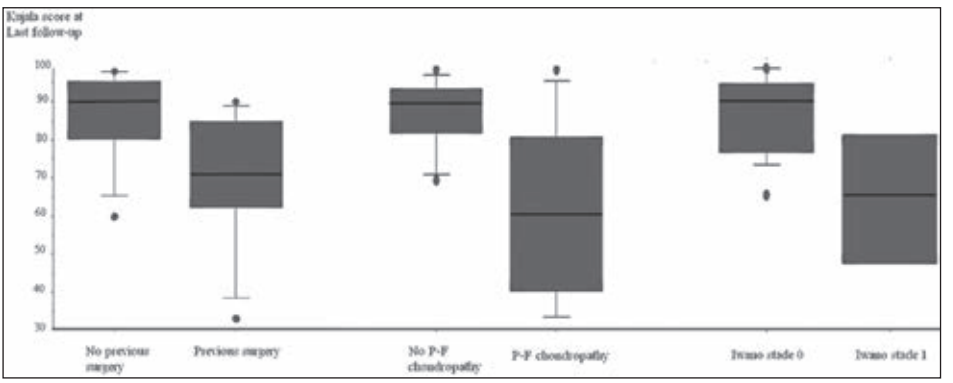
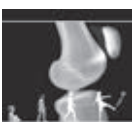


Fig. 4: Kujala score at last follow-up according to pre-operative status. Two groups were constituted for each of the 3 criteria analyzed (previous surgery, patellofemoral chondropathy, and patellofemoral osteoarthritis according to the Iwano system [10]).



All patients operated on for painfree instability (n=7) had slight pain at last follow-up. The pain was generally localized around the tibial tubercle screw sites. All patients, except 1 who complained of pre-operative pain, (n=11) reported significant pain improvement at last follow-up (Table 3).

The operation failed to stabilize the patellofemoral joint in 2 cases. One had a fall, sustaining dislocation of the patella, managed conservatively without further episodes of instability. One patient had recurrent patello-femoral instability; she had had 3 tibial tubercle transpositions before the operation but was still in pain and unstable, and remained unchanged after the index operation; she was the only patient who declared she was disappointed by the operation.

Radiological outcome

Mean trochlear groove height (BC) changed from 4.8mm (range, 0 to 8mm) pre-operatively to -0.8mm (range, -8 to 6mm) post-operatively, and mean trochlear ridge height (AC) from 9.1mm (range, 5 to 13mm) to 3.4 mm (-4 to 11mm). Mean PTA on Merchant view changed from 14° (range, 6° to 26°) to 6° (range, -1° to 24°). There was no significant difference in PTA correction according to associated MPFL reconstruction. Six patients showed radiological signs of patellofemoral osteo-arthritis at last follow-up. Radiological changes appeared in 3 patients and the 3 cases of pre-operative Iwano grade 1 [16] scored as grade 2 at last follow-up.

DISCUSSION

The most important finding of the present study was that recession wedge trochleoplasty is feasible as an additional procedure for patellar instability with major trochlear dysplasia. The ideal indication for recession wedge trochleoplasty is painful patellofemoral instability with major trochlear dysplasia (grade B, C or D on Dejour's classification, with trochlear prominence >5mm), or when other procedures have failed to provide patellofemoral stability. Depression trochleo-plasty seems to prevent future patellar dislocation and is effective in reducing anterior knee pain in these difficult primary cases or in revision when other realignment procedures have failed. It also provides improvement in radiographic measurements, including a decrease in trochlear prominence and patellar tilt angle. The present results are similar to those of several investigators in terms of improved subjective short-term outcome scores [2, 6, 17-21] (Table 4). However, comparison with other series is difficult since surgical procedures and follow-up varied, numbers were often small and patients were sometimes treated for pain rather than instability [6, 21]. It is not possible to assess the role of trochleoplasty in patellofemoral stability, as it was never

Table 3: Further postoperative procedures, clinical findings at follow-up, and satisfaction scores.

No.	Post trochleoplasty procedures		Clinical findings at follow-up			Subjective assessment	IKDC	KOOS	Kujala	Pain		PF arthritis (Iwano)	
	Removal of screws	Other	Apprehension	Recurrent instability	Crepitus					Pre-op	Last FU	Pre-op	Last FU
1.	No	None	+	0	+	Satisfied	62	81	88	No	Slight	0	0
2.	No	None	0	0	+	Satisfied	55	59	69	Moderate	Slight	0	0
3. L	Yes	None	0	0	0	Very satisfied	84	96	84	No	Slight	0	0
R	Yes	None	0	0	0	Very satisfied	83	95	92	No	Slight	0	0
4.	Yes	Athrolysis	0	0	0	Very satisfied	76	84	96	No	Slight	0	0
5.	No	None	0	0	0	Very satisfied	68	72	71	Moderate	Slight	0	1
6.	Yes	No	0	0	+	Satisfied	36	49	60	Moderate	Slight	1	2
7.	No	None	0	0	+	Satisfied	66	65	84	Moderate	Slight	1	2
8.	Yes	TT pseudarthrosis	0	0	0	Satisfied	75	62	79	No	Slight	0	0
9.	Yes	None	0	0	+	Very satisfied	71	67	90	No	Slight	0	1
10.	Yes	None	+	0	+	Satisfied	60	52	69	Moderate	Slight	0	0
11.	No	None	0	0	0	Satisfied	78	66	98	No	Slight	0	1
12.	No	None	0	0	+	Very satisfied	92	95	98	No	Slight	0	0
13.	Yes	No	0	0	+	Satisfied	82	72	90	Moderate	Slight	0	0
14.L	Yes	Trochlear exostosectomy	+	0	+	Satisfied	72	75	85	Moderate	Slight	0	0
R	Yes	None	+	+	+	Satisfied	61	65	60	Moderate	Slight	0	0
15.	Yes	None	++	+	+	Disappointed	21	23	33	Severe	Severe	1	2
16.	No	None	0	0	0	Satisfied	N/A	N/A	N/A	Moderate	Slight	N/A	N/A
17.	No	None	+	0	0	Satisfied	69	83	94	Moderate	Slight	0	0

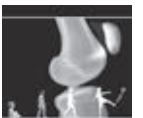


Table 4: Literature review: trochleoplasty for major dysplastic trochlea.

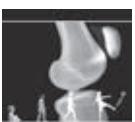
Author(s)	Year	N	F-U (months)	Previous surgery(%)	Indication		Surgical technique	Additional procedures(%)	Results		Complications	
					Pain(%)	Instability(%)			Satisfaction(%)	Failure(%)	Pain(%)	Stiffness(%)
Masse[15]	1978	18	40	?	61	100	Deepening trochleoplasty	?	?	0	11	17
Reynaud [16]	1995	40	33	50	?	97	Dejour trochleoplasty	100	77	2	?	7
Gougeon et al.[10]	1998	51	40	41	?	80	Albee (75%) deepening(25%)	?	?	2	?	11
Goutallier et al.[12]	2002	12	48	92	100	0	Recession wedge trochleoplasty	84	67	8	83	0
Verdonk et al.[25]	2005	13	18	77	100	54	Dejour trochleoplasty	23	77	0	46	46
Von Knoch et al.[26]	2006	45	96	33	77	100	Bereiter trochleoplasty	?	100	0	91	0
Donell et al. [8]	2006	15	36	60	?	100	Modified Dejour trochleoplasty	100	80	0	?	33
Utting et al. [24]	2008	59	24	30	?	100	Bereiter trochleoplasty	49	92	0	15	2
Our study	2010	20	34	40	60	100	Recession wedge trochleoplasty	100	94	10	100	5

isolated and associated abnormalities were always corrected in the same surgical step. We think, however, that correcting trochlear depth abnormality plays a major role in stabilizing the patella by facilitating proper entrance of the patella into the trochlea. Depression trochleoplasty was found to enable PTA correction even when the MPFL was not reconstructed, and is effective in revision when other procedures had failed, as reported by Goutallier *et al.* [6]. Thus, MPFL reconstruction should not be necessary when recession wedge trochleoplasty has been performed, as the reduction in trochlear prominence prevents lateral misdirection and facilitates patellar sliding into the trochlear recess. The instability recurrence rate of around 10% (n=2) in the present series was acceptable, given that in 1 of the cases trauma was implicated in the recurrent patellar dislocation, and the other was a difficult multi-operated knee in which three previous stabilization procedures had already failed.

Deepening trochleoplasty is often not attempted because of surgeons' limited familiarity with this demanding surgical technique. There is, moreover, no reliable landmark to determine where to locate the new trochlear groove. In comparison, recession wedge trochleoplasty requires assessment of the abnormal geometry, but is not difficult to master. Wedge recession is identical in principle to deepening trochleoplasty, except that a wedge rather than a trench of bone is removed so as to create a new sulcus. It was first described by Goutallier and early results were encouraging [6]. The aim is to lower the subchondral bone of the trochlear groove at the anterior cortex of the femoral shaft without modifying its shape.

A study performed on fetuses by Glard *et al.* suggested that the anatomic characteristics of the patellar groove were integrated into the genome during the course of evolution [22]. This would be in favor of a genetic origin for patellar groove dysplasia. Postnatally, the position of the patella in relation to the trochlea plays a major role in the final shape of the patella and trochlea, which develop congruent articulating surfaces [23, 24]. Moreover, there is a difference between the bony and cartilage morphology of the patellofemoral joint [25, 26], so that congruent cartilaginous articulation may coexist with underlying bony incongruence. From this point of view, lowering without deepening the groove facilitates the entry of the patella into its groove with respect to patellofemoral congruence. The aim of the procedure is to diminish the central bump responsible for patellar misdirection and lateral subluxation and to guarantee adequate trochlear depth, and maximal hyaline cartilage conservation without affecting patellofemoral cartilage congruence.

The risks associated with deepening trochleoplasty include breaking the osteochondral flap, distal detachment, and excessive thinning of the flap, decreasing blood supply. There are also concerns about articular cartilage viability following trochleoplasty. Recession wedge trochleoplasty should decrease the risk of chondral damage and necrosis. Since the dysplastic segment of the trochlea is lifted as a single osteochondral block and there is no need to fashion a new groove by cutting the osteochondral flap, it is possible to conserve more subchondral bone, thus decreasing the risk of possible severe irreversible articular and subchondral injury, especially in older patients where cartilage is less flexible. Moreover, the wedge and trochlear recess are flat and complementary, whereas in deepening trochleoplasty, the osteochondral flap might not tally

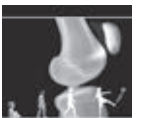


perfectly with the V-shaped recipient bed. Areas of slight cohesion between cut surfaces, and the use of suture instead of screws to secure the flap could slow down osseointegration. However, these considerations are theoretical, as chondrolysis has in fact never been observed in deepening trochleoplasty. Articular cartilage is avascular, and keeping thick flaps protects subchondral bone and theoretically allows early vascularization through the cancellous bone. Schöttle, studying cartilage viability after Bereiter trochleoplasty [27], found that tissue in the trochlear groove remained viable, conserving distinctive hyaline architecture and composition with only a few minor changes in the calcified layers.

The present study, however, has several weaknesses. In the absence of a control group, it was not possible to know whether the additional procedure provided any additional benefit. It was not possible to assess the role of trochleoplasty in the patellofemoral stability, as it was never isolated but systematically associated to other corrections within the same surgical step. The study was retrospective, with short-term follow-up, and patients were heterogeneous. Some had already had several operations which had failed, or already showed patellofemoral degeneration at the time of surgery. Although multi-operated patients had lower functional scores at last follow-up than those operated for the first time, they too were satisfied with their result. None of the patients was completely pain-free at last follow-up, although symptoms and functional activity improved. Patients operated for pain-free instability were satisfied overall, although all reported slight pain at last follow-up. This pain was mostly difficult to attribute directly to the trochleoplasty, due to the multiplicity of associated procedures. However, trochleoplasty should be considered with caution in this indication, not being actually necessary for consistent success with surgical treatment of recurrent patellar dislocation despite dysplastic trochlea. Two authors reported the results of reconstruction of the medial patellofemoral ligament in patients with trochlear dysplasia [28, 29]: there was no significant association between severity of dysplasia and Kujala score in either study. Finally, depression trochleoplasty is not without disadvantages. It reduces but does not abolish the bump and convex dome shape of the trochlea, and the groove created is not deep enough for the lateral facet to block any further tendency of the patella to dislocate. In the present series, it failed to stabilize the patella in 2 cases, and 2 complications were surgery-related and required surgical revision: 1 patient required arthrolysis for postoperative knee stiffness and the other required arthroscopic exostosectomy near the trochlea for a persistent ridge. However, these 2 complications were not directly related to the specific surgical technique of depression trochleoplasty, and the rate of complications and failure remained low and quite similar to other trochleoplasty series [6, 17, 20, 21]. While recession wedge trochleoplasty was effective in reducing anterior knee pain, it did not seem to halt the progression of patellofemoral arthritis, although follow-up was too short for any definite conclusions to be drawn. At last follow-up, 6 knees showed osteoarthritic changes in the patellofemoral compartment according to the Iwano classification, which was similar to the results obtained with deepening trochleoplasty [4]; however, MRI or CT to determine postoperative cartilage status were not systematically performed at last follow-up, and these results must be interpreted with caution. Future studies with larger numbers of patients and long-term follow-up would be needed to confirm that surgical correction of trochlear dysplasia can slow down late patellofemoral degeneration.

CONCLUSIONS

Despite encouraging clinical results, correction of trochlear dysplasia is usually not attempted because it is technically difficult, introduces a significant potential for complications and is usually not necessary for a successful outcome. However, this procedure should be considered in case of painful instability with severely dysplastic trochlea (trochlear dysplasia grade B, C or D on Dejour's classification, with trochlear prominence >5mm) or in revision



when other procedures have failed to obtain patellofemoral stability. Recession wedge trochleoplasty is a simple and safe procedure which seems to be effective in providing patellofemoral stability and pain relief. Our attitude is to perform recession wedge trochleoplasty never in isolation but in association to realignment of the extensor apparatus according to the "à la carte" surgery concept.

REFERENCES

[1] ALBEE F. The bone graft wedge in the treatment of habitual dislocation of the patella. *Med Rec 1915; 88: 257-9.*

[2] MASSE Y. Trochleoplasty. Restoration of the intercondylar groove in subluxations and dislocations of the patella. *Rev Chir Orthop 1978; 64: 3-17.*

[3] DEJOUR D, SAGGIN P. The sulcus deepening trochleoplasty. The Lyon's procedure. *Int Orthop 2010; 34: 311-16.*

[4] VON KNOCH F, BOHM T, BURGI ML, VON KNOCH M, BEREITER H. Trochleaplasty for recurrent patellar dislocation in association with trochlear dysplasia. A 4- to 14-year follow-up study. *J Bone Joint Surg (Br) 2006; 88: 1331-35.*

[5] BLØND L, SCHÖTTLE PB. The arthroscopic deepening trochleoplasty. *Knee Surg Sports Traumatol Arthrosc 2010; 18: 480-5.*

[6] GOUTALLIER D, RAOU D, VAN DRIESSCHE S. Retro-trochlear wedge reduction trochleoplasty for the treatment of painful patella syndrome with protruding trochleae. Technical note and early results. *Rev Chir Orthop 2002; 88: 678-85.*

[7] DEJOUR H, WALCH G, NEYRET P, ADELEINE P. Dysplasia of the femoral trochlea. *Rev Chir Orthop 1990;76: 45-54.*

[8] GRELSAMER RP, BAZOS AN, PROCTOR CS. Radiographic analysis of patellar tilt. *J Bone Joint Surg (Br) 1993 ;75(5): 822-24.*

[9] CATON J, DESCHAMPS G, CHAMBAT P, LERAT JL, DEJOUR H. Patella infera. A propos of 128 cases. *Rev Chir Orthop. 1982; 68(5): 317-25.*

[10] GOUTALLIER D, BERNAGEAU J, LECUDONNEC B. The measurement of the tibial tuberosity. Patella groove distance: technique and results. *Rev Chir Orthop. 1978; 64(5): 423-8.*

[11] DEJOUR D, LE COULTRE B. Osteotomies in patello-femoral instabilities. *Sports Med Arthrosc 2007; 15: 39-46.*

[12] ORNETTI P, PARRATTE S, GOSSEC L, TAVERNIER C, ARGENSON JN, ROOS EM, GUILLEMIN F, MAILLEFERT JF. Cross-cultural adaptation and validation of the French version of the Knee injury and Osteoarthritis Outcome Score (KOOS) in knee osteoarthritis patients. *Osteoarthritis Cartilage 2008; 16: 423-28.*

[13] KUJALA UM, JAAKKOLA LH, KOSKINEN SK, TAIMELA S, HURME M, NELIMARKKA O. Scoring of patellofemoral disorders. *Arthroscopy 1993; 9(2): 159-63.*

[14] BRITTBERG M, WINALSKI CS. Evaluation of cartilage injuries and repair. *J Bone Joint Surg (Am) 2003; 85-A Suppl 2: 58-69.*

[15] DAVIS DK, FITHIAN DC. Techniques of medial retinacular repair and reconstruction. *Clin Orthop Relat Res 2002; 402: 38-52.*

[16] IWANO T, KUROSAWA H, TOKUYAMA H, HOSHIKAWA Y. Roentgenographic and clinical findings of patellofemoral osteoarthrosis. With special reference to its relationship to femorotibial osteoarthrosis and etiologic factors. *Clin Orthop Relat Res 1990; 252: 190-7.*

[17] DONELL ST, JOSEPH G, HING CB, MARSHALL TJ. Modified Dejour trochleoplasty for severe dysplasia: operative technique and early clinical results. *Knee 2006; 13: 266-73.*

[18] GOUGEON F, VANOVERMEIRE P, MIGAUD H, DEBROUCKER MJ, SPIERS A, DUQUENNOY A. Résultats après 3 ans de recul de 51 trochléoplasties pour instabilité frontale fémoropatellaire. *Rev Chir Orthop 1996; 87(S2): 85-6.*

[19] REYNAUD P. Les trochleoplasties –creusement. *8e Journees Lyonnaises de chirurgie de genou 1995: 176-90.*

[20] UTTING MR, MULFORD JS, ELDRIDGE JD. A prospective evaluation of trochleoplasty for the treatment of patellofemoral dislocation and instability. *J Bone Joint Surg (Br) 2008; 90: 180-5.*

[21] VERDONK R, JANSEGERS E, STUYTS B. Trochleoplasty in dysplastic knee trochlea. *Knee Surg Sports Traumatol Arthrosc 2005; 13: 529-33.*

[22] GLARD Y, JOUVE JL, GARRON E, ADALIAN P, TARDIEU C, BOLLINI G. Anatomic study of femoral patellar groove in fetus. *J Pediatr Orthop. 2005; 25(3): 305-8.*

[23] TARDIEU C, DUPONT JY. Origine des dysplasies de la trochlea femorale. *Rev Chir Orthop 2001; 87: 373-83.*

[24] FUCENTESE SF, VON ROLL A, KOCH PP, EPARI DR, FUCHS B, SCHOTTLE PB. The patella morphology in trochlear dysplasia: a comparative MRI study. *Knee 2006; 13: 145-50.*

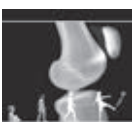
[25] SHIH YF, BULL AM, AMIS AA. The cartilaginous and osseous geometry of the femoral trochlea groove. *Knee Surg Sports Traumatol Arthrosc 2004; 12: 300-6.*

[26] STAEUBLI HU, BOSSHARD C, PORCELLINI P, RAUSCHNING W. Magnetic resonance imaging for articular cartilage: cartilage-bone mismatch. *Clin Sports Med 2002; 21: 417-33.*

[27] SCHÖTTLE PB, SCHELL H, DUDA G, WEILER A. Cartilage viability after trochleoplasty. *Knee Surg Sports Traumatol Arthrosc 2007; 15: 161-7.*

[28] CHRISTIANSEN SE, JACOBSEN BW, LUND B, LIND M. Reconstruction of the medial patellofemoral ligament with gracilis tendon autograft in transverse patellar drill holes. *Arthroscopy 2008; 24: 82-7.*

[29] THAUNAT M, ERASMUS PJ. The favourable anisometry: an original concept for medial patellofemoral ligament reconstruction. *Knee 2007; 14: 24-8.*



DEEPENING TROCHLEOPLASTY: THE LYON PROCEDURE

P.R.F. SAGGIN, P.G. NTAGIOPOULOS, P. FERRUA, D. DEJOUR

INTRODUCTION

Trochlear dysplasia is one of the major factors causing patellar instability. This anatomical abnormality is present in 96% of the people who present patellar dislocations [1]. Trochleoplasty is the procedure designed to correct the abnormal shape of the trochlea improving patellar tracking and avoiding instability.

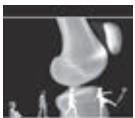
To understand the principles of modifying trochlear shape, its function must be understood. The lateral facet of the trochlea is oriented obliquely in both sagittal and coronal planes. It deviates anteriorly and laterally from the bottom of the groove. The articulating opposed lateral patellar surface follows this orientation. As the patella engages the trochlea in early flexion, a posterior directed force (patello-femoral reaction force) pushes the patella against the trochlea, and as a result of the articulating surfaces configuration, a medial vector is created, directing patellar tracking.

INDICATIONS

Trochleoplasty is indicated in patients with high-grade trochlear dysplasia and patellar instability, particularly when abnormal tracking of the patella is observed, after the second or third documented dislocation.

Image evaluation is essential to understand the trochlear dysplasia and to allow its classification. Dysplastic trochleae are shallow, flat or even convex. This is represented on lateral X-rays by the crossing sign – the groove line reaches (or crosses) the line representing the facets. Two other features typical of dysplastic trochleae on lateral views are the supratrochlear spur, located on the superolateral aspect of the trochlea, and the double contour sign, representing the medial hypoplastic facet. Based on these signs, aided by computed tomography (CT) axial views, trochlear dysplasia may be classified in four types 2:

- **Type A:** presence of crossing sign in the lateral true view. The trochlea is shallower than normal ones, but still symmetric and concave.
- **Type B:** crossing sign and trochlear spur. The trochlea is flat in axial images.
- **Type C:** presence of crossing sign and, in addition, the double-contour sign can be found on the lateral view, representing the medial hypoplastic facet. There is no spur. In axial views, the lateral facet is convex and the medial hypoplastic.
- **Type D:** combines all the mentioned signs: crossing sign, supratrochlear spur and double-contour sign. In the axial view, there is clear asymmetry of the facets height, also referred to as a cliff pattern (fig. 1).



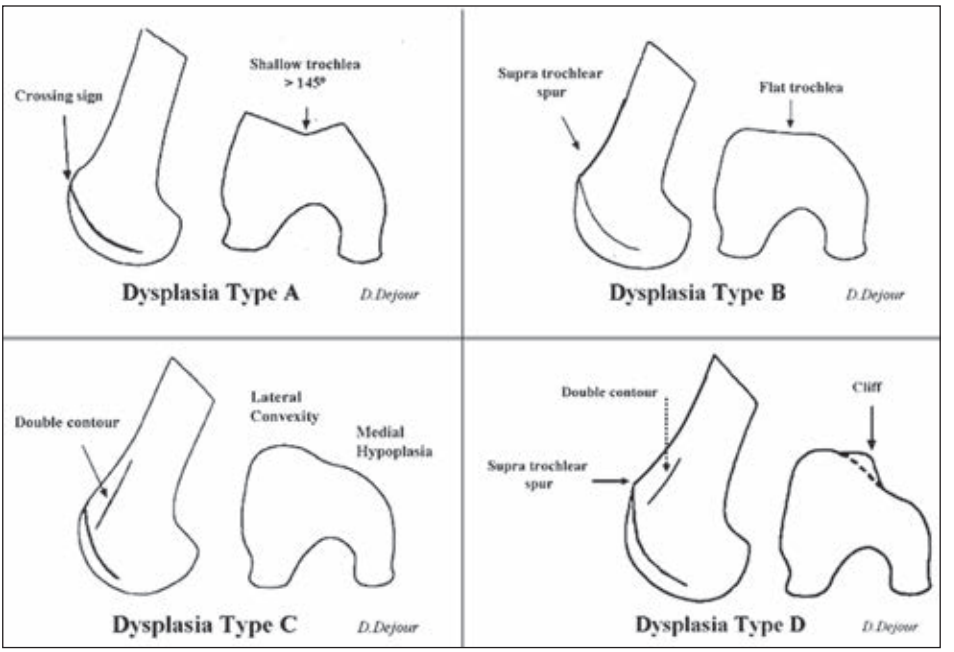


Fig. 1 : Trochlear dysplasia classification (D. Dejour).

Trochleoplasty is indicated in dysplasia types B and D, when there is prominence of the trochlea. Type A is low grade trochlear dysplasia and should not be responsible for instability, if present. Type C represents a subtype of trochlear dysplasia not suited for sulcus deepening trochleoplasty since the trochlea is already hypoplastic and bone removal is contraindicated.

Open growth plates are a contraindication to trochleoplasty. Early (or established) arthritis is another contraindication since trochleoplasty can worsen the cartilage status.

To achieve successful outcomes, associated abnormalities should also be addressed. TT-TG is corrected when trochleoplasty is carried out since the trochlear groove is moved laterally from its native location.

SURGICAL TECHNIQUE

Positioning

The patient is positioned lying supine. A lateral pad applied to the proximal thigh and a distal support under the foot allow the knee to be kept flexed by approximately 80 degrees. Full range of motion is possible during the procedure.

Exposure

A straight midline incision is performed from the superior patellar limit to the tibiofemoral articulation with the knee flexed. The arthrotomy is performed through the medial capsule. Proximally, the *vastus medialis obliquus* is split in line with its fibers 4cm

proximally into the muscle belly. After inspection, the patella is retracted laterally, providing adequate trochlear exposure.

Before starting the osseous procedure, the synovium must be removed from the edge of the trochlea. It is incised with a scalpel on the cartilage edge and a periosteal elevator is used to retract it away from the trochlear edge.

Trochlear planning

A line deviating 3 to 6 degrees laterally is drawn proximally from the intercondylar notch representing the bottom of the new groove. The lateral margins are also drawn from the intercondylar notch through the *sulcus terminalis* of each condyle. The proximal part of the new groove can be positioned according to the TT-TG value in order to correct a possible malalignment (fig. 2).

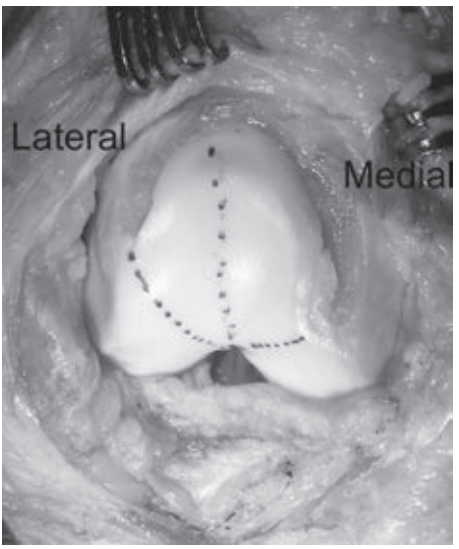


Fig. 2 : The new trochlea is planned over the native one. The central line deviating slightly laterally represents the bottom of the groove. Note that cortical bone has already been removed from the osteochondral edge.

Bone removal

A strip of cortical bone (corresponding to the amount of the bump) from the anterior femoral cortex till the cartilage edge must be removed, using a thin osteotome. This creates the access to the under surface of the trochlea and constitutes the first step to bring the new trochlea to a normal position (not prominent) and eliminate the bump.

The cancellous bone that lies under the trochlear cartilage must also be removed to reshape and reposition the new trochlea. Osteotomes and curettes are helpful in the procedure, but a drill with a specific depth guide is used for this purpose. This depth guide is set to allow bone removal from the cartilage undersurface producing a uniform cartilage-bone shell of 5 millimeters. It avoids cartilage damage and thermal necrosis which could result from excessive bone removal (fig. 3). The shell produced must be sufficiently compliant to allow modeling. Without the guide, bone must be removed especially from where the new planned sulcus will lie. All bone that projects beyond the anterior femoral cortex should be removed (fig. 4).

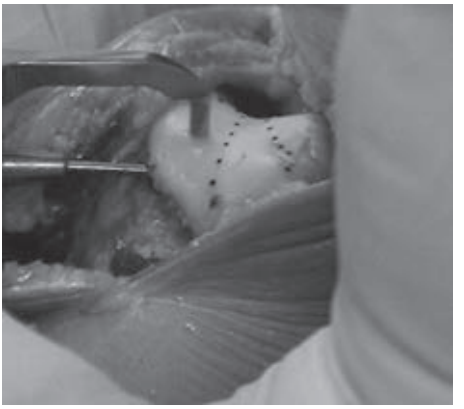
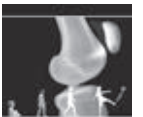


Fig. 3 : Cancellous bone removal with a drill and a specific guide.



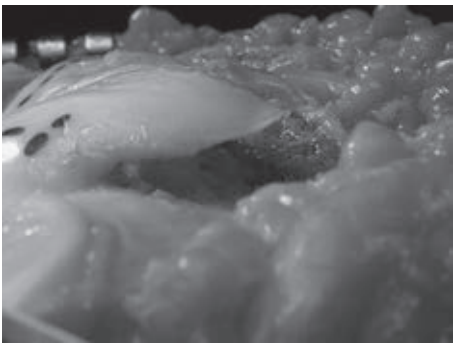


Fig. 4 : The bone from the undersurface of the trochlea has been removed. All bone that projects beyond the anterior cortex of the femur must be resected in order to eliminate the bump.

The cartilage-bone flap should sit flush on the underlying bone bed. The bone bed should be deepened in its central portion to recreate and adequate the groove. Once the flap is adequately modeled over the bone bed and the trochlear conformation is satisfactory, fixation is performed.

Fixation

Absorbable vycril sutures are used to fix the cartilage-bone flap to the underlying bone bed. One suture is passed from each facet and tied over the respective medial and lateral gutters; this allows pulling down the new groove with similar pressure of both facets on the cancellous bone, promoting a perfect healing (fig. 5).

Fig. 5 : After fixation, trochlear anatomy resembles that of normal knees.

The synovium that was formerly dissected away from the osteochondral margin is sutured back to it. This protects the patellar cartilage from the femoral bone and minimizes blood loss through the exposed cancellous bone.

Patellar tracking check-up: After satisfactory trochlear shape is achieved, the associated planned procedures are performed. We routinely perform an associated medial patellofemoral ligament reconstruction. After the complete procedure is done, patellar tracking and stability are checked.

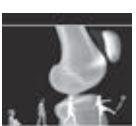
Closure

Closure of the medial retinaculum is the final step. No drains are installed.

Post-op protocol

Immobilization or weight restriction are not necessary. Early mobilization on a continuous passive motion device improves cartilage nutrition and helps further trochlear modeling by the patella. Immediate weight bearing is allowed with an extension brace and flexion must be regained without forced or painful postures. Early rehabilitation goals are pain and edema reduction and range of motion recovery. The brace is removed when the quadriceps strength allows the patient to walk. Quadriceps strengthening with weights on the foot or the tibial tubercle is prohibited in the initial phase. After 45 days, cycling with weak resistance and weight bearing proprioceptive exercises may be initiated. From the 4th to the 6th month, running can be reinitiated and quadriceps reinforcement with open kinetic chain exercises between 0 and 60 degrees and minor loads are allowed. Sports return is allowed after the 6th month.

Control X-rays are obtained postoperatively immediately and at 6 weeks. Six months after the procedure, a CT scan is performed to document the obtained correction.



COMPLICATIONS

Complications include trochlear necrosis, cartilage damage, incongruence with the patella, and hypo or hypercorrection. Trochlear necrosis seems not to occur, since the cartilage nutrition is not dependent on the underlying bone. Schottle [3] performed biopsies in three patients after trochleoplasty, showing cartilage cell viability and flap healing. He concluded that the risk of cartilage damage is low.

Incongruence with the patella is another possible complication, especially because patellar dysplasia is common in this population, but the long term outcomes are not known. Hypo or hypercorrection, along with missed associated abnormalities may cause failure of the procedure.

RESULTS

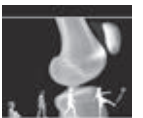
Trochleoplasty results are generally good in terms of stability and function, while the results for pain are less clear.

Fucentese *et al.* [4], using CT scans pre and post-operatively, demonstrated that trochleoplasty lateralized the trochlear groove and medialized the patella, increased the trochlear depth, decreased the lateral patellar inclination angle, decreased the sulcus angle and increased the lateral trochlear slope, thus recreating a more normal anatomy. Amis *et al.* [5] studied the patellar tracking in normal knees, after simulating dysplasia and again after performing a trochleoplasty and concluded that trochleoplasty increased stability and provided small but significant changes in patellar tracking.

Two series reviewing clinically deepening trochleoplasty were published in the *"10e Journées Lyonnaises de Chirurgie du Genou"* in 2002: The first group included 18 patients who had failed patellar surgery for instability, with a mean age of 24 years at surgery. The mean follow up was 6 years (2 to 8 years, no patients lost to follow up). The new surgery was 6 times indicated for pain and 12 times for recurrence of instability. The average number of surgeries before the trochleoplasty was 2. The deepening trochleoplasty was associated to a tibial tubercle medialization in 8 patients, in 6 to a tibial tubercle distalization and in 18 to a *vastus medialis obliquus* advancement. All patients were reviewed clinically with the IKDC form and radiographically. Sixty five percent were satisfied or very satisfied. The knee stability was rated 13 times A and 5 times B. Twenty eight percent of the patients had residual pain, and this was correlated to the cartilage status at surgery. Two patients developed patellofemoral arthritis. The mean patellar tilt was 35° (18°-48°) in the preoperative setting, and improved to 21°(11°-28°) with the quadriceps relaxed and 24° (16°-32°) with the quadriceps contracted after the surgery.

In the second group there were 44 patients. They had no antecedents of patellofemoral surgery. The mean follow up was 7 years (2 to 9 years). Twenty two tibial tubercle medializations, 26 distalizations and 32 vastus medialis obliquus advancements were associated at the time of surgery. These patients were also reviewed clinically with the IKDC form and radiographically. Eighty five percent were satisfied or very satisfied. The knee stability was rated 31 times A and 13 times B. Five percent had residual pain, but this was not correlated to the cartilage status at surgery. No patellofemoral arthritis was noted. The mean patellar tilt preoperatively was 33 ° (24°-52°), and improved postoperatively to 18° (9°-30°) with the quadriceps relaxed and 22° (14°-34°) with the quadriceps contracted.

Other authors also reviewed deepening trochleoplasty. Verdonk *et al.* [6] described 13 procedures with a mean follow-up of 18 months. Patients were assessed using the Larsen-Lauridsen score considering pain, stiffness, crepitus, flexion and loss of function. Seven patients scored poorly, three fairly and three well. On a subjective scoring system, however, six patients rated the result as very good, four as good and one as satisfactory. Only two patients found the result inadequate and would never undergo the procedure again. Thus, 77% were satisfied with the procedure. Donnel *et al.* [7] described 15 patients (17 knees) submitted to deepening trochleoplasty with a mean follow-up of 3 years.



Trochleoplasty was indicated if there was a boss greater than 6mm, and associated procedures performed as required. Of the 17 knees, 9 had undergone previous surgery for patellar instability. The boss height was reduced, postoperatively, from an average of 7.5mm to 0.7mm. Tracking became normal in 11 knees and six had a slight J-sign. Seven knees had mild residual apprehension. Seven patients were very satisfied, six were satisfied, and two were disappointed. The Kujala score improved from an average of 48 to 75.

CONCLUSION

Deepening trochleoplasty is indicated in patients with high-grade trochlear dysplasia (types B or D) and abnormal patellar tracking. It is part of the "menu à la carte" used to the correct each of the anatomical abnormalities, one by one. The technical procedure is highly demanding and prone to complications. It is, however, effective in providing stability and achieves satisfactory clinical results.

LITERATURE

[1] DEJOUR H, WALCH G, NOVE-JOSSERAND L, GUIER C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc. 1994; 2(1): 19-26.*

[2] DEJOUR D, LE COULTRE B. Osteotomies in patello-femoral instabilities. *Sports Med Arthrosc. Mar 2007; 15(1): 39-46.*

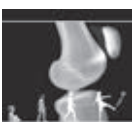
[3] SCHOTTLE PB, SCHELL H, DUDA G, WEILER A. Cartilage viability after trochleoplasty. *Knee Surg Sports Traumatol Arthrosc. Feb 2007; 15(2): 161-7.*

[4] FUCENTESE SF, SCHOTTLE PB, PFIRRMANN CW, ROMERO J. CT changes after trochleoplasty for symptomatic trochlear dysplasia. *Knee Surg Sports Traumatol Arthrosc. Feb 2007;15(2): 168-74.*

[5] AMIS AA, OGUZ C, BULL AM, SENAVONGSE W, DEJOUR D. The effect of trochleoplasty on patellar stability and kinematics: a biomechanical study in vitro. *J Bone Joint Surg Br. Jul 2008; 90(7): 864-69.*

[6] VERDONK R, JANSEGERS E, STUYTS B. Trochleoplasty in dysplastic knee trochlea. *Knee Surg Sports Traumatol Arthrosc. Oct 2005; 13(7): 529-33.*

[7] DONELL ST, JOSEPH G, HING CB, MARSHALL TJ. Modified Dejour trochleoplasty for severe dysplasia: operative technique and early clinical results. *Knee. Aug 2006; 13(4): 266-73.*



LES TROCHLÉOPLASTIES DE RELÈVEMENT

C. MAYER, G. PASQUIER, F. GOUGEON, F. RÉMY, H. MIGAUD

INTRODUCTION

A l'origine de la chirurgie de l'instabilité patellaire, nous pouvons citer les travaux de Drew et Albee [1] qui, au début du siècle, ont ébauché les premières bases, puis plusieurs techniques chirurgicales ont été proposées pour corriger les différentes anomalies anatomiques marquant cette pathologie.

Le principal facteur anatomique de l'instabilité fémoro-patellaire a été identifié comme étant la dysplasie de la trochlée.

Deux types de trochléoplasties pour corriger cette anomalie ont été développés, soit la trochléoplastie de relèvement de la facette latérale de la trochlée décrite par Albee, soit la trochléoplastie de creusement de la gorge de la trochlée décrite par Masse.

Nous rapportons ici les résultats des patients ayant eu une trochléoplastie de relèvement qui ont été opérés entre 1988 et 1995 dans le service d'orthopédie du CHRU de Lille.

Cette cohorte présente un recul moyen de 16 ans.

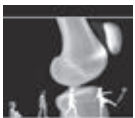
Le but de ce travail était d'évaluer à long terme les résultats de cette trochléoplastie sur l'instabilité fémoro-patellaire et accessoirement de voir l'apparition d'arthrose.

MATERIEL ET METHODE

Tous les patients ont été revus par le même examinateur qui n'avait pas participé aux interventions.

Nous avons revu 31 patients (37 genoux), au recul moyen de 16 ans (11 ; 21), avec un sex-ratio (H/F) de 0,23, ayant eu une trochléoplastie de ce type entre 1988 et 1995, à l'âge de 28 ans en moyenne (6 ; 16). Vingt-huit patients étaient porteurs d'une instabilité rotulienne objective (luxations récidivantes), neuf porteurs d'une instabilité potentielle. Toutes les trochlées étaient dysplasiques et selon la classification de D. Dejour, il y avait 10 types A, 2 types B, 23 types C, et 2 types D. Douze patients étaient en échec d'une transposition isolée de la tubérosité tibiale et gardaient une instabilité rotulienne.

Par une voie d'abord antérieure et puis une arthrotomie latérale, l'intervention consistait, en un relèvement, de 5 mm en moyenne, de la berge externe de la trochlée, gardant les charnières médiales et inférieures, comblés avec une autogreffe prise aux dépens du tibial ipsilatéral (tubercule de Gerdy). La fixation de l'ostéotomie était assurée par une ostéosuture. Les genoux étaient ensuite immobilisés dans une attelle amovible en extension. La rééducation était entreprise dès le lendemain de



l'opération, initialement par un réveil musculaire puis par une mobilisation passive et douce du genou.

10 genoux n'avaient pas eu de geste sur la tubérosité. Une médialisation pure selon la technique d'Elmslie-Trillat a été faite chez 14 patients, un abaissement isolé de la tubérosité a été réalisé dans un seul cas, et une médialisation associée à un abaissement de la tubérosité dans 10 cas. Pour deux patients, une deuxième transposition de tubérosité tibiale antérieure a dû être réalisée.

Une plastie interne du muscle vaste médial a été utilisée dans 18 cas, 13 patients ont eu une plastie du muscle Sartorius et 6 patients n'ont pas eu de plastie musculaire interne. 7 patients ont été opérés d'une plastie du muscle vaste latéral.

Nous avons évalué, le degré de satisfaction (quatre réponses possibles : très satisfait, plutôt satisfait, déçu, mécontent), la question : "Le referiez-vous, en sachant les suites ?" (deux réponses possibles : oui ou non) et la fiche d'évaluation fonctionnelle lilloise de l'articulation fémoro-patellaire [6].

Cette fiche était cotée sur 100 points, qui se répartissaient pour presque la moitié (45 points) sur les deux plaintes principales de la pathologie fémoro-patellaire : l'instabilité (30 points) et la douleur (15 points) pondérée par trois points en moins en cas de prises d'antalgiques. Le reste des questions évaluait la marche (8 points), le gonflement (5 points), les blocages (5 points), les escaliers (8 points), la course (4 points), l'accroupissement (5 points), la position à genoux (5 points) et l'activité fonctionnelle dans la vie courante (5 points) et au travail avec le sport (5 points).

Un bilan radiographique standard a été réalisé en préopératoire ainsi qu'en postopératoire qui comportait un cliché genou de face en appui monopodal, genou en extension, un genou de face en schuss, un genou de profil strict à 30° de flexion et un cliché axial en défilé fémoro-patellaire 30° de flexion.

Un bilan tomodensitométrique a été réalisé en pré et postopératoire mesurant la T.A-G.T., la pente trochléenne externe ou obliquité de la joue latérale de la trochlée, l'antéversion fémorale et torsion tibiale externe, la mesure de l'angle trochléen (la valeur normale de l'angle était de 145° calculée sur la coupe à l'arc roman) et la bascule rotulienne quadriceps décontracté et contracté.

RÉSULTATS

Trente-sept genoux ont pu être revus, chez trente et un patients, avec un recul moyen de 16 ± 2,4 ans, (11 ; 20).

Résultats cliniques

Satisfaction

92 % des interventions sur les genoux à la révision, étaient évaluées par les patients de très satisfaisants (57 %) ou de satisfaisants (35 %).

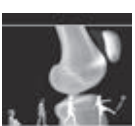
Deux patients étaient déçus, (une hypoesthésie dans le territoire du nerf saphène et un pour des accrochages au fémoro-patellaire) et un seul patient était mécontent de son genou, il souffrait d'une fibromyalgie et de pathologies digestives lourdes.

A la question "le referiez-vous, en sachant les suites ?", la grande majorité des genoux le referait, seuls deux patients (deux genoux) ne le conseilleraient pas.

Scores fonctionnels lillois

La moyenne est à 68/100, avec très bon résultat : 10 genoux, bon résultat : 5 genoux, moyen résultat : 7 genoux, résultat passable : 5 genoux, mauvais résultat : 10 genoux (27 %).

Soit autant de "très bon" et "bon" résultats (41 %) que de résultats "passable" et "mauvais", à la révision (41 %).



La stabilité

Au recul, nous retrouvons : genou stable : 18 genoux, dérobement occasionnel pendant le sport : 11 genoux, dérobement occasionnel pendant la vie courante : 3 genoux, dérobement fréquent pendant la vie courante : 4 genoux, au moins une luxation : 1 genou, plus de 2 luxations : 0 genou (0 %).

La seule récidive de luxation a été observée chez un patient qui présentait des séquelles d'une méningite avec des crises d'hypertonie.

Il y a une amélioration significativement pour la stabilité, elle a augmenté de 3 niveaux (3,4 ± 1,7), les patients sont PLUS stables à la révision (p=0,03).

Tous les patients se sont trouvés améliorés du point de vue de la stabilité, sauf deux genoux qui se sont trouvés inchangés. Ces 2 genoux avaient été repris, l'un par une prothèse fémoro-patellaire et l'autre par une ostéotomie réalisée dans un autre centre. 54 % des genoux ont gagné quatre niveaux et plus.

Douleur

La répartition se faisait comme suit : aucune : 12 genoux, légère, occasionnelle : 12 genoux, modéré mécanique : 6 genoux, importante mécanique : 4 genoux, constante sévère : 3 genoux.

A la révision, 64 % des genoux étaient indolores ou ne présentaient que des douleurs légères. 3 genoux présentaient des douleurs sévères, il s'agissait d'un patient fibromyalgique, d'un autre avec la maladie d'Ehler-Danlos et d'un patient présentant des douleurs au niveau de l'aileron latéral sans arthrose sur la fémoro-patellaire à la radiographie.

Tous les genoux pouvaient prendre les escaliers sauf 2, qui étaient des reprises, une par prothèse fémoro-patellaire et l'autre par une OTV.

Il y a une baisse significative de la douleur entre la révision à 16 ans et le préopératoire. Le gain moyen était de 1 niveau (0,92 ± 1,7) : les patients sont MOINS douloureux à la révision (p=0,02).

Blocage

68 % des genoux ne présentaient jamais de blocage, 24 % avaient plutôt des accrochages et trois genoux (8 %) avaient des blocages occasionnels (tous repris dans les suites).

Résultats radiologiques

36 genoux ont été revus avec des radiographies, cela est dû au fait qu'une patiente était enceinte à la révision. 4 genoux avaient eu une prothèse fémoro-patellaire et donc, les mesures de la saillie, de la profondeur et la quantification de l'arthrose n'ont pas pu être faites sur leurs radiographies.

La saillie a diminué, et la profondeur a augmenté.

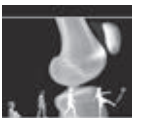
La hauteur rotulienne moyenne calculée selon l'indice de Caton-Deschamps était de 1,02 ± 0,15 mm (0,6 ; 1,3). La différence par rapport au préopératoire est de 0,13.

Arthrose fémoro-patellaire

27 genoux présentaient une arthrose fémoro-patellaire, soit 84 % des genoux non repris par prothèse fémoro-patellaire.

Nous retrouvions selon la classification d'IWANO :
- *Stade 1 :* 12 genoux (38 %)
- *Stade 2 :* 8 genoux (25 %)
- *Stade 3 :* 4 genoux (13 %)
- *Stade 4 :* 3 genoux (9 %)
- *Pas de signe dégénératif :* 5 genoux (16 %)

Nous observons une forte proportion d'arthrose fémoro-patellaire, mais, pour 62 % d'entre eux, à un stade peu évolué (stades 1 + 2).



Du point de vue localisation, 28 % avaient une atteinte fémoro-patellaire médiale, 31 % une atteinte latérale, et 25 % étaient touchés de manière globale au niveau fémoro-patellaire.

En comptant les prothèses fémoro-patellaires comme arthrosique, on arrive à 86 % d'arthrose fémoro-patellaire à la révision.

19 genoux avaient des ostéophytes au niveau de la fémoro-patellaire, localisés juste au niveau de la rotule pour 9 genoux (28 %) et de manière bilatérale (rotule et trochlée) chez 10 genoux (31 %).

Il y a une différence significative concernant l'arthrose fémoro-patellaire à la révision ($p<10^{-4}$) : les patients sont plus arthrosiques au niveau de l'articulation fémoro-patellaire à la révision.

Devant le nombre important d'arthrose fémoro-patellaire, il n'a pas pu être mis en évidence de corrélation statistique avec les antécédents, le type d'instabilité, l'âge à l'intervention, l'obliquité de la berge externe, à la différence d'obliquité entre la révision à 16 ans et le préopératoire, l'angle trochléen à la révision calculé au scanner, différence angle trochléen entre la révision à 16 ans et le préopératoire ou l'indice de satisfaction.

Arthrose fémoro-tibial

Sur le cliché de face, 15 genoux (42 %) avaient un début d'arthrose, n'atteignant jamais le stade 2 d'Ahlbäck.

L'atteinte était médiale isolée chez 10 genoux avec pour 9 genoux un pincement <50 % et pour 1 genou, un genou avec un ostéophyte fémoral. L'atteinte était latérale isolée chez 4 genoux avec pour 2 genoux un pincement <50 %, et pour 1 genou, 2 genoux avec des ostéophytes fémoraux. L'atteinte était médiale et latérale chez 1 genou un pincement <50 % des deux compartiments.

Résultats tomographiques

TA-GT

La TA-GT était de 12,2 ± 4,9 mm (0 ; 20). La différence par rapport à la mesure préopératoire est de - 2,2 mm.

Bascule rotulienne décontractée (TDM)

La bascule rotulienne avec le quadriceps décontracté était de 8° ± 6 (0 ; 27). La différence par rapport au préopératoire était de - 9,2°.

Bascule rotulienne contractée (TDM)

La bascule rotulienne avec le quadriceps contracté était de 13° ± 7 (0 ; 37).

Angle trochléen

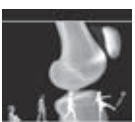
L'angle trochléen mesuré au scanner était de 141° ± 9 (extrêmes : 123 ; 156). La différence par rapport au préopératoire était de - 28°. Il restait 8 patients avec des angles trochléens supérieurs à 145°.

Obliquité joue latérale de la trochlée (pente externe)

L'obliquité de la joue latérale de la trochlée mesurée au scanner était de 16,5° ± 6,5 (3 ; 28). La différence par rapport au préopératoire est de + 10,2°.

Complications

20 genoux (54 %) ont eu des complications.
7 genoux douloureux (3 repris par une prothèse fémoro-patellaire, 2 genoux pour une OTV,



1 maladie d'Ehlers-Danlos, 1 fibromyalgie), 5 genoux raides (3 reprises par mobilisation et deux reprises par arthrolyse sous arthroscopie), 1 dérobement repris par une prothèse fémoro-patellaire, 4 hypoesthésies, 1 granulome, 1 rupture de l'insertion du muscle vaste interne post-traumatique, 1 hématome.

Les patients qui ont eu des complications à la révision ont significativement une moins bonne augmentation du score de stabilité (p=0,024). Les patients repris sont ceux qui ont eu le plus de complications ($p<10^{-4}$). 65 % des patients ayant eu une complication ont été repris.

Reprise

1re reprise

13 genoux (35 %) ont eu une reprise avec 3 mobilisations, 2 arthrolyses sous arthroscopie, 4 prothèses fémoro-patellaires, 1 exérèse de granulome, 1 arthroscopie exploratrice (Ehlers-Danlos), 1 évacuation d'hématome, 2 OTV.

2e reprise

Une mobilisation sous anesthésie générale, 6 semaines après avoir mis une prothèse fémoro-patellaire.

Un abaissement de tubérosité tibiale antérieure associé à une plastie du vaste externe, réalisée après avoir réalisé une mobilisation sous AG.

Les reprises sont liées de manière significative à l'âge du patient lors de l'intervention : les patients les plus âgés lors de l'intervention ont plus de chance d'être repris (p=0,03) ; les deux tiers des patients repris avaient plus de 30 ans, et 60 % des plus de 30 ans ont été repris.

Il y a une tendance chez les patients repris à avoir un gain de stabilité plus faible, 3 contre 5 (p=0,06).

Echecs

Nous avons considéré comme échec, les reprises par prothèses fémoro-patellaires, ou un score fonctionnel inférieur à 70 (mauvais ou passable).

Nous avons retrouvé 15 genoux considérés comme des échecs, avec 4 reprises par une prothèse fémoro-patellaire, et 11 genoux ayant des scores fonctionnels insuffisants.

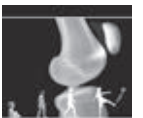
Il y a significativement plus d'échecs chez les patients avec des antécédents fémoro-patellaire, et chez les patients avec un âge à l'intervention supérieur à 30 ans.

DISCUSSION

La série possède un recul important, comparé à la littérature sur l'instabilité de rotule, c'est même le plus long, pour nous, sur les trochléoplasties [2]. Il est difficile de comparer ces résultats car les indications dans les études ne sont pas toutes identiques, les gestes associés et les techniques de rééducation sont très variés [7], mais nos résultats sont comparables voire meilleurs en terme de reprise et stabilité [3, 5]. Il existe un contraste important entre des résultats subjectifs jugés favorablement par les patients et des résultats radiographiques plus inquiétants. L'utilisation de cette technique dans des cas de dysplasie modérée a un effet stabilisateur efficace comme le montre cette série mais au prix d'un fréquent retentissement articulaire osseux qui explique qu'on mette en balance ce traitement avec une stabilisation ligamentaire pure.

CONCLUSION

Après une bonne analyse de la pathogénie de l'instabilité fémoro-patellaire, la trochléoplastie peut être proposée, dans certain cas, avec de bons résultats, même en cas d'antécédent de chirurgie sur le genou.



Cette étude a pu mettre en évidence une complète régression de l'instabilité rotulienne. D'autre part, nous avons constaté une amélioration des douleurs qui se maintient avec le temps.

L'évolution arthrosique, tant redoutée, a été constatée chez la plupart des genoux, de manière légèrement supérieure aux études précédentes sur le traitement de l'instabilité fémoro-patellaire. Mais elle est restée peu importante au niveau de la gravité et sans conséquence au niveau du résultat sur la stabilité et les douleurs.

Il faut aussi prendre en compte l'évolution naturelle de l'instabilité fémoro-patellaire [5].

Il est généralement admis que l'existence d'une arthrose fémoro-patellaire n'est pas toujours douloureuse, mais il est admis que les trochléoplasties de relèvement doivent être évitées car elles sont arthrogènes.

Il peut encore rester des indications aux trochléoplasties de relèvement, notamment en cas de dysplasie sans éperon (grades A et C de D. Dejour).

LITTÉRATURE

[1] ALBEE F. The bone graft wedge in the treatment oh habitual dislocation of the patella. *Med. Rec. 1915;7(88): 257-9.*

[2] KOETER S, PAKVIS D, VAN LOON C, *and al.* Trochlear osteotomy for patellar instability: satisfactory minimum 2-year results in patients with dysplasia of the trochlea. *Knee Surg Sports Traumatol Arthrosc. 2007;15: 228-32.*

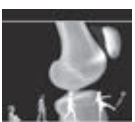
[3] KURODA R, KAMBIC H, VALDEVIT A, *and al.* Distribution of patellofemoral joint pressures after femoral trochlear osteotomy. *Knee Surg, Sports Traumatol, Arthrosc 2002;10: 33-7.*

[4] MAENPAA H, LEHTO MU. Patellofemoral osteoarthritis after patellar dislocation. *Clin Orthop. 1997; 339: 156-62.*

[5] NOMURA E, INOUE M. Cartilage lesions of the patella in recurrent patellar dislocation. *Am J sports Med. 2004. 32(2): 498-502.*

[6] RÉMY F, BESSON A, GOUGEON F, *and al.* Evaluation fonctionnelle de l'instabilité fémoro-patellaire par un score sur 100 points. *Rev Chir Orthop Repatrice Appar Mot, 1999; Supp 3: 92.*

[7] WEIKER GT, BLACK KP. The anterior femoral osteotomy for patellofemoral instability. *Am J Knee Surg. 1997;10: 221-27.*



DOES PATELLOFEMORAL OSTEOARTHRITIS MAY BE IDIOPATHIC?

A. PINAROLI

INTRODUCTION

The term arthritis refers to full thickness loss of articular cartilage and concomitant inflammation, excluding biological causes of wear and damage of cartilage (inflammatory diseases and infection).

Patellofemoral arthritis is so characterized by pain and other clinical symptoms, almost due to cartilage damages and dysfunctions of the extensor mechanism.

Lately, patellofemoral arthritis is also characterized by radiographic findings, including joint space narrowing, cysts and osteophyte formations.

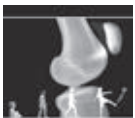
As it is clear that the patello femoral compartment can be involved in diffuse form of knee arthritis, isolated patellofemoral arthritis may not be so rare [9, 12].

Some mechanical causes are likely to include all conditions leading to isolated patellofemoral arthritis.

PATHOPHYSIOLOGY AND ETIOLOGY OF PATELLOFEMORAL OSTEOARTHRITIS

The patellofemoral joint includes the entire extensor mechanism of the knee (quadriceps tendon, patella and patellar tendon). The patella is a sesamoïd bone that acts as a marker for the alignment of the hole extensor mechanism. The trochlear groove and an arch of articular cartilage around the intercondylar notch make up the femoral side of the joint [5].

The primary soft tissue static stabilizers are the medial and lateral patellofemoral and patellotibial complexes. Patellofemoral stability is also dependent on limb alignment (varus and valgus) as well as rotational variances in femoral version. Furthermore, the relation of the knee to pelvic position and strength has been added as an important stabilizer of the knee and the patellar elements. This complex orchestration of factors often challenges to understanding patellofemoral joint function [12].



The movements of the patellofemoral joint are complex [5, 9]. One part or another of the patellar cartilage remains loaded throughout the entire flexion-extension cycle, excepted in the first degrees of flexion (the patella here rests on the synovium of the supracondylar fat pad). The distal portion of the patella is loaded as the knee flexes, and the contact area migrates proximally with progressive flexion until 90°, after which the contact area moves back toward the central aspect and lateral facet of the patella, while the medial facet lies in contact with the synovium overlying the anterior cruciate ligament.

Most activities involving knee flexion take place in a closed-kinetic-chain mode, including bending down, rising from a chair and ascending stairs, where the forces across the patellofemoral joint increase as the knee flexes from 0 to 90°, as do the contact pressures [9].

A improper fit between the two mating areas leads to an abnormal stress distribution and eventually blunt trauma.

So, while primary osteoarthritis is signaled by an imbalance between the synthesis and degradation of the matrix components, mechanical destruction of cells and matrix due to blunt trauma may be the cause of secondary osteoarthritis.

Furthermore, the articular cartilage of the patella consists of a solid phase, made up mostly of collagen and glycosaminoglycans, and a fluid phase. The solid phase is slightly permeable and when a load is applied to the articular surface, the fluid slowly redistributes itself within the solid matrix. Disruption of the articular surface by cracks, fissures or crevices leads to a loss of pressure within the fluid phase. High stresses are then borne by the collagen fibers, which become more prone to breakdown [6, 9].

Moreover, the ability of collagen to withstand high stresses has a variable genetic component, wich accounts for the wide range of clinical responses to a given joint load among different patients [9].

Etiologic factors, independently or associated, are then logical causes of isolated patellofemoral arthritis [5, 8, 9, 12].

Extensor mechanism malalignment

"Malalignment" is a general term encompassing conditions that lead to a poor positioning and poor tracking of the patella. An improper fit of the mating surfaces leads to abnormal distribution of pressure, which in turn can lead to arthritis.

Patellofemoral dysplasia

Patellofemoral dysplasia is highly correlated with patellofemoral arthritis, and there even is a specific correlation between arthritis and the various forms of dysplasia, especially for trochlear dysplasia [8].

Patella alta, as an isolated factor, was not found to predispose to arthritis [8].

Instability (symptomatic medio-lateral displacement, including dislocation)

Blunt traumas occuring during dislocations associated with patellofemoral dysplasia in most cases can explain the occurence of patellofemoral arthritis.

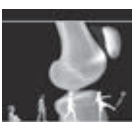
Trauma

Direct blunt trauma can lead to degenerative changes in articular cartilage.

Articular fractures of the patella and the trochlea can be expected to pose the same risk of arthritis as other intra-articular fractures.

Obesity

Pressures across the patellofemoral joint during closed-kinetic-chain activities increase with the person's weight.



Obesity (BMI greater than 30) has been found to predispose a person to patellofemoral arthritis [3, 11].

DIAGNOSIS OF ISOLATED PATELLO FEMORAL OSTEOARTHRITIS

Pain is the primary symtom but can be particularly challenging to link to patellofemoral joint because a large number of conditions can refer pain to the anterior aspect of the knee, and can even be referred from elsewhere in the knee [7] or from distant sites such as hip or spine [9, 13].

A patient with isolated patellofemoral arthritis typically describes anterior knee pain when rising from a seated position and/or going dowstairs or upstairs, standing on one leg with the knee semi-flexed, running with short steps, squatting down [5, 9]. Sometimes, there may be pseudolocking due to "kissing" lesions, or even true locking from osteophytes [5].

Clinical presentation is somewhat non specific: crepitus on grinding the patella or during knee movement and effusion are common [8, 12].

A key sign of symptomatic patellofemoral arthritis on the physical examination is tenderness of the lateral (or occasionally medial) facet of the patella [9].

Abnormal patellar tracking and patella tilt can be signs of instability. Abnormal torsional signs of the lower limb can explain malalignment.

A complete examination is mandatory, including hip and spine.

A routine series of radiographs is necessary, including standing anteroposterior, 45° postero-anterior flexion weightbearing, lateral and skyline view (Merchant view) radiographs [2].

Narrowing of the patellofemoral joint space on the 45° skyline view is the most informative radiograph to classify the site and stage of arthritis, and to identify possible osteophytes, cysts or erosions (saw tooth arthritis [1]).

When maltracking is suspected, a CT scan can be usefull.

MRI doesn't bring specific informations for patellofemoral arthritis.

DISCUSSION

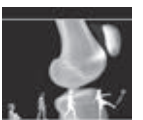
The first step in diagnosing patellofemoral arthritis is to affirm that anterior knee pain is related to patellofemoral joint. Neither symptoms nore clinical examination findings are specific, and, on the other hand, quite specific signs are sometimes painless (eg crepitation). Although, pain arising from the medial compartment may be confused with patellofemoral arthritis [7].

The second step, relating pain to patellofemoral joint damage, is not that easy because pain sometimes exists without any cartilage lesions, probably due to dysfunctions of the extensor mechanism, mostly treated by physiotherapy [4, 13].

Nevertheless, limited lesions of the articular cartilage are difficult to diagnose and injected CT scan can be usefull. But direct relation between cartilage lesion and pain is not so clear: arthroscopic findings show often patellar or trochlear chondral lesions without any patellofemoral complaint [4].

When painfull patellofemoral osteoarthritis is clinically diagnosed, radiographic findings are necessary to stage the degree of arthritis, its site, and also to eliminate arthritis of another compartment. Routine radiographs are mostly sufficiant, especially lateral [2] view and 45° skyline view.

Once the isolated character of patellofemoral arthritis is done, its etiology must be understandable, because it can help its prevention, or the type of treatment.



Etiologies such as trauma (patella or trochlea fractures) dysplasia or instability are well known risk factors.

Obesity is a classical risk factor for developping arthritis on the lower limb, but even if patellofemoral joint is first evolved, arthritis may probably occur in the other compartments sooner or later.

On the same way, genetic factors affecting cartilage resistance may probably follow the same evolution.

No prospective study has been undertaken to verify that patients who have malalignment have a greater predisposition to arthritis, but patients who have patellofemoral arthritis often demonstrate malalignment [8].

The greatest prevalence of chondral wear is on the lateral facet, more commonly than on the central or medial aspect of the patella, suggesting that there is some degree of tilt or malalignment in the etiology of patellofemoral arthritis [12], theory well demonstrated by some authors with an MRI study [10].

CONCLUSION

Even if patellofemoral compartment is commonly involved in knee osteoarthritis, isolated patellofemoral arthritis is not so rare.

Secondary patellofemoral arthitis is well known, especially after trauma (patella fracture).

Patellofemoral dypslasia and instability are also well documented risk factors for patellofemoral arthritis.

So primary patellofemoral osteoarthritis seems to occur very rarely. It is hereby related to extensor mechanism malalignment, and can probably not be called idiopathic.

LITERATURE

[1] ANBARASU A, LOUGHRAN CF. Saw-tooth patello-femoral arthritis. *Clinical Radiologics, 2000; 55: 767-9.*

[2] BHATTACHARYA R, KUMAR V, SAFAWI E. *and al.* The knee skyline radiograph: its usefulness in the diagnosis of patello-femoral osteoarthritis, *International Orthopaedics, 2007; 31: 247-52.*

[3] COOPER C, MCALINDON T, SNOW S. *and al.* Mechanical and constitutional risk factors for symptomatic knee osteoarthritis: différences between medial tibiofemoral and patellofemoral disease. *Journal of Rheumatology, 1994; 21: 307-313.*

[4] DEVEREAUX MD, LACHMAN SM. Patello-femoral arthralgia in athletes attending a sports injury clinic, *British Journal of Sports Medicine, 1984; 18: 18-21.*

[5] DONELL ST, GLASGOW M. Isolated patellofemoral osteoarthritis, *The Knee, 2007; 14: 169-76.*

[6] GOODFELLOW J, HUNGERFORD DS, WOODS C. Patello-femoral joint mechanics and pathology. *Journal of Bone and Joint Surgery, 1976; 58-B: 291-9.*

[7] GOUTALLIER D, DELÉPINE G, DEBEYRE J. The patello-femoral joint in osteoarthritis of the knee with genu varum. *Revue de Chirurgie Orthopédique et Réparatrice de l'Appareil Locomoteur, 1979; 65: 25-31.*

[8] GRELSAMER RP, DEJOUR D, GOULD J. The pathophysiology of patellofemoral arthritis, *Orthopedic Clinics of North America, 2008; 39: 269-74.*

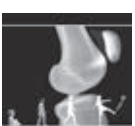
[9] GRELSAMER RP, STEIN DA Current concepts review : Patellofemoral arthritis, *Journal of Bone and Joint Surgery, 2006; 88-A: 1849-60.*

[10] KALICHMAN L, ZHANG Y, NIU J. *and al.* The association between patellar alignment on magnetic resonance Imaging and radiographic manifestations of knee osteoarthritis, *Arthritis Research & Therapy, 2007; 9: R26.*

[11] McALINDON T, ZHANG Y, HANNAN M. *and al.* Are risk factors for patellofemoral and tibiofemoral knee osteoarthritis different? *Journal of Rheumatology, 1996; 23: 332-7.*

[12] MINKOWITZ RB, BOSCO JA. Patellofemoral arthritis, *Bulletin of the NYU Hospital for Joint Diseases, 2009; 67: 30-8.*

[13] QUILTY B, TUCKER M, CAMPBELL R. *and al.* Physiotherapy, including quadriceps exercises and patellar taping, for knee osteoarthritis with predominant patello-femoral joint involvement: randomized controlled trial, *Journal of Rheumatology, 2003; 30: 1311-17.*



IMAGING OF PATELLOFEMORAL JOINT OSTEOARTHRITIS

C. LAPRA, S. CHOMEL, M. BAKIR

Imaging of the patellofemoral joint (PFJ) is both customary and innovative: the narrowed joint space, the very definition of osteoarthritis pathology, is routinely detected using standard radiological assessment. Early degenerative chondral lesions are detected using slice imaging: arthro-CT scans, commonly used in France, MRI and its new acquisition sequences or even MRI arthrography with an intra-articular injection of Gadolinium.

Joint impairment involves the lateral compartment of the PFJ in 90% of cases and encompasses four etiologies:
- Primary patellofemoral joint osteoarthritis (OA),
- Patellofemoral joint OA with instability: the patient has a history of patellar dislocation,
- Traumatic patellofemoral joint OA: history of patellar fracture,
- Articular chondrocalcinosis.

Imaging procedures are used to study this etiological framework, which is particularly crucial for determining the treatment.

QUESTION 1: IS THERE DEGENERATIVE IMPAIRMENT OF THE PATELLOFEMORAL JOINT?

Standard radiological assessments

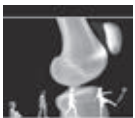
Standard radiological assessments include front views extended in monopodal stance, schuss, lateral view extended in 30° of flexion, axial view of the patella at 30° of flexion.

The Iwano classification describes the four stages of joint space impairment on the axial view of the patella (fig. 1).
- *Stage 1:* mild OA joint space at least 3mm.
- *Stage 2:* moderate OA; joint line narrowing > 3mm.
- *Stage 3:* severe OA; joint line narrowing < 3mm
- *Stage 4:* very severe OA, joint surfaces entirely touch each other.

Radiological assessments are aimed at detecting certain etiological factors:
- Chondrocalcinosis is, in particular, characterized by a jagged, uneven aspect of the joint surfaces. Meniscal calcifications are not specific to articular chondrocalcinosis.
- Patellar fracture sequelae are generally revealed during medical questioning; on the X-ray, the patella appears enlarged transversally.

Patellofemoral chondral lesions: MRI and arthro-CT scan

MRI and arthro-CT scans are indicated in the case of diagnostic doubt about early arthrosis, without joint space narrowing upon first assessment or with radiological anomalies that do not explain the pain [15, 16]. Given the development of cartilage repair techniques, there is increasing interest in detecting early chondral lesions.



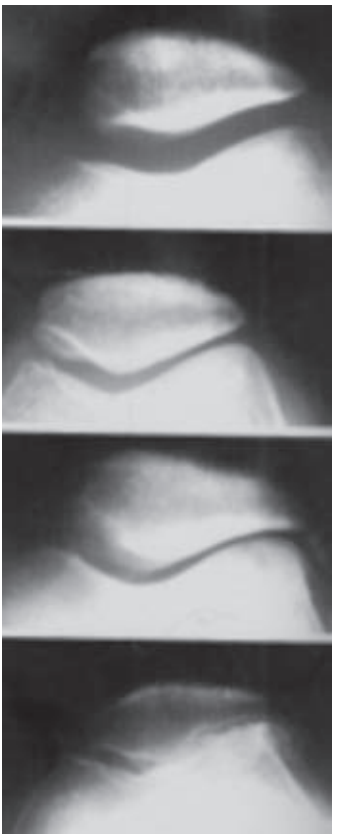


Fig. 1: Iwano classification *(source : traité de chirurgie du genou, Ph. Neyret, G. Demey, E. Servien, S. Lustig).*

Many studies do not show any clear evolutive link between the cartilage disease "chondromalacia patellae" and patellofemoral arthrosis. However, in athletes, PF cartilage lesions are considered as related more to trauma or micro-trauma than to over-use [3]. Dupont [12] considers that cartilage lesions in children and athletic teens are more frequently unnoticed than they are actually rare [11].

As viscosupplementation has proven effective in common arthrosis, authors suggest wider use in athletes as soon as patellofemoral chondral lesions appear in athletes; hence the usefulness of detecting such lesions early.

What can be expected from a patellofemoral MRI focused on the cartilage?

Due to its superficial location and the thickness of its cartilage, the patellofemoral joint lends itself well to MRI evaluation and it is the joint whose cartilage has been studied most.

The purpose of the MRI sequences we have is to:
- clearly demarcate the joint surface of the trochlear and patellar cartilage,
- define the thickness of the cartilage,
- explore the deep layer and the osseous portion: tide-mark and subchondral bone,
- differentiate the cartilage from the intra-articular fluid to detect superficial irregularities or notches of varying depths that may reach the subchondral bone,
- determine the biochemical composition of the lamellar structure of the cartilage so as to detect intra-articular anomalies, without surface lesions.

Contribution of 3D MRI sequences:
- What are the specifications for an MRI sequence? The "ideal" MRI sequence would be the one that combines short examination time, a good signal-to-noise ratio, very good spatial resolution, 3D study enabling ultra-fine sections and multiplanar reconstructions, specific signal from the cartilage differentiating it from the articular fluid, specific signal from structural modifications. Like any technique, MRIs are a compromise!
- 3D Fast Spin-Echo (FSE) sequences including submillimeter sections have been improved thanks to so-called parallel imaging enabling shorter acquisition times and improved spatial

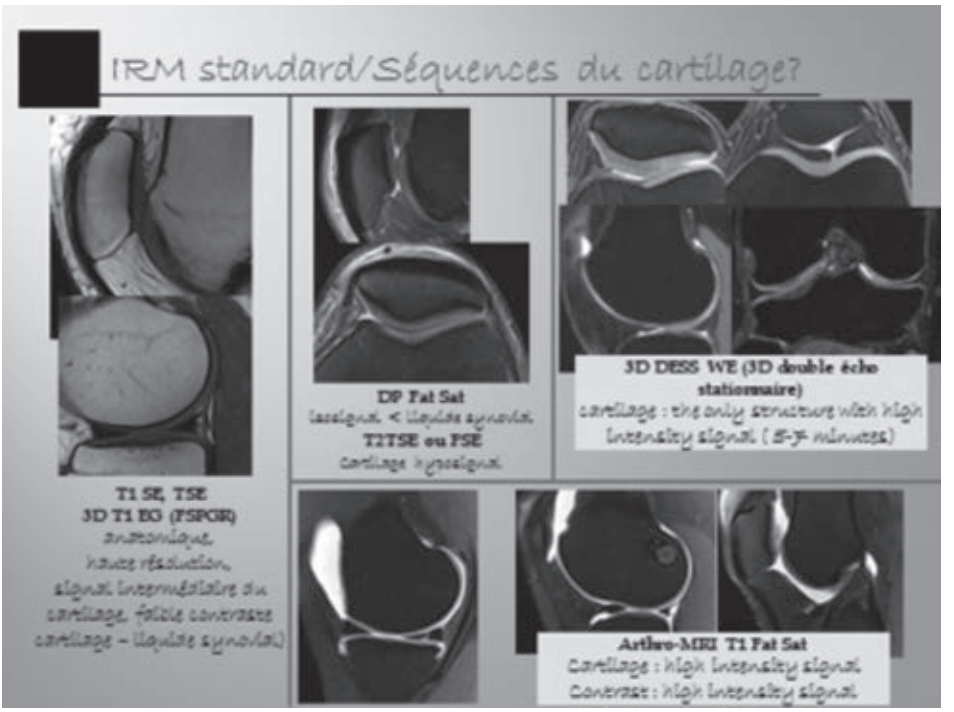


Fig. 2: Source : Chomel S MD Imaging Cartilage of the knee, EPU January 2012 Lyon.

resolution. The result of the sequences is to acquire one plane and then, like with a scan, create reconstructions in other planes. To create high-quality multiplanar reconstructions, MRI sequences must be as isotropic as possible.

Comparison of the cartilage signal based on sequences used [10, 24, 28] (fig. 2)

As a result, several analyses can be used to study the cartilage

- Morphological analysis, which can be compared to Outerbridge [22] and International Cartilage Repair Society (ICRS) arthroscopic classifications,
- Volume analysis,
- More recent "biochemical" analysis.

Morphological analysis (fig. 3, 4, 5)

Exams with injections: arthro CT-scan (fig. 6a and b).

MRI Arthrography (fig. 7 a,b): although the intra-articular injection may sometimes make it easier to grade the chondral anomalies, it makes the exam more invasive.

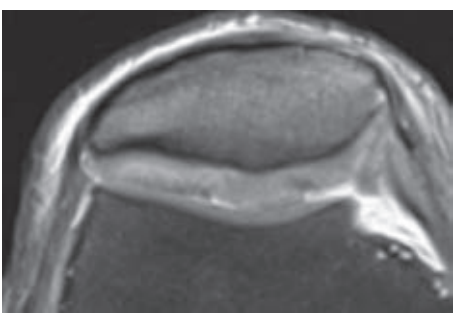
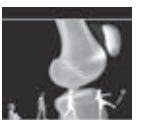


Fig. 3: ICRS Grade 2



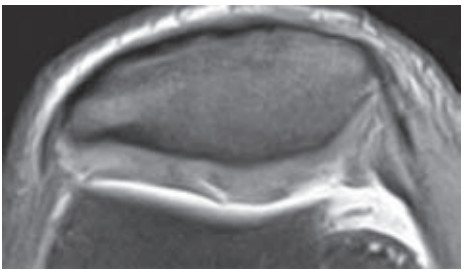


Fig. 4: ICRS Grade 3

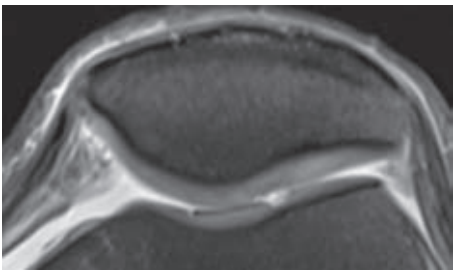


Fig. 5: ICRS Grade 4 on the lateral trochlea

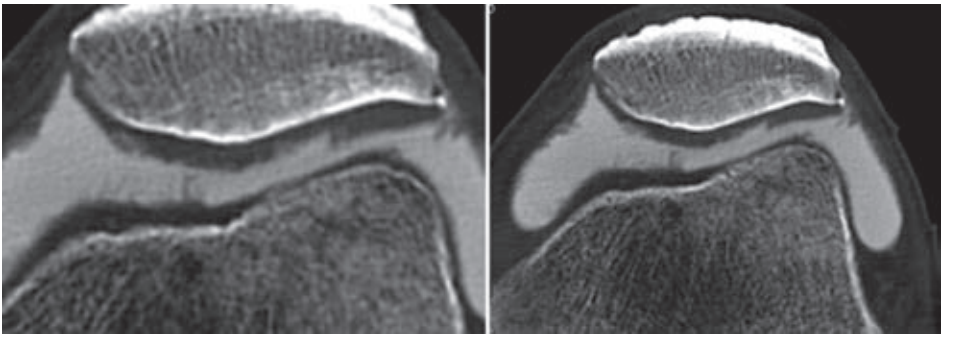


Fig. 6

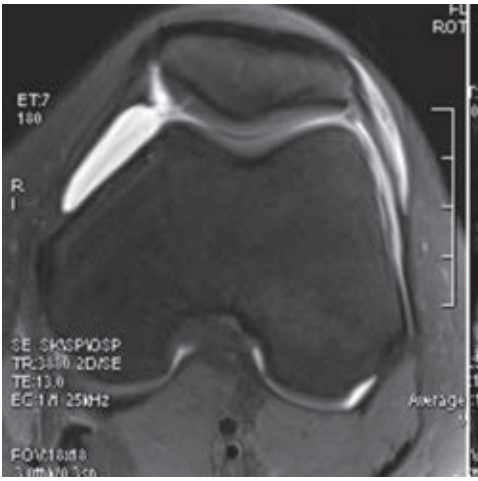


Fig. 7a: Proton density MR.

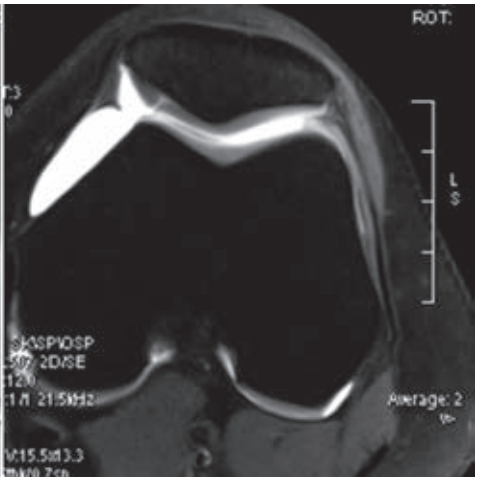


Fig. 7b: MR arthrography: grade 3 on medial patella

"*Biochemical" analysis*

On the views shown above (fig. 8), regular chondral thinning and signal anomalies within the cartilage can be noted; there is no deep chondral erosion [7, 28].

T2 mapping [29] can be used in current practice; it consists of a T2 multi-echo sequence of 10 slices of 3-mm thickness. It indirectly provides information about the extracellular matrix. The different layers of cartilage are characterized by their biochemical composition and in particular their water content which provides different T2 values for the deep, middle and upper zones.

Principle of T2 mapping:
- Measurement of T2 relaxation time of the cartilage at a given level with echo times (TE).
- T2 relaxation time reflects the ability of free water to move inside the cartilaginous matrix.
- Pathological cartilage: decrease in glyco-aminoglycan content, and in size of proteo-glycans, increase in water movement.
- Increase of T2 in pathological cartilage compared to healthy cartilage.
- Creation of mapping and fused image (fig. 9).

The T2 mapping sequences do not explore chondral ulcers (fig. 10)

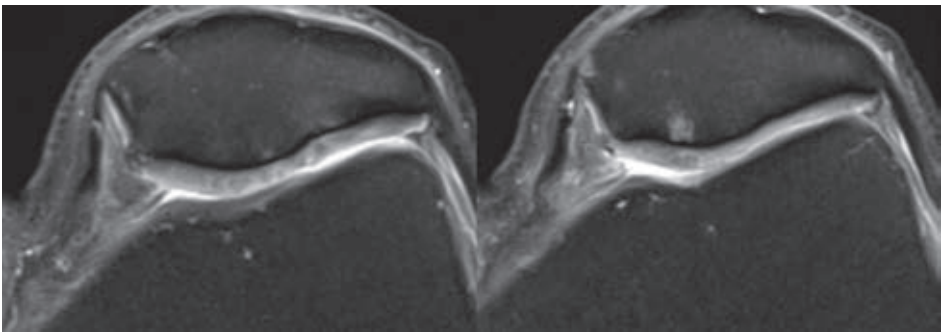


Fig. 8: Proton-density fat saturation (PDFS)

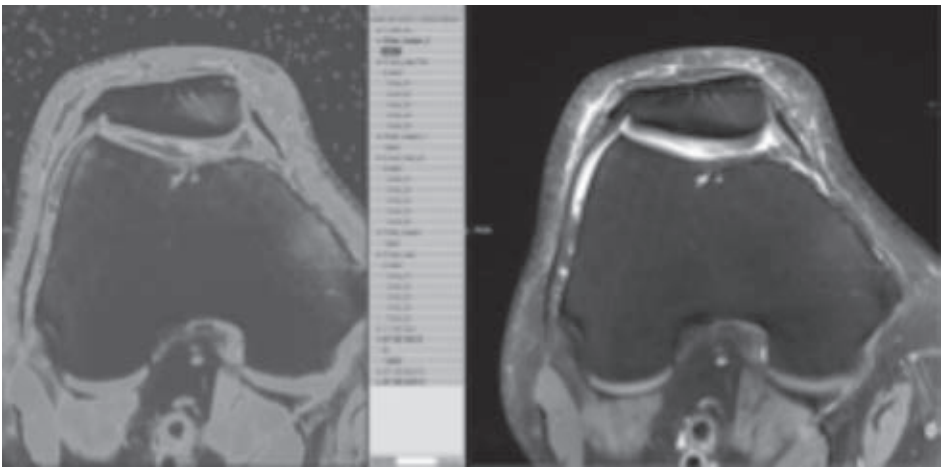
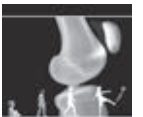


Fig. 9: T2 mapping



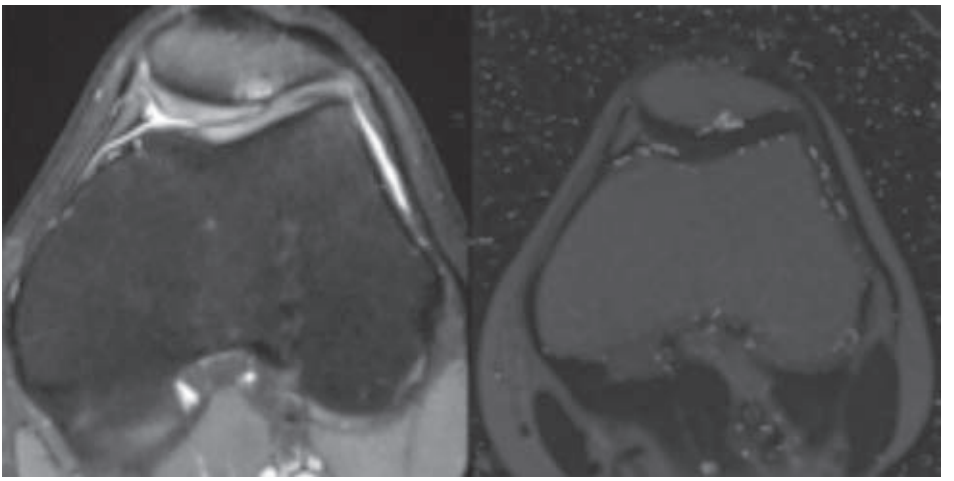


Fig. 10: There is cartilage thinning of the lateral patellar facet, not visible on the T2 map.

Delayed gadolinium-enhanced MRI of cartilage (dGEMRIC) [30]: these sequences consist of an intra-articular injection of a double dose of gadolinium, associated with the patient walking for 15 minutes. The examination is done after 90 minutes. It is a "molecular" study of the cartilage that is not routinely done. In healthy cartilage, gadolinium (Gd) does not penetrate cartilage; Gd fixation increases if the glycoaminoglycan content decreases.

Associated lesions: synovitis, subchondral bone edema

Many studies have shown that the pain of arthrosis was often linked to secondary lesions associated with edema of the subchondral bone due to trabecular bone microcracks and synovial inflammation, which explains the evolution in spurts [14, 18, 32].

<u>*Synovitis*</u> (fig. 11)

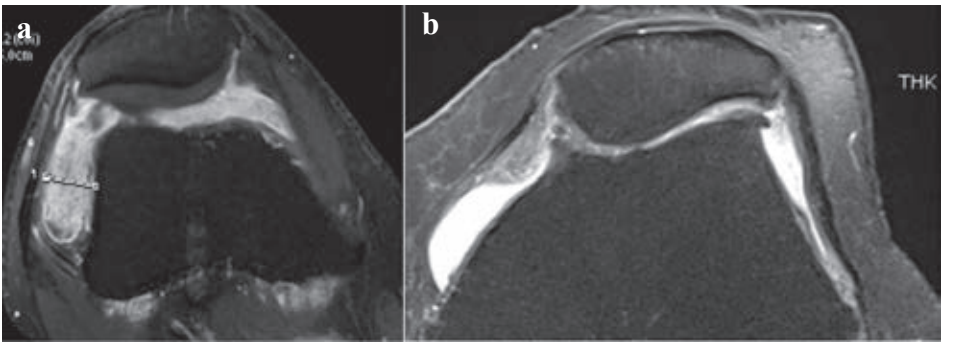
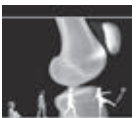


Fig. 11: Same patient, MRI. - a: Right knee MRI with injection: synovitis. b: On the left knee (Proton density), the joint space is more impaired but there is less synovial inflammation.



Subchondral bone

A lateral patellofemoral friction syndrome may also be associated with degenerative cartilage lesions, pre-disposed by a pre-existing morphological anomaly of the joint, fostering instability. This syndrome has also been described as responsible for anterior knee pain, in the absence of arthritic lesions [2, 8, 26] (fig. 12).

Future areas of development for cartilage MRI will move towards ultra-high-field (7 Tesla) MRIs, T1 mapping, molecular imaging (sodium) (fig. 13)

QUESTION 2: IS THERE AN ANATOMICAL PREDISPOSITION TO INSTABILITY?

Trochlear dysplasia is a frequent predisposing factor for lateral patellofemoral; it is present in 80% of the cases of patellofemoral OA. Articular morphology findings are especially important for the treatment of patellofemoral OA.

The imaging assessment looks for:
- Signs of trochlear dysplasia:
  - On the lateral view, crossing sign,
  - Trochlear boss,

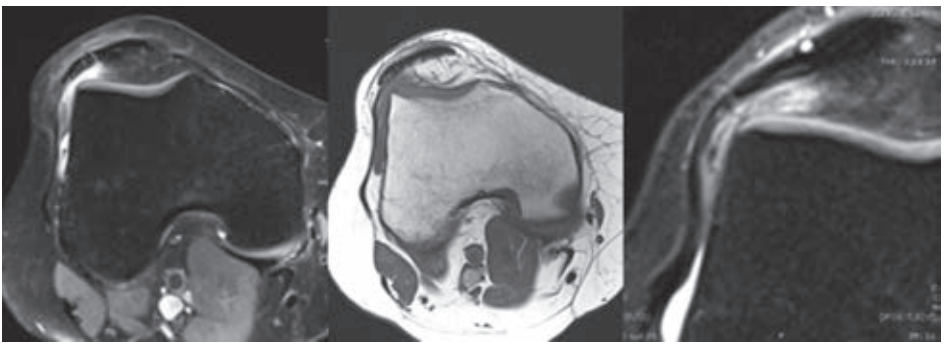


Fig. 12

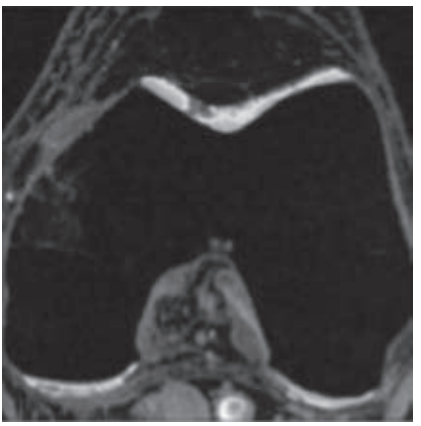
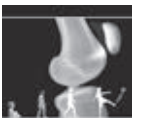


Fig. 13: T1 Mapping (source : Siemens)



- On CT scan or MRI, a deficient lateral trochlear slope or insufficient trochlear depth, the lateral trochlear inclination index can be both measured on CTsan or MRI [4].
- Patella alta on the lateral X-ray,
- Lateral tilt of the patella (axial, lateral view, MRI or CT-scan measurement of the patellar tilt),
- Measurement of the TT-GT distance on CT-scan or MRI [15, 31] (fig. 14 a,b).

Trochlear dysplasia can be studied by CT-scan or MRI [4, 17, 23, 31], different studies validate the instability measurements validated with the CT-scan or MRI. Thanks to its 3D reconstructions, scans make it possible to thoroughly explore the trochlear anatomy and the insufficient depth of the trochlear groove.

To conclude, imaging assessments must be capable of answering two main questions:
- In the case of pain indicating the patellofemoral joint, are there degenerative chondral lesions, or even proven osteoarthritis? If so, what is causing the pain: a subchondral edema? Lateral patellofemoral friction? Associated synovitis?
- Are there anatomical predispositions to instability? The traditional criteria shown on the lateral view (trochlear dysplasia, patellar tilt, patellar height) were also found on the latest generation MRI and 3D and 2D CT scans.

The "best radiological exam" will undoubtedly remain the most exhaustive in terms of information provided for the therapeutic treatment. As such, MRIs have become almost mandatory!

Fig. 14a: Patellofemoral Joint OA on axial view

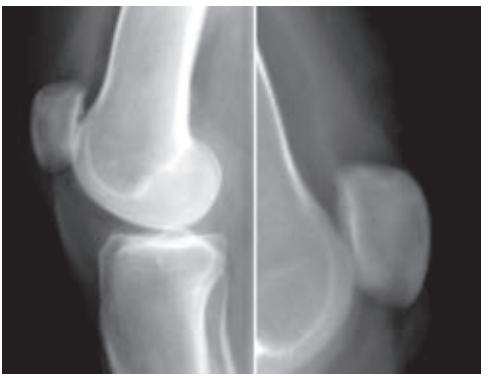


Fig. 14b: trochlear dysplasia

LITERATURE

[1] ARENDT EA, FITHIAN DC, COHEN E. Current concepts of lateral patella dislocation. *Clin Sports Med. 2002 Jul;21(3): 499-519. Review.*

[2] BARBIER-BRION B, LERAIS JM, AUBRY S, LEPAGE D, VIDAL C, DELABROUSSE E, RUNGE M, KASTLER B. Magnetic resonance imaging in patellar lateral femoral friction syndrome (PLFFS): prospective case-control study. *Diagn Interv Imaging. 2012 Mar; 93(3):e171-82.*

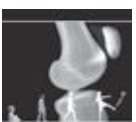
[3] BRITTBERG M, WINALSKI CS. Evaluation of cartilage injuries and repair. *J Bone Joint Surg Am. 2003; 85-A Suppl 2: 58-69.*

[4] CARILLON Y, ABIDI H, DEJOUR D, FANTINO O, MOYEN B, TRAN-MINH VA. Patellar instability: assessment on MR images by measuring the lateral trochlear inclinaison. Initial experience. *Rgy 2000 Aug; 216(2): 582-5.*

[5] CATON J. Method of measuring the height of the patella. *Acta Orthop Belg. 1989; 55(3): 385-6.*

[6] COTTEN A, BOUTRY N, DEMONDION X, BERA-LOUVILLE A. Imagerie musculosquelettique : Pathologies locorégionales. *Masson mars 2008.*

[7] CHU CR, WILLIAMS A, TOLLIVER D, KWOH CK, BRUNO S 3rd, IRRGANG JJ. Clinical optical coherence tomography of early articular cartilage degeneration in patients with degenerative meniscal tears. *Arthritis Rheum. 2010 May; 62(5): 1412-20.*



[8] CHUNG CB, SKAF A, ROGER B, CAMPOS J, STUM X, RESNICK D. Patellar tendon-lateral femoral condyle friction syndrome. MR imaging in 42 patients. *Skeletal Radiol 2001 Dec ; 30(12): 694-7.*

[9] DEJOUR H, WALCH G, NOVE-JOSSERAND L, GUIER C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc. 1994; 2(1): 19-26.*

[10] DISLER DG, McCAULEY TR, KELMAN CG, FUCHS MD, RATNER LM, WIRTH CR, HOSPODAR PP. Fat-suppressed three-dimensional spoiled gradient-echo MR imaging of hyaline cartilage defects in the knee: comparison with standard MR imaging and arthroscopy. *AJR Am J Roentgenol. 1996 Jul; 167(1): 127-32.*

[11] DRAPER CE, BESIER TF, GOLD GE, FREDERICSON M, FIENE A, BEAUPRE GS, DELP SL. Is cartilage thickness different in young subjects with and without patellofemoral pain? *Osteoarthritis Cartilage. 2006 Sep; 14(9): 931-7. Epub 2006 Apr 27.*

[12] DUPONT P, THEMAR NOEL C, BENSAHEL H. Chondropathies rotuliennes de l'enfant et de l'adolescent. *Sciences et Sports, 1, 75-80.*

[13] GOUTALLIER D, BERNAGEAU J, LECUDONNEC B. The measurement of the tibial tuberosity. Patella groove distanced technique and results (author's transl). *Rev Chir Orthop Reparatrice Appar Mot. 1978 Jul-Aug; 64(5): 423-8.*

[14] GRELSAMER RP, DEJOUR D, GOULD J. The pathophysiology of patellofemoral arthritis. *Orthop Clin North Am. 2008 Jul; 39(3): 269-74.*

[15] KRAMER J, WHITE LM, RECHT MP. MR imaging of the extensor mechanism. *Semin Musculoskelet Radiol. 2009 Dec; 13(4): 384-401.*

[16] LAREDO JD Imagerie ostéo-articulaire-arthrographie, arthroscanner, arthro-IRM. *Sauramps 2011.*

[17] LIPPACHER S, DEJOUR D, ELSHARKAWI M, DORNACHER D, RING C, DREYHAUPT J, REICHEL H, NELITZ M. Observer agreement on the Dejour trochlear dysplasia classification: a comparison of true lateral radiographs and axial magnetic resonance images. *Am J Sports Med. 2012 Apr; 40(4): 837-43.*

[18] LOEUILLE D, RAT AC, GOEBEL JC, CHAMPIGNEULLE J, BLUM A, NETTER P, GILLET P, CHARY-VALCKENAERE I. Magnetic resonance imaging in osteoarthritis: which method best reflects synovial membrane inflammation? Correlations with clinical, macroscopic and microscopic features. *Osteoarthritis Cartilage. 2009 Sep; 17(9): 1186-92.*

[19] MALGHEM J, MALDAGUE B. Depth insufficiency of the proximal trochlear groove on lateral radiographs of the knee: relation to patellar dislocation. *Radiology. 1989 Feb; 170(2): 507-10.*

[20] NEYRET P, ROBINSON AH, LE COULTRE B, LAPRA C, CHAMBAT P. Patellar tendon length--the factor in patellar instability? *Knee. 2002 Feb; 9(1): 3-6.*

[21] NEYRET P, VERDONK P, AÏT SI SELMI T, MÜLLER W. Chirurgie du genou : My Knee Practice. *Masson novembre 2006.*

[22] OUTERBRIDGE RE, DUNLOP JA.The problem of chondromalacia patellae. *Clin Orthop Relat Res. 1975 Jul-Aug; (110): 177-96.*

[23] PFIRMANN CW, ZANETTI M, ROMERO J, HODLER J. Femoral trochlear dysplasia: MR findings. *Radiology 2000 Sep; 216(3): 858-64.*

[24] RECHT MP, PIRAINO DW, PALETTA GA, SCHILS JP, BELHOBEK GH. Accuracy of fat-suppressed three-dimensional spoiled gradient-echo FLASH MR imaging in the detection of patellofemoral articular cartilage abnormalities. *Radiology. 1996 Jan; 198(1): 209-12.*

[25] SHELLOCK FG, MINK JH, DEUTSCH AL, FOO TK. Kinematic MR imaging of the patellofemoral joint: comparison of passive positioning and active movement techniques. *Radiology. 1992 Aug; 184(2): 574-7.*

[26] SUBHAWONG TK, ENG J, CARRINO JA, CHHABRA. Supralateral Hoffa's fat pad edema. *Am J Roentgenol 2010 Dec; 195(6): 1376-73.*

[27] TETSUO HIGUCHI, YUJI ARAI, HISATAKE TAKAMIYA,TATSUYA MIYAMOTO, DAISAKU TOKUNAGA, TOSHIKAZU KUBO. An analysis of the medial patellofemoral ligament length change pattern using open-MRI. *Knee Surg Sports Traumatol Arthrosc (2010), 18: 1470-75.*

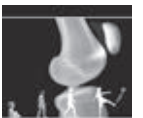
[28] WACKER FK, BOLZE X, FELSENBERG D, WOLF KJ. Orientation-dependent changes in MR signal intensity of articular cartilage: a manifestation of the "magic angle" effect. *Skeletal Radiol. 1998 Jun; 27(6): 306-10.*

[29] WILLIAMS A, QIAN Y, CHU CR. UTE-T2$*$ mapping of human articular cartilage in vivo:repeatability assessment. *Osteoarthritis Cartilage. 2011 Jan; 19(1): 84-8.*

[30] WILLIAMS A, GILLIS A, MCKENZIE C, PO B, SHARMA L, MICHELI L, MCKEON B, BURSTEIN D. Glycosaminoglycan distribution in cartilage as determined by delayed gadolinium-enhanced MRI of cartilage (dGEMRIC): potential clinical applications. *AJR Am J Roentgenol. 2004 Jan; 182(1): 167-72.*

[31] WITTSTEIN JR, BARTLETT EC, EASTERBROOK J, BYRD JC. Magnetic Resonance Imaging evaluation of patellofemoral malignanment. *Arthroscopy 2006 Jun; 22(6): 643-9.*

[32] ZAFFAGNINI *et al.* Patellofemoral Pain, Instability, and Arthritis, Clinical Presentation, Imaging and Treatment. *Springer-Verlag 2010.*



PLACE DU TRAITEMENT MÉDICAL ET DE LA KINÉSITHÉRAPIE DANS L'ARTHROSE FÉMOROPATELLAIRE ISOLÉE

B. QUELARD

L'arthrose fémoropatellaire isolée est une affection à part entière, bien distincte des autres arthroses du genou dans sa pathogenèse. Elle n'est pas rare. Dans l'étude radiologique réalisée par Duncan *et al.* en 2006 [7], 24 % des patients de plus de 50 ans souffrant de gonalgies présentaient des lésions dégénératives isolées du compartiment fémoropatellaire. Elle touche une population majoritairement féminine, généralement plus jeune que celle des arthroses fémoro-tibiales.

Sur le plan histologique, l'arthrose fémoropatellaire isolée ne possède pas de spécificité propre. Elle associe des lésions du cartilage plus ou moins profondes et plus ou moins étendues, une condensation de l'os sous-chondral, des ostéophytes et une inflammation de la membrane synoviale plus ou moins marquée. Sur le plan clinique sa symptomatologie est proche de celle de l'arthrose fémoro-tibiale. La douleur siège généralement à la face antérieure du genou, elle est de type mécanique conduisant progressivement à une limitation des activités les plus contraignantes pour le système extenseur (descente des escaliers, marche sur terrain en dénivelé, accroupissement…), à une réduction du périmètre de marche puis à une diminution de la mobilité articulaire et au déficit de la fonction musculaire.

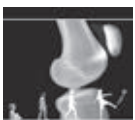
L'intensité de la douleur et la difficulté à réaliser les activités de la vie quotidienne sont étroitement liées à la sévérité du stade évolutif de l'arthrose [8, 9, 17] objectivée radiologiquement par la réalisation d'un défilé fémoropatellaire à 30° de flexion. La classification d'Iwano *et al.* en 4 stades de sévérité croissante en fonction de la réduction de l'interligne fémoropatellaire est la plus utilisée en France. Il faut en moyenne 18 ans pour passer d'un stade I au stade IV [5], ce qui explique que l'arthrose fémoropatellaire isolée soit considérée comme longtemps bien tolérée.

Sur le plan thérapeutique, la prise en charge de l'arthrose du genou fait l'objet de recommandations internationales [13, 22], peu appliquées dans notre pratique quotidienne. La méconnaissance de la maladie fait partie des raisons pour lesquelles ces recommandations sont faiblement suivies [11].

Après un rappel de la physiopathologie de l'arthrose et de la pathogénèse de l'arthrose fémoropatellaire isolée nous envisagerons la place du traitement non chirurgical dans la prise en charge de cette pathologie.

PHYSIOPATHOLOGIE DE L'ARTHROSE

Les mécanismes physiopathologiques à l'origine de l'arthrose ne sont pas encore totalement



élucidés. Ils sont complexes, mettant en jeu de nombreuses substances chimiques (facteurs de croissances, cytokines, prostaglandines…) synthétisées par les cellules du cartilage (chondrocytes), de la membrane synoviale (synoviocytes) et de l'os sous chondral. Ces médiateurs chimiques ont pour le rôle d'activer ou d'inhiber la production d'enzymes protéolytiques spécifiques (métalloprotéases…) responsables de la dégradation de la matrice extra-cellulaire constituée essentiellement d'eau (70 à 80 %), de microfibrilles de collagène de type II et de protéoglycanes.

Les études menées *in vitro* et chez l'animal ont montré que des pressions excessives appliquées sur les chondrocytes ou les surfaces articulaires jouent un rôle prépondérant dans le déclenchement des processus conduisant à l'arthrose [15, 18].

A la phase initiale, ces processus consistent en une prolifération et une activation des chondrocytes dans le but de réparer les lésions induites par les agressions mécaniques répétées. La prolifération excessive des chondrocytes aboutit à leur dédifférenciation. Ces chondrocytes anormaux fabriquent une matrice anormale constituée de collagène de type I dont les propriétés mécaniques sont inférieures à celle du type II.

A la phase d'état la chondrolyse l'emporte, entretenue par le stress mécanique et le stress "biochimique" induit par la constitution de 3 boucles physiopathologiques : cartilagino-cartilagineuse, synovio-cartilagineuse et ostéo-cartilagineuse (fig. 1).

Une fois les processus de chondrolyse enclenchés, la dégradation du cartilage est inéluctable, auto entretenue par les pressions s'exerçant sur une matrice qualitativement affaiblie.

PATHOGENÈSE DE L'ARTHROSE FÉMOROPATELLAIRE ISOLÉE

Les anomalies architecturales de la trochlée et à un degré moindre de la rotule ainsi qu'un mauvais alignement du système extenseur représentent les principaux facteurs de risque spécifiques à cette localisation de l'arthrose [12, 14, 5]. Dans les études réalisées par Dejour [5] et par Guilbert [10] pour le symposium de la SOFCOT de 2003 une dysplasie de la trochlée était présente chez 78 % et 83 % des patients de leur série respective, 42 % avait une dysplasie de la rotule.

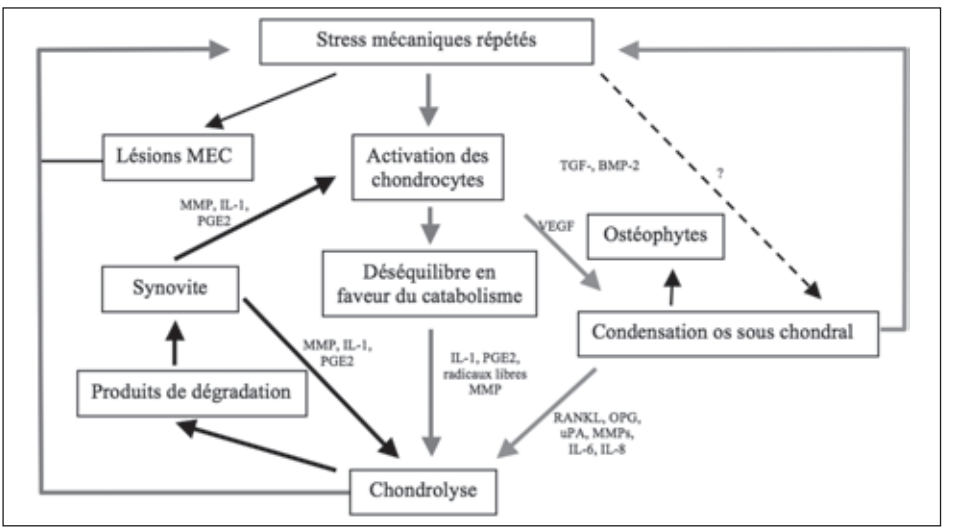


Fig. 1 : Boucles physiopathologiques de l'arthrose : cartilagino-cartilagineuse, synovio-cartilagineuse et ostéo-cartilagineuse

Les facteurs traumatiques (fracture de rotule, microtraumatismes sportifs ou professionnels répétés), les facteurs métaboliques (chondrocalcinose) et les facteurs iatrogènes (hypermédialisation de la tubérosité tibiale) sont également des facteurs de risque. La surcharge pondérale n'est par contre qu'un facteur aggravant.

Quelles soient congénitales ou acquises, les anomalies morphologiques de l'articulation fémoropatellaires modifient la cinématique de la rotule et la répartition des pressions. Les lésions cartilagineuses générées par les stress mécaniques répétés feront le lit de l'arthrose. La topographie de ces lésions varie en fonction du facteur étiologique.

Une fois l'arthrose installée, la dégradation du cartilage peut se faire de façon lentement progressive ou par poussées avec des phases de chondrolyse alternant avec des phases plus ou moins longues de stabilité. Les premières manifestations cliniques des lésions dégénératives sont les douleurs. Elles ne sont pas induites par le cartilage lui-même qui n'est pas innervé, mais par l'inflammation synoviale ou les hyperpressions s'exerçant sur l'os sous chondral. Elles peuvent avoir également pour origine des excès de pression intra-osseuse (œdème de l'os spongieux) [21]. Ces douleurs vont entraîner une baisse d'activités et une perte de la mobilité articulaire conduisant à une atrophie et à des raideurs musculaires qui majorent les contraintes articulaires par perte de la fonction musculaire d'amortisseur. Ainsi s'établit un cercle vicieux auto-aggravant entre contraintes mécaniques, douleurs et baisse d'activités (fig. 2).

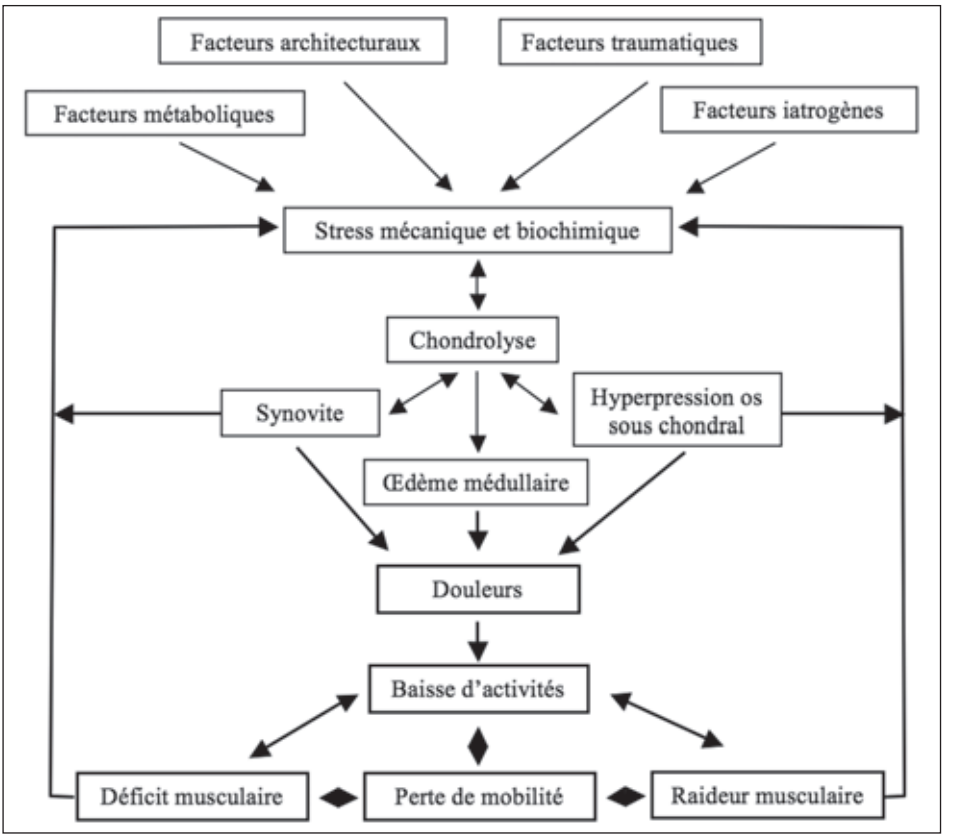
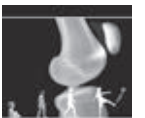


Fig. 2 : Pathogenèse de l'arthrose fémoropatellaire isolée



PLACE DU TRAITEMENT MÉDICAL ET KINÉSITHÉRAPIQUE

Les moyens thérapeutiques dont nous disposons n'ont guère évolué au cours de ces 10 dernières années malgré une meilleure connaissance de la physiopathologie de cette affection. Ils restent purement symptomatiques, aucun traitement n'étant capable aujourd'hui de promouvoir la régénération d'un cartilage qualitativement normal.

La prise en charge thérapeutique de la gonarthrose fait l'objet de recommandations établies par des sociétés d'experts européens *(European League Against Rheumatism* [13]) ou internationaux (*Osteoarthritis Research Society International* [22]). Aucune de ces recommandations n'est spécifique à l'arthrose fémoropatellaire isolée. Toutefois, la plupart lui est applicables.

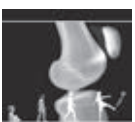
Le traitement de l'arthrose fémoropatellaire isolée est longtemps et avant tout non chirurgical, la chirurgie s'adressant aux arthroses évoluées radiologiquement, douloureuses et invalidantes, réfractaires au traitement médical. L'association de mesures thérapeutiques non pharmacologiques et pharmacologiques est la première recommandation formulée par les experts pour une prise en charge optimale.

Les mesures thérapeutiques non pharmacologiques sont la clef de voûte de la prise en charge de l'arthrose. Elles reposent principalement sur l'éducation du patient et la rééducation fonctionnelle. Elles visent à freiner la progression des lésions structurales de l'articulation en rompant le cercle vicieux : augmentation des contraintes mécaniques – douleurs – baisse d'activités. Elles doivent être mises en œuvre dès les premiers stades de l'arthrose et prolongées durant toute son évolution car il existe une relation entre l'efficacité d'un programme d'exercices et son observance [16].

L'éducation conditionne l'observance du traitement [16]. Elle doit informer le patient sur l'importance de supprimer les activités sportives qui engendrent des poussées inflammatoires et des douleurs, de conserver une activité physique régulière [3] en priorisant celles qui génèrent moins de contraintes sur l'articulation fémoropatellaire (marche et cyclisme sur sol plat, natation), de réaliser régulièrement des exercices d'assouplissements musculaires afin de prévenir la perte de mobilité et notamment l'installation d'un flexum. Elle vise également à lui enseigner les règles d'économie articulaire : lutter contre la surcharge pondérale, éviter le port de charges lourdes, les marches sur terrains à fort dénivelé, les marches prolongées sur terrains accidentés, les montées et descentes d'escaliers répétées, le port de talons hauts…

La rééducation a pour objectifs d'entretenir les amplitudes articulaires, d'améliorer la fonction quadricipitale ainsi que la stabilité du genou. Elle repose sur les principes que l'application de stimulations cycliques modérées favorise la synthèse de protéoglycanes par les chondrocytes [2], qu'une raideur du quadriceps et/ou des ischio-jambiers augmente les contraintes fémoropatellaires et qu'une plus grande force de quadriceps prévient la perte de cartilage au niveau du compartiment fémoropatellaire externe, diminue les douleurs et améliore les capacités physiques des patients [1]. Elle doit tenir compte de la biomécanique de l'articulation fémoropatellaire : dans les amplitudes comprises entre 0 et 60° de flexion les exercices en chaîne en cinétique ouverte sont plus contraignants que les exercices en chaîne cinétique fermée mais au-delà la chaîne cinétique fermée devient plus contraignante [20].

En pratique, la rééducation associe un rodage articulaire à faible résistance (vélo de rééducation, pédalage en piscine…), des étirements et surtout l'apprentissage d'auto-étirements des différentes chaines musculaires (quadriceps, psoas-iliaque, ischio-jambiers, triceps), un travail de renforcement du quadriceps (contractions isométriques du quadriceps en chaîne cinétique ouverte genou en extension, électrostimulation des vastes, travail du vaste interne au biofeedback, renforcement progressif et infra douloureux du quadriceps en chaîne cinétique fermée dans le secteur 0/60°), un travail proprioceptif en chaîne cinétique fermée. Elle va permettre d'établir un programme personnalisé



d'exercices à effectuer au long cours plusieurs fois par semaine. Aucun programme d'exercices n'a prouvé sa supériorité, l'essentiel étant qu'il soit adapté à la gravité de l'arthrose ainsi qu'aux capacités et aux besoins fonctionnels du patient [16]. Il est recommandé d'élaborer le programme sous la conduite d'un kinésithérapeute puis de le poursuivre seul, à domicile [6].

Les autres mesures non pharmacologiques ont un but essentiellement antalgique. Elles regroupent les aides techniques à la marche (cannes), les semelles absorbantes, la cryothérapie, la neurostimulation électrique transcutanée. Elles font partie des mesures recommandées par l'OARSI dans le cadre de la gonarthrose. La contention par taping est controversée [19].

Les mesures thérapeutiques pharmacologiques visent à soulager la douleur en agissant sur le stress "biochimique". Elles sont complémentaires aux mesures précédentes et font appel à des produits dont l'effet est soit immédiat (antalgiques, anti-inflammatoires non stéroïdiens (AINS), injections intra-articulaires de corticoïdes) soit différé ("anti-arthrosiques" symptomatiques d'action lente, injections intra-articulaires d'acide hyaluronique). Le paracétamol et les AINS sont les médications les plus utilisés en première intention. Les AINS doivent être prescrits à la dose minimale efficace et leur utilisation au long cours doit être évitée [22]. Les injections intra-articulaires de corticoïdes sont indiquées en cas de douleurs modérées à sévères et de poussées inflammatoires. Concernant les "anti-arthrosiques" ils permettraient une diminution, généralement modeste mais statistiquement significative, des douleurs et de la consommation d'AINS après une période de latence de quelques semaines. Leurs effets se maintiendraient 3 à 4 mois après l'arrêt du traitement. Quant au traitement par l'acide hyaluronique il comporte 3 injections espacées d'une semaine. Son action antalgique débute en général 4 semaines après l'injection initiale et pourrait se prolonger jusqu'à un an.
Dans chaque classe de médicaments il existe de très nombreuses spécialités sur le marché. A notre connaissance aucune n'a apporté la preuve de son efficacité dans l'arthrose fémoropatellaire isolée en dehors du Synvisc® [4].

La prise en charge thérapeutique des premiers stades évolutifs de l'arthrose fémoropatellaire isolée est médicale. Elle a pour principaux objectifs de soulager la douleur, d'entretenir la mobilité articulaire, la fonction musculaire et les capacités fonctionnelles du patient, de ralentir la progression des lésions et de retarder la chirurgie prothétique. Elle fait appel à différentes modalités thérapeutiques dont les effets se potentialisent en agissant sur les stress mécaniques et biochimiques responsables de la dégradation progressive des surfaces articulaires.

RÉFÉRENCES

[1] AMIN S, BAKER K, NIU J *and al.* Quadriceps strength and the risk of cartilage loss and symptom progression in knee osteoarthritis. *Arthritis Rheum 2009; 60(1): 189-98.*

[2] BADER DL, SALTER DM, CHOWDHURY TT. Biomechanical influence of cartilage homeostasis in health and disease. *Arthritis 2011: 979032.*

[3] BOSOMWORTH NJ. Exercise and knee osteoarthritis: benefit or hazard? *Can Fam Physician. 2009; 55(9): 871-8.*

[4] CLARKE S, LOCK V *and al.* Intra-articular hylan G-F20 (Synvisc) in the management of patellofemoral osteoarthritis of the knee (POAK). *Knee, 2005. 12(1): 57-62.*

[5] DEJOUR D, ALLAIN J. Histoire naturelle de l'arthrose fémoropatellaire isolée. *Revue de Chirurgie Orthopédique Suppl. 2004; 90: 89-93.*

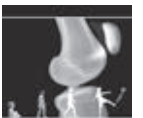
[6] DELARUE Y, DE BRANCHE B, ANRACT P *and al.* Supervised or unsupervised exercise for the treatment of hip and knee osteoarthritis. Clinical practice recommendations. *Ann Readapt Med Phys. 2007;50(9): 759-68, 747-58.*

[7] DUNCAN R, HAY EM, SAKLATVALA J *and al.* Prevalence of radiographic osteoarthritis--it all depends on your point of view. *Rheumatology (Oxford). 2006; 45(6): 757-60.*

[8] DUNCAN R, PEAT G, THOMAS E *and al.* How do pain and function vary with compartmental distribution and severity of radiographic knee osteoarthritis? *Rheumatology (Oxford). 2008; 47(11): 1704-7.*

[9] DUNCAN R, PEAT G, THOMAS E, *and al.* Does isolated patellofemoral osteoarthritis matter? *Osteoarthritis Cartilage. 2009; 17(9): 1151-5.*

[10] GUILBERT S, GOUGEON F, MIGAUD H *and al.* Evolution de l'arthrose fémoropatellaire isolée : devenir à 9 ans de recul moyen de 80 genoux non opérés. *Revue de Chirurgie Orthopédique Suppl. 2004; 90: 86-89.*



[11] HENROTIN Y, CHEVALIER X. Recommandations sur la prise en charge de l'arthrose de la hanche et du genou. Pour qui ? Pourquoi ? Pour quoi faire ? *Presse Med. 2010; 39(11): 1180-8.*

[12] HUNTER DJ, ZHANG YQ, NIU JB *and al.* Patella malalignment, pain and patellofemoral progression: the Health ABC Study. *Osteoarthritis Cartilage. 2007; 15(10): 1120-7.*

[13] JORDAN KM, ARDEN NK, DOHERTY M *and al.* EULAR Recommendations 2003: an evidence based approach to the management of knee osteoarthritis: Report of a Task Force of the Standing Committee for International Clinical Studies Including Therapeutic Trials (ESCISIT). *Ann Rheum Dis. 2003 Dec; 62(12): 1145-55.*

[14] KALICHMAN L, ZHANG Y, NIU J *and al.* The association between patellar alignment and patellofemoral joint osteoarthritis features--an MRI study. *Rheumatology (Oxford). 2007; 46(8): 1303-8.*

[15] LEE DA, NOGUCHI T, FREAN SP *and al.* The influence of mechanical loading on isolated chondrocytes seeded in agarose constructs. *Biorheology. 2000; 37(1-2): 149-61.*

[16] MAZIÈRES B, THEVENON A, COUDEYRE E *and al.* Adherence to, and results of, physical therapy programs in patients with hip or knee osteoarthritis. Development of French clinical practice guidelines. *Joint Bone Spine. 2008; 75(5): 589-96.*

[17] NEOGI T, FELSON D, NIU J *and al.* Association between radiographic features of knee osteoarthritis and pain: results from two cohort studies. *BMJ. 2009, 21; 339: b2844.*

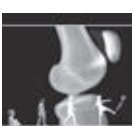
[18] PISCOYA JL, FERMOR B, KRAUS VB, *and al.* The influence of mechanical compression on the induction of osteoarthritis-related biomarkers in articular cartilage explants. *Osteoarthritis Cartilage. 2005; 13(12): 1092-9.*

[19] RICHETTE P, SAUTREUIL P, COUDEYRE E *and al.* Usefulness of taping in lower limb osteoarthritis. French clinical practice guidelines. *Joint Bone Spine. 2008; 75(4): 475-8.*

[20] STEINKAMP LA, DILLINGHAM MF, MARKEL MD *and al.* Biomechanical considerations in patellofemoral joint rehabilitation *Am J Sports Med. 1993; 21(3): 438-44.*

[21] YUSUF E, KORTEKAAS MC, WATT I *and al.* Do knee abnormalities visualised on MRI explain knee pain in knee osteoarthritis? A systematic review. *Ann Rheum Dis. 2011 J; 70(1): 60-7.*

[22] ZHANG W, NUKI G, MOSKOWITZ RW, *and al.* OARSI recommendations for the management of hip and knee osteoarthritis: part III: Changes in evidence following systematic cumulative update of research published through January 2009. *Osteoarthritis Cartilage. 2010; 18(4): 476-99.*



PATELLOFEMORAL CARTILAGE DEFECTS – IS THERE ALWAYS AN OSTEOARTHRITIC TERMINUS?

K.F. ALMQVIST, A.A.M. DHOLLANDER, P. VERDONK, J. VICTOR

CARTILAGE LESIONS

Cartilage defects in the knee joint are frequently present. In 1000 consecutive patients who underwent an arthroscopy, 61% had a chondral or osteochondral lesion, and 19% presented with a focal (osteo)chondral defect [9]. The incidence of cartilage defects in the patellofemoral joint is around 35% with 69% on the patella, 22% on the trochlea and 8% on the both.

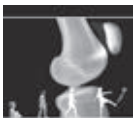
Articular cartilage lesions of the knee are known for their limited potential to heal spontaneously. Persistent defects in the knee will frequently become symptomatic and many progress toward secondary osteoarthritis (OA), affecting daily living and quality of life [2, 4, 7]. On imaging, these lesions are often associated with bone-marrow edema (BME) on MRI. The understanding of the relationship of structural changes in an affected joint and the subsequent development of OA could lead to new treatment strategies to prevent and treat this debilitating condition [6]. Treatment modalities of joint surface lesions aim to restore pain-free joint function by promoting the formation of repair tissue that has the structure and durability of natural hyaline-like articular cartilage [5, 12]. Interventions intended to re-establish the cartilage surface by tissue repair include marrow stimulation techniques such as microfracture (MF) [15], mosaicplasty [8] or regenerative approaches such as autologous chondrocyte implantation (ACI) [2] and other variations on chondrocyte-based therapies. While MF consists of a single-step arthroscopic procedure, ACI requires an arthroscopic intervention to obtain a good-quality cartilage sample for expansion and a subsequent mini-arthrotomy to implant the expanded chondrocytes.

OSTEOARTHRITIS

Osteoarthritis is a chronic disorder characterized by softening and disintegration of articular cartilage, with reactive remodeling phenomena, osteoblastic activity in subchondral bone, new growth of cartilage and of bone (osteophytes) at the joint margins, and capsular fibrosis. Venous congestion and microvascular hyperpression in subchondral bone has repeatedly been reported [11]. Osteoarthritis is the most common joint disease and one of the most frequent causes of physical impairment [10].

Clinical forms of osteoarthritis

The disease can be subdivided into *primary* and *secondary* osteoarthritis. In *primary* or idiopathic osteoarthritis the physiological and biochemical characteristics of the tissues are normal at the onset of the disease, but the joint



is compromised by its function as a weight bearing joint. With abnormal use or increasing age, the involved joint will present the common anatomical changes of osteoarthritis. In postural primary osteoarthritis the joint is subjected to increased loading due to static abnormalities with degenerative changes as a result, e.g. in the knee due to trochleodysplasia or patella alta.

In secondary osteoarthritis the joint tissues are abnormal at the onset of the disease. This condition can occur when the articular cartilage has previously been affected by transient but recurrent inflammation of the synovial membrane as seen in haemochromatosis, in chondrocalcinosis or in burned-out rheumatoid arthritis. Inherited disorders of articular cartilage collagen molecules and metabolic disorders such as ochronosis, acromegaly and chondrocalcinosis can also be the underlying cause of this subgroup of osteoarthritis. Furthermore, secondary osteoarthritis will be present in joints in which the subchondral bone has previously been affected e.g. after intra-articular fractures or chondral trauma. Secondary osteoarthritis has a higher incidence in patients who also present primary osteoarthritis.

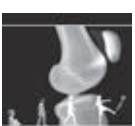
Trauma to a joint with articular incongruity and recurrent instability will also generate osteoarthritic changes. The development of clinical and radiological osteoarthritis in these weight bearing joints is more frequent in people with generalized osteoarthritis than in those without.

Genetic, metabolic and endocrine (primary) factors will alter the physical properties of articular cartilage determining who will be a candidate for developing osteoarthritis, whereas trauma or increased stress determine when and where osteoarthritis will occur.

Since the progression of the disease is slow, the symptoms occur at an advanced age, at variable intervals after the initiating event. The degenerative changes are more related to impact loading than to frictional wear.

Defining and diagnosis of early knee OA [14]

Usually the diagnosis of knee OA can be made by history and physical examination including signs/symptoms of knee pain with joint stiffness, joint crepitus and functional limitations of the knee, typically in a population above 45-50 years old. Diagnosis is confirmed by conventional radiographs demonstrating changes such as joint space narrowing, osteophytes, subchondral bone sclerosis and cysts. The radiograps are graded according to Kellgren (II-IV) or in the patellofemoral joint graded according to Iwano. Early OA of the knee is somewhat more complicated as the patient show limited and sporadic signs/symptoms, only becoming manifest under certain conditions such as after long term loading (jogging or other sports activities…). This early form of knee OA is thought to be a process that displays a number of tissue related phenomena leading to the loss of homeostasis of the knee, and in most cases leading to established OA. It is the clinical recurrence of pain and discomfort of the knee, short periods of stiffness, with in between long periods of very little clinical manifestations eg. slight swelling of the joint, probably due to spontaneous adaption of the patient, that sets the clinician to perform additional investigations (radiographs, ultrasound, MR or arthroscopy). In these cases the history and the clinical examination often suggest a local problem of mechanical nature. Classical radiographs in general are quite disappointing in this sense that, certainly if no earlier X-rays are available as in most cases, very few specific signals are seen. Some joint space narrowing in one compartment, some hints for the formation of what may probably become an osteophyte, and thus typically at best qualified by a Kellgren I or Iwano I, could be seen. It reveals very little of potentially many more tissue processes in the joint. In addition, the robustness of this last scoring categories is however difficult, and studies reveal the quite poor intra- and inter-reader reproducibility of this scoring system.



New advanced imaging techniques are rapidly improving in quality, in particular MR and arthroscopy, revealing a spectrum of alterations in the joint tissue that definitely identify more pathology in the joint than thought, and loss of joint homeostasis. The tissue changes in the knee (in this case the PFJ) typically shows changes in cartilage morphology (joint surface fibrillation and single or multiple cartilage defects), more diffuse cartilage loss, bone marrow changes, slight subchondral sclerosis and cysts, synovitis and some presence of joint fluid… In these cases, MR and arthroscopy are to a certain extent complementary thereby providing an overall assessment of the knee and its structural changes. It is with these tools available in daily clinical practice, that it is clear that in some patients early OA can be diagnosed. Early OA would typically combine slight clinical signs and symptoms as described above but also displaying a number of structural changes that are not seen on conventional X-rays, and yet revealing by advanced imaging changes identifying a knee that could progress and develop established OA. With the current knowledge of the development of these technologies, it is surprising that the current OA definition has not changed since 1986 to capture early OA. Luyten *et al.* [14] suggested that in view of an increasing number of clinical investigations and trials with advanced imaging techniques, it would be useful to define early OA (with advanced imaging and arthroscopy components of this classification). Having this new classification system would be useful to discriminate early OA from frank established OA, and from patients with isolated (traumatic) cartilage damage with an otherwise healthy joint. It is suggested that there is a potentially different (clinical) outcome for different treatment approaches, so studied patient group should be more specified in the future.

Classification criteria early knee OA [14]

The ACR criteria for OA of the knee have been published by Altman *et al.* [1], mostly the combination of clinical and classical radiographic findings. It includes one of the following three findings (age above 50 years, joint stiffness with activity less than 30min, crepitus of the involved joint), together with radiographic changes i.e. osteophytes and joint space narrowing on standardized X-rays. These classification criteria have a 91% sensitivity and 86% specificity.

The definition of classification criteria for symptomatic early knee osteoarthritis is certainly a challenge. However, these classification criteria would be use in the future based on the fact that the patient cannot be classified as established OA by the current used classification system. To make it clinically relevant would still imply the combination of symptoms/signs and structural changes. However, strict radiographic criteria as defined by Kellgren or Iwano will not suffice to capture an early OA patient. Therefore, new classification criteria allowing other methods of structural assessment such as MR and/or arthroscopy could be proposed.

As suggested above, and in view of the current classification criteria for established OA, Luyten *et al.* proposed the following [14]: A patient can be classified as having early OA of the knee based on the following clinical and imaging criteria:

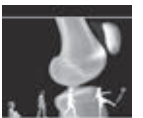
Clinical Criteria

Pain in the knee *(e.g. at least 40mm on VAS scale, and/or duration e.g. for >10 days in the last 3 months, or knee pain at least one day during the last 4 weeks)*.

+ two of the three following criteria:

Imaging Criteria

1) Iwano grade I-II (probable osteophytes and/or joint space narrowing).
2) Arthroscopic findings: ICRS grade 1-2.
3) MR findings demonstrating articular cartilage degeneration and/or subchondral bone marrow lesions…



TREATMENT OF A CARTILAGE DEFECT IN THE PATELLOFEMORAL JOINT

The question is now: does the treatment of cartilage lesions in the patellofemoral joint prevent or slow down the appearance of osteoarthritis in this joint?

Treatment of cartilage defects (conservative or surgical) in the PFJ is performed when there are symptoms such as pain and locking.

The cause of the lesion has to be diagnosed: is it:

1/ due to an underlying structural (morphologic) deformity or soft tissue insufficiency causing recurrent dislocation of the patella or mechanical overload of the joint, or

2/ induced by a blunt trauma or a traumatic dislocation of the patella without any underlying structural deformities.

In the first case the underlying deformity or insufficiency must be addressed to optimize the cartilage repair procedure. This is complex surgery that could be performed in a one or two-stage procedure. Rotational abnormality, patella alta, excessive patella tilt, high grade trochlea dysplasia and/or ligamentous insufficiency must be addressed when one believes they are producing increased or abnormal load on the repaired cartilage. After these correcting procedures the cartilage defect in the PFJ could be addressed by different types of cartilage restoring procedures that will be discussed below.

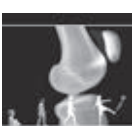
In the second case eg. a blunt trauma, different cartilage procedures have been described. Due to disappointing results after microfracturing patellofemoral chondral lesions, alternative treatment methods in this compartment are preferred. For patella chondral lesions > 2cm^2, different techniques of ACI can be used [13]. For the trochlea, ACI as well or autologous osteochondral transplantation could be done. In long-term follow-up studies with ACI in the PFJ a promising clinical outcome has been noted with improved symptoms [16] and a disappearance of the initially present BME.

CONCLUSION

We need a classification system that includes advanced imaging which is already a part of our current clinical practice. This would allow for a category of early osteaoarthritis.

Resurfacing techniques may have success only if patellofemoral joint kinematics are optimized.

There are no studies today showing that these resurfacing techniques will reduce the incidence of PF OA, although the reduction of pain and symptoms keeps us optimistic.



REFERENCES

[1] ALTMAN R, ASCH E, BLOCH D, *et al.* Development of criteria for the classification and reporting of osteoarthritis – classification of osteoarthritis of the knee. *Arthritis and Rheumatism 1986; 29: 1039-49.*

[2] BRITTBERG M, LINDAHL A, NILSSON A, *et al.* Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. *N Engl J Med. 1997; 331(14): 889-95.*

[3] BUCKWALTER JA. Articular cartilage injuries and potential for healing. *Orthop Sports Phys Ther. 1998; 28(4): 192-202.*

[4] BUCKWALTER JA, MANKIN HJ. Articular cartilage: degeneration and osteoarthritis, repair, regeneration, and transplantation. *Instr Course Lect. 1998; 47: 487-504.*

[5] CHEN F, FRENKEL S, DI CESARE P. Chondrocyte transplantation and experimental therapeutic options for articular cartilage defects. *Am J Orthop. 1997; 26(6): 396-406.*

[6] DING C, JONES G, WLUKA AE, *et al.* What can we learn about osteoarthritis by studying a healthy person against a person with early onset of disease? *Curr Opin Rheumatol. 2010; 22(5): 520-7.*

[7] GELTER AC, HOCHBERG MC, MEAD LA, *et al.* Joint injuries in young adults and risk for subsequent knee and hip osteoarthritis. *Am Intern Med. 2000; 133(5): 321-8.*

[8] HANGODY L, KISH G, KENPATI Z, *et al.* Arthroscopic autogenous osteochondral mosaicplasty for the treatment of femoral condylar articular defects: a preliminary report. *Knee Surg Sports Traumatol Arthrosc. 1997; 5(4): 262-7.*

[9] HJELLE K, SOLHEIM E, STRAND T, *et al.* Articular cartilage defects in 1,000 knee arthroscopies. *Arthroscopy 2002; 18(7): 730-4.*

[10] HUTTON CW. Treatment, pain and epidemiology of osteoarthritis. *Curr Opin Rheumatol. 1990; 2: 765-9.*

[11] IMHOF H, SULZBACHER I, GRAMPP S, *et al.* Subchondral bone and cartilage disease: a rediscovered functional unit. *Invest Radiol. 2000; 35: 581-8.*

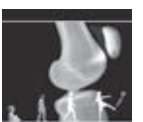
[12] JACKSON DW, SIMON TM. Chondrocyte transplantation. *Arthroscopy 1996; 12(6): 732-8.*

[13] KREUZ PC, STEINWACHS MR, ERGGELET C, *et al.* Results after microfracture of full-thickness chondral defects in different compartments in the knee. *Osteoarthritis and Cartilage 2006; 14:1119-25.*

[14] LUYTEN FP, DENTI M, KON E, *et al.* Definition and classification of early osteoarthritis of the knee. *Knee Surg Sports Traumatol Arthrosc. 2012; 20(3): 401-6.*

[15] STEADMAN JR, RODKEY WG, RODRIGO JJ. Microfracture: surgical technique and rehabilitation to treat chondral defects. *Clin Orthop Relat Res. 2001; 391: S362-69.*

[16] VASILIADIS HS, LINDAHL A, GEORGOULIS AD, *et al.* Malalignement and cartilage lesions in the patellofemoral joint treated with autologous chondrocyte implantation. *Knee Surg Sports Traumatol Arthrosc. 2011; 19(3): 452-7.*



CELL-BASED THERAPY IN ARTICULAR CARTILAGE LESIONS OF THE KNEE

N. NAKAMURA, H. YOSHIKAWA, K. SHINO

INTRODUCTION

The articular cartilage plays an important role in load transmission and decreasing joint friction on articular joints including the knee. Damage or degeneration of this tissue potentially gives rise to several clinical symptoms such as pain and eventually develops osteoarthritis [1]. Nonetheless, it is widely accepted that chondral injury does not usually heal spontaneously due to its avascular surroundings and unique matrix organization. Therefore, a variety of approaches have been tested to improve cartilage healing [1, 2]. Among them, chondrocyte-based therapies have been focused in terms of their feasibility to such incurable lesions.

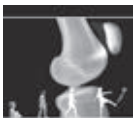
Brittberg *et al.* [3] for the first time described the successful repair of femoral condyle defects of the knee using implantation of autologous cultured chondrocytes. Hereafter, a number of studies followed this procedure and suggested that autologous cartilage implantation (ACI) is an effective procedure for cartilage defects of the knee (See this review). This procedure, however, may have limitations including the sacrifice of undamaged cartilage within the same joint and the in vitro expansion of the cells. Due to the degenerative change in cartilage with aging, the availability of the cells may be limited in elderly individuals [4].

To overcome such potential problems, stem cell therapy has been tested in regenerative tissue repair. Mesenchymal stem cells (MSCs) have the capability to differentiate into a variety of connective tissue cells including bone, cartilage, tendon, muscle, and adipose tissue [5]. These cells may be isolated from various tissues such as bone marrow, skeletal muscle, synovial membrane, adipose tissue, and umbilical cord blood. Although stem cells have potential feasibility to restore articular surface, there are several issues especially associated with safety concerns in clinical application and therefore, as compared with chondrocyte-based therapies, the number of stem cell-based therapies are limited. The purpose of this study was to assess the effectiveness and safety of cell-based therapy, including chondrocyte-based and stem cell-based therapies to clinically significant, symptomatic defects of the knee.

METHODS

Selection of Studies and Data Evaluation

We searched the MEDLINE (1994 to April 2011) to identify all English-language studies evaluating the effect of cell-based therapies for



chondral lesions of the knee. We reviewed all the clinical trials of cell-based therapy for symptomatic chondral lesions of the knee including randomized controlled trials, prospective comparative studies, systematic reviews and case series studies.

Three review authors selected studies for inclusion independently. Records retrieved by the initial search were scanned by review authors to exclude obviously irrelevant studies. Full text articles were retrieved and reviewed independently by the authors. Differences of opinion were resolved by discussion among the authors.

Data from the studies were evaluated independently using the rating system of the *Journal of Bone and Joint Surgery* "Levels of Evidence for Primary Research" for therapeutic studies as published in the Arthroscopy Journal Instructions for Authors. The scoring, specifically Levels I and II, was supported by the methods for methodological quality assessment by the Cochrane Database Systematic Review [6] and Schulz *et al.* [7]. All differences of opinion between the authors were resolved by discussion. Original therapeutic studies of Levels I and II and recent important systematic reviews (Levels I-III) were reviewed in this study.

Analyses

Statistical analyses were not undertaken due to clinical and methodological heterogeneity in the available studies and reviewed studies were described individually.

RESULTS

Twelve randomized controlled trials (RCTs) and three prospective comparative studies, comparing ACI with any other type of cartilage repair surgery, were identified. One prospective comparative study on the outcome of ACI according to the sports activity level and one prospective comparative study on the outcome of ACI according to the accelerated rehabilitation were identified.

Based on these methodological quality assessments, we evaluated five RCTs as Level I and the other RCTs as Level II. In addition, five prospective comparative studies were classified as Level II. We summarize the Level I and II prospective comparative studies in Table.

DISCUSSION

Since year 1994, over 80 studies on cell-based cartilage repair have been published, while majority of the studies are Level IV study which have no control group. Most of the Level IV studies reviewed in here reported promising results, however, there appear lot of room to introduce bias and may not be appropriate to draw any specific conclusion to evaluate the feasibility of the treatment procedures to cartilage repair. Several treatment options are currently performed for chondral lesions including stimulation of a repair process by the penetration of the subchondral bone such as microfracture and drilling, and replacement of the damaged articular surface by osteochondral graft [1, 2]. Since cell-based therapy is generally time and cost-consuming procedures with potential risk accompanying cell culture, it should have significant advantage over such conventional treatment options in clinical application. Therefore, comparative studies are required. However, we could identify only 15 prospective comparative studies including 12 RCTs. All RCTs dealt with ACI, and three of them compared ACI with osteochondral grafting procedures. While these 3 studies had substantial flaws which could introduce bias, we over all found no difference between the cell-based studies and osteochondral grafting. Only one study [9] reported statistically significant results that ACI is superior based on ICRS arthroscopic evaluation. However, only 30% of the total number randomized received arthroscopy. This study also showed that ACI gave better clinical outcomes than mosaicplasty but this was only observed at medial femoral condylar defects and appears to have been based on an unplanned subgroup analysis of participants. When taking into account all participants, significant differences might not be clearly noted.

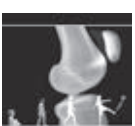


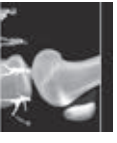
Table: Selected demographic data and results from included prospective controlled studies

Study	Journal	Year	Level	Total enrolled participants	ACI (number)	Control (number)	patient age (years)	primary clinical outcome	followw up (months)	Results
Bentley et al	JBJS Br	2003	II	100	ACI-P:8 ACI-C:46	OAT:42	31.3	Mod Cinn Stanmore score ICRS arthroscopic grading	19	Mod Cinn: superior to OAT in MFC
Horas et al	JBJS Am	2003	II	40	ACI-P:20	OAT:20	33.4	Lysholm score Meyers rating scale Tegner activity score Biopsy	24	Lysholm: inferior to OAT
Dozin et al	CJSM	2005	II	47	ACI-P:22	OAT:25	28.7	Lysholm score IKDC	36	no difference
Knutsen et al	JBJS Am	2004	I	80	ACI-P:40	MFx:40	32.3	Lysholm score Tegner activity score VAS SF-36 Biopsy	24	SF-36: inferior to MFx in physical component score
Knutsen et al.	JBJS Am	2007	I	80	ACI-P:40	MFx:40	32.3	Lysholm score Tegner activity score ICRS VAS SF-36	60	no difference
Saris et al.	AJSM	2008	I	118	characterized ACI:57	MFx:61	33.9	KOOS Biopsy	18	no difference better histology at 12 months
Saris et al.	AJSM	2009	I	118	characterized ACI:57	MFx:61	33.9	KOOS	36	Superior to MFx at 36 months
Vanlauwe et al.	AJSM	2010	I	118	characterized ACI:57	MFx:61	33.9	KOOS	60	no difference
Kon et al.	AJSM	2009	II	50	ACI-Hyalograft C:40	MFx:40	29.8	IKDC Tegner activity score	60	IKDC: superior to MFx at 5 yeas
Visna et al	ACB	2004	II	68	MACI:25	abrasion:25	30.8	Lysholm score IKDC	12	Lysholm and IKDC: superior to abrasion
Gooding et al	Knee	2006	II	80	ACI-P:33	ACI-C:35	30.5	Mod Cinn ICRS arthroscopic grading Biopsy (ICRA)	24	no difference
Bartlett et al	JBJS Br	2005	II	91	ACI-C:44	MACI:47	33.5	Mod Cinn Stanmore score VAS ICRS arthroscopic grading Biopsy	24	no difference
Kreuz et al	AJSM	2007	II	118	ACI-P with low activity:69	ACI-P with high activity:49	35	Mod Cinn ICRS	36	Mod Cinn and ICRS: inferiorr to high activity
Ebert et al	Oseto Cartilage	2008	II	62	MACI with FWB at 2months:31	MACI with FWB at 3months:31	35	KOOS SF-36 VAS VICON motion analysis system	3	KOOS: superior to FWB 3months in knee pain

MFC = lateral femoral condyle; JBJS Br = Journal of Bone and Joint Surgery–British Edition; JBJS Am = Journal of Bone and Joint Surgery–American Edition; ACB = Acta Chirurgica Belgica; AJSM = American Journal of Sports Medicine; CORR = Clinical Orthopaedics and Related Research; CJSM = Clinical Journal of Sports Medicine; Osteoarthritis Cartilage; Osteoarthritis and Cartilage

ACI = autologous chondrocyte implantation; OAT = osteochondral autograft transplantation; MFx = microfracture;, MACI = matrix-induced autologous chondrocyte implantation; ACI-C = autologous chondrocyte implantation-collagen cover; ACI-P = autologous chondrocyte implantation-periosteal cover

ICRS = International Cartilage Repair Society clinical score; Mod Cinn = Modified Cincinnati score; VAS = visual analog scale; Stanmore score; SF-36 PC = SF-36 Physical Component score; Lysholm score; IKDC = International Knee Documentation Committee subjective score; ICRA = International Cartilage Repair Society cartilage repair assessment; KOOS = Knee Injury and Osteoarthritis Outcome Score



Among several surgical interventions to chondral lesions, microfracture technique is presumably most widely performed procedure. In this regard, the result of Level I RCT [10, 11], showing no significant difference in clinical outcome or histological results between ACI and microfracture might have considerable clinical relevance. Conversely, recent Level I RCT demonstrated that ACI using cultured chondrocytes with higher chondrogenic differentiation potentials resulted in better structural repair by histomorphometry and overall histologic evaluation at 12 months [12]. Knee injury and Osteoarthritis Outcome Score (KOOS) at 12 to 18 months after characterized ACI was comparable with microfracture, however, their longer follow-up (3 year) results demonstrated that ACI group showed better KOOS than microfracture group [13]. However, such significance disappeared at 5 year followup [14]. These series of reports of the RCT suggest the importance of long term followup post ACI and moreover of the accumulation of evidence for the evaluation of this new therapy.

Also, recent prospective comparative study revealed that arthroscopic second-generation ACI using Hyalograft C showed significantly better improvement of the International Knee Documentation Committee objective and subjective scores than microfracture at 5-year follow-up [15]. These results suggest that the progress in cell culture technique as well as the optimization of scaffold development might improve the clinical results of new generation ACI. Additional RCTs of ACI with microfracture with more patient number and with longer follow-up will be required to draw a definitive conclusion.

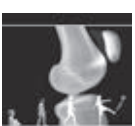
There was one Level II RCT of matrix-guided ACI versus collagen-covered ACI [16].

No significant difference was found between the two interventions in terms of clinical score, arthroscopic scoring or histological assessment. Based on the results no advantage of using collagen I/III based scaffold in ACI was demonstrated. This study did not provide enough information on blind participants or outcome assessors, which should be taken into consideration. Further high quality RCT will be required.

There was one Level II RCT comparing periosteum covered ACI (ACI-P) versus type I/III collagen covered ACI (ACI-C) for the osteochondral defect of the knee [17]. There was no significant difference in the clinical outcome, while significant number of patients who had the periosteum-covered ACI required shaving of a hypertrophied graft. Based on the results, the authors concluded that there is no advantage in using periosteum. However, there was difference in the patients' profile between these groups. The osteochondral defects were located at patella in 61% of ACI-P group and at femoral condyles in 74% of ACI-C group. There was a report showing that there was a difference in the clinical result of ACI between the femoral condyle and patella [18]. Therefore, regional difference might influence the result and there might be caution required to interpret the results.

In addition to the comparative studies with respect to the treatment procedures, there was s prospective comparative study to test the influence of sports activity level and postoperative rehabilitation program on the outcome of ACI. The patients with high sports activity level showed significantly better results in the ICRS and Cincinnati scores than the patients with lower activity level. The result suggests physical training might contribute to the improvement of long-term clinical results [19]. Moreover, there was randomized controlled study to test the influence of accelerated rehabilitation on the outcome of MACI. Accelerated postoperative rehabilitation with 4 week-earlier full weight bearing than the conventional program resulted in better pain relief effect [20].

As compared with ACI, the number of published stem cell therapy is limited. There were only two Level II prospective comparative studies published using stem cell therapy approach. One study reported the comparison of mesenchymal stem cell implantation (MSCI) plus high tibial osteotomy (HTO) with cell-free scaffold transplantation plus HTO [21]. There was no significant difference in clinical



outcome between the groups in this relatively short-term followup study, although there was better arthroscopic and histological score obtained by the MSCI group. There was no randomization process in this study and the followup period was short (less than two years in average). The other study prospectively compared the patients with ACI versus bone marrow derived stem cell therapy in chondral lesions. Two year followup resulted in no significant difference detected in terms of clinical scores (IKDC, Lysholm, Tegnar and SF36) or of histological evaluation [22]. The authors claimed that bone marrow derived stem cell therapy could be an alternative to ACI with potential advantage of less invasiveness (avoidance of repetitive surgery) and of cost effectiveness. Future accumulation of the results of more comparative studies will be required to evaluate the significance of stem cell therapies targeting chondral lesions.

Literature review so far provides no evidence for biological regeneration of hyaline cartilage by any intervention in animal experiments and current available therapies for cartilage resurfacing are still far from regenerative, and rarely restore full function. Therefore, we still need to wait for future advances in the understanding of the biology of cartilage and stem cells, and further technological advancements in therapeutic development.

The limitation of this study was the lack of quantitative evaluation of the quality of the papers. Due to the limited number of the studies included, we only evaluated the studies by the JBJS rating system. Further accumulations of the publications would enable more quantitative evaluation of the treatment effects. Finally, the relevance of articular cartilage repair procedures should be in the long term (10-20 years) and not the short term. We do not have any evidence-based studies on cell-based therapy for articular cartilage lesions for very long-term outcomes. Further longer-term follow-up is needed in relation to the preventive effect from the progression in osteoarthritis.

LITERATURE

[1] BUCKWALTER JA. Articular cartilage injuries. *Clin Orthop Relat Res. 2002;402: 21-37.*

[2] HUNZIKER EB. Articular cartilage repair: basic science and clinical progress. A review of the current status and prospects. *Osteoarthritis Cartilage. 2002;10: 432-63.*

[3] BRITTBERG M, LINDAHL A, NILSSON A, OHLSSON C, ISAKSSON O, PETERSON L. Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. *New England Journal of Medicine 1994; 331(14):889-95.*

[4] HICKERY MS, BAYLISS MT, DUDHIA J, LEWTHWAITE JC, EDWARDS JC, PITSILLIDES AA. Age-related changes in the response of human articular cartilage to IL-1alpha and transforming growth factor-beta (TGF-beta): chondrocytes exhibit a diminished sensitivity to TGF-beta. *J Biol Chem. 2003; 278: 53063-71.*

[5] PITTENGER MF, MACKAY AM, BECK SC, JAISWAL RK, DOUGLAS R, MOSCA JD, *et al.* Multilineage potential of adult human mesenchymal stem cells. *Science. 1999;284: 143-7.*

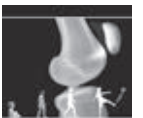
[6] WASIAK J, CLAR C, VILLANUEVA, E. AUTOLOGOUS cartilage implantation for full thickness articular cartilage defects of the knee. *Cochrane Database Syst Rev. 2006 ;3 :CD003323 16856003.*

[7] SCHULZ KF, CHALMERS I, HAYES RJ, ALTMAN DG. Empiral evidence of bias. Dimensions of methodological quality associated with estimates of treatment effects in controlled trials. *JAMA 1995; 273(5): 408-12.*

[8] NAKAMURA N, MIYAMA T, ENGEBRETSEN L, YOSHIKAWA H, SHINO K. Systematic Review. Cell-Based Therapy in Articular Cartilage Lesions of the Knee. *Arthroscopy, 2009, 25: 531-52.*

[9] BENTLEY G, BIANT LC, CARRINGTON RW, AKMAL M, GOLDBERG A, WILLIAMS AM, SKINNER JA, PRINGLE J. A prospective, randomised comparison of autologous chondrocyte implantation versus mosaicplasty for osteochondral defects in the knee. *J Bone Joint Surg Br. 2003 85:223-30.*

[10] KNUTSEN G, ENGEBRETSEN L, LUDVIGSEN TC, DROGSET JO, GRONTVEDT T, SOLHEIM E, STRAND T, ROBERTS S, ISAKSEN V, JOHANSEN O. Autologous chondrocyte implantation compared with microfracture in the knee. A randomized trial. *J Bone Joint Surg Am. 2004 86-A: 455-64.*



[11] KNUTSEN G, DROGSET JO, ENGEBRETSEN L, GRØNTVEDT T, ISAKSEN V, LUDVIGSEN TC, ROBERTS S, SOLHEIM E, STRAND T, JOHANSEN O. A randomized trial comparing autologous chondrocyte implantation with microfracture. Findings at five years. *J Bone Joint Surg Am. 2007 Oct;89(10): 2105-12.*

[12] SARIS DB, VANLAUWE J, VICTOR J, HASPL M, BOHNSACK M, FORTEMS Y, VANDEKERCKHOVE B, ALMQVIST KF, CLAES T, HANDELBERG F, LAGAE K, VAN DER BAUWHEDE J, VANDENNEUCKER H, YANG KG, JELIC M, VERDONK R, VEULEMANS N, BELLEMANS J, LUYTEN FP. Characterized chondrocyte implantation results in better structural repair when treating symptomatic cartilage defects of the knee in a randomized controlled trial versus microfracture. *Am J Sports Med. 2008 Feb; 36(2): 235-46.*

[13] SARIS DB, VANLAUWE J, VICTOR J, ALMQVIST KF, VERDONK R, BELLEMANS J, LUYTEN FP, TIG/ACT/01/2000&EXT STUDY GROUP. Treatment of symptomatic cartilage defects of the knee: characterized chondrocyte implantation results in better clinical outcome at 36 months in a randomized trial compared to microfracture. *Am J Sports Med. 2009 Nov; 37 Suppl 1:10S-19S.*

[14] VANLAUWE J, SARIS DB, VICTOR J, ALMQVIST KF, BELLEMANS J, LUYTEN FP; TIG/ACT/01/2000&EXT STUDY GROUP. Five-year outcome of characterized chondrocyte implantation versus microfracture for symptomatic cartilage defects of the knee: early treatment matters. *Am J Sports Med. 2011 Dec;39(12):2566-74.*

[15] KON E, GOBBI A, FILARDO G, DELCOGLIANO M, ZAFFAGNINI S, MARCACCI M. Arthroscopic Second-Generation Autologous Chondrocyte Implantation Compared With Microfracture for Chondral Lesions of the Knee. Prospective Nonrandomized Study at 5 Years. *Am J Sports Med. 2009 Jan; 37(1): 33-41.*

[16] BARTLETT W, SKINNER JA, GOODING CR, CARRINGTON RW, FLANAGAN AM, BRIGGS TW, BENTLEY G. Autologous chondrocyte implantation versus matrix-induced autologous chondrocyte implantation for osteochondral defects of the knee: a prospective, randomised study. *J Bone Joint Surg Br. 2005 May;87(5): 640-5.*

[17] GOODING CR, BARTLETT W, BENTLEY G, SKINNER JA, CARRINGTON R, FLANAGAN A. A prospective, randomised study comparing two techniques of autologous chondrocyte implantation for osteochondral defects in the knee: Periosteum covered versus type I/III collagen covered. *Knee 2006 Jun; 13(3): 203-10. Epub 2006 Apr 27.*

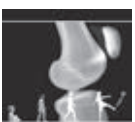
[18] PETERSON L, MINAS T, BRITTBERG M, NILSSON A, SJOGREN-JANSSON E, LINDAHL A. Two- to 9-year outcome after autologous chondrocyte transplantation of the knee. *Clin Orthop Relat Res. 2000 May;(374):212-34.*

[19] KREUZ PC, STEINWACHS M, ERGGELET C, LAHM A, KRAUSE S, OSSENDORF C, MEIER D, GHANEM N, UHL M. Importance of sports in cartilage regeneration after autologous chondrocyte implantation: a prospective study with a 3-year follow-up. *Am J Sports Med. 2007 Aug; 35(8): 1261-8.*

[20] EBERT JR, ROBERTSON WB, LLOYD DG, ZHENG MH, WOOD DJ, ACKLAND T. Traditional vs accelerated approaches to post-operative rehabilitation following matrix-induced autologous chondrocyte implantation (MACI): comparison of clinical, biomechanical and radiographic outcomes. *Osteoarthritis Cartilage. 2008 Oct; 16(10):1131-40. Epub 2008 Apr 22.*

[21] WAKITANI S, IMOTO K, YAMAMOTO T, SAITO M, MURATA N, YONEDA M. Human autologous culture expanded bone marrow mesenchymal cell transplantation for repair of cartilage defects in osteoarthritic knees. *Osteoarthritis Cartilage. 2002 Mar; 10(3): 199-206.*

[22] NEJADNIK H, HUI JH, FENG CHOONG EP, TAI BC, LEE EH. Autologous bone marrow-derived mesenchymal stem cells versus autologous chondrocyte implantation: an observational cohort study. *Am J Sports Med. 2010 Jun; 38(6): 1110-6.*



THE PARTIAL LATERAL FACETECTOMY

E. GANCEL, Y. YERCAN, G. DEMEY, S. LUSTIG, E. SERVIEN, P. NEYRET

INTRODUCTION

Radiographic prevalence of isolated patello-femoral arthritis was 14% in women and 15% in men older than 60 years old according to McAlindon [12]. Open and arthroscopic procedures, which include subchondral drilling, realignment procedures, patellectomy, patello-femoral arthroplasty and total knee replacement could be purposed. The purpose of the study was to investigate the intermediate-term results of partial lateral patella facetectomy in patients with lateral isolated patellofemoral arthritis.

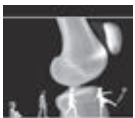
METHODS

We reviewed partial lateral facetectomy in 24 knees in 19 patients (14 female and 5 male). These patients were operated between 2004 and 2009. The mean follow-up was 61.8 months (range 36 to 90) with 3 years follow-up at least. All patients had had failed conservative treatment. All patients had physical examination and standard radiographs before surgery and at each follow-up. These included standard weight bearing AP view, posterior-anterior weight bearing view in 45° of knee flexion, a lateral view in 30° of knee flexion and an axial view in 30° of knee flexion. We used the Iwano radiographic evaluation scale [8] to grade the severity of the patellofemoral osteoarthritis. We used the Knee Society Score [7] before surgery and at each follow-up.

The inclusion criteria were: isolated lateral patellofemoral osteoarthritis, lateral patella pain on physical examination and anterior tuberosity-trochlear groove smaller than 16mm. Patients with osteoarthritis in medial or lateral compartment, medial or central patellofemoral osteoarthritis, history of fracture, anterior tuberosity-trochlear groove bigger than 16mm were excluded.

SURGICAL TECHNIQUE

With the patient supine and under tourniquet control, the knee was approached through a lateral parapatellar incision. A lateral retinacular release was done from the inferior to the superior pole of the patella. We did not injure the vastus lateralis. With the knee extended, the patella and trochlear groove were observed for cartilage lesions and checked for patellofemoral congruency. About 1 to 1.5cm of lateral border of the patella including osteophytes and 1 to 2mm of cartilage were resected with an oscillating saw. We did a meticulous hemostasis with bone wax if necessary. Low-molecular-weight heparin was used prophylactically for venous thromboembolism in all patients. Range of motion and isometric quadriceps exercises



were initiated as soon as possible and weight bearing was allowed the first week post-operatively.

RESULTS

On the 19 patients who underwent partial lateral facetectomy, 3 were lost of follow-up. One patient required total knee arthroplasty during our follow-up (2 years after the first surgery).

The total knee society score improved from a mean of 68.6 points (range 25 to 80) preoperatively to a mean of 84.6 points at the time of the final follow-up (range 70 to 100). The functional score improved from 89.8 points (range 26 to 100) before surgery to 93.5 points at last follow-up (range 50 to 100).

Patellofemoral arthritis was present in all knees preoperatively. Five knees had *stage 1* patello-femoral osteoarthritis, eight had a *stage 2*, six a *stage 3* and seven a *stage 4*. At latest follow-up, five knees had a stage 2, six a stage 3 and nine a stage 4 (fig. 1). Before surgery, no patient had radiological osteoarthritis on tibiofemoral component. During follow-up, 2 patients had tibiofemoral osteoarthritis which one received a total knee arthroplasty.

DISCUSSION

Many operative techniques have been advocated for patellofemoral arthritis. Anterior or anteromedialization of tibial tubercle [6, 9, 19, 20], lateral retinacular release [1], partial lateral facetectomy of the patella [13, 14, 18, 21, 22], patellofemoral joint replacement [2, 10] or total knee arthroplasty [4, 5, 11, 15, 16, 17] could be purposed. We think that the partial lateral facetectomy of the patella is a reliable surgical method compared with other choices in selected cases.

Mc Caroll [13] was the first to describe the results of partial lateral facetectomy in 57 patients. Martens [14] obtained 90% good results in 20 patients treated by this procedure. Yercan [22] described good results with a 8 years mean follow-up. For him, ideal candidates for this procedure are relatively young and active patients who have High physical demands and want to maintain their active lifestyles. According to Wetzels [21], partial lateral patellectomy satisfied in half of the cases at 10 years follow-up.

Anterior elevation or anteromedialization of tibial tubercle could be purposed to decrease patellofemoral contact joint pressure but there's no consensus on the optimal elevation. High rate of complication is described [3].

Partial facetectomy can be associated with tibial tubercle medialization if anterior tuberosity-trochlear groove was higher than 16mm. We didn't associated facetectomy with reconstruction of medial patellofemoral ligament.

Arthroplasty (patellofemoral arthroplasty or total knee replacement) can treated patello-femoral osteoarthritis. Patellofemoral arthro-plasty shows better results recent years but this

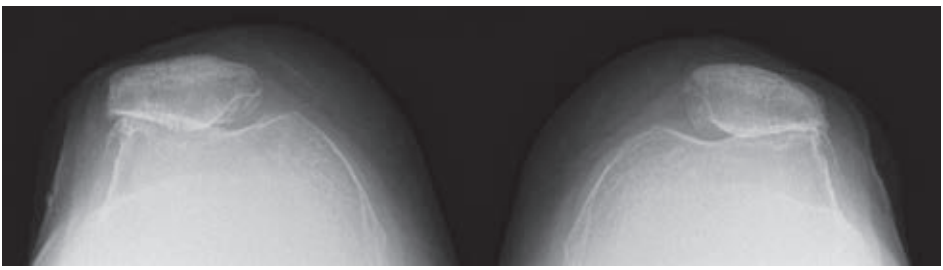
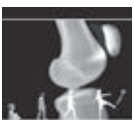


Fig. 1 : Patellofemoral osteoarthritis grade four at 6 years follow-up.



surgical technique is difficult to master for inexperienced surgeons. Total knee arthroplasty shows good results for patellofemoral osteoarthritis. Particular technical difficulties such as patellar tracking are well known by surgeons to avoid these pitfalls. According to Yercan, total knee arthroplasty may be a good surgical method of choice for patients who are elderly, severely disabled, bilaterally involved, and for patients who engage in only mild physical activities. However, in young and more active patients who have isolated patello-femoral osteoarthritis, total knee arthroplasty is not the best solution for a durable result.

CONCLUSION

Partial lateral facetectomy is a simple and reliable treatment to treat isolated patellofemoral osteoarthritis. On failure, this technique has the advantage of not compromising the establishment of a total knee arthroplasty.

LITERATURE

[1] ADERINTO J, COBB AG. Lateral release for patello–femoral osteoarthritis. *Arthroscopy 18 : 399-403. 2002.*

[2] ARGENSON JN, FLECHER X, PARATTE S, AUBANIAC JM. Patellofemoral arthroplasty: an update. *Clin Orthop Relat Res; 440: 50-53. 2005.*

[3] BELLEMANS J, CAUWENBERGHS F, BRYS P, VICTOR J, FABRY G. Fracture of the proximal tibia after Fulkerson anteromedialtibial tubercle transfer. A report of four cases. *Am J Sports med; 26: 300-2. 1998.*

[4] DALURY DF. Total knee replacement for patellofemoral disease. *J Knee Surg; 18(4): 274-7. 2005.*

[5] DEJOUR D, BARBOSA J, JACQUOT N, NEYRET P. Résultats des prothèses totales de genou dans l'arthrose fémoro-patellaire isolée. *Rev Chir Orthop; 90: 111-3. 2004.*

[6] FULKERSON JP. Anteromedialization of the tibial tuberosity for patellofemoral malalignment. *Clin Orthop Relat Res; 177: 176-81. 1983.*

[7] INSALL JN, DORR LD, SCOTT RD, SCOTT WN. Rationale of the Knee Society clinical rating system. *Clin Orthop Relat Res; 248 : 13-14. 1989.*

[8] IWANO T, KUROSAWA H, TOKUYAMA H, HOSHIKAWA Y. Roentgenographic and clinical findings of patellofemoral osteoarthritis. *Clin Orthop relat Res; 253 : 190-7. 1990.*

[9] JENNY JY, SADER Z, HENRY A, JENNY G, JAEGER JH. Elevation of the tibial tubercle for patellofemoral pain syndrome. An 8-to15-year follow-up. *Knee Surg Sports Traumatol Arthrosc ; 4(2): 92-96. 1996.*

[10] KOOJIMAN HJ, DRIESSEN AP, VAN HORN JR. Long-term results of patellofemoral arthroplasty. A report of 56 arthroplasties with 17 years of follow-up. *J Bone Joint Surg Br; 85(B) : 836-40. 2003.*

[11] LASKIN RS, VAN STEIJN M. Total knee replacement for patients with patellofemoral arthritis. *Clin Orthop Relat Res;8(6): 89-95. 1999.*

[12] McALINDON TE, SNOW S, COOPER C, DIEPPE PA. Radiographic patterns of osteoarthritis of the knee joint in the community: The importance of the patello-femoral joints. *Ann Rheum Dis 51: 844-849. 1992.*

[13] McCAROLL JR, O'DONOGHUE DH, GRANA WA. The surgical treatment of chondromalacia of the patella. *Clin Orthop relat Res; 175: 130-4. 1983.*

[14] MARTENS M, DE RYCKE J. Facetectomy of the patella in patellofemoral osteoarthritis. *Acta Orthop Belg ; 56: 563-7. 1990.*

[15] MEDING JB, WING JT, KEATING EM, RITTER MA. Total knee arthroplasty for isolated patellofemoral arthritis in younger patients. *Clin Orthop Relat Res; 464: 78-82. 2007.*

[16] MONT MA, HAAS S, MULLICK T, HUNGERFORD DS. Total knee arthroplasty for patellofemoral arthritis. *J Bone Joint Surg Am; 84(11): 1977-81. 2002.*

[17] PARVIZI J, STUART MJ, PAGNANO MW, HANSSEN AD. Total knee arthroplasty in patients with isolated patellofemoral arthritis. *Clin Orthop Relat Res; 392: 147-52. 2001.*

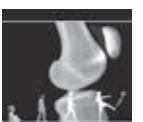
[18] PAULOS LE, O'CONNOR DL, KARISTINOS A. Partial lateral patellar facetectomy for treatment of arthritis due to lateral patellar compression syndrome. *Arthroscopy; 24(5) : 547-53. 2008.*

[19] RADIN E, PAN H. Long-term follow-up study on the Maquet procedure with special reference to the causes of failure. *Clin Orthop Relat Res; 290: 253-8. 1993.*

[20] SAKAI N, KOSHINO T, OKAMOTO R. Pain reduction after anteromedial displacement of the tibial tuberosity: 5-year follow-up in 21 knees with patellofemoral arthrosis. *Acta Orthop Scand; 67 (1): 13-15. 1996.*

[21] WETZELS T, BELLEMANS J. Patellofemoral osteoarthritis treated by partial lateral facetectomy: results at long-term follow-up. *Knee. 2011. In press.*

[22] YERCAN HS, AÏT SI SELMI T, NEYRET P. The treatment of patellofemoral osteoarthritis with partial lateral facetectomy. *Clin Orthop Relat Res; 436: 14-19. 2005.*



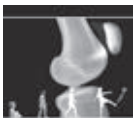
PATELLOFEMORAL ARTHROPLASTY

R.J. DE JONG, H.P.W. VAN JONBERGEN, A. VAN KAMPEN

The debate on patellofemoral arthroplasty (PFA) is becoming more intriguing as our knowledge on the subject continues to grow. The advantages of PFA above total knee arthroplasty to address symptomatic isolated patellofemoral osteoarthritis (PFOA) seem obvious; PFA targets only the involved (patellofemoral) compartment, sparing the tibiofemoral cartilage, menisci, and cruciate ligaments. Knee kinematics and propriocepsis are better preserved which are strong arguments favoring PFA above TKA for treating PFOA. Skepticism is losing ground as more promising, including long-term, PFA results appear [1-5] and the results of total knee arthroplasty after conversion are not negatively affected [6]. Despite these reassuring findings there are still certain issues that need to be addressed. Choosing the right patient and the right implant is the hallmark to successful treatment of PFOA. To date this remains difficult. The low incidence of PFOA and PFA is the reason that level l evidence on the subject is not, and is not likely to become, available. Which makes proving the many theories about PFA, for example: "Do differences in etiology play a role on PFA outcome?", a big challenge. Also declaring superiority of one PFA design over another is skating on thin ice when it is not backed up by a randomized controlled trial. On top of that the low volumes of PFA does not give every surgeon the chance of getting familiar with the surgical procedure and the many pitfalls of implantation of this technical high demanding prosthesis. These issues: choosing the implant, choosing the patient, the role of etiology and the enhanced risk on surgical error (and its consequences) need answering. These answers ideally will have to come from combined efforts of orthopedic surgeons throughout the world who are playing the field of patellofemoral pathology. In the meantime we hope that this overview can facilitate your future decision making on PFA.

Patient selection is crucial in PFA, especially pre-existent femorotibial OA has a negative effect on PFA succes rates. Symptomatic isolated patellofemoral osteoarthritis is the common indication for PFA. Although being a distinct entity PFOA can act as a precursor of generalized knee OA [22] making clear that when considering PFA the surgeon must inform the patient of the possibility of conversion to TKA. In patients with knee osteoarthritis, isolated patellofemoral joint osteoarthritis occurs in approximately 4% to 24% of patients [7-9]. Two retrospective case series noted that isolated patellofemoral osteoarthritis represented approximately 5% of patients undergoing total knee replacement [10, 11].

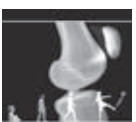
Isolated patellofemoral osteoarthritis can be considered a distinct entity from femorotibial osteoarthritis. Systemic and local factors influencing both the development and maintenance of patellar cartilage differ from those in the femorotibial joint [12]. In addition,



PFOA is disparately distributed across the patellofemoral joint itself, with the patella side demonstrating more severe degeneration earlier in the disease process compared to the juxtaposed femoral groove [13, 14]. Several hypotheses have been suggested based on these observations, including differences in load duration, chondrocyte metabolism, and histological, material and compositional properties [13]. The etiology of PFOA has proposedly three major pathologic pathways: posttraumatic, history of instability and primary (or idiopathic) [18, 19]. Long-term studies showed no difference in PFA survival between these diagnostic groups [1], although no prospective data are available. Post-traumatic PFOA can result from direct trauma to the patellar cartilage, non-anatomic reduction of displaced patellar fractures, excessive callus formation in comminuted fractures or suboptimal refixation of the patellar tendon after partial patellectomy [20]. Patellofemoral arthroplasty cohort studies included patients with end-stage post-traumatic osteoarthritis in 12-32% [1, 21]. Although patellar dislocation can result in traumatic joint cartilage lesions, the risk factors for instability itself also predisposes a patient to the development of osteoarthritis [12]. These risk factors include deficiencies in one of three anatomical structures that stabilize the patellofemoral joint: trochlear groove geometry, the medial and lateral retinacula (including the medial patellofemoral ligament (MPFL)) and the alignment of the extensor apparatus (including the quadriceps muscles, patellar tendon, and tibial tuberosity). Primary isolated patello-femoral osteoarthritis is diagnosed when no history of trauma or findings of instability are noted. This subgroup is probably over-represented in clinical studies due to unrecognized instability.

When asymptomatic, the natural course of PFOA can be benign, as shown by Guilbert et al. in a cohort of patients treated nonoperatively for isolated patellofemoral osteoarthritis; 90% had not been operated upon after a follow-up of 9 years [23]. They concluded that PFOA is well tolerated as long as the femorotibial compartments were not involved in the arthritic process. If symptomatic PFOA is not well tolerated and the indication for PFA is set, there still is the possibility that OA progresses femorotibial. This occurs less in posttraumatic PFOA then in patients after a history of PF instability or with idiopathic (primary) PFOA.

The diagnosis of PFOA is based on a typical history of anterior knee pain after prolonged sitting or upon rising from a chair, and pain when descending and ascending stairs. The pain usually is less severe when walking on level ground. Clinical findings are not specific and include quadriceps wasting, pain, and crepitus emanating from the anterior compartment [15]. Compared to the operative findings of patellofemoral osteoarthritis, patellofemoral crepitus as a sign has a sensitivity of 89% and specificity of 82% [16]. Findings of patello-femoral instability are common. Regardless of the presenting age or etiology, symptomatic PFOA is rare [17]. Radiological findings of PFOA using the lateral and axial (skyline) view typically include patellofemoral joint space narrowing without signs of femorotibial osteoarthritis. The skyline view has been suggested to be more sensitive than the lateral view for assessing patellofemoral osteoarthritis, though over 75% of orthopedic surgeons in the United Kingdom do not use the skyline view in the routine investigation of knee osteoarthritis [24]. Compared to intra-operative findings, no significant differences in terms of sensitivity and specificity were found between lateral (sensitivity 82%, specificity 65%) and skyline (sensitivity 79%, specificity 80%) radiographs [16]. A clinical study comparing lateral and skyline radiographic views with intra-operative findings demonstrated that a normal skyline radiograph can be used to reliably exclude clinically significant (Collins grade 4) patellofemoral osteoarthritis [25]. Magnetic resonance imaging (MRI) studies have shown that joint space narrowing in axial view radiographs has a high specificity for cartilage defects detected by MRI in the same joint [26]. A significant association has been found between the presence of an osteophyte in the patellofemoral joint and knee pain; other MRI findings, including focal or diffuse cartilaginous abnormalities, subchondral cysts, and bone marrow edema were not associated with knee symptoms [27]. In the majority of cases,

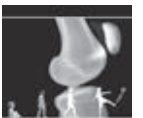


patellofemoral osteoarthritis can be reliably assessed using conventional radiographs alone. However, the exclusion of significant degenerative changes in both femorotibial compartments is important, especially when patellofemoral arthroplasty is considered. For this reason technetium-99m bone scans can be used. A bone scan gives more accurate information on femorotibial and PF involvement because of its higher sensitivity for detecting OA compared to conventional radiographs. This is often very useful in the decision making process.

Examining conventional radiographs of patients with isolated patellofemoral osteoarthritis, the majority (70-90%) showed lesions on the lateral side of the trochlea [15, 28], demonstrating degenerative changes due to lateral malalignment with subluxation. A small number of cases (<10%) showed medial facet and medial trochlea disease. The precise etiology of this condition is unknown but it might suggest that there is some kind of medial overload causing these changes, varus-valgus alignment may influence which compartment is affected [29]. The remaining cases demonstrated symmetrical patterns of wear affecting equally both the medial and lateral facets of the patella and trochlea groove [28].

Implant designs for PF prosthesis to treat PFOA already have seen a long history. The need for a PF prosthesis was fed by unsuccessful non-operative treatment modalities and insufficient surgical procedures to address PFOA and PF pain, such as patellectomy, lateral release and/or realignment procedures. Joint-preserving surgical treatment generally results in insufficient, unpredictable, or only short-term improvement [1]. The first attempt to replace the PF joint surface was a Vitallium patella resurfacing prosthesis introduced by McKeever in 1948 [30]. Concerns about the trochlea in patella resurfacing led to the development of PFA as we know it today. In 1975, Lubinus introduced the patella glide bearing total prosthesis [31]. The early experiences showed that the prosthesis did relieve retropatellar pain. However, reports of medium-term results described satisfactory, good or excellent results in only 45% to 64% of cases, prompting some authors to discontinue the use of this prosthesis [28, 32]. The unconstrained anatomical implant, which was narrow and short, was suspected of making the patella susceptible to malalignment and impingement [28]. Because of the relative large radius of the curvature, placing the trochlear component in flexion was often necessary, leaving the proximal edge of the prosthesis offset from the anterior femoral shaft, resulting in snapping and catching [33]. Blazina *et al.* had started using the Richards patellofemoral prosthesis in 1974 and reported about the favorable short-term results of 57 replacements in 1979 [34]. The non-anatomic trochlear component was highly constrained with a deep central groove, and the polyethylene patellar component had a longitudinal ridge. In reviewing the failures, Blazina *et al.* noted areas of concern, such as the tracking of the patellar prosthesis when coming in and out of the trochlear groove proximally and distally, resulting in similar catching phenomena as were seen with the Lubinus design. Suggestions for a more shallow trochlear groove and adapt the shape of the patellar component accordingly were made [34]. Despite these concerns, the Richards prosthesis has been widely used since that time. The number of re-operations for patellar maltracking is high in all reported series [1, 35]. Recently, we published long-term outcomes of the Richards type II prosthesis in two studies. With a median of 13.3 [1] and 9.2 [36] years of follow-up in 181 [1] and 33 [36] knees; the survival, with revision for any reason as the endpoint, was determined at 84% at 10 years and 69% at 20 years [1] and 88% and 80% respectively [36]. In Table 1, we summarized these results, which are comparable to earlier published results on this PFA design [1, 2, 3, 36].

The current concept of different designs of PFA is to restore or recreate a PF joint with preservation of as much bone stalk as possible and not to make alterations to knee kinematics. There is a wide diversity of PF implants, from constrained types to less constraint designs. All but one PFA prosthesis are cemented and all consist of a polyethylene patellar component and a metal (Cobalt-Chromium) trochlea component. It is beyond the scope of this overview to review all available PFA designs in detail. It suffices to state that the problems



Study	Number of knees/ patients	FU	Age	Satisfaction (good/excellent)	Survival	Conversion to TKA (%)
Jonbergen [1]	181/161	13.3 (2-30.6)	52	N/A	84% at 10 years 69% at 20 years	13 (23/181)
Hoogervorst [36]	33/24	9.2 (2-20)	48 (33-82)	95%	88% at 10 years 80% at 20 years	18 (6/33)

Table 1: Long-term PFA results, recent own publications on Richards II prosthesis

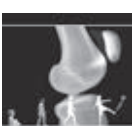
encountered in the first designs, such as catching and malalignment/-tracking remain a challenge today, and that each specific design often comes with its own specific problems. When searching available literature on the different designs of PFA the following observations are made: first of all, many recent designs do not have long-, middle- or even short-term results. Making evidence based decisions to choose between PFA implants based on available literature therefore impossible. There is only one type (Richards I-III (Smith & Nephew, Memphis, TN, USA)) of which long-term results are available (Table 1) [1, 2, 3, 36]. Obviously further research is warranted. Secondly, in most cases of failure and/or additional surgery the problems are caused by mechanisms that can be contributed to either specific design problems, surgical errors or inadequate patient selection. Especially not recognized pre-existent femorotibial OA and pre-existing PF instability are factors that worsen PFA mid-term outcome. In most of these cases conversion to TKA was inevitable. We summarized the encountered problems with PFA as we found them in the available literature in Table 2, which provides on overview and an insight in the complex task of creating a well-functioning prosthesis.

Despite of these problems, promising results have been published with the early PF prosthesis with 10-year survival rates of approximately 80%. As to make a choice between available PF prosthesis based on current knowledge, literature slightly favors the Avon and Kine-Matic prosthesis, based on the low prosthesis related revision rate and high survival rates. The differences in outcome measures between studies do not allow objective comparison and no definitive conclusions can be made.

An interesting question is if there is something like one PFA solution. As briefly mentioned earlier one theory is that the three different etiologies of PFOA require different approaches in regard to PFA. When you look at PFOA with underlying PF instability, it is tempting to make an analogy to TKA; in unstable knees more constrained (PS, hinged) types of TKA are used. Hence, a more constrained type of PFA (e.g. Richards) might be more suitable for PFOA with underlying instability. Using the same analogy, posttraumatic, stable, PFOA is probably better addressed with a less constrained type of PFA, which allows the PF joint to retain its own alignment characteristics. Although a tempting thought, current data does not suggests different outcomes in PFA when different etiologies are compared. Prospective research is needed.

Another interesting fact is that up till now no one has tried replacing just the trochlear surface; without a patellar component. This is a tested and proved concept in TKA and might be an option in PFA.

Conclusively PFA is here to stay; since its introduction many patients have been helped with good results. There is room for improvement however. The low patient numbers and the diversity of underlying causes make it a challenge for the future to come up with either one perfect fit for all types of PFOA or the perfect customization possibilities to meet the knees' needs.



Encountered Problems	Design, surgical error, patient selection error
Femorotibial OA	Patient selection error
Persistent patellofemoral instability	Design, surgical error, patient selection error
Malpositioning	Surgical error
Effusion	Not related
«Hooking/catching/snapping» coming from deep flexion at transition cartilage to prosthesis	Design: LCS/Richards/Lubinus/KineMatch. Surgical error
Stiffness/arthrofibrosis	Design: Autocentric
"Catching" on transition proximal femur/prosthesis around extension	Design: Autocentric/Lubinus. Surgical error
Medial/lateral «catching»	Design: Avon/Lubinus. Surgical error
ACL irritation	Design: Lubinus
"Toggling"	Design: Richards
Loosening of Trochlea component	Rarely encountered, no obvious cause
Loosening of Patellar component	Rarely encountered, no obvious cause
Wear	Rarely encountered, no obvious cause
Breaking PE	Design: Kinematch/LCS
Dislocation PE/Patella	Design: LCS

Table 2: Encountered problems in PFA

LITERATURE

[1] VAN JONBERGEN H.P. *et al.* Long-term outcomes of patellofemoral arthroplasty. *J Arthroplasty, 2010. 25(7): p. 1066-71.*

[2] KOOIJMAN HJ, AP DRIESSEN, JR VAN HORN Long-term results of patellofemoral arthroplasty. A report of 56 arthroplasties with 17 years of follow-up. *J Bone Joint Surg Br, 2003. 85(6): p. 836-40.*

[3] CARTIER P, JL SANOUILLER, A KHEFACHA. Long-term results with the first patellofemoral prosthesis. *Clin Orthop Relat Res, 2005(436): p. 47-54.*

[4] SISTO DJ, VK SARIN. Custom patellofemoral arthroplasty of the knee. *J Bone Joint Surg Am, 2006. 88(7): p. 1475-80.*

[5] ACKROYD CE, B CHIR. Development and early results of a new patellofemoral arthroplasty. *Clin Orthop Relat Res, 2005(436): p. 7-13.*

[6] VAN JONBERGEN HP, DM WERKMAN, A VAN KAMPEN. Conversion of patellofemoral arthroplasty to total knee arthroplasty: A matched case-control study of 13 patients. *Acta Orthop, 2009. 80(1): p. 62-6.*

[7] BARRETT JP. Correlation of Roentgenographic Patterns and Clinical Manifestations of Symptomatic Idiopathic Osteoarthritis of the Knee. *Clin Orthop Relat Res, 1990(253): p. 179-83.*

[8] MCALINDON TE *et al.* Radiographic patterns of osteoarthritis of the knee joint in the community: the importance of the patellofemoral joint. *Ann Rheum Dis, 1992. 51(7): p. 844-9.*

[9] DAVIES AP. The Radiologic Prevalence of Patellofemoral Osteoarthritis. *Clin Orthop Relat Res, 2002(402): p. 206-12.*

[10] LASKIN RS, M VAN STEIJN. Total knee replacement for patients with patellofemoral arthritis. *Clin Orthop Relat Res, 1999(367): p. 89-95.*

[11] THOMPSON NW *et al.* Total knee arthroplasty without patellar resurfacing in isolated patellofemoral osteoarthritis. *J Arthroplasty, 2001. 16(5): p. 607-12.*

[12] HINMAN RS, KM CROSSLEY. Patellofemoral joint osteoarthritis: an important subgroup of knee osteoarthritis. *Rheumatology (Oxford), 2007. 46(7): p. 1057-62.*

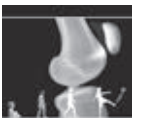
[13] CLARK AL. Osteoarthritis: what we have been missing in the patellofemoral joint. *Exerc Sport Sci Rev, 2008. 36(1): p. 30-7.*

[14] LONNER JH, JG JASKO, RE BOOTH Jr. Revision of a failed patellofemoral arthroplasty to a total knee arthroplasty. *J Bone Joint Surg Am, 2006. 88(11): p. 2337-42.*

[15] IWANO T *et al.* Roentgenographic and clinical findings of patellofemoral osteoarthrosis. With special reference to its relationship to femorotibial osteoarthrosis and etiologic factors. *Clin Orthop Relat Res, 1990(252): p. 190-7.*

[16] BHATTACHARYA R *et al.* The knee skyline radiograph: its usefulness in the diagnosis of patello-femoral osteoarthritis. *Int Orthop, 2007. 31(2): p. 247-52.*

[17] KRAJCA-RADCLIFFE JB, TP COKER. Patellofemoral arthroplasty. A 2- to 18-year followup study. *Clin Orthop Relat Res, 1996(330): p. 143-51.*



[18] ARGENSON JN, JM GUILLAUME, JM AUBANIAC. Is there a place for patellofemoral arthroplasty? *Clin Orthop Relat Res, 1995(321): p. 162-7.*

[19] VAN JONBERGEN HP, RW POOLMAN, A. VAN KAMPEN. Isolated patellofemoral osteoarthritis. *Acta Orthop, 2010. 81(2): p. 199-205.*

[20] HAKLAR U *et al.* Arthroscopic inspection after the surgical treatment of patella fractures. *Int Orthop, 2009. 33(3): p. 665-70.*

[21] ARGENSON JN *et al.* Patellofemoral arthroplasty: an update. *Clin Orthop Relat Res, 2005. 440: p. 50-3.*

[22] MAZZUCA SA *et al.* Risk factors for progression of tibiofemoral osteoarthritis: an analysis based on fluoroscopically standardised knee radiography. *Ann Rheum Dis, 2006. 65(4): p. 515-9.*

[23] GUILBERT S. Evolution de l'arthrose femoro-patellaire isolee: devenir a 9 ans de recul moyen de 80 genoux non-operes. *Symposium SOFCOT 2003, 2003.*

[24] VINCE AS, AK SINGHANIA, MM GLASGOW. What knee X-rays do we need? A survey of orthopedic surgeons in the United Kingdom. *Knee, 2000. 7(2): p. 101-4.*

[25] McDONNELL SM *et al.* Skyline patellofemoral radiographs can only exclude late stage degenerative changes. *Knee, 2011. 18(1): p. 21-3.*

[26] BOEGARD T *et al.* Correlation between radiographically diagnosed osteophytes and magnetic resonance detected cartilage defects in the tibiofemoral joint. *Ann Rheum Dis, 1998. 57(7): p. 401-7.*

[27] KORNAAT PR *et al.* Osteoarthritis of the knee: association between clinical features and MR imaging findings. *Radiology, 2006. 239(3): p. 811-7.*

[28] TAURO B *et al.* The Lubinus patellofemoral arthroplasty. A five- to ten-year prospective study. *J Bone Joint Surg Br, 2001. 83(5): p. 696-701.*

[29] ELAHI S *et al.* The association between varus-valgus alignment and patellofemoral osteoarthritis. *Arthritis Rheum, 2000. 43(8): p. 1874-80.*

[30] McKEEVER DC. Patellar prosthesis. *J Bone Joint Surg Am, 1955. 37-A(5): p. 1074-84.*

[31] LUBINUS HH. Patella Glide Bearing Total Replacement. *Orthopedics, 1979. 2(2).*

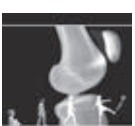
[32] SMITH AM. Treatment of patello-femoral arthritis using the Lubinus patello-femoral arthroplasty: A retrospective review. *Knee, 2002(9): p. 27-30.*

[33] LONNER JH. Patellofemoral arthroplasty: pros, cons, and design considerations. *Clin Orthop Relat Res, 2004(428): p. 158-65.*

[34] BLAZINA ME *et al.* Patellofemoral replacement. *Clin Orthop Relat Res, 1979(144): p. 98-102.*

[35] LONNER JH. Patellofemoral arthroplasty. *J Am Acad Orthop Surg, 2007. 15(8): p. 495-506.*

[36] HOOGERVORST P, DE JONG RJ, VAN KAMPEN A. Long-term results of the Richards II patellofemoral arthroplasty - Survivorship, functional results and patient satisfaction. *Submitted, 2012.*



COMPARATIVE RESULTS OF TKA AND PFA FOR ISOLATED PATELLOFEMORAL OSTEOARTHRITIS

D.L. DAHM

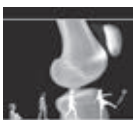
INTRODUCTION

There has been a renewed interest in treatment options for patients with isolated advanced patellofemoral arthritis. Reported surgical options for early stage disease and/or patellar malalignment include arthroscopic debridement with or without lateral retinacular release, tibial tubercle elevation or anterior medialization, cartilage stimulation techniques (such as microfracture or abrasion arthroplasty), and cartilage replacement procedures [1-3]. For advanced patellofemoral arthritis, patellectomy and patellar resurfacing have been described but have fallen out of favor due to generally unsatisfactory results [2-5].

Total knee arthroplasty (TKA) is an accepted method of treatment for advanced isolated patellofemoral arthritis [6-9]. Although TKA has been reported to provide pain relief and functional improvement in this population, its indications and benefits relative to patello-femoral arthroplasty (PFA) remain controversial [10]. A paucity of literature exists with regards to comparison of results of TKA versus PFA using modern implants. In this comparative retrospective study, we hypothesized that patients who underwent PFA for treatment of isolated advanced patellofemoral arthritis would have similar outcomes to patients treated with TKA for the same pathology.

METHODS

All patients who underwent knee arthroplasty at our institution between January 2003 and December 2005 were reviewed. From this Joint Arthroplasty Registry, we selected only those patients diagnosed with patellofemoral arthritis. Patients were further screened for isolated disease using the following criteria: a Kellgren and Lawrence score less than or equal to 2 at the tibial femoral joint and an Iwano score greater than 2 at the patellofemoral joint, determined radiographically by consensus of two of the authors [11, 12]. Patellofemoral arthroplasties were performed using the Avon patellofemoral prosthesis (Stryker Howmedica Osteonics, Mahwah NJ). A single surgeon performed all but two of the patellofemoral arthroplasties included in this study and none of the total knee arthroplasties. The total knee arthroplasties were performed by surgeons who did not have experience with patello-femoral arthroplasty. A total of 8 surgeons used one of two modern total knee arthroplasty designs (Zimmer, Warsaw IN of Sigma Orthopedics Inc., Johnson & Johnson, Warsaw IN). Thirteen designs were posterior cruciate ligament (PCL substituting) and 9 were PCL retaining implants. All procedures were performed using a standard medial parapatellar arthrotomy.



Patient demographics were collected for both PFA and TKA groups. Preoperative and postoperative *Knee Society Clinical Rating System* (KSS) scores were calculated from standardized questionnaires given prospectively to all patients as part of surgery and at regular intervals thereafter. Preoperative Tegner activity scores and *University of California, Los Angeles* (UCLA) activity scores were obtained retrospectively for patient records. Postoperative Tegner and UCLA scores that were not obtainable through chart reviews were obtained by telephone interview. Preoperative and postoperative radiographs were reviewed. Trochlear dysplasia was assessed using the Dejour classification method [13]. Patellar position was determined using the Insall Salvati index [14].

STATISTICS

Compared comparisons between the PSA and TKA groups were performed using Wilcoxon signed-rank tests. Multivariate regression was used to analyze the outcome effects of any independent variable showing its significant difference between the groups. All regression models were analyzed for power and fit with significance set at .05. The statistical analysis was performed using JMP 6 statistical discovery software (SAS, Inc., Cary, NC).

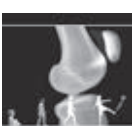
RESULTS

Between January 2003 and December 2005, 3500 patients underwent a knee arthroplasty procedure at our institution. We identified 205 knees (5.8%) coded in our database for patellofemoral arthritis. After detailed radiographic review, 45 knees (1.3%) with isolated patellofemoral arthritis were identified. There were 23 knees (0.7%) that underwent PFA and 22 knees (0.6%) that underwent TKA.

Mean follow-up was 29 months (range, 24 to 49 months) in the PFA group and 27 months (range, 24 to 33 months) in the TKA group. There were no statistically significant differences between the groups in regards to gender, race, body mass index, average number of prior knee surgeries, smoking status, or employment status. Mean age at the time of surgery was 60 years (range, 39-81 years) in the PFA group and 69 years (range, 44-83 years) in the TKA group ($p=0.01$). There were no statistically significant differences between the PFA and TKA groups in relation to mean preoperative Kellgren and Lawrence score, mean preoperative Iwano score or the presence/absence of trochlear dysplasia. Mean preoperative KSS scores, KSS function scores, Tegner scores, and UCLA scores were not statistically different between groups. Likewise, mean preoperative range of motion was similar between groups. Mean postoperative Knee Society clinical rating systems scores were 89 and 90 in the PFA and TKA cohorts respectively. Mean UCLA scores were 6.6 in the PFA group and 4.2 in the TKA group ($p<0.0001$). Mean blood loss ($p=0.03$) and hospital stay ($p=0.001$) were significantly lower among PFA patients. Linear regression analysis showed that blood loss, hospital stay, and functional outcomes were not affected by age as an independent variable. No significant complications occurred in the PFA group. There was one deep vein thrombosis in the TKA group; additionally, one patient in the TKA group required a manipulation under anesthesia. At last follow-up, no patient in either group had required revision knee arthroplasty.

DISCUSSION

The surgical treatment of advanced symptomatic patellofemoral arthritis remains somewhat controversial. Satisfactory results have been reported for both PFA and TKA in this setting [6-10, 15]. In the present study we retrospectively compared the clinical and functional outcomes of patients who underwent either PFA or TKA for treatment of isolated patellofemoral arthritis. Demographically the two cohorts were remarkably similar. Radiographs were reviewed carefully to ensure that only patients treated for isolated patellofemoral arthritis were included. Although the TKA



patients were significant older than the PFA patients (mean, 69 vs 60 yeas, respectively), a well powered regression analysis demonstrated that age was an independent variable had no effect on postoperative KSS score, KSS function score, Tegner score, UCLA score, or satisfaction at final follow-up. It should be emphasized that strict radiographic criteria were utilized in our study in order to include only patients with isolated patellofemoral arthritis. Although it is possible that with longer follow-up we will see deterioration of results in the group treated with patellofemoral arthritis, the early benefits of improved function, return to higher activity, and less morbidity seemed to outweigh the risk of revision for tibiofemoral arthritis progression.

CONCLUSIONS

In our study patients who underwent modern PFA for treatment of isolated patellofemoral arthritis were compared with a cohort who underwent total knee arthroplasty for the same diagnosis during the same time period. Patients treated with PFA demonstrated similar results with respect to pain relief, but showed improved function and return to activity when compared with the patients treated with TKA. PFA patients also experienced less blood loss, fewer complications, and shorter hospital stay following surgery. Our results indicate that PFA is a less invasive treatment option for patients with isolated patellofemoral arthritis, yielding early outcomes that compare favorably to TKA.

LITERATURE

[1] FEDERICO DJ, REIDER B. Results of isolated patellar debridement for patellofemoral pain in patients with normal patellar alignment. *Am J Sports Med 1997; 25(5): 663-9.*

[2] FULKERSON JP. Alternatives to patellofemoral arthroplasty. *Clin Orthop Relat Res 2005;(436): 76-80.*

[3] MAQUET P. Advancement of the tibial tubercle. *Clin Orthop Relat Res 1976;(115): 225-30.*

[4] ACKROYD CE, POLYZOIDES AJ. Patellectomy for osteoarthritis. A study of eighty-one patients followed from 2 to 22 years. *J Bone Joint Surg 1978; 60B(3): 353-7.*

[5] ARCIERO RA, TOOMEY HE. Patellofemoral arthroplasty: A 3-9 year follow-up study. *Clin Orthop Relat Res 1988; (236): 60-71.*

[6] LASKIN RS, VAN STEIJN M. Total knee replacement for patients with patellofemoral arthritis. *Clin Orthop Relat Res 1999; 3670: 89-95.*

[7] MEDING JB, WING JT, KEATING EM, RITTER MA. Total knee arthroplasty for isolated patellofemoral arthritis in younger patients. *Clin Orthop Relat Res 2007; (46): 78-82.*

[8] MONT MA, HAAS S, MULLICK T, HUNGERFORD DS. Total knee arthroplasty for patellofemoral arthritis. *J Bone Joint Surg 2002; 84A(11): 1977-81.*

[9] PARVIZI J, STUART MJ, PAGNANO MW, HANSSEN AD. Total knee arthroplasty in patients with isolated patello-femoral arthritis. *Clin Orthop Relat Res 2001;(392): 147-52.*

[10] LONNER JH. Patellofemoral arthroplasty: pros, cons, and design considerations. *Clin Orthop Relat Res 2004;(428): 158-65.*

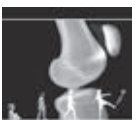
[11] KELLGREN JH, LAWRENCE JS. Radiological assessment of osteo-arthrosis. *Ann Rheum Dis 1957;16(4): 494-502.*

[12] IWANO T, KUROSAWA H, TOKUYAMA H, HOSHIKAWA Y. Roentgenographic and clinical findings of patellofemoral osteoarthrosis. With special reference to its relationship to femorotibial osteoarthrosis and etiologic factors. *Clin Orthop Relat Res 1990;(252): 190-7.*

[13] TECKLENBURG K, DEJOUR D, HOSER C, FINK C. Bony and cartilaginous anatomy of the patellofemoral joint. *Knee Surg Sports Traumatol Arthrosc 2006; 14(3): 235-40.*

[14] INSALL J, SALVATI E. Patella position in the normal knee joint. *Radiology 1971;101(1): 101-4.*

[15] MERCHANT AC. Early results with a total patelloefmoral joint replacement arthroplasty prosthesis. *J Arthroplasty 2004; 19(7): 829-36.*



RÉSULTATS ET CAUSES D'ÉCHEC DES PROTHÈSES FÉMOROPATELLAIRES
The patellofemoral arthroplasties: results and etiologies of failures

J. CHOUTEAU

INTRODUCTION

Les premières prothèses patellaires ont été implantées en 1955 par Mc Keever [27] qui avait dessiné un bouclier en vitalium vissé sur la patella. Par la suite, plusieurs nouveaux dessins ont été proposés pour réaliser une arthroplastie fémoropatellaire [8, 26, 42, 45].

L'arthroplastie fémoropatellaire reste un sujet de controverse du fait de résultats très variables dans la littérature et d'un taux d'échec précoce parfois élevé.

Nous rapportons les résultats et les causes d'échec des prothèses fémoropatellaires (PFP) en envisageant deux types d'implants : les prothèses fémoropatellaires de première génération dites de resurfaçage (sans coupe, positionnement supposé anatomique dans la trochlée) et les prothèses fémoropatellaires anatomiques à coupes (coupe de la trochlée et dessin similaire à celle d'une PTG).

RÉSULTATS

Evaluation clinique

Paxton rapporte que les deux scores cliniques les plus adaptés à l'évaluation postopératoire des PFP seraient le Short-Form 36 (SF 36) et le Knee Injury and Osteoarthritis Outcome Score (KOOS) [35].

Influence de l'étiologie de l'arthrose

Quels que soient les scores utilisés, toutes les séries ont pour point commun de meilleurs résultats cliniques dans le groupe des arthroses fémoropatellaires sur dysplasie.

Gadeyne [16] rapporte 68,2 % de bons et très bons résultats dans le groupe dysplasie contre 44,4 % dans le groupe arthrose primitive (tableau 1). En comparant les résultats avant l'intervention et à la révision, 74 % des patients avaient été améliorés pour la marche, 65 % pour les escaliers et 77 % sur le plan de la douleur. Hassaballa [17] a montré que se mettre à genou est toujours très difficile après PFP mais amélioré par la prothèse (28 % d'amélioration).

Argenson [5], avec une prothèse similaire (Prothèse Autocentric DePuy®), retrouvait des résultats comparables avec 73 % de bons résultats dans le groupe dysplasie contre 54 % dans le groupe des arthroses primitives. Leadbetter [23] sur une série de 30 genoux à 2 ans de recul retrouvait 83 % de bons à très bons résultats avec des scores plus élevés dans le groupe arthrose sur instabilité sur dysplasie

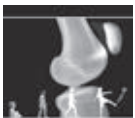


Tableau 1 : Résultat clinique global des PFP

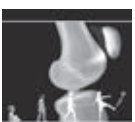
		Type de Prothèse	n	Recul	Bons ou très bons
Arciero	1988	Richards - CFS-Wrigh	31 ou 36	5	72 %
Ackroyd C	2007	AVON	109	5	80 %
Blazina	1979	Richards	57	2 ans	81 %
Bauchu/Bousquet	1991	Serf	117		56 %
Board	2004	Lubinus	17	1 an 1/2	53 %
Butler	2009	Performa	22	5	
Cartier	1990	Richards II/III	70	4	91 %
Cossey	2006	Avon (Naviguée)	4	1	100 %
De Cloedt	1999	Autocentrique	45	6	63 %
De Winter	2001	Richards II	26	11	62
Goutallier	1999	Guepar	28	5.5	79 %
Grammont	1989	Autocentrique	45	0.5 – 8	88 %
Kooijman	2003	Richards	56	17	86 %
Krajka	1996	Blazina	16	5.8	88 %
Leadbetter	2009	Avon	79	3	84 %
Merchant	2004	LCS PFJ	15	3.75	93 %
Sisto	2006	Custom Kinamed	25	6	100 %
Smith	2002	Lubinus	45	3 - 9	64 %
Starks	2009	AVON	37	2	100 %
Tauro	2001	Lubinus	76	7.5	45 %
Van Wagenberg	2009	Autocentric	24	4.8	30 %
Werkman	1991	Bechtol	84		80 %
Witvoet	1994	Guepar	78	5	75 %

fémoropatellaire. Lotke [25] rapporte 60 à 80 % de bons résultats postopératoires, résultats grevés par l'instabilité patellaire et le conflit externe en flexion.

Lors du symposium de la SOFCOT de 2003, il a été montré que les PFP donnaient un meilleur résultat dans les cas suivant : absence de surcharge pondérale, âge < 65 ans, activité régulière persistante, bonne mobilité pré opératoire, pas de valgus/varus excessif, grade IV d'Iwanno et en cas d'arthrose sur instabilité plutôt qu'arthrose essentielle (tableau 2).

Tableau 2 : Résultats globaux postopératoires SOFCOT 2003

	Perdus de vue	n	Recul	Score F-P	IKS genou	IKS fonction	Arthrose F-T
PFP	37	174	8 (1-22)	73	83	68	21 : 10 %
PTG	0	47	5 (2-12)	78	88	75	



Résultats globaux des PFP et taux de survie

Prothèse de première génération

Prothèse Autocentric® (Depuy)

En prenant comme critère d'échec le changement de l'implant et quelle que soit la cause de cette révision, la courbe de survie à cinq ans était de 82 % pour Gadeyne [16] et à dix ans de 62 %. La moyenne de survie de la prothèse était de 11 ans. En excluant les reprises par PTG pour dégradation des compartiments fémorotibiaux, la courbe de survie à cinq ans était de 83,5 % et de 79,1 % à dix ans et la moyenne de survie de la prothèse était de 12,7 ans. Argenson [5] rapporte les résultats de 66 PFP Autocentric® (fig. 1) implantées entre 1972 à 1990. L'âge moyen à l'implantation était de 57 ans. A un recul de 16.2 ans, 14 genoux avaient été repris pour progression de l'arthrose fémorotibiale avec changement par PTG à 7.3 ans en moyenne plutôt dans les cas d'arthrose fémoropatellaire primitive que sur les dysplasies. 11 implants avaient été repris pour instabilité et descellement à 4.5 ans postopératoire en moyenne. Enfin, étaient rapportés une fracture rotule, 3 infections, 6 raideurs postopératoires traités par 2 mobilisations sous AG et 6 PTG, 5 sections aileron et patellectomie externe pour tilt et conflit externe. Le taux de survie de l'implant était de 58 % à 16 ans. Avec le même type d'implant (prothèse Autocentric® Depuy), Van Wagenberg [41] sur 24 genoux à 4.8 ans de recul rapporte des résultats très péjoratifs avec 87.5 % de prothèses reprises dont 29 % par PTG.

Prothèse de type Lubinus®

Board [9] sur 17 cas d'implants de type Lubinus à 19 mois postopératoire retrouve 53 % de bons à très bons résultats, 35 % reprise globale et 24 % reprise par PTG. Smith [37] sur 45 PFP de type Lubinus rapporte 64 % de bons et excellents résultats et 17 % de non satisfaits. Ackroyd [1] sur 76 PFP Lubinus rapporte un taux de survie si révision prothétique de 65 %. Si en plus une douleur sévère ou moyenne à 8 ans était comptée comme un échec chirurgical le taux de survie chutait alors à 48 %.

Prothèse Richards I®

Cartier [11], sur 70 PFP Richards I opérées entre 1975/1991 (âge moyen à l'implantation de 60 ans), retrouve 75 % des implants toujours en place à 10 ans et 91 % bons à très bons résultats (principale indication prothétique dysplasie 70 %, 13 reprises au total, dont 8 PTG). Kooijman [19], sur une série de 56 PFP Richards I à 15 à 21 ans de recul, retrouve 86 % de résultats satisfaisants à 17 ans. La cause majeure des reprises était la progression de l'arthrose fémorotibiale (23 % des patients).

Fig. 1 : Types prothétiques : Prothèse Autocentric® (Depuy) à gauche, Prothèse Avon® (Strycker Howmedica Osteonics, New Jersey) à droite.

Prothèse de deuxième génération

Prothèse Richards II® (Smith Nephew Richards, Memphis, TN)

De Winter [13] retrouve 62 % de bons et très bons résultats avec une prothèse de type Richards II.

Prothèse Avon® (Strycker Howmedica Osteonics, New Jersey) (fig. 1)

Les résultats de cette prothèse sont les plus favorables de la littérature. Ackroyd [2] a présenté les résultats d'une série prospective de 109 PFP de type Avon à 5 ans. Le taux de survie était de 95,8 % sans aucun descellement prothétique. Leadbetter [21] avec un implant identique sur une étude multicentrique de 79 genoux à 3 ans de recul retrouve 84 % de bons et excellents résultats et 90 % des patients sans douleurs dans la vie quotidienne. Odumenya [34] sur 50 Avon à 5.3 ans rapporte un taux de survie de 100 %. Enfin, Starks [38] sur une série de 37 Avon à 2 ans trouve lui aussi 100 % très bons et bon résultats et aucune reprise, mais 22 % de progression de l'arthrose fémorotibiale interne sans traduction clinique.

Prothèse LCS PFA® (Depuy)

Merchant [28] avec la prothèse LCS PFA retrouve 93 % d'excellents et bons résultats à 3,75 ans.

Reprise des prothèses fémoropatellaires

Dans la littérature, les prothèses fémoropatellaires donnent de 45 à 100 % de bons à très bons résultats. Les reprises, notamment pour les implants de premières générations, ont été fréquemment réalisées [4].

Le détail des séries est rapporté dans le tableau 3. Sur 17 cas d'implants de type Lubinus à 19 mois postopératoire Board [9] retrouve 35 % de reprise globale (24 % par PTG).

Tableau 3 : Pourcentage de révision des prothèses fémoropatellaires

	Prothèse	n	Recul	Révisions	PTG
Arciero (1988)	Richards	31	5	9 %	9 %
Blazina (1979)	Richards I/II	55	2 ans	50 %	9 %
Board (2004)	lubinus	17	1.5	35 %	24 %
Bousquet (Bauchu) (1991)	Serf	117		17 % (41 % pb course rot.)	
Kooijman (2003)	Richards	56	17	15,5 %	18 %
Cartier (1990/2005)	Richards	72	4	7 %	5 %
De Cloedt (1999)	Autocentrique	45	6	8 : 19 %	12 %
Krajka (1996)		16	2 - 18	6 %	
Goutallier (1999)	Guepar	28	5.5	14 %	3 %
Grammont (1989)	Autocentrique	45	0.5 – 8	11 %	2 %
Minas 2008	LCS/ Avon/ Sigma custom	41	5	22 %	22 %
Nicol (2006)	Avon	103	7.1	14 %	
Odumenya (2010)	Avon	50	5.1	0 %	0%
Smith (2002)	Lubinus	45	3 - 9	19 %	11 %
Sisto (2006)	Custom Kinamed	25	6	0 %	0 %
Starks (2009)	AVON	37	2	0 %	0 %
Tauro (2001)	Lubinus	76	7.5	28 %	6.5 %
Van jonbergen 2009	Richards I/II	196	30	11,7 %	100 % des révisions
Van Wagenberg 2009	Autocentric	24	4.8	87.5 %	29%
Witvoet (1994)	Guepar	78	5	34 % (11 TTA)	15 %

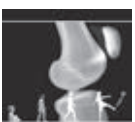


Tableau 4 : Pourcentage de révision des prothèses fémoropatellaires (SOFCOT 2003)

	PTG	Changement ou ablation	Transposition	Autres	Total
%	21 %	13 %	2 %	2 %	38 %
(n)	(44)	(27)	(5)	(5)	(81)

La série du symposium de la SOFCOT 2003 rapporte 38 % de reprise chirurgicale dont 21 % de changement par PTG, 13 % de changement ou ablation et 2 % d'ablation (tableau 4).

CAUSES D'ÉCHEC

La prothèse fémoropatellaire doit respecter un cahier des charges contraignant. Elle doit pouvoir s'adapter aux variations anatomiques sans générer de conflits. Le caractère asymétrique des joues de la trochlée peut entraîner des conflits entre le débord de l'implant et la patella (Gadeyne [16], Ackroyd [1, 2], Amis [3]).

Jess et Lonner [24] évoquent l'importance du choix de l'implant qui serait à l'origine de la plupart des complications ; Ils rapportent un taux de 17 % avec la prothèse Lubinus® dite de première génération contre 4 % avec la prothèse Avon (Strycker Howmedica Osteonics, New Jersey) dite de deuxième génération.

Selon Tauro [39], l'inconvénient principal des PFP de type Lubinus® première génération serait une mauvaise course rotulienne ("maltracking") source de douleur et d'usure prématurée.

Les prothèses à coupe ont plusieurs avantages. Elles optimisent la fiabilité et la reproductibilité de la mise en place, elles autorisent la rotation externe et la translation externe du composant fémoral réduisant ainsi les contraintes sur la berge externe.

Une malposition prothétique peut se traduire par un débord antérieur du carter (fig. 2), une bascule rotulienne sur malposition du bouton avec pour conséquence subluxation et hyperpression externe source de douleur et de descellement plus ou moins précoces [2, 16].

Pour Gadeyne [16], l'axe horizontal du carter était plus élevé chez les patients réopérés ($p = 0{,}015$), ce qui correspondait à un carter trochléen posé en rotation médiale excessive (fig. 2). La berge latérale était alors en superstructure à l'origine de conflits symptomatiques.

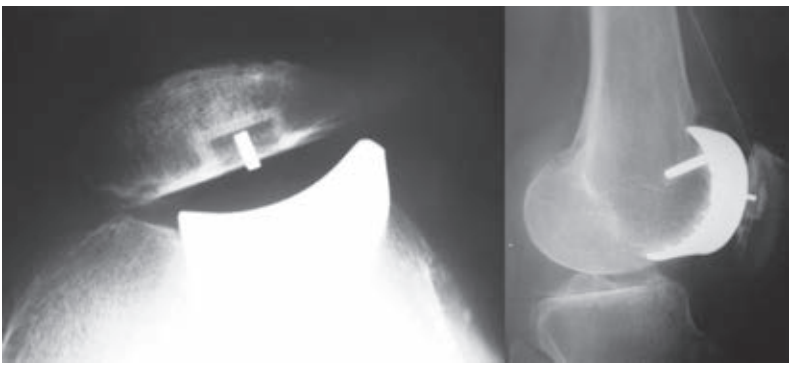


Fig. 2 : Malposition prothétique avec composant fémoral implanté en rotation interne à gauche et surépaisseur du carter à droite.

Le débord antérieur du carter est également plus élevé dans le groupe des reprises (p = 0,004). Lorsque le carter est implanté en creusant insuffisamment la trochlée, il est posé en superstructure et l'espace antérieur du genou subit alors des contraintes excessives à l'origine des symptômes.

Lonner [24] rapporte que l'instabilité fémoropatellaire postopératoire serait le résultat d'un mauvais équilibrage des tissus mous (conséquence d'un mauvais positionnement des implants avec mauvais alignement de la rotule et douleur antérieure).

Influence du type prothétique

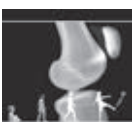
L'utilisation d'un métal back a été délétère au fonctionnement des prothèses de Bousquet [7], puisqu'au recul de huit ans, 48 % des patients avaient été réopérés pour changement prothétique. Arumilli [6] rapporte les résultats de la prothèse LCS PFA avec rotule métal back cimentée. Il retrouve des échecs précoces sur cette rotule mobile avec usure et contact métal/métal, luxation du PE et fracture du PE l'ayant conduit à abandonner ce type d'implant. Merchant [28, 29] rapporte des résultats plus encourageant avec la même prothèse. L'étude porte sur 15 cas au recul de 3,5 ans avec 93 % de bons résultats.

Lonner [24] rapporte une analyse des différents dessins d'implants pour expliquer les échecs. La trochlée profonde et très contrainte des PFP Richards I & II (Smith Nephew Richards, Memphis, TN) les prédispose à une mauvaise course patellaire et à des conflits entre implant patellaire et berges de la trochlée précipitant les causes de reprise chirurgicale. Cela a été confirmé par la publication de de Winter [13] qui, à une moyenne de 11 an de recul, trouvait que 7 implants Richards II sur une série de 26 avaient nécessité une reprise pour instabilité et mauvais positionnement 2 patients traités par transposition de la TTA, 3 par patellectomie et 2 par prothèse totale. Kooijman [19] ont rapporté, quant à eux, 86 % de survie à long terme sur 45 PFP. Cependant, une chirurgie secondaire au niveau des tissues mous était nécessaire précocement chez 18 % des patients, et le changement prothétique réalisé chez 7 patients pour accrochage, instabilité et douleur.

La PFP de type Lubinus a aussi été associée à un haut taux de reprise pour dysfonction fémoro patellaire. Lonner [24] rapporte 17 % de complication (contre 4 % avec une prothèse de type Avon). Board [9] a retrouvé 18 % subluxation patella, 18 % raideur, 1 infection, 12 % progression arthrose et a arrêté la pose de cet implant. Tauro [39] a montré un taux de 55 % de résultats non satisfaisant avec nécessité de révision pour 21 genoux (28 %) à une moyenne de 7,5 ans. 32 % présentaient une instabilité prothétique même si elle n'était pas toujours symptomatique. 15 genoux ont été repris avec bon résultats : 10 par PTG, 10 par une autre PFP à dessin différent. Smith [37] a enfin revu 45 implants Lubinus avec des résultats décevants. Dans cette série, 5 genoux ont été repris par PTG. Dans la série de Lonner [24], les douleurs postopératoires sont passées de 17 % avec la prothèse Lubinus à 4 % avec la prothèse Avon. Hendrix [18] avec une série de reprise de PFP de type Lubinus par PFP de type Avon retrouve, comme cause d'échec, une malposition initiale de la trochlée et du bouton avec subluxation et usure du bouton rotulien. Ackroyd [1] sur 76 PFP de type Lubinus à 7.5 ans de recul retrouve seulement 45 % bons résultats. La cause majeure d'échec était un mauvais engagement du PE dans 32 % des cas avec 28 % de révision (PFP ou PTG, 1/3 des révisions sur arthrose fémorotibiale interne).

Complications précoces

Les arthroplasties fémoropatellaires sont émaillées d'un taux de complications précoces et secondaires plus important que celui des arthroplasties totales de genou sur arthrose fémoropatellaire isolée [16]. Les complications postopératoires précoces les plus fréquemment rapportées sont : la luxation de rotule, la fracture de rotule, l'infection, l'algodystrophie, la phlébite, l'accrochage, la subluxation et les douleurs de type rotule (douleur antérieure par augmentation de l'espace antérieur : surépais-



seur de l'implant ("overstuff") par insuffisance de resurfaçage ou de coupe [4]). L'épaisseur initiale de la patella ainsi que l'épaisseur restante étaient plus importantes dans le groupe des mauvais résultats [31].

Les instabilités fémoropatellaires postopératoires précoces sont moins fréquentes sur implants récents [15]. En cas de patella alta, une distalisation de la TTA permettrait un meilleur engagement et diminuerait le risque d'instabilité, tout comme la correction d'une TAGT excessive [15]. La rotule basse avec index de Caton-Deschamps à moins de 0.8 serait une contre indication à la PFP [15].

Leadbetter [23] sur une étude de 30 genoux à 2 ans rapporte un notching fémoral antérieur, 4 raideurs avec mobilisation sous anesthésie et deux ruptures du tendon quadricipital. Ackroyd [1] sur une série de 306 PFP Avon à 2 ans de recul retrouve 4 % de douleurs antérieures, 5 % complications précoces : dont 1.6 % mob sous AG.

Lors du symposium de la SOFCOT 2003 [14], les complications précoces locales représentaient 16 % des cas ayant imposé 4 % de reprises (tableau 5). Le taux de complications précoces est élevé et représente un argument de poids contre cette intervention.

Complications tardives

Globalisation de l'arthrose fémorotibiale

Le pourcentage de révision prothétique suite à une progression de l'arthrose fémorotibiale varie de 0 % à 70 mois pour Krajka [20] à 22 % à 204 mois pour Kooijman [19].

Il s'agit du principal critère d'échec dans la littérature comme rapporté par Ackroyd [2] (28 % dont 4 % de conversion en PTG).

La dégradation de l'articulation fémorotibiale est surtout le fait des arthroses primitives. Leadbetter [22] sur une étude rétrospective de 12 publications préalables répertorie un taux global d'échec de 24 % sujet à la progression de l'arthrose majoritairement puis à une anomalie au niveau du système extenseur. Une importante déviation axiale du membre devrait faire contre-indiquer la PFP en raison du risque important d'évolution arthrosique ultérieure. Witvoet [43, 44] contre indique la PFP dans les désaxations frontales en varus supérieures à 5° et en valgus supérieures à 8°. Nicol [33] sur une étude prospective de 103 PFP Avon à 7,1 ans de recul rapporte 14 genoux repris (14 %), 12 % à cause de la progression de l'arthrose fémorotibiale. Le délai moyen de révision était de 55 mois (14 à 95). 17 % des PFP sur genou sans dysplasie montraient des signes de progression de l'arthrose fémorotibiale, aucun si dysplasie. Il retrouvait moins de révision chez les patients opérés sur arthrose sur dysplasie fémoropatellaire ($p<0.01$).

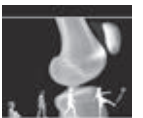
Van jonbergen [40], sur une série de 196 PFP Richards I/II à 30 mois de recul, rapporte 11,7 % de révision par PTG dans 100 % cas.

Leadbetter [21] retrouve 13 échecs cliniques, 1 fracture rotule, une arthrofibrose repris par PTG dans 5 cas.

Ackroyd [1] sur une série de 306 PFP Avon à 2 ans de recul retrouve 3.6 % de révision pour arthrose fémorotibiale, principale complication tardive. Il ne rapporte pas de complication tardive due à l'implant avec une stabilité du résultat fonctionnel et douloureux à 5 ans.

Tableau 5 : Complications précoces des prothèses fémoropatellaires (Sofcot 2003)

	Algodystrophie	Raideur	Infection	Hématome	Nécrose cutanée	Total
Prothèses fémoropatellaires	13 (6 %)	9 (4 %)	7 (3 %)	5 (2 %)	3 1 %	34 (16 %)



Cartier [11] avec une prothèse de type Richards rapporte comme principale cause d'échec la détérioration fémorotibiale si l'HKA excédait 180 ± 3°.

Minas [30] rapporte les résultats d'une série de 41 implants variés (9 LCS PFA, 8 AVON, 27 Sigma customized) avec 22 % d'échec par reprise par PTG expliquée par une progression arthrose. Les douleurs antérieures étant plus spécifiquement retrouvées sur les LCS PFA.

Smith [37] rapporte 19 % de reprise par PTG ou nouvelle PFP (progression arthrose FT (3/5), instabilité (1/5), douleurs antérieurs (1/5)).

Descellement

Gadeyne [16] rapporte un taux de 24 % d'échec (11 prothèses) repris par PTG à 74 mois (2 pour descellement aseptique sur malposition des pièces (débord antérieur du carter, malposition du bouton). 2 pour mauvais engagement sur bouton posé en bascule interne (fig. 2), 4 cas de décompensation arthrose fémoro-tibiale globale).

Kooijman [19] rapporte un taux de descellement de 2 %.

Autres complications de PFP

Les complications tardives retrouvées lors du symposium de la SOFCOT 2003 sont rapportées dans le tableau 6. Dans la littérature elles conduisent fréquemment à une reprise chirurgicale [4, 8, 11, 39] notamment en cas d'accrochage en flexion, d'instabilité chronique fémoropatellaire postopératoire, de défaut d'engagement (jusqu'à plus de 40 dans la série de Bauchu [7]) d'infection ou d'arthrose fémorotibiale. Enfin, Leadbetter [23] rapporte un taux d'épanchement chronique de 33 %. Il insiste sur le fait de ne pas mettre le composant en rotation interne et en recurvatum.

VOIES D'AVENIR

Les résultats montrent une sensibilité des PFP à un défaut de pose et surtout des dessins parfois inappropriés. L'optimisation des dessins d'implants et de l'instrumentation de pose devra être obtenue grâce à des études biomécaniques fines [32].

La chirurgie assistée par ordinateur

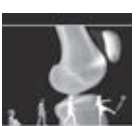
La chirurgie assistée par ordinateur a montré sa fiabilité et sa reproductibilité pour atteindre les objectifs de pose pour l'implantation des prothèses totales de genou. Elle pourrait aussi le permettre pour les PFP. Cossey [12] rapporte les résultats à 1 an de 4 prothèses de type Avon en prenant comme repères per opératoires : les épicondyles fémoraux, le centre du genou, la ligne de Whiteside, les centres de la hanche du genou et de la cheville. Il conseille d'implanter le composant trochléen en léger flexum pour éviter toute surépaisseur. Il constate 100 % de bons et excellents résultats, ne déplore aucun échec précoce et aucune instabilité postopératoire.

Les implants et guides sur mesure

Les techniques d'implants sur mesure 3D obtenus par scanner préopératoire fémoropatellaire

Tableau 6 : Complications tardives des prothèses fémoropatellaires (SOFCOT 2003)

	Instabilité	Accrochage	Fracture rotule	Rupture implant	Arthrose fémorotibiale	Descellement	Usure	Total
PFP	11 (5 %)	12 (6 %)	10 (5 %)	6 (3 %)	21 (10 %)	32 (15 %)	35 (17 %)	99 (47 %)
PTG	0	0	1 (2 %)	0	0	0	0	1 (2 %)



(dit : Custom fit CT 3D) ont pour but d'optimiser la couverture des surfaces osseuses sans entraîner d'hyper structure. Sisto [36] rapporte les résultats encourageant de 25 implants sur mesure à partir de scanner préopératoire (Implants Custom Kinamed®). A un recul moyen de 73 mois, il retrouve 100 % de bons et excellent résultats et aucun échec pour reprise. Butler rapporte 22 cas de PFP à 5 ans (prothèse performa custom fit Biomet®, custom fit sur CT scan 3D) [10]. Il déplore une révision à 18 mois, 2 raideurs postopératoires traitées arthroscopiquement. Radiologiquement, aucun descellement des implants de la trochlée mais un bouton rotulien rompu.

CONCLUSION

Les résultats et les causes d'échec des prothèses fémoropatellaires apparaissent très contrastés selon le type d'implant utilisé et les séries.

Les meilleurs résultats ont été obtenus pour les prothèses fémoropatellaires anatomiques récentes dites à coupe.

Une sélection rigoureuse des patients augmente le taux de bons et très bons résultats (absence de surcharge pondérale, âge < 65 ans, activité régulière persistante, bonne mobilité préopératoire, pas de valgus/varus excessif, grade IV Iwanno, arthrose sur instabilité fémoropatellaire plutôt qu'arthrose essentielle fémoropatellaire).

L'amélioration et l'optimisation du dessin des implants anatomiques à coupe se fera grâce à des études biomécaniques poussées au niveau de la trochlée et du bouton rotulien [3]. L'utilisation de la navigation ou de guides d'implantation dédiés au patient pourrait être le gage d'une meilleure fiabilité et d'une meilleure reproductibilité des techniques de pose [15] pour permettre une amélioration des résultats postopératoires [3].

RÉSUMÉ

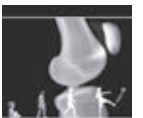
L'arthroplastie fémoropatellaire reste un sujet de controverse du fait de résultats très variables dans la littérature et d'un taux d'échec précoce parfois élevé.

Nous rapportons les résultats et les causes d'échec des prothèses fémoropatellaires (PFP) en envisageant deux types d'implants : les prothèses fémoro patellaires de première génération dites de resurfaçage (sans coupe, positionnement supposé anatomique dans la trochlée) et les prothèses fémoropatellaires anatomiques à coupes (coupe de la trochlée et dessin similaire à celui d'une PTG).

Les résultats et les causes d'échec des prothèses fémoropatellaires apparaissent très contrastés selon le type d'implant utilisé et les séries. Les prothèses de première génération ont des taux d'échec avec reprise allant jusqu'à 87 %. Les meilleurs résultats ont été obtenus pour les prothèses fémoropatellaires anatomiques récentes dites à coupe avec plusieurs séries rapportant un haut niveau de satisfaction des patients, des scores fonctionnels postopératoires élevés et l'absence de complications postopératoires attribuables à l'implant.

Une sélection rigoureuse des patients augmente le taux de bon et très bon résultats (absence de surcharge pondérale, âge < 65 ans, activité régulière persistante, bonne mobilité préopératoire, pas de valgus/varus excessif, grade IV Iwanno, arthrose sur instabilité plutôt qu'arthrose essentielle).

L'amélioration et l'optimisation du dessin des implants anatomiques à coupe se feront grâce à des études biomécaniques poussées au niveau de la trochlée et du bouton rotulien. L'utilisation de la navigation ou de guides d'implantation dédiés au patient pourrait être gage d'une meilleure fiabilité et d'une meilleure reproductibilité des techniques de pose permettant une augmentation de la satisfaction postopératoire.



SUMMARY

The patellofemoral replacement (PFR) is still a subject of controversies because of non consistent reported results in the literature and because of high rate of early failures.

We report the results and the etiologies of failures of the PFR. We studied separately two different designs: the so called first generation of PFR design (characterized by resurfacing the patello femoral joint) and the recent optimized designs of PFR with anterior femoral bone cut (similar to TKA).

The PFR results and etiologies of failures change with the type of implant and design used. The first generation of PFR showed high failure rate leading to high revision rate up to 87%. The best results have been obtained with recent PFR requiring anterior femoral bone cut. Several series showed high patients satisfaction rate, high post operative functional scores and no post operative complications caused by the implants.

Strict preoperative criteria of patient selection increase significantly the results (no overweight patient, patient age under 65 yrs, frequent physical activity, good pre operative ROM, no excessive preoperative varus/valgus malalignment, grade IV from the Iwanno's classification, patellofemoral osteoarthritis on dysplasia rather than without dysplasia).

Optimization and increase in anatomical implant designs will be obtained by means of biomechanical studies on the trochlea and on the patellar implants. Navigation and patient specific 3D customized cutting guide could allow an increase in the reproducibility and reliability of the procedure and lead to higher patient postoperative satisfaction.

REFERENCES

[1] ACKROYD CE, CHIR B. Development and early results of a new patellofemoral arthroplasty. *Clin Orthop Relat Res. 2005 Jul(436): 7-13.*

[2] ACKROYD CE, NEWMAN JH, EVANS R, ELDRIDGE JD, JOSLIN CC. The Avon patellofemoral arthroplasty: five-year survivorship and functional results. *J Bone Joint Surg Br. 2007 Mar; 89(3): 310-5.*

[3] AMIS AA, SENAVONGSE W, DARCY P. Biomechanics of patellofemoral joint prostheses. *Clin Orthop Relat Res. 2005 Jul(436): 20-9.*

[4] ARCIERO RA, TOOMEY HE. Patellofemoral arthroplasty. A three- to nine-year follow-up study. *Clin Orthop Relat Res. 1988 Nov(236): 60-71.*

[5] ARGENSON JN, FLECHER X, PARRATTE S, AUBANIAC JM. Patellofemoral arthroplasty: an update. *Clin Orthop Relat Res. 2005 Nov; 440: 50-3.*

[6] ARUMILLI BR, NG AB, ELLIS DJ, HIRST P. Unusual mechanical complications of unicompartmental low contact stress mobile bearing patellofemoral arthroplasty: a cause for concern? *Knee 2010 Oct; 17(5): 362-4.*

[7] BAUCHU P. Résultats des prothèses fémoropatellaires *Thèse médecine, Faculté de médecine de Saint-Étienne. 1991.*

[8] BLAZINA ME, FOX JM, DEL PIZZO W, BROUKHIM B, IVEY FM. Patellofemoral replacement. 1979. *Clin Orthop Relat Res. 2005 Jul(436): 3-6.*

[9] BOARD TN, MAHMOOD A, RYAN WG, BANKS AJ. The Lubinus patellofemoral arthroplasty: a series of 17 cases. *Arch Orthop Trauma Surg. 2004 Jun; 124(5): 285-7.*

[10] BUTLER JE, SHANNON R. Patellofemoral arthroplasty with a custom-fit femoral prosthesis. *Orthopedics. 2009 Feb; 32(2): 81.*

[11] CARTIER P, SANOUILLER JL, KHEFACHA A. Long-term results with the first patellofemoral prosthesis. *Clin Orthop Relat Res. 2005 Jul(436): 47-54.*

[12] COSSEY AJ, SPRIGGINS AJ. Computer-assisted patellofemoral arthroplasty: a mechanism for optimizing rotation. *J Arthroplasty. 2006 Apr; 21(3): 420-7.*

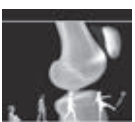
[13] de WINTER WE, FEITH R, VAN LOON CJ. The Richards type II patellofemoral arthroplasty: 26 cases followed for 1-20 years. *Acta Orthop Scand. 2001 Oct;72(5):487-90.*

[14] DEJOUR D, BARBOSA J, JACQUOT N, NEYRET P. Résultats des prothèses totales du genou dans l'arthrose fémoropatellaire isolée. Symposium sous la direction de J Allain et D Dejour. *Rev Chir Orthop 2004; 90(Suppl 5): 111-3.*

[15] FARR J 2nd, BARRETT D. Optimizing patellofemoral arthroplasty. *Knee 2008 Oct; 15(5): 339-47.*

[16] GADEYNE S, BESSE JL, GALAND-DESME S, LERAT JL, MOYEN B. Résultats de la prothèse fémoropatellaire autocentrique : à propos d'une série continue de 57 prothèses. *Rev Chir Orthop Reparatrice Appar Mot. 2008 May; 94(3): 228-40.*

[17] HASSABALLA MA, PORTEOUS AJ, NEWMAN JH. Observed kneeling ability after total, unicompartmental and patellofemoral knee arthroplasty: perception versus reality. *Knee Surg Sports Traumatol Arthrosc. 2004 Mar; 12(2): 136-9.*



[18] HENDRIX MR, ACKROYD CE, LONNER JH. Revision patellofemoral arthroplasty: three- to seven-year follow-up. *J Arthroplasty 2008 Oct; 23(7): 977-83.*

[19] KOOIJMAN HJ, DRIESSEN AP, VAN HORN JR. Long-term results of patellofemoral arthroplasty. A report of 56 arthroplasties with 17 years of follow-up. *J Bone Joint Surg Br. 2003 Aug; 85(6): 836-40.*

[20] KRAJCA-RADCLIFFE JB, COKER TP. Patellofemoral arthroplasty. A 2- to 18-year followup study. *Clin Orthop Relat Res 1996 Sep(330): 143-51.*

[21] LEADBETTER WB, KOLISEK FR, LEVITT RL, BROOKER AF, ZIETZ P, MARKER DR, *et al.* Patellofemoral arthroplasty: a multi-centre study with minimum 2-year follow-up. *Int Orthop 2009 Dec; 33(6): 1597-601.*

[22] LEADBETTER WB, RAGLAND PS, MONT MA. The appropriate use of patellofemoral arthroplasty: an analysis of reported indications, contraindications, and failures. *Clin Orthop Relat Res. 2005 Jul(436): 91-9.*

[23] LEADBETTER WB, SEYLER TM, RAGLAND PS, MONT MA. Indications, contraindications, and pitfalls of patellofemoral arthroplasty. *J Bone Joint Surg Am. 2006 Dec; 88 Suppl 4: 122-37.*

[24] LONNER JH. Patellofemoral arthroplasty: pros, cons, and design considerations. *Clin Orthop Relat Res. 2004 Nov(428): 158-65.*

[25] LOTKE PA, LONNER JH, NELSON CL. Patellofemoral arthroplasty: the third compartment. *J Arthroplasty. 2005 Jun; 20(4 Suppl 2): 4-6.*

[26] LUBINUS H. Patella glide bearing total replacement. *Orthopedics. 1979: 119-27.*

[27] Mc KEEVER D. Patellar prosthesis. *J Bone Joint Surg Am. 1955; Oct;37-A(5): 1074-84.*

[28] MERCHANT AC. Early results with a total patellofemoral joint replacement arthroplasty prosthesis. *J Arthroplasty. 2004 Oct; 19(7): 829-36.*

[29] MERCHANT AC. A modular prosthesis for patellofemoral arthroplasty: design and initial results. *Clin Orthop Relat Res 2005 Jul(436): 40-6.*

[30] MINAS T. Patellofemoral replacement: the third compartment. *Orthopedics 2008 Sep; 31(9): 920-2.*

[31] MOFIDI A, BAJADA S, HOLT MD, DAVIES AP. Functional relevance of patellofemoral thickness before and after unicompartmental patellofemoral replacement. *Knee. 2011 Apr 11.*

[32] MORRA EA, GREENWALD AS. Patellofemoral replacement polymer stress during daily activities: a finite element study. *J Bone Joint Surg Am. 2006 Dec; 88 Suppl 4: 213-6.*

[33] NICOL SG, LOVERIDGE JM, WEALE AE, ACKROYD CE, NEWMAN JH. Arthritis progression after patellofemoral joint replacement. *Knee. 2006 Aug; 13(4): 290-5.*

[34] ODUMENYA M, COSTA ML, PARSONS N, ACHTEN J, DHILLON M, KRIKLER SJ. The Avon patellofemoral joint replacement: Five-year results from an independent centre. *J Bone Joint Surg Br. 2010 Jan; 92(1): 56-60.*

[35] PAXTON EW, FITHIAN DC. Outcome instruments for patellofemoral arthroplasty. *Clin Orthop Relat Res. 2005 Jul(436): 66-70.*

[36] SISTO DJ, SARIN VK. Custom patellofemoral arthroplasty of the knee. *J Bone Joint Surg Am. 2006 Jul; 88(7): 1475-80.*

[37] SMITH AM, PECKETT WR, BUTLER-MANUEL PA, VENU KM, D'ARCY JC. Treatment of patello-femoral arthritis using the Lubinus patello-femoral arthroplasty: a retrospective review. *Knee. 2002 Feb; 9(1): 27-30.*

[38] STARKS I, ROBERTS S, WHITE SH. The Avon patellofemoral joint replacement: independent assessment of early functional outcomes. *J Bone Joint Surg Br. 2009 Dec; 91(12): 1579-82.*

[39] TAURO B, ACKROYD CE, NEWMAN JH, SHAH NA. The Lubinus patellofemoral arthroplasty. A five- to ten-year prospective study. *J Bone Joint Surg Br. 2001 Jul; 83(5): 696-701.*

[40] VAN JONBERGEN HP, WERKMAN DM, VAN KAMPEN A. Conversion of patellofemoral arthroplasty to total knee arthroplasty: A matched case-control study of 13 patients. *Acta Orthop. 2009 Feb; 80(1): 62-6.*

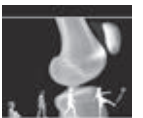
[41] VAN WAGENBERG JM, SPEIGNER B, GOSENS T, DE WAAL MALEFIJT J. Midterm clinical results of the Autocentric II patellofemoral prosthesis. *Int Orthop. 2009 Dec; 33(6): 1603-8.*

[42] WITVOET J. L'état actuel des prothèses fémoropatellaires. Conférences d'enseignement 1994. *Cahiers d'enseignement de la Sofcot n° 46. Exp Scient Fr, Paris: 7992.*

[43] WITVOET J. L'état actuel des prothèses fémoropatellaires. In: Conférences d'enseignement 1994 *Cahiers d'enseignement de la Sofcot n° 46 Exp Scient Fr, Paris, 1991, 7992.*

[44] WITVOËT J, BENSLAMA R, ORENGO P, AUBRIOT J, BROUTART J, LE BALC'H T. La prothèse fémoro patellaire Guépar. Description. Résultats initiaux. *Rev Chir Orthop Reparatrice Appar Mot. 1983; 69 Suppl 2: 156-8.*

[45] WITVOËT J, BENSLAMA R, ORENGO P, AUBRIOT J, BROUTART J, LE BALC'H T. La prothèse fémoro patellaire Guépar. Description. Résultats initiaux. *Rev Chir Orthop Reparatrice Appar Mot. 1983; 69 Suppl 2: 156-8.*



THE TREATMENT EVOLUTION OF PATELLOFEMORAL DEGENERATION, ARTHRITIS, AND ARTHROPLASTY: An Historical Account of How a Forgotten Joint Became the Center of World Attention

W.B. LEADBETTER

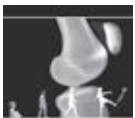
"The longer we keep studying the subject… the more we are in danger of soon knowing nothing certain about it!"

Mark Twain

The evolution of surgical treatment options for symptomatic patellofemoral chondral degeneration and its promoting causes has paralleled the evolution of all scientific and surgical thought [1]. It is a history fraught with at best, misconception and at worst, dogma. It is a history dominated by a multifactorial elusive symptom – refractory knee pain. It is a history characterized by a progression from reliance solely on the physical exam and the intraoperative appearance of the joint surface to one of intensive histologic, biochemical, biomechanical, anatomic and radiologic study. It is a history in which operative approach has evolved from a philosophy of unknowing expedient extirpation and expendibility, ie patellectomy, to one of critical conservation. And it is a history in which the French and Lyonnaise Orthopaedic tradition has figured prominently. Two technologies that have had great influence on present day thinking of the salvage of the painful worn patella have been total knee arthroplasty and biological articular cartilage restoration and/or transplantation. Ironically, total knee arthroplasty has played the role of both stimulating the development of isolated patellofemoral arthroplasty as well as setting a "gold standard" against which attempts at isolated patellofemoral replacement are often criticized [2]. In this article, when and how some of these sentinel historical influences have evolved and contributed to present day surgical beliefs will be explored.

In the Beginning… the Patellofemoral Pre-Dawn Age

In 1803, Hey coined the term "internal derangement" (often usage includes mechanical derangement) to denote any disorder of the knee joint [1]. It was a term that served it's purpose as there were no radiologic tools or operative options to further define the structural implications of the diagnosis. Undoubtly, many of these derangements involved arthritic disease. With the advent of radiographs, arthritis was recognized to present in two prevalent forms – atrophic and hypertrophic. The former was later recognized as most often rheumatologic and the latter the result of abnormal loads and forces leading to subchondral sclerosis and osteophytosis. Furthermore, this so called osteoarthritis became considered as a degenerative process



associated with aging, although the two tissue pathologies are similar, but not entirely identical [3]. Hence, young patients (a class increasingly difficult to define) may display premature patellofemoral wear. In 1908, Budinger excised degenerative patella articular chondral tissue which he described as "chondropathy" (fig. A). In 1928, Aleman from Scandanavia refined this physical description in the operating room as "chondromalacia patella post-traumatica", a post injury softening of the patellar articular surface [1]. Cadaveric studies of Owre (1936) and Hirsch (1941) would introduce the term, "chondromalacia patellae" as a unique entity [4]. No long after, Cox (1945) and Bronitsky (1947) would include patella cartilage fibrillation and softening in the potential causes of patellofemoral pain syndromes [4]. Chondromalacia patellae would become synonymous with patellofemoral arthralgia. Lacking insight into the functional cost of patellectomy and the secondary deleterious effects on tibiofemoral joint durability, standard of care drifted toward a "when in doubt, cut it out" salvage for the damaged patella. Despite isolated satisfactory outcomes, patellectomy did not account for pain arising from trochlea disease and there was a significant pattern of persistent impairment after patellectomy [5]. Otherwise, in many ways, it was the age of the "forgotten patella".

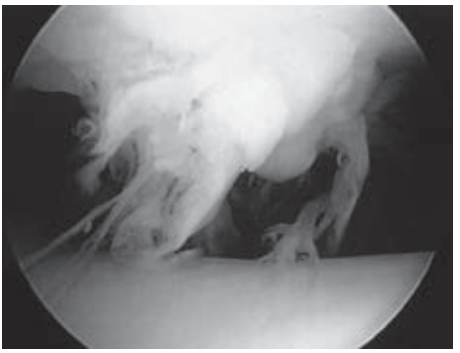


Fig. A: Chondromalacia patellae (arthroscopic view): Often treated by the euphemistic method of chondroplasty. While studies have shown short term relief of pain, functional improvement is unpredictable. The concept is limited in the same way that mowing a lawn down to dirt can eliminate weeds, ie nonrestorative.

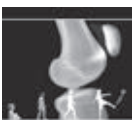
That said, some surgeons were impressed enough with the disability of patellofemoral degenerative wear to advocate patella resurfacing with skin, fat pad, or fascia.

In 1955, the earliest attempt at prosthetic replacement of the degenerative patella surface was reported by McKeever [6] (fig. B). This metal hemiarthroplasty was used sparingly with improvements by Worrell. Primitive design and inherent failure of the trochlea cartilage surface limited its application. The idea would not be seriously reconsidered until 1979.

Fig. B: McKeever prosthesis

The Age of Awakening- the Patellofemoral Renaissance (circa 1960s-early 1990's)

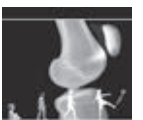
A resurgence in surgical interest in salvaging the painful patellofemoral joint began to stir in the 1960's. With the publication of their article on the diagnosis and treatment for recurrent patellofemoral instability, Trillat and Dejour called attention to the importance of extensor malalignment as a source of anterior knee pain and its potential correction by redirection [7]. Hughston was another early voice, commenting in 1960 and reiterating in 1984 that recurrent subluxation of the patella was often overlooked in the diagnosis of medial knee joint pain [8]. Shortly after, a young American graduate



orthopedic surgeon came to visit Professor Ficat in Toulouse. His name was David S. Hungerford. Trained in England and Germany, Dr Hungerford provided a timely intellectual vector between European and North American orthopedics. The emerging concepts to which he was introduced regarding the importance of lower extremity alignment, the "Law of Knee Valgus", and the implication to the forces exerted upon the knee and patellofemoral joint by malalignment would form a foundation of thought for much of his career. In addition, Ficat would impress upon his protege the potential role of intraosseous hypertension not only in avascular necrosis of the femoral head, but as a source of patella pain. Dr Hungerford would return to assume a leadership position at The Johns Hopkins Hospital, Baltimore. There he began applying much of what he had learned in his travels. Two young physicians who came under his influence were Dr Leadbetter and Dr Fulkerson. Ficat and Hungerford would culminate their collaboration in the publication in 1977 of the most comprehensive treatise up to the time on disorders of the patellofemoral joint [4]. The patella was no longer forgotten.

It is important to consider historical developments in one discipline in the overall context of technological and societal trends of the same period. The 1970's and 80's were a dynamic era in the treatment of knee disorders with respect to: 1) increased diagnostic capability, eg. arthroscopy, CT, arthrography, and MRI; 2) the development of reliable knee arthroplasty, both total and partial; and 3) the rise of aerobic fitness exercise and wellness awareness. Patients developed increasing expectation of improved functional outcomes with every newly announced advance. Coincidently, this stimulated the birth and growth of a whole new orthopaedic subspecialty-orthopaedic sports medicine. Of course, in modern Europe there was a long association of medicine and sport dating back to the reinstitution of the Olympic Games with the founding of the Federation Internationale de Medicine du Sport (FIMS) in 1928. However, in North America the professional recognition of the subspecialty began in 1964. It was then that Dr Jack Hughston started the planning of what would formally become in 1972 the American Orthopaedic Society for Sports Medicine. With the blooming of running exercise, recreational sport, and women's athletics, anterior knee pain soon became the most common knee complaint in the outpatient orthopedic clinic worldwide.

During this period many surgeons made contributions to defining the pathology of chondromalacia, to the correction of patellofemoral instability and to identifying the factors contributing to progressive patello-femoral articular degeneration. Ficat wrote extensively about the pathology of degenerative cartilage and fibrillation. He wrote, "the causes of disorders of articular cartilage are the same as those of degenerative joint disease… the three main etiological factors are trauma, structural disorders, and mechanical problems, such as dysplasia, patellar instability, and joint overload" [9]. In 1978, he introduced his concept of lateral retinacula release for "lateral hyperpressure syndrome" [10]. The approach was facilitated by Merchant and Metcalf; however Ficat forewarned that not all chondral disease was either progressive or symptomatic and that the surgery of the chondromalacia is essentially a surgery of the pain and therefore, always keeps a bit of mystery [9]. Unfortunately, arthroscopic lateral release remains to this day one of the most over utilized and misapplied of operations. Radin summed up the operative strategy of the time when he wrote, "cartilage fibrillation does not necessarily progress. There appears to be different mechanical factors involved in the initiation and the progression of cartilage changes in osteoarthrosis. This means there is some rationale behind what we see clinically happening after successful osteotomy or other operations that lower the stress on degenerating joints, and it means that if we can do something about the level of stress and, therefore, the level of bone remodeling in patients with fibrillation, we may be able to keep that cartilage from further degenerating" [12]. With such observations, reducing symptom producing instability and the unloading of injured or degenerative patello-femoral cartilage became a prime surgical motive. Chambat reported the long term results of distal medial realignment for instability [11].



Maquet introduced controversy with his anterorization tibial tubercle osteotomy for knee arthritis [13]. Fulkerson improved upon these concepts with his anteromedialization tibial tubercle osteotomy (AMZ) [14].

However, despite Radin's hopes, there was a growing number of patients who failed to achieve relief from the available operative solutions. Total knee arthroplasty was in it's infancy and was not considered an option for the arthritically disabled, often younger (age less than 50 years old) patient. And so, beginning in 1974, Blazina launched the modern age of patellofemoral arthroplasty with the Richard I and II prostheses. The average patient age in his series was 39 years (range, 19- 81) [15]. At the same time in France, Cartier began his series using the both the Richards II and later the Richards III patellofemoral prosthesis. As a salvage procedure, these early experiences were encouraging. In 2005, he would report on his experience with 70 patients, average age 60 years (range, 36-81). There was a prosthetic survivorship of seventy-five percent at average follow-up of 10 years [16]. Yet, during this period prosthetic replacement of the patellofemoral joint remained the unpopular step child of total knee arthroplasty primarily because of reported high revision rates due to design deficiencies and difficult patient selection with regard to risk of tibiofemoral joint arthritic progression.

We will arbitrarily close this renaissance period with two developments. The first is the diagnosis, classification, and treatment implications of trochlear dysplasia. First described by Ficat and later refined by Henri Dejour, Phillipe Neyret, and David Dejour, the recognition of trochlear dyplasia proved to have significant implications for both the successful long term outcome and prosthetic evolution of patellofemoral prosthetic design [17, 18]. Argenson called attention to the presence of trochlear dyplasia as a key factor in predicting the success of patellofemoral arthroplasty with respect to progressive tibiofemoral joint involvement. It was theorized that because this mechanical cause of premature patellofemoral degeneration seemed independent of more genetically predisposed tricompartment arthritis, patellofemoral dysplasia would tend to be a marker for the selection of the so called isolated patellofemoral arthroplasty case [19]. Others would agree [20, 21]. The second development was Dye's recognition that radionuclear bone imaging could be a useful tool in dynamically visualizing Wolf's Law. His concept of the comfortable knee and patellofemoral joint functioning in a homeostatic load/use envelope and the association of patellofemoral pain with supraphysiologic load in the absence of other structural damage helped clarify an old dilemma as to why isolated chondromalacia was not always symptomatic [22] (fig. C). As a corollary, while an inactive nuclear bone scan can be seen in the painful degenerative patellofemoral joint, an active nuclear bone scan is not pathognomonic of a degenerative diagnosis as some would claim.

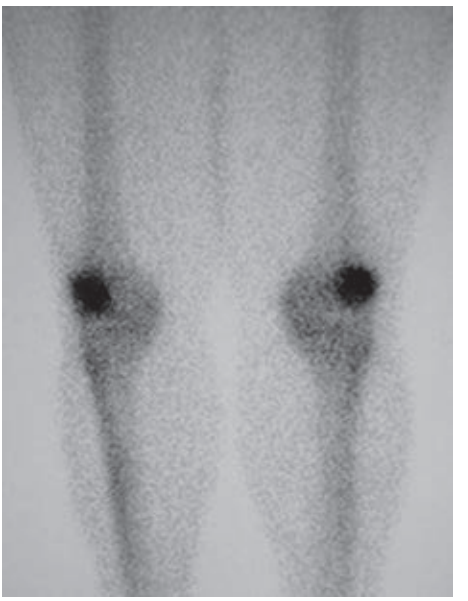


Fig. C: Typical radionucleotide scan revealing loss of bone homeostasis of the patellae. Plain radiographs were normal; however, such patients may or may not have patellofemoral degeneration.

Moving Forward - The Age of Scientific Discovery (circa 1994 - the Present)

In 1994, Brittenberg and Peterson fired the biological restoration shot that was heard around the world with their publication of the autologous cartilage cell transplantion procedure [23]. However, despite early optimism biological restoration of the patellofemoral joint has proven challenging due to in large part it's inherently hostile biomechanical environment (fig. D) (Table 1). A major step in meeting the challenge of defining the operative approach to symptomatic patellofemoral joint occurred with the founding by Fulkerson and Dupont in 1995 of the International Patellofemoral Study Group (IPSG). What has followed has been a plethora of monographs and papers addressing all aspects of anterior knee pain and patellofemoral arthritis that together have revolutionized thinking on the subject (Table 2). After years of lateral side strategy, a major focus during this era has been the recognition of the importance of the medial patellofemoral ligament in reducing extensor instability [24]. It is a time for patella conserving interventions such as partial lateral facetectomy [25]. It is a time when the results of total knee arthroplasty have drawn increasing scrutiny [2, 20]. There has been a renewed interest in patellofemoral arthroplasty as a salvage procedure to address advanced degeneration in younger active patients who are unwilling to accept the risks and potential revision of a total joint (fig. E). This trend has set up a philosophical divide between the traditional "total joint surgeon" and the (for lack of a better

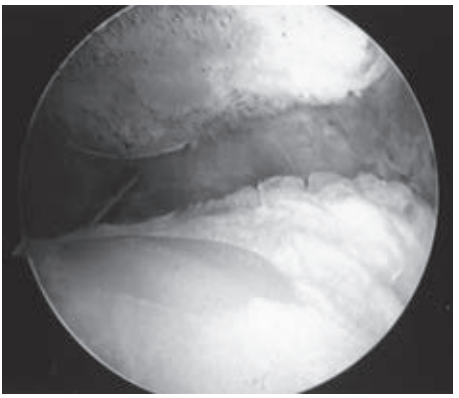


Fig. D: Arthroscopic view of advanced patellofemoral chondral degeneration. Note uncontained bipolar lesions unfavorable to present restorative procedures.

Table 1: The Hostile Biomechanical Nature of the Patellofemoral Joint

1.	Intrinsically Lax
2.	Incompletely Congruent
3.	Dysplastic Prone
4.	Overloaded/Overused
5.	Highly dependent upon balancing opposing extrinsically generated forces that remain clinically unmeasurable

Table 2: Current Operative Approaches for Patellofemoral Arthritis

1.	Arthroscopic debridement/ chondroplasty
2.	Microfracture articular restoration
3.	Lateral release
4.	Soft tissue realignment of the extensor mechanism
5.	Osteotomies of the tibial tubercle
6.	Mosaicplasty/autologous chondrocyte implantation/biodegradable scaffold
7.	Partial lateral facetectomy
8.	Patellofemoral arthroplasty
9.	Total knee arthroplasty

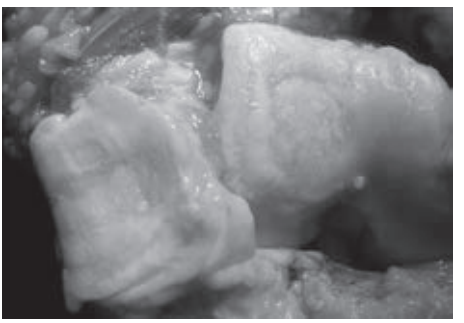
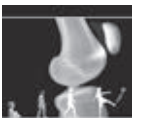


Fig. E: Patellofemoral arthritis with trochlear dyplasia



term) "comprehensive knee surgeon". However, the advent of second and third generation prosthetic design predicated upon years of total joint design, refinements in operative technique, and improved selection criteria have improved the outcomes of isolated patellofemoral arthroplasty [26, 27]. Likewise, biological restoration is maturing in effectiveness with the combination of unloading concepts and improved biotechnology [28]. Yet, as we enter the 21st millennium, the term "chondromalacia patellae" remains firmly fixed in the clinical diagnostic lexicon. Indeed, there still remains what Grelsamer has dubbed a "Tower of Babble" when one reads the literature of patellofemoral pain and arthritis. Teige has summarized the salient issues in this fashion: 1) bone architecture dictates where the force vectors acting on the patella will be directed; 2) abnormal skeletal alignment may alter the displacement forces acting on the patellofemoral joint, causing ligament failure with subsequent instability; 3) skeletal malalignment may also increase patellar facet loading leading to arthrosis; 4) Increased joint loading with the addition of subluxation may further increase unit loading. Pain results from this excess load and tension in the soft tissues or compression of the articular surfaces; 5) treatment depends on the primary pathology [30].

LITERATURE

[1] FITHIAN DC. A historical perspective of anterior knee pain. *Sports Med and Arthroscopy Rev. 2001; 9: 273-81.*

[2] DAHM DL, AL-RAYASHI W, DAJANI K *et al.* Patellofemoral arthroplasty versus total knee arthroplasty in patients with isolated patellofemoral osteoarthritis. *Am J Orthop. 2010; 39(10): 487-91.*

[3] KEUTTNER KE, SCHLEYERBACH R, PEYRON J *et al.* Articular Cartilage and Osteoarthritis: *Workshop Conference Hoechst Werk Kalle-Albert, Ed. Raven Press, New York. 1991.*

[4] FICAT RP, HUNGERFORD DS. Disorders of the Patello-femoral Joint. *Williams and Wilkins, Baltimore, MD, USA, 1977.*

[5] LENNOX IA, COBB AG, KNOWLES J, BENTLEY G. KNEE function after patellectomy. A 12- to 48-year follow-up. *Journal Bone and Joint surgery 1994; 76 Br: 485-7.*

[6] McKEEVER DC. Patellar prosthesis. *J Bone Joint Surg 1955; 37A:1074-84.*

[7] TRILLAT A, DEJOUR H, COUTTE A. Diagnostic et traitement des subluxation recidivantes de la rotule. *Rev Chir Orthop. 1964; 50: 813-24.*

[8] HUGHSTON JC, WALSH WM, PUDDU G. Patellar subluxation and dislocation. *Saunders Monographs in Clinical Orthopedics. Vol. 5 Philadelphia. WB. Saunders, 1984.*

[9] PICKETT JC, RADIN EL. Chondromalacia of the Patella. Eds. *Williams and Wilkins, Baltimore, 1983.*

[10] FICAT P. The syndrome of lateral hyperpressure of the patella. *Acta orthopaedica Belgica 1978; 44: 65-76.*

[11] CHAMBAT P, DEJOUR H. The results of transplantation of the tibial tubercle after follow-up of 10 years or more. *Orthop Trans 1980;4: 124.*

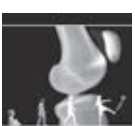
[12] RADIN EL. Chondromalacia as a separate entity: new perspectives on osteoarthrosis. *Bulletin of the Hospital for Joint Diseases 1979; 40: 84-9.*

[13] MAQUET P. Advancement of the tibial tuberosity. *Clin Orthop Relat Res 1976; 115: 225-30.*

[14] FULKERSON JP. Anteromedialization of the tibial tuberosity for patellofemoral malalignment. *Clin Orthop 1983; 177: 176-81.*

[15] BLAZINA ME, FOX JM, DEL PIZZO W, *et al.* Patellofemoral replacement. *Clin Orthop 1979; 144: 98-102.*

[16] CARTIER P, SANOUILLER J-L, KHEFACHA A. Long – term results with the first patellofemoral prosthesis. *Clin Orthop Relat Res 2005; 436: 47-54.*



[17] DEJOUR H, WALCH G, NEYRET P. et al. La dysplasia de la trochlee femorale. *Rev Chir Orthop.1990, 76: 45-54.*

[18] LIPPACHER S, DEJOUR D, ELSHARKAWI M, *et al.* Observer agreement on the Dejour trochlear dysplasia classification - a comparison of true lateral radiographs and axial magnetic resonance images. *Am J Sports Med 2012; 40(4): 837-43.*

[19] ARGENSON JN, GUILLAUME JM, AUBANIAC JM. Is there a place for patellofemoral arthroplasty? *Clin Orthop Relat Res 1995; 321: 162-7.*

[20] LEADBETTER WB, MONT MA. Patellofemoral arthroplasty: a useful option for recalcitrant symptomatic patellofemoral arthritis. *Seminars in Arthroplasty 2009; 20(3); 148-60.*

[21] NEWMAN JH. Patellofemoral arthritis and its management with isolated patellofemoral replacement: a personal experience. *Orthopedics 2007; 30(suppl 8): 58-61.*

[22] DYE SF. The pathophysiology of patellofemoral pain: a tissue homeostasis perspective. *Clin Orthop Relat Res 2005; 436: 100-10.*

[23] BRITTENBERG M, LINDAHL A, OHLSSON C. *et al.* Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. *N Engl J Med 1994; 331: 889-95.*

[24] SALEH KJ, ARENDT EA, ELDERIDGE J, *et al.* Symposium: operative treatment of patellofemoral arthritis. *J Bone Joint Surg 2005; 87A; 659-71.*

[25] YERCAN HS, SELMIO TAS, NEYRET P. The treatment of patellofemoral osteoarthritis with partial lateral facetectomy. *Clin Orthop Relat Res 2005; 436: 14-19.*

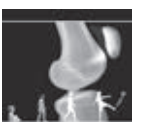
[26] LEADBETTER WB, RAGLUND PS, MONT MA. The appropriate use of patellofemoral arthroplasty-an analysis of reported indications, contraindications, and failures. *Clin Ortho Relat Res 2005: 436; 91-9.*

[27] ACKROYD CE, JOSLIN C, NEWMAN JH. The Avon patellofemoral arthoplasty: The five year results and survivorship. *J Bone Joint Surg Br. 2007; 89: 310-15.*

[28] FARR J. Autologous chondrocyte implantation and anteromedialization in the treatment of patellofemoral chondrosis. *Orthop Clin N Am 2008; 39: 329-35.*

[29] GRELSAMER RP. Patellar nomenclature: the Tower of Babel revisited. *Clinical orthopaedics and related research 2005: 60-5.*

[30] TEIGE RA. Patellofemoral syndrome a paradigm for current surgical strategies. *Orthop Clin N Am 2008; 39: 287-311.*



THE PRINCIPLES OF AN IDEAL PATELLOFEMORAL ARTHROPLASTY

M. ODUMENYA, S.J. KRIKLER, A.A. AMIS

INTRODUCTION

Isolated patellofemoral arthritis can be successfully treated with patellofemoral arthroplasty (PFA). The outcome is determined by the prosthesis design, surgical technique and patient selection.

Historically, poor patient selection was thought to be the main cause of unsatisfactory outcomes following PFA. Whilst this most certainly was partly to blame, prosthetic design and failure to restore or correct patellofemoral biomechanics may have played a more significant role in the high revision rates. Biomechanical analysis of the older inlay designs and an appreciation for the causes and effects of malpositioning and misalignment have aided the development of contemporary second and third generation PFAs. These newer onlay designs, coupled with improved surgical instrumentation, have shown better clinical outcomes, with the majority of failures relating to the progression of tibiofemoral arthritis rather than surgical complications. Greater understanding of the variations in pattern of progression of the underlying pathologies, such as trochlear dysplasia and idiopathic patellofemoral arthritis, has resulted in surgeons adopting more stringent selection criteria. This, coupled with appreciation of biomechanical abnormalities requiring surgical correction, has also contributed to lower failure rates.

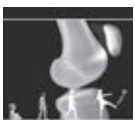
The aim of this article is to highlight the design issues of previous patellofemoral arthroplasties and how the problems encountered with these implants have influenced the development of newer second and third generation prostheses. The ideal design features that are required to provide improved clinical outcomes based on biomechanical and material theory as well as patient selection will also be discussed.

PAST PATELLOFEMORAL PROSTHESIS DESIGN ISSUES

In the past, the most frequent complaints following patellofemoral arthroplasty were of patellar instability manifesting as snapping, clunking or subluxation and anterior knee pain [1-4]. These symptoms, most commonly associated with the older first-generation patellofemoral arthroplasties, were almost certainly related to the flawed designs of their trochlear components.

The critical features of trochlear component geometry are the degree of constraint, the dimensions of both medial-lateral width and proximal extension of the anterior flange, and the sagittal radius of curvature.

The Richards (or Blazina) I and II (Smith & Nephew Richards Inc., Memphis, Tennessee)



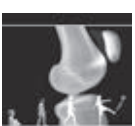
prostheses had a deep constraining trochlear design which required exact alignment of the patellar and trochlear components; failure to do so resulted in maltracking and catching of the patellar component on the rim of the trochlea. Due to the unforgiving nature of these prostheses, a high number of additional operations have been reported. De Winter *et al.* [2] found, at a mean of eleven years follow-up, that 27% of patients (7 out of 26) had required further surgery to treat misalignment or maltracking; two had patellar realignment, three had patellectomy and two had total knee arthroplasty. Kooijman *et al.* [5] reported, at a mean of seventeen years follow-up, 27 reoperations in 25 out of 45 PFA, of which 26% involved corrective surgery for either patellofemoral symptoms such as catching or prosthesis malpositioning.

The Lubinus (Waldemar Link, Hamburg, Germany) PFA was also associated with a high number of patellofemoral complications. This prosthesis had a narrow medial-lateral width and a deep constraining groove in the axial plane (see fig. 1A). The anterior flange did not extend proximally, leaving the patella to articulate with the anterior femoral cortex in full knee extension before engaging with the trochlear component in the initial 30° of flexion (see fig. 2A). Unfortunately, this transition from the femoral articular cartilage to the prosthesis was not smooth due to its shape in the sagittal plane not matching that of the distal femur. Therefore, to avoid impingement of the intercondylar aspect of the trochlear component on the tibia or anterior cruciate ligament when the knee reached full extension, the component had to be inserted with an offset from the anterior femoral cortex. It was this offset position that caused the patella to catch and sublux on the proud proximal anterior flange at 30° of tibiofemoral flexion [3, 4, 6], resulting in poor clinical outcomes. Of the seventy-six Lubinus arthroplasties reviewed by Tauro et al. [4] twenty four had patellar misalignment and a further 21 knees required revision surgery of which 15 were for patellar maltracking. Therefore 51% of knees had patellofemoral dysfunction, matching the high rate of unsatisfactory clinical outcome, of 55% of knees.

The developers of newer prostheses have taken into consideration these design flaws of the past as signified by the improvements in clinical performance and lower rates of patellofemoral dysfunction.

SECOND AND THIRD GENERATION PATELLOFEMORAL PROSTHESES

The most distinctive design difference between first-generation and newer PFAs is the design of the trochlear component. The first-generation prostheses had a characteristic inlay design, in which the implant was inset into the trochlea. These prostheses were significantly smaller, thinner in depth and narrower in width with a deeper, more constraining groove than more recent designs. The philosophy behind these designs was to preserve bone and avoid overstuffing the patellofemoral joint, allowing for simple revision if required. In contrast, the second- and third-generation prostheses are onlay designs which have thicker and wider trochlear components. The aim of the increased thickness is to restore normal trochlear offset, that is, the offset that existed before arthritis if the trochlea was not dysplastic, in order to maintain soft tissue tensions and the mechanical advantage of the extensor mechanism. Unlike the inlay design, the shape of the trochlea does not completely govern the positioning of the onlay device. Instead, the trochlea can be placed in the correct position in the presence of abnormal trochlear morphology without the risk of the component sitting prominent relative to the adjacent articular surface. This still requires the surgeon to avoid step-like discontinuities of the surface at the distal end of the prosthetic articulation, which can cause patellar catching and clunking. The anterior flange of onlay designs extends far more proximally and covers up to and beyond the articular portion of the anterior femoral cortex, allowing the patellar component to remain in contact with the trochlear component in full knee extension.



A number of authors have reported significant clinical improvements and a reduction in patellofemoral complications following the revision of Lubinus prostheses to the Avon Patello-Femoral Joint Replacement System (Stryker® Howmedica Osteonics, Allendale, New Jersey), a second-generation PFA [6, 7]. A retrospective case series found that 17% of patients with the Lubinus prosthesis suffered from either subluxation, catching or severe pain. This rate was significantly reduced to 4% following conversion to the Avon [6]. Another study replaced 14 failed Lubinus prostheses with Avon implants for patellar component wear and trochlear component malposition. The authors reported substantial improvement in functional outcomes and pain, with no cases of misalignment or instability at five years follow-up [7]. These improvements are probably attributable to the trochlear design of the Avon.

The Avon, unlike the Lubinus or Richards, has an anterior flange which is straight near extension, in the sagittal plane, and then a radius of curvature distally, through an arc close to 90°; this shape is closer to the normal geometry of the distal femur (see fig. 2B). This allows for the trochlear prosthesis to lie flush within the intercondylar notch, medially, laterally and on the surface of the anterior femoral cortex. The broader sulcus angle of the Avon (approximately 125°) compared with the Lubinus (approximately 110°) is less constraining in extension and thus less sensitive to slight patellar tilt or subluxation (see fig. 1A-B). This concept is based on previous biomechanical studies [8, 9] performed during the development phase of the Kinemax total knee replacement which suggested that the patella is unconstrained in full extension and its movement is dictated by the quadriceps, mainly vastus lateralis, and hence there is often a lateral tilt. The patellar component has enough freedom to find its course smoothly along the trochlear groove, whereas the narrower Lubinus prosthesis was much less forgiving.

The Avon prosthesis was based on the Kinemax Plus total knee replacement, with a symmetrical component; one geometric design exists for both left and right. The justification [10] for this design was that the prosthesis aligns with the mechanical axis, not the anatomical one, and therefore sided prostheses are not necessary. The functional outcomes for this prosthesis are excellent, the incidence of patellar maltracking is less than 1% and survivorship has been reported as 100% at mean 5 year follow-up [11] and 94% at 8-14 year follow-up [12]. Despite these encouraging results, other authors have postulated that the lack of asymmetry

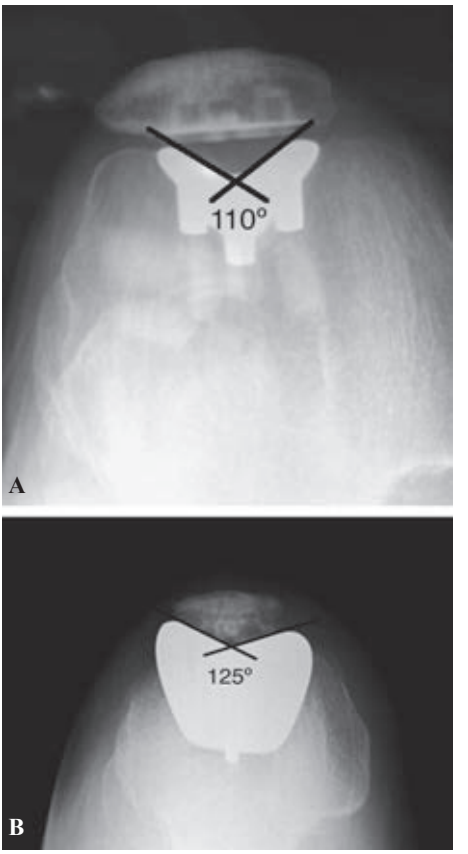
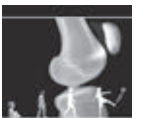


Fig. 1A-B : [A] The skyline radiograph shows the trochlear sulcus angle of the Lubinus prosthesis is approximately 110°. [B] The skyline radiograph shows the broader trochlear sulcus angle of the Avon prosthesis is approximately 125°.
Permission to use image granted by copyright owners Lippincott Williams & Wilkins. Lonner JH, Patellofemoral arthroplasty: pros, cons and design considerations. Clin Orthop 2004; 428: 158-65.



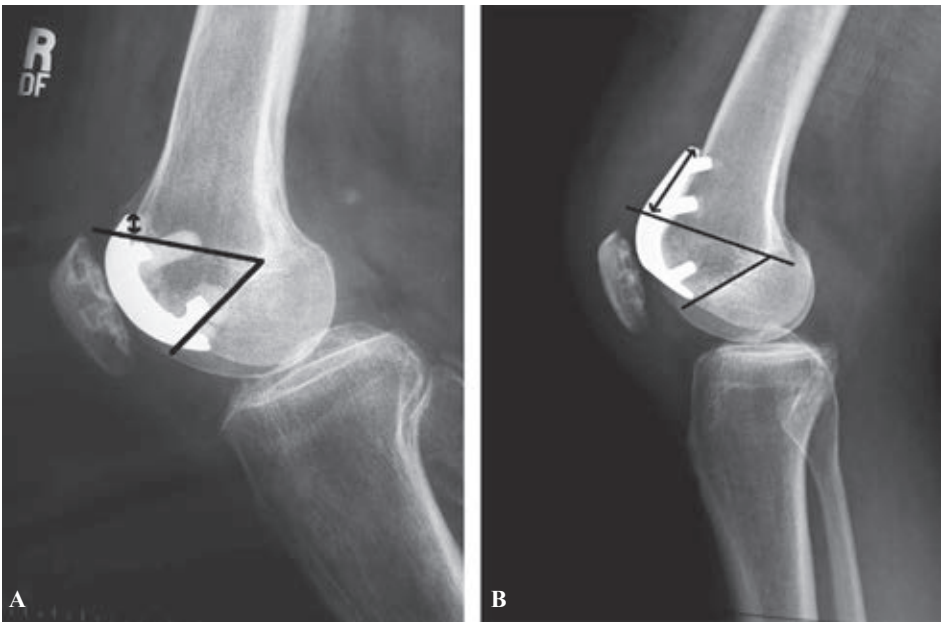


Fig. 2A-B : [A] The short anterior flange of the Lubinus prosthesis causes the patella to catch or sublux as it moves from the native femoral articular surface to the prosthesis in the initial 30° of knee flexion. [B] The Avon prosthesis has a much longer proximal extension of the anterior flange ensuring the patellar component remains in articulation with the trochlear component in full extension.
Permission to use image granted by copyright owners Lippincott Williams & Wilkins. Lonner JH, Patellofemoral arthroplasty: pros, cons and design considerations. Clin Orthop 2004; 428: 158-65.

means the position of the trochlear prosthesis in the coronal plane varies considerably and may affect the pattern of patellar tracking [13] especially in patients with a propensity to track laterally. Amis *et al.* [14] also established that lateral patellar shift is well controlled with a steep lateral trochlear facet and confirmed the difference in loading profile of the lateral and medial facet (60:40) which suggests the lateral facet should be relatively larger to accommodate this. The third-generation designs such as the FPV® Patello-Femoral Replacement (Wright Medical Technology Inc., Arlington, Tennessee), Journey® PFJ (Smith & Nephew®, Memphis, Tennessee) and Zimmer Gender Solutions PFJ have been designed as asymmetrical sided prostheses based on these biomechanical concepts.

Currently, there are no published data reporting mid to long-term outcomes on these prostheses, but the short-term results are promising [15, 16] Although these are all asymmetrical designs, each implant has a unique selling point. The FPV trochlear prosthesis was designed to emulate the normal anatomical trochlea. The lateral facet of the trochlear groove is steeper and has a larger surface area than the medial facet. In contrast to the Avon, the sulcus angle is even wider, measuring 140°. The sagittal arc of curvature is 90°, similar to the Avon, and therefore it matches the distal femur. Although shorter than the Avon, the proximal extension of the anterior flange is long enough to remain in contact with the patella in full knee extension. Due to the congruent nature of the trochlear and patellar components, accurate positioning of both components is paramount. Unlike the FPV, the Journey PFA, another asymmetrical third-generation prosthesis, based on the GENESIS® II Total Knee System (Smith & Nephew

(Reconstructive) Ltd, Memphis, USA), offers patellar resurfacing as optional. The unique oxidised zirconium material of the trochlear component has a significantly lower coefficient of friction compared with cobalt chrome and therefore the risk of wear will theoretically be considerably reduced. The Zimmer® Gender Solutions™ PFA System offers a guided milling system for preparation of the trochlea which allows accurate depth control and contouring, thus avoiding the pitfalls of freehand technique. In addition, it is the only PFA that incorporates anatomical differences between genders into the design. The trochlear sulcus angle for four out of five of the available sizes is greater to accommodate the larger Q angle found in females who are more predisposed to develop patellofemoral arthritis, presumably for this very reason. Whilst these innovations appear fairly logical and plausible it is important to bear in mind that there is no clinical evidence to suggest that they are of any benefit or advantage. Similarly, customised prostheses developed as a solution to the issue of size limitations with standard prostheses have not been shown to be of clinical or cost effective benefit. Future robust studies are required to investigate these theoretical advantages.

The most recent Report from the National Joint Registry for England and Wales [17] revealed the Avon as the most popular PFA, with the lowest revision rate at 5 years reported as 7% compared with 12% for the other brands (FPV, Journey, Zimmer Natural-Knee® II PFJ and Zimmer Gender Solutions PFJ).

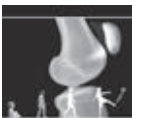
Generally these more advanced designs have better results than the first-generation prostheses. Fewer failures secondary to patellofemoral dysfunction have been reported, with progression of tibiofemoral degeneration being the most significant mode of failure. Complications such as loosening and anterior knee pain are also far less common [18, 19].

INSTRUMENTATION

Studies have shown the importance of aligning the trochlear prosthesis in external rotation to improve patellar tracking and minimise forces causing lateral translation [20]. A jig that sets the external rotation using femoral alignment is ideal, and freehand sculpting of the trochlea may result in less accurate preparation. Ideal instrumentation will provide accurate orientation and consistent depth of the entire cut surface to allow the prosthesis to sit flush on the anterior surface of the distal femur, and have a smooth transition from the prosthetic surface to the native femoral articular surface.

Most current PFA systems are fully instrumented. They claim not to require freehand bone preparation and therefore offer consistent, accurate alignment and precise bone cuts. However, it is difficult to provide instrumentation which can accommodate the deficient bone which is often encountered in patients with advanced patellofemoral degenerative changes or dysplasia.

The current instrumentation systems are not without limitations. For instance, the FPV patellar spring loaded clamp is only designed to be used with a medial parapatellar approach. Surgeons intending to perform a lateral parapatellar approach may attempt to overcome this predicament by using the contralateral patella component in reverse. However this risks erroneous alignment which will have a negative impact on patellar tracking. Currently, most of the systems do not offer a short intramedullary anterior cut guide. In patients who have existing intramedullary fixation, the guide is unable to contact the distal femur and therefore the risk of notching and incorrect external rotation are high. Another example is the milling device and cutting jig used in the Zimmer Gender Solutions PFJ system. Although a very useful instrument it can be challenging (particularly in the presence of hard bone) to maintain orientation of the mill within the guided tracks (see fig. 3A-B). Loss of perpendicular mill alignment relative to the jig could result in loss of the “set position”, the jig rising off the bone and ultimately an uneven, shallow bone preparation. This will offset component positioning and potentially affect the patellofemoral biomechanics. Furthermore, once the jig is pinned into place the depth of the



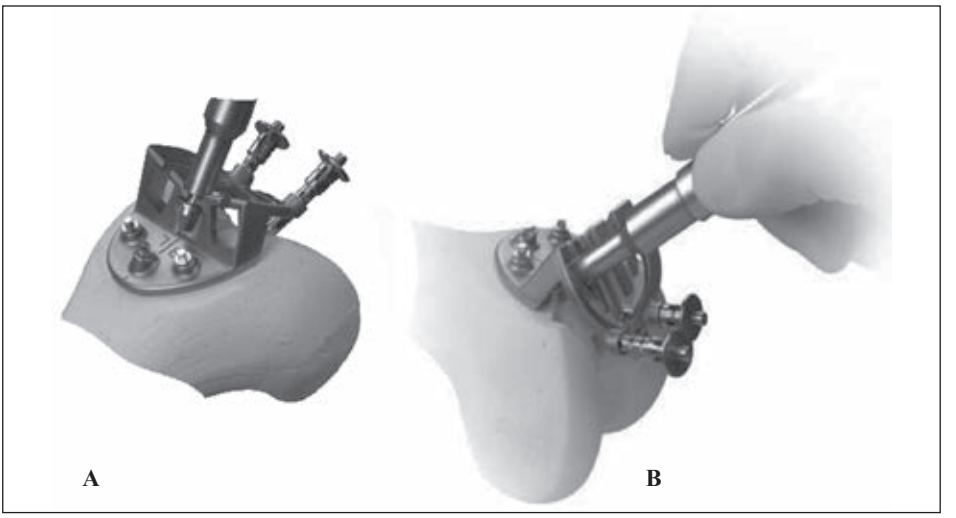


Fig. 3A-B : Zimmer PFJ milling guide. [A] Burr guard of milling handpiece placed within the central track of the milling guide. [B] Milling handpiece held perpendicular to milling guide. Slight deviation from this position due to toggling or binding may potentially result in loss of alignment and an inaccurate resection.
Permission to use images granted by copyright owners© 2012 Zimmer Inc.

femoral resection is set and not adjustable. Unfortunately, such inflexibility does not reflect real life clinical practice in which gross variability is encountered. In reality, when confronted with a severely worn, irregularly shaped distal femur, such standard instrumentation will not produce the ideal surface to fit a standard trochlear component. In many cases, some hand sculpting of the distal femur is required to get the standard implant to sit perfectly. For these reasons, the Journey PFA system offers the surgeon the option of adjusting the femoral resection depth after the jig is fixed in position and thus may minimize the need for the additional freehand fashioning (see fig. 4A-C).

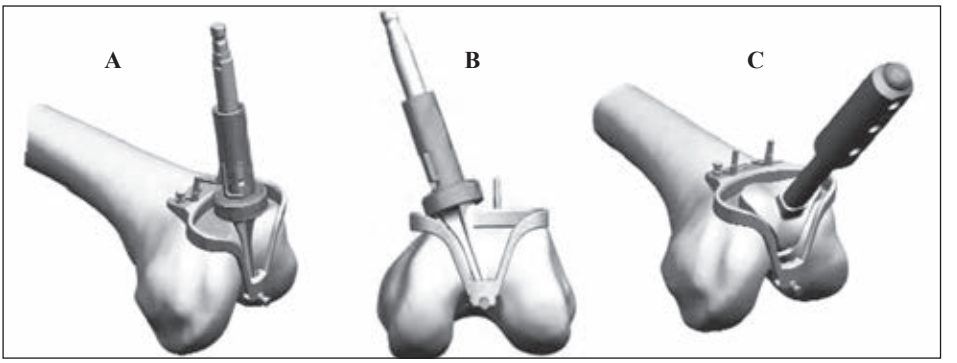
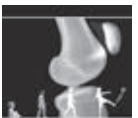


Fig. 4A-C : Journey PFJ reamer guide. [A] Yellow standard ream sleeve on reamer shaft is used to achieve sufficient depth by placing the reamer tip into the round indentation in the reamer guide and reaming from medial-lateral-medial. [B] Completion of reaming is indicated when the reamer sleeve is in continuous contact with the reamer guide throughout the range of motion. [C] Trochlear depth gauge is used to determine adequacy of reaming depth. If more bone is required, other sleeves are available to increase the depth and width of reamed surface area.
Permission to use images granted by copyright owners Smith & Nephew®.



Despite these relatively minor issues, these instrumented systems are far superior compared with those of the past. Systems which required significant freehand sculpting of the femur were daunting to surgeons trained to perform knee surgery with sophisticated guides and cutting blocks, and there was a feeling that these systems required a long learning curve.

It is plausible to believe that the high reproducibility that more modern instruments provide together with theoretically better prosthetic designs would have a positive impact on clinical outcomes. However, to date, no other prosthesis has equalled or exceeded the excellent functional results [11, 12] or revision rate [11, 12, 17] of the Avon. Time will tell whether these newer prostheses are able to improve on these highly commendable results.

BIOMECHANICAL AND MATERIAL SCIENCE CONSIDERATIONS

The ideal PFA should attempt to reproduce patellofemoral biomechanics and correct co-existing pathology causing instability.

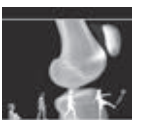
The patellofemoral joint has a complex configuration. Knowledge of the joint reaction forces, kinematics and stability are required to create an effective PFA. Patients suitable for PFA often have instability symptoms secondary to the maltracking or malalignment pathology that may have caused the arthritis to occur. Therefore, it is of paramount importance that the articular geometry of the femoral and patellar components provides stability in the medial-lateral direction (transverse plane).

The patella is pulled laterally in the final 20° of knee extension and disengages from the proximal trochlear groove. The line of motion is estimated as the direction of the force vector of the vastus lateralis, the largest muscle of the quadriceps. This direction of motion is reversed during early knee flexion, where the patella moves ‘relatively medially’ and the lateral facets of both patella and trochlear groove engage. In patients with patellar maltracking the patella tends to displace laterally. The severity of displacement depends on the integrity of the surrounding soft tissue stabilisers and bony alignment. A femoral component with a prominent proximal lateral facet could prevent this lateral displacement by causing the maltracking patella to engage, capture and maintain engagement as it passes into the distal portion of the trochlear groove in deep flexion.

The mechanics of medial-lateral stability is of crucial importance for reasons already discussed. However, the other degrees of freedom that make up the complex movement pattern of the patella must be considered. This includes rotation in the sagittal plane (that is: flexion-extension) and rotation and translation in the coronal plane [21]. During the initial 30° of tibiofemoral flexion there is a lag discrepancy between flexion occurring at the tibiofemoral joint and that of the patella due to the distal translation of the patella that occurs (10° of patellar flexion at 20° of tibiofemoral flexion). Beyond this, the patella rotates around the arc of the femoral articular geometry in the sagittal plane, with 55° of patellar flexion at 90° tibiofemoral flexion [14]. A femoral component with a low-profile anterior flange would enable this motion to occur smoothly and thus avoid the ‘catching’ or ‘snapping’ symptoms caused by a bulky anterior flange that forces the patella into extension during initial engagement.

In deep flexion femoral roll-back occurs and the distal end of the patella is raised out of the trochlear groove, bridging the intercondylar notch. Therefore the transition between the trochlear component and normal articular surface should be smooth, i.e. no step, to avoid ‘clunking’ during knee motion.

In patients with severe misalignment symptoms such as lateral patellar tilt, chronic lateral subluxation or lateral maltracking, the increased prominence of the lateral facet of the trochlear component may not be sufficient to correct the abnormality. Soft tissue reconstruction such as medial patellofemoral ligament reconstruction or even tibial tuberosity anteromedialisation may be necessary to correct the pathology.



A previous study [14] assessed the pre- and post-operative tracking kinematics in vitro of four patellofemoral arthroplasties: Avon, Blazina, Leicester (Corin Group, Cirencester, England) and Lubinus (see fig. 5A-D). The Avon and Leicester implants had tracking paths that most resembled the native knee. Unsurprisingly, the Blazina, had a comparatively linear pattern following engagement into the V-shaped trochlear groove. The Lubinus demonstrated an inconsistent pattern in some of the specimens. Assessment of these specimens identified abrupt changes in patellar tilt indicating the transition point as the patellar component moved from articulating with the trochlear component to the native articular surface.

Patellofemoral joint reaction forces occur as a result of the tension in the extensor mechanism. The force vectors in the sagittal plane are composed of the quadriceps and patellar tendon tensions (see fig. 6). The result is a force applied by the patella posteriorly onto the anterodistal femur. In the coronal plane this force has a lateral component caused by the Q angle. Due to the compressive nature of the force, loosening is unlikely to occur during point loading however, as the force moves position with increasing knee flexion, large forces will be applied closer to the proximal edge of the patella. Numerous fixations pegs cemented into the patellar bone will resist potential displacement of the component.

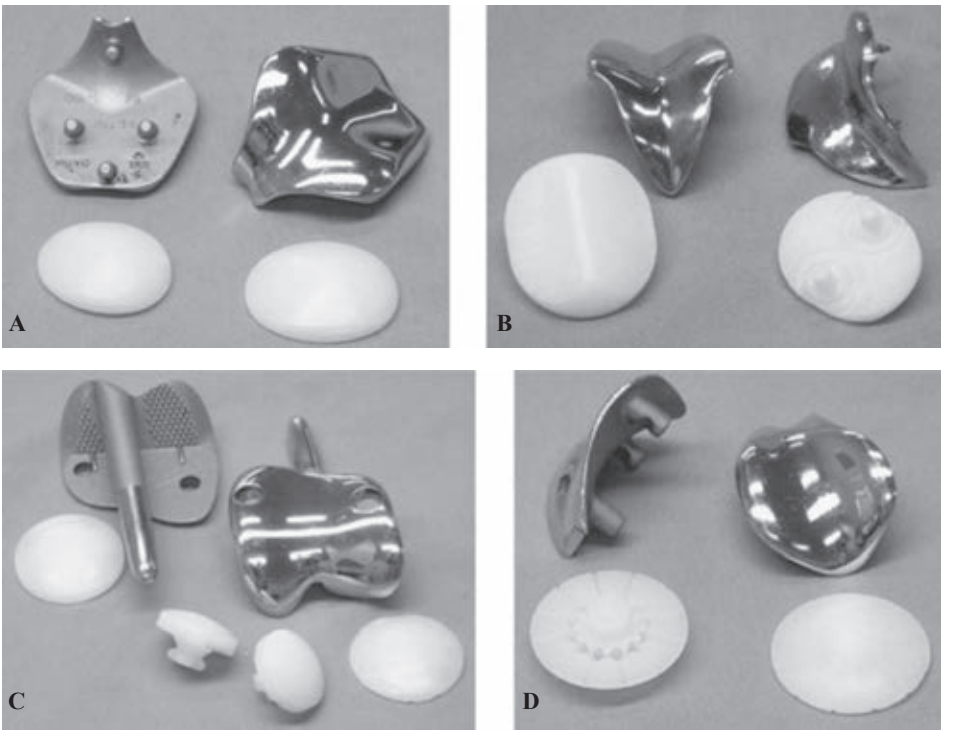


Fig. 5A-D : Patellofemoral joint prostheses: [A] Avon prosthesis, [B] Blazina prosthesis, [C] Leicester prosthesis and [D] Lubinus prosthesis.

Permission to use image granted by copyright owners Lippincott Williams & Wilkins. Amis AA, Senavongse W, Darcy P. Biomechanics of patellofemoral joint prostheses. Clin Orthop 2005; 436: 20-9.

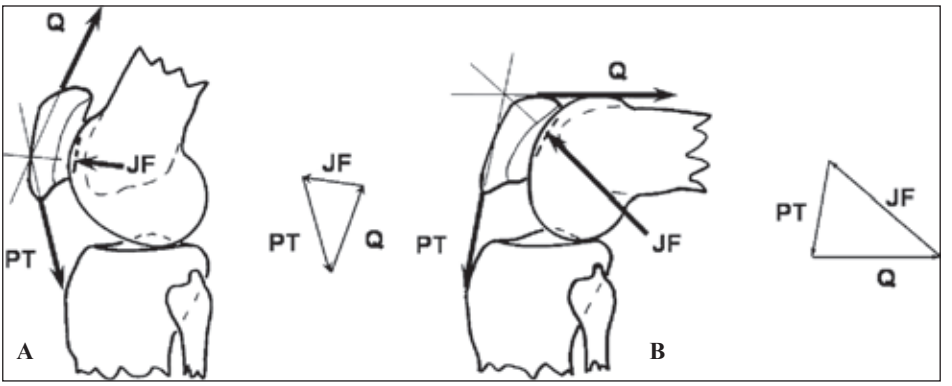


Fig. 6 : Forces acting on the patella in the sagittal plane. Quadriceps tension (Q), patellar tendon tension (PT) and joint force (JF). The JF moves proximally across the patella as the knee flexes, rising significantly with increase in knee flexion for the same PT.

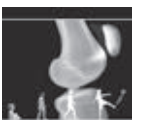
Permission to use image granted by copyright owners Lippincott Williams & Wilkins. Amis AA, Senavongse W, Darcy P. Biomechanics of patellofemoral joint prostheses. Clin Orthop 2005; 436: 20-9.

Trochlear Component

It is not uncommon for patients with isolated patellofemoral arthritis to have associated abnormal patellofemoral biomechanics. Trochlear dysplasia combined with an increased tibial tuberosity-trochlear groove distance [22] results in increased pressure on the lateral aspect of the patellofemoral joint. This predisposes the patella to sublux laterally. A trochlear prosthesis with a steep, relatively long prominent lateral facet would prevent the patella subluxing, ensure contact in full extension and engagement of the patella in early knee flexion. If the lateral facet is too prominent soft tissue impingement will occur, therefore a balance must be found between constraint and conformity.

In mid knee flexion, internal rotation of the tibia will have reduced the Q angle and consequently the lateral force, whilst the posterior force is increased due to the angle closing between the quadriceps and patellar tendon in the sagittal plane. During this range of movement the patellar component should be stable; a trochlear component with a single radius of curvature is sufficient to allow this motion to take place effectively. The trochlear component should fit flush over all aspects of contact with the distal femur, avoiding the need to place it in flexion or hyperextension, where it will catch the patella or impinge onto the anterior cruciate ligaments, respectively [23]. In deep knee flexion the patellar component articulates with the articular surfaces of the femoral condyles. The transition point from prosthesis to articular cartilage must be even to avoid any catching of the patella. All current prostheses extend to the intercondylar notch but not beyond. There is an assumption that the articular cartilage adjacent to the prosthesis is normal, however this is not true for all cases. A wider distal end may provide adequate coverage for the more extensive wear patterns but potentially at the expense of damage to the menisci when the knee is extended, and preventing the later addition of a unicompartmental arthroplasty in the presence of progression of tibiofemoral arthritis. This means that the patellar component must be compatible with the articular cartilage.

In the native knee the trochlea is asymmetrical. The lateral articular facet is approximately 50% larger than the medial to accommodate the higher load. It therefore seems logical that a femoral component should mimic this geometry, particularly when considering a significant number of patients will have a history of patellar instability with a tendency to track laterally.



Patellar Component

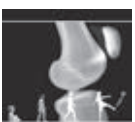
In broad terms there are two types of patellar component design in current use: the non-anatomical dome-shaped (axisymmetrical) 'button' and the conforming asymmetrical component which is either functional or anatomical.

The Avon and FPV both have conforming geometry. The most recent Avon patella component is anatomical, mimicking the seven facets of the natural patella. The design is based on the Kinemax patella and has a central crest offset towards the medial side by 3mm. The medial and lateral sides each have a superior, central and inferior facet. The medial side is shorter than the lateral, and so the medial edge is thicker than the lateral, due to the offset crest. This edge is smoothed to create an articular surface similar to the odd facet. In deep knee flexion the patella rotates internally and this surface articulates with the lateral aspect of the medial femoral condyle. In the original design the medial edge scored the medial condyle, hence the change. The designer recommends leaving a 1-2mm rim of cancellous bone so that a new rim of fibrous tissue (a 'patellar meniscus') can form and protect the component, and to use a smaller size thus reducing the degree of wear of the button by widening the fibrous tissue rim. The FPV patellar component is sided and facetted with an off-centre longitudinal ridge that becomes increasingly more medialised from proximal to distal. The base is oval shaped to minimise the uncovered resection surface of the patella. The two proposed advantages of such a design are the increased surface area compared to the dome shaped patellar component and the greater resistance to lateral subluxation. However, one could argue that the FPV patellar component needs to be more resistant against such a broad sulcus angle compared to the Avon. The main concern with these components is achieving the correct alignment. If fixed in the wrong orientation the component will not articulate in the desired fashion and the patient may become symptomatic.

The axisymmetric button does not need to be fixed in a particular rotation, therefore fixation is less challenging. However, contact pressures are known to be higher with an axisymmetric patellar component compared to a more anatomical bearing surface. Previous studies have shown that the contact stresses at the button-trochlear interface exceed the yield strength of the *ultra high molecular weight polyethylene* (UHMWPE) causing plastic deformation [24, 25]. *In vivo* some of the load may be transmitted to the adjacent fibrous tissue, so the contact force may actually be lower than previously measured *in-vitro*.

The Low Contact Stress (LCS) patellofemoral prosthesis (Depuy Orthopaedics, Warsaw, Indiana) had a modular two-part patellar component, consisting of a metal plate for bone fixation and a mobile polyethylene bearing [26]. The concept was that the patella would be self-aligning within the trochlear groove to enhance tracking. The early (two-year) results from an independent centre were discouraging in that 31% (17 out of 51) had already undergone revision of which only two were related to tibiofemoral disease progression [27]. This study reported a dismal 46% (95% CI 30 to 63) survival at three years (severe pain/revision endpoint). The high failure rate was most likely due to the modular patellar component. Multiple complications have been reported including dissociation of the polyethylene from the metal base and loss of mobility. Significant metallosis has also been found in a number of revised knees as a result of metal-on-metal articulation of the trochlea with the metal base of the patellar component when it became exposed at extremes of motion [27].

It is crucial that there is overlap of the patellar and trochlear articular surfaces in full knee extension. The amount of overlap should be greater than 30%; if it is less than 30% there is a risk of subluxation [28]. If this is not achieved, patella alta is created and the patella may not engage in the initial stages of knee flexion. Once flexion commences the patella becomes increasingly more engaged within the trochlear groove. At approximately 90° knee flexion the patella is most stable, beyond this it begins to internally rotate and the odd facet articulates with the lateral aspect of the medial femoral



condyle. In deeper flexion, the patella articulates with the femoral condyles with the risk of cartilage wear but in the presence of a 'patellar meniscus', it bears against the fibrous tissue rim of the patella and thus prevents polyethylene wear.

Material Science

The ideal trochlear component should be highly resistant to abrasive wear and have a low coefficient of friction. The Avon and FPV are both made from cobalt chrome (CoCr). The Journey PFJ consists of oxidised Zirconium (OXINIUM®; Smith & Nephew, Memphis, USA). This bearing material has been reported as nearly 5000 times more resistant to abrasion than CoCr [29]. OXINIUM® also has a significantly lower coefficient of friction against UHMWPE due to its more hydrophilic surface compared with cobalt chrome [30]. This means the synovial fluid has a greater propensity for the surface of the prosthesis and disperses evenly, and therefore acts as a more effective sliding lubricant. For these reasons, the manufacturers have suggested that resurfacing of the patella is optional. The use of UHMWPE for patellar components is standard practice. Issues of wear are related more to the tracking motion of the patellar component rather than the yield strength. Despite advances in materials for implants, there are no clinical results of PFA which better a polyethylene patellar component articulating against a CoCr trochlea, with both components fixed using PMMA bone cement.

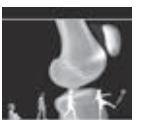
PATIENT SELECTION

Whilst considering the ideal PFA, there must be a degree of deliberation over the ideal patient. Patellofemoral arthroplasty should not be carried out in patients with inflammatory joint arthropathy or chondrocalcinosis. A popular belief growing amongst some surgeons is to reserve this procedure for patients with trochlear dysplasia with or without a degree of patellar tilt or subluxation. A study supporting this principle showed that PFA was most effective in patients with trochlear dysplasia [31]. These findings were further corroborated by a later study [28] which found that none of the patients, at a mean of 7.1 years follow-up, who had patellofemoral arthritis secondary to trochlear dysplasia, required revision surgery for progression of tibiofemoral arthritis compared to 17% (5 out of 30 knees) of those without this underlying diagnosis. Those with severe misalignment due to an abnormal Q angle or maltracking will require additional procedure(s) at the time of PFA to ensure correct patellofemoral biomechanics are restored. The complex nature of such combined surgery requires a surgeon experienced in both arthroplasty and patellar instability.

CONCLUSION

The ideal PFA consists of a trochlear component that is able to engage the patella within the trochlear groove during the full range of flexion-extension without over-constraining the patella in knee extension, and also possess a sagittal shape congruent with the distal femur. The patellar component design is dependent to a degree on the geometry of the trochlear component. There are advantages to both axisymmetric and conforming designs, but the ease of use of an axisymmetrical button may be outweighed by the superior bearing performance of a more conforming design in a younger patient. Modular components should be avoided, given the significant complication rate. To ensure good fixation both components should have an adequate number of pegs appropriately positioned to optimise fixation and this should be consistent between sizes to so the best fit can be achieved.

The ultimate goal is for an improved clinical outcome including greater patient satisfaction. Ideally, this should be comparable to total knee arthroplasty while bearing in mind that for some patients, PFA may be "bridging surgery" to avoid total knee arthroplasty early in life. For others it may be a permanent solution. Tibiofemoral degeneration should be the most common reason for revision surgery.



LITERATURE

[1] BLAZINA ME, FOX JM, DELPIZZO W, *et al.* Patellofemoral replacement. Clin Orthop 1979;144:98-102.

[2] DE WINTER W, FEITH R, VAN LOON CJM. The Richards type II patellofemoral arthroplasty: 26 cases followed for 1-20 years. *Acta Orthop Scand 2001;72:487-90.*

[3] SMITH A, PECKETT W, BUTLER-MANUEL P, *et al.* Treatment of patellofemoral arthritis using the Lubinus patellofemoral arthroplasty: a retrospective review. *Knee 2002;9:27-30.*

[4] TAURO B, ACKROYD CE, NEWMAN JH, *et al.* The Lubinus patellofemoral arthroplasty: a five- to ten-year prospective study. *J Bone Joint Surg [Br] 2001;83-B:696-701.*

[5] KOOIJMAN HJ, DRIESSEN A, VAN HORN JR. Long-term results of patellofemoral arthroplasty: a report of 56 arthroplasties with 17 years of follow-up. *J Bone Joint Surg [Br] 2003;85-B:836-40.*

[6] LONNER JH. Patellofemoral arthroplasty: pros, cons, and design considerations. *Clin Orthop 2004;428:158-65.*

[7] HENDRIX MRG, ACKROYD CE, LONNER JH. Revision patellofemoral arthroplasty: three- to seven-year follow-up. *J Arthroplasty 2008;23:977-83.*

[8] WALKER PS. Design of Kinemax total knee replacement bearing surfaces. *Acta Orthop Belg 1991;57 Suppl II:108-13.*

[9] HSU HP, WALKER PS. Wear and deformation of patellar components in total knee arthroplasty. *Clin Orthop 1989;246:260-66.*

[10] WRIGHT J, EWALD FC, WALKER PS, *et al*. Total knee arthroplasty with the kinematic prosthesis. Results after five to nine years: a follow-up note. *J Bone Joint Surg [Am] 1990;72-A:1003-09.*

[11] ODUMENYA M, COSTA ML, PARSONS N, *et al.* The Avon patellofemoral joint replacement: five year results from an independent centre. *J Bone Joint Surg [Br] 2010;92-B:56-60.*

[12] ACKROYD CE, NEWMAN JH, EVANS R, *et al.* The Avon patellofemoral arthroplasty: five-year survivorship and functional results. *J Bone Joint Surg [Br] 2007;89-B:310-15.*

[13] LONNER JH. Patellofemoral arthroplasty: The impact of design on outcomes. *Orthop Clin N Am 2008;39:347-54.*

[14] AMIS AA, SENAVONGSE W, DARCY P. Biomechanics of patellofemoral joint prostheses. *Clin Orthop 2005;436:20-29.*

[15] BEITZEL K, SCHÖTTLE PB, COTIC M, *et al*. Prospective clinical and radiological two-year results after patellofemoral arthroplasty using an implant with an asymmetric trochlea design. *Knee Surg Traumatol Arthrosc May 2012. [Epub ahead of print].*

[16] MOFIDI A, BAJADA S, HOLT MD, *et al.* Functional relevance of patellofemoral thickness before and after unicompartmental patellofemoral replacement. *Knee 2012;19:180-84.*

[17] National Joint Registry of England and Wales 8th Annual Report 2011.

[18] LASKIN RS, VAN STEIJN M. Total knee replacement for patients with patellofemoral arthritis. *Clin Orthop 1999;367:89-95.*

[19] MONT MA, HAAS S, MULLICK T, *et al.* Total knee arthroplasty for patellofemoral arthritis. *J Bone Joint Surg [Am] 2002;84-A:1977-81.*

[20] ACKROYD CE. Development and early results of a new patellofemoral arthroplasty. *Clin Orthop 2005;436:7-13.*

[21] RHOADS D, NOBEL P, REUBEN J, *et al.* The effect of femoral component position on patellar tracking after total knee arthroplasty. *Clin Orthop 1990;260:43-51.*

[22] SCHÖTTLE PB, FUCENTESE SF, PFIRRMANN C, et al. Trochleaplasty for patellar instability due to trochlear dysplasia: a minimum 2-year clinical and radiological follow-up of 19 knees. *Acta Orthop 2005;76:693-8.*

[23] BOARD TN, MAHMOOD A, RYAN WG, *et al.* The Lubinus patellofemoral arthroplasty: a series of 17 cases. *Arch Orthop Trauma Surg 2004;124:285-7.*

[24] McNAMARA JL, COLLIER JP, MAYOR M, *et al.* A comparison of contact pressures in tibial and patellar total knee components before and after service in vivo. *Clin Orthop 1994;299:104-11.*

[25] TAKEUCHI T, LATHI VK, KHAN AM *et al*. Patellofemoral contact pressures exceed the compressive yield strength of UHMWPE in total knee arthroplasties. *J Arthroplasty 1995;10:363-8.*

[26] MARCACCI M, ZAFFAGNINI S, LO PRESTI M, *et al*. Treatment of chronic patellar dislocation with a modified Elmslie-Trillat procedure. *Arch Orthop Trauma Surg 2004;124:250-7.*

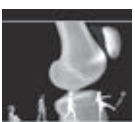
[27] CHARALAMBOUS CP, ABIDDIN Z, MILLS SP, *et al*. The low contact stress patellofemoral replacement: high early failure rate. *J Bone Joint Surg [Br] 2011;93-B:484-9.*

[28] MONK AP, DOLL HA, GIBBONS CL, *et al.* The path-anatomy of patellofemoral subluxation. *J Bone Joint Surg [Br] 2011;93-B:1341-7.*

[29] HUNTER G, LONG M. Abrasive wear of oxidized Zr-2.5Nb, CoCrMo and Ti-6Al-4V against bone cement. *In: Transactions of the Sixth World Biomaterials Congress. Minneapolis: Society For Biomaterials, USA, 2000:835.*

[30] DAVIDSON J, ASGLAN C, MISHRA A, *et al.,* editors. Zirconia (ZrO2)-coated zirconium-2.5Nb alloy for prosthetic knee applications. *Bioceramics Vol. 5: Kyoto: Kobunshi Kankokai, 1992:389-401.*

[31] ARGENSON JNA, GUILLAUME JM, AUBANIAC JM. Is there a place for patellofemoral arthroplasty? *Clin Orthop 1995-321:162-7.*



IMPORTANCE AND RADIOGRAPHIC IDENTIFICATION OF THE FEMORAL INSERTION IN MEDIAL PATELLOFEMORAL LIGAMENT RECONSTRUCTION

P.B. SCHOETTLE

Since biomechanical studies have shown the MPFL as the main restraint against lateral patellar displacement [4, 6], MPFL-reconstruction became a widely accepted technique to restore normal patellar tracking and stability [5, 9, 10]. Although the clinical outcome studies after MPFL reconstruction report promising results related to stability, there are some cases, reporting about increased pain or loss of function postoperatively [8, 11, 13, 15, 16]. Main reason therefore seems to be a non-anatomical reconstruction, since the importance of correct graft positioning for ligament reconstruction has been recognized already in 1938 by Palmer [12], and its influence on the clinical outcome is well known in ACL reconstruction [1]. While the patellar insertion, where the medial facet can be prepared completely, even with relatively small skin incisions, the femoral insertion, which is described to be close to the medial epicondlye and the adductor tubercle can be difficult to palpate, not only when covered by soft tissue, but also in skinny patients.

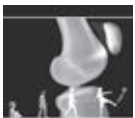
Since biomechnical studies [2, 7] have shown the influence of a non-anatomical femoral insertion onto the patelllofemoral pressure, this insertion became the key point in MPFL reconstruction.

The reason for this estimated increase of medial patellofemoral pressure is founded by the idea that a too proximal fixation point would lead to an increased distance to the patella, when the knee flexes, and vice versa for a too posterior attachment [2], a proper tunnel placement is necessary to restore physiological kinematics and pressure postoperatively.

However, although numerous studies have focused the MPFL anatomy in preparation studies, guidelines for a intraoperative use for minimal invasive surgery is missing. Compared to ACL reconstruction, where radiographic guidelines for proper tunnel placement were given to improve clinical results [3], same guidelines are mandatory for an anatomical MPFL reconstruction, achieving not only stability, but also full range of motion/function without presence of patellofemoral pain by increasing the retropatellar pressure [2, 7, 15].

In this work, a proper radiographic landmark is demonstrated to identify the anatomical femoral MPFL insertion intraoperatively or to use it as a postoperative control.

To initially verify this point, a number of eight cadavers have been prepared and the center of the femoral MPFL insertion have been identified and marked with a radiodense ball with a small diameter. Then, a straight lateral view, with both posterior condyles projected in the same plane, were taken and the position of



the ball was determined. When realizing that all points were situated in the same area, the following reference lines were determined as orientation: a first line in extension of the posterior femoral cortex towards distal to measure the anteroposterior position *(line 1)*, a second line intersecting the contact of the posterior femoral condyle with the posterior cortex *(line 2)*, and a third line intersecting the most posterior point of the Blumensaat line *(line 3)*, both perpendicular to line 1, measuring the proximo-distal position (fig. 1 and 2) [14].

Anterior-posterior position: The insertion marker was located anterior to the posterior cortical extension line in nearly all specimens, with a mean location of 1.3 ± 1.7mm anterior to line 1.

Proximal-distal position: In all specimens the marker ball was midway between line 2 and line 3. The mean location was 2.5mm ± 0.8 distal to line 2. However, since all points were within 5mm of each other, it was possible to draw a 5mm diameter circle containing all marker locations.

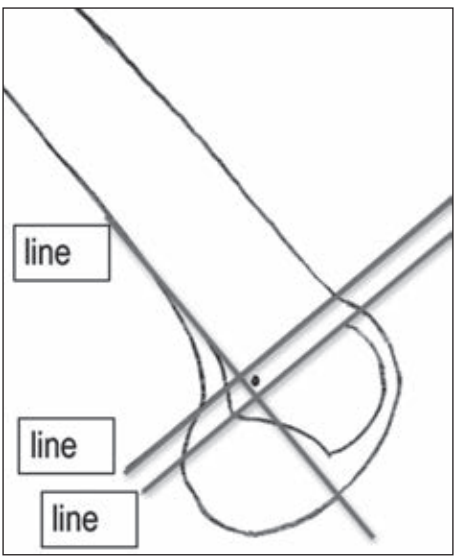


Fig. 1: Schematic drawing of a distal femur with the reference lines, seen in a straight lateral view. The circle is identifying the region, where an insertion would be anatomical.

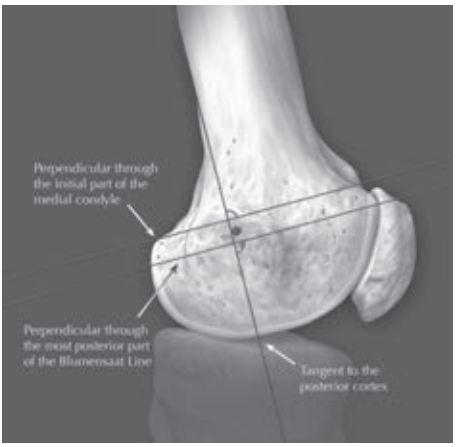


Fig. 2: Anatomical drawing with the same reference lines to show the relation to the gross anatomy, i.e. the medial epicondyle and the adductor tubercle.

Although one can say that the mean position we determined is not valid for every knee as the distance of the single points is up to 5mm in the proximal-distal direction, recent studies have shown 15 that a distance of 5mm or less from the anatomical femoral MPFL insertion is not changing the MPFL isometry. Therefore, it is recommended to use this radiographic landmark intraoperatively due to the following benefits.

For intraoperative use, it is recommended to first prepare the laminar patellar insertion and to identify the anatomical MPFL layer, just in between the joint capsula and the vastus medialis obliquus muscle. A clamp is inserted into this layer down to the femur, where the tubercle and epicondyle is palpated. In this area, a little skin incision of 3 millimeters is performed and a guide wire is drilled into the medial distal femur, in the area of the bony landmarks, until it has a sechure fixation. Afterwords, a cannulated drill (according to the graft diameter with a minimum of 5mm) is inserted over the guide wire down to the bone, and the straight lateral view is taken with the use of a fluoroscope. The drilled insertion point is exactly there, where the cannulated drill attaches to the bone. As described before, this point should be anterior to the elongation of the posterior cortex, distal to the origin of the posterior medial condyle and proximal to the most posterior point of the Blumesaat line

(fig. 3). If this point is deviating obviously or the cannulated drill is inside the anatomical insertion area only by 50% (fig. 4), the guide wire has to be removed and reinserted towards the desired direction, until an anatomical positioning is achieved.

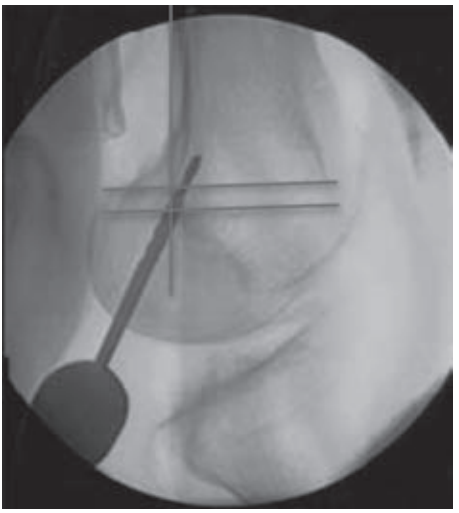


Fig. 3: Intraoperative view by fluoroscope at the correct position. The insertion point is identified at the point, where the cannulated drill is attaching the bone (anterior to the posterior condyle line, distal to the perpendicular through the inition of the medial condyle and proximal to the most posterior point of the Blumensaat line).

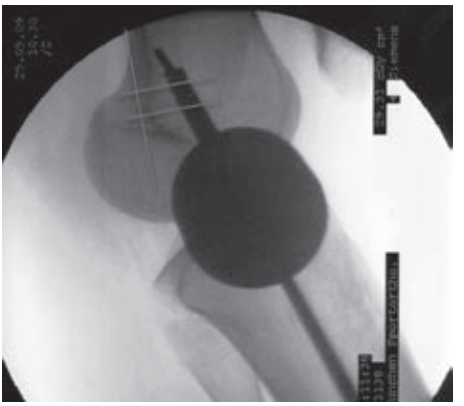


Fig. 4: Non anatomical position: the cannulated drill attaches the bone too far anterior and proximal

Besides the possibility of a very small skin incision at the femoral side with the maximal length of the screw diameter, used for the femoral fixation, the femoral insertion becomes reproducible and the risk of a non-anatomical reconstruction can be minimized drastically. Especially in patients, where the medial aspect of the distal femur is covered with soft tissue, a radiological identification of the insertion point is highly recommended and is simplifying the operation.

LITERATURE

[1] ALLEN CR, GIFFIN JR, HARNER CD. Revision anterior cruciate ligament reconstruction. *Orthop Clin North Am, 34(1): 79-98, 2003.*

[2] AMIS AA, FIRER P, MOUNTNEY J, SENAVONGSE W, THOMAS NP. Anatomy and biomechanics of the medial patellofemoral ligament. *Knee, 10(3): 215-20, 2003.*

[3] BERNARD M, HERTEL P, HORNUNG H, CIERPINSKI T. Femoral insertion of the ACL. Radiographic quadrant method. *Am J Knee Surg, 10(1): 14-21; discussion 21-2, 1997.*

[4] CONLAN T, GARTH WP Jr, LEMONS JE. Evaluation of the medial soft-tissue restraints of the extensor mechanism of the knee. *J Bone Joint Surg Am, 75(5): 682-93, 1993.*

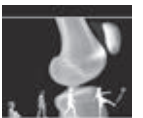
[5] DAVIS DK, FITHIAN DC. Techniques of medial retinacular repair and reconstruction. *Clin Orthop Relat Res, (402): 38-52, 2002.*

[6] DESIO SM, BURKS RT, BACHUS KN. Soft tissue restraints to lateral patellar translation in the human knee. *Am J Sports Med, 26(1): 59-65, 1998.*

[7] ELIAS JJ, COSGAREA AJ. Technical Errors During Medial Patellofemoral Ligament Reconstruction Could Overload Medial Patellofemoral Cartilage: A Computational Analysis. *Am J Sports Med, 2006.*

[8] ELLERA GOMES JL, STIGLER MARCZYK LR, CESAR DE CESAR P, JUNGBLUT CF. Medial patellofemoral ligament reconstruction with semitendinosus autograft for chronic patellar instability: a follow-up study. *Arthroscopy, 20(2): 147-51, 2004.*

[9] HAUTAMAA PV, FITHIAN DC, KAUFMAN KR, DANIEL DM, POHLMEYER AM. Medial soft tissue restraints in lateral patellar instability and repair. *Clin Orthop, (349): 174-82, 1998.*



[10] NOMURA E, HORIUCHI Y, KIHARA M. Medial patellofemoral ligament restraint in lateral patellar translation and reconstruction. *Knee, 7(2): 121-7, 2000.*

[11] NOMURA E, INOUE M. Surgical technique and rationale for medial patellofemoral ligament reconstruction for recurrent patellar dislocation. *Arthroscopy, 19(5): E47, 2003.*

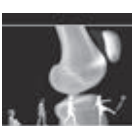
[12] PALMER I. Om injuries to the ligaments of the ankle joint. A clinical study. *Acta Chir Scand, 53 [Suppl](53 [Suppl]): 1-282, 1938.*

[13] SCHOTTLE PB, FUCENTESE SF, ROMERO J. Clinical and radiological outcome of medial patellofemoral ligament reconstruction with a semitendinosus autograft for patella instability. *Knee Surg Sports Traumatol Arthrosc, 13(7): 516-21, 2005.*

[14] SCHOTTLE PB, SCHMELING A, ROSENSTIEL N, WEILER A. Radiographic landmarks for femoral tunnel placement in medial patellofemoral ligament reconstruction. *Am J Sports Med, 35(5): 801-4, 2007.*

[15] SMIRK C, MORRIS H. The anatomy and reconstruction of the medial patellofemoral ligament. *Knee, 10(3): 221-7, 2003.*

[16] STEENSEN RN, DOPIRAK RM, MCDONALD WG 3rd The anatomy and isometry of the medial patellofemoral ligament: implications for reconstruction. *Am J Sports Med, 32(6): 1509-13, 2004.*



THE LONG-TERM RESULTS OF PATELLO-FERMORAL ARTHROPLASTY

S. PARRATTE, M. OLLIVIER, X. FLECHER, J.M. AUBANIAC, J.N. ARGENSON

INTRODUCTION

Isolated osteoarthritis of the patellofemoral joint can be a cause of severe anterior knee pain and may limit daily activities such as standing up from a chair or climbing stairs. Although physical therapy and other nonoperative methods have a role in the treatment, surgery may be necessary in patients with persistent knee pain and several surgical options have been proposed. These include debridement of the patellofemoral joint, elevation of the anterior tibial tubercle, spongialization of the patella, prosthetic resurfacing of the patella alone, or patellectomy. Total knee arthroplasty (TKA) has also been reported for patients with arthritis confined to the patellofemoral joint.

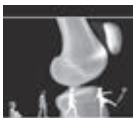
Patellofemoral arthroplasty (PFA), defined as resurfacing both sides of the patellofemoral joint, has been proposed as a treatment option. There have been several studies published on PFA during the past 25 years and the reported results at mid-term follow-ups are variable with favorable results ranging from 72 to 85%. We published our experience with PFA in 1995, with 84% of satisfactory results at mid-term follow-up (range 2-10 years). In this paper, the long terms results will be presented as well as the quality of the outcomes according to the pre-operative diagnosis.

MATERIAL AND METHODS

We retrospectively reviewed 66 consecutive patients (66 knees) who had isolated unilateral PFA between 1972 and 1990. Nine patients died for causes unrelated to the knee surgery and all of these patients had well functioning PFA implanted knees at the time of death. The remaining 57 patients were evaluated at a mean follow-up of 16.2 years (range, 12-20 years). There were 31 women and 26 men studied who had a mean age at operation of 57 years (range, 21-82 years). The etiologies of osteoarthritis were: instability with a history of patellar dislocation (21 patients), and posttraumatic osteoarthritis primarily attributable to patellar fractures (18 patients), and primary osteoarthritis of the patellofemoral joint (18 patients).

At operation all patients had clinical and radiographic evidence of severe osteoarthritis of the patellofemoral joint with preserved medial and lateral tibiofemoral joints. Eighteen patients had a previous surgical procedure on the involved joint, which included 12 cases of patellar open reduction and internal fixation and six cases of elevation of the anterior tibial tubercle.

The indication for PFA was failure for previous nonoperative or surgical treatment.



All patients receved an autocentric patello-femoral prosthesis (DePuy, Warsaw, IN) with a cemented polyethylene (PE) patella and a Co-Cr femoral component. The design characteristics and the surgical technique regarding this device have been described in a previous paper. Concomitant procedures done at the time of PFA included eight osteotomies (three cases of upper tibial osteotomy for varus correction, and five cases of lower femoral osteotomy for valgus correction aiming in every cases for neutral alignment) and five realignments of the anterior tibial tuberosity for medialization.

Clinical knee scores were calculated using the Knee Society Score, and follow-up radiographs were assessed for signs of loosening and complete degeneration of the tibiofemoral joint. The clinical and radiographic evaluations were done by a single observer independent from the operating surgeons. Revision of the PFA for any reason was considered an end point for survival.

RESULTS

Degeneration of the unreplaced tibiofemoral joint was the most frequent cause of failure of PFA. Revision surgery was done in 14 of the 57 living patients for progression of osteoarthritis in the tibiofemoral compartment and the PFAs were converted to TKA at an average of 7.3 years (range, 1-12 years) after the implantation of the patellofemoral prostheses.

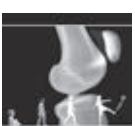
Progressive degeneration was noted mainly in the primary osteoarthritis etiologic group (8 of 18 patients) compared with the two other groups (three of 21 patients in the instability group and three of 18 patients in the post-traumatic group). Eleven patients were revised for loosening after an average follow-up of 4.5 years (range, 1-10 years) with an equal occurrence in the three groups. All the revisions for loosening were done on the femoral component except in one patient who had a patellectomy for patellar fracture. Among these 11 revisions for loosening three were caused by infection, in patients who had at least one previous surgery. Early aseptic loosening in three patients with uncemented femoral components were treated successfully with insertion of a cemented femoral component. The remaining four patients who had revision surgery for loosening of their PFA components had conversion to TKA. The knees of six patients were revised for stiffness; two were treated by manipulation under anesthesia and four (these patients were part of the posttraumatic etiologic group) required conversion to TKA. Additionally, five patients required a secondary lateral release with resection of the lateral patellar facet (which was not done routinely at the beginning of our experience) and had good results. The cumulative survival rate for the all group, including the 9 patients died of unrelated causes, was 58% at 16 years [95% confidence intervals (CI)].

Most patients had a substantial and persistent improvement in knee function for patients affected by isolated patellofemoral arthritis and treated by PFA. For the 29 of 57 patients (51%) who still had their original PFA prostheses in place at the time of follow-up the mean Knee Society pain score had improved from 53.1 points preoperatively (range, 43-70 points) to 78.5 points (range, 60-100 points) at the final follow-up. The mean Knee Society function score improved from 40.6 points (range, 10-80 points) preoperatively to 81.2 points (range, 40-100 points) at final follow-up. Twenty-one patients had no complaints of pain whereas 11 complained of moderate pain.

Twenty patients had a range of motion (ROM) greater than 105°, 10 had a ROM between 90° and 105°, and two had a ROM less than 90°. The radiographic analysis of the knees with the PFA still in place showed no evidence of mechanical failure or loosening and no detrimental degeneration of the tibiofemoral joint.

DISCUSSION

The long-term evaluation of PFA presented in this study showed a high rate of conversion to TKA after a 16 years' average followup. The most frequent reason for failure was progression of arthritis in the tibiofemoral joint, occurring

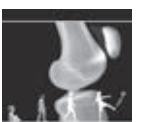


mainly in the primary osteoarthritis etiologic group. Despite the absence of statistical group-to-group comparison, which may constitute one of the limitations of this study, we think our best results were seen in patients with a pre-operative diagnosis of instability and corrected alignment of the extensor mechanism. The clinical evaluation presented in this study also confirmed that PFA can provide a significant improvement in knee function for patients affected by isolated patellofemoral arthritis.

This long-term study of 12 to 20 years showed that 29 out of 66 patients implanted with PFA required conversion to TKA. In the only available study with a comparative long-term follow-up published by Kooijman *et al.*, the authors also concluded that patellofemoral unicompartmental arthroplasty has a high revision rate. In that study of 45 knees with an average follow-up of 15.6 years the authors noted seven revisions related to the arthroplasty and 12 revisions for tibiofemoral osteoarthritis progression. Authors of both studies show that progressive osteoarthritis in the unreplaced tibiofemoral compartment was the major cause of failure in knees implanted with PFA.

Progression of osteoarthritis in the unreplaced femorotibial compartment also was also noted. In our study we did try to identify the etiology of the patello-femoral degeneration and we found that this progression of osteoarthritis occurred mainly in the group of primary patellofemoral arthritis. Patients with a history of patellar instability or patellar fracture did not tend to have progressive osteoarthritis changes in the tibiofemoral joint. In patients with primary patellofemoral osteoarthritis, careful preoperative radiographs should determine whether there is tibiofemoral malalignment. If significant frontal plane malalignment is present, this should either be corrected or be considered a relative contraindication to PFA. In this study, we think our best results were seen in patients with a neutral or slightly valgus mechanical axis and none of the patients with concomitant osteotomy had revision for tibiofemoral degeneration. The need for lateral retinacular release or realignment of the extensor mechanism at the time of PFA also has been noted in the studies of Tauro *et al.* and Krajca-Radcliffe and Coker. Cartier *et al.* and Mertl *et al.* used the elevation of the anterior tibial tubercle as part of the procedure. We think that this not required for most of the patient and the problem is most of the time on the femoral side and this issue can be addressed without any action on the anterior tibial tuberosity, specially with the new generation of implants. During surgery the importance of precise alignment of the femoral component, avoiding flexion and internal rotation, may also contribute to achieve correct patella tracking as noted previously.

Regarding the number of secondary procedures after PFA compared with the outcome after TKA for the treatment of isolated patellofemoral osteoarthritis, we favor TKA as the first option in older patients. Laskin *et al.* and Parvizi *et al.* reported good results after TKA for isolated patellofemoral arthritis in patients averaging 67 or 70 years old, but they also noted a high incidence of lateral retinacular release or extensor mechanism realignment procedures and the number of patients with postoperative patellar tilt found in this group of patients. Mont *et al.* noted only one poor result after 30 cases of TKA done for patellofemoral arthritis in patients averaging 73 years old. Despite the high rate of conversion to TKA at a 12- to-20 year followup presented in this study we consider PFA to be a valuable option for middle-aged carefully selected patients. The ideal candidate should have a knee with isolated patellofemoral arthritis and no important frontal tibiofemoral deformity. When the arthritis is secondary to patellofemoral instability, attention should be taken to obtain a correct alignment of the extensor mechanism. For patients younger than 60 years, we think PFA can be a useful "temporary" procedure that easily can be converted to TKA if needed. We believe all the components of the PFA should be cemented and the PE patellar button should be compatible with conventional TKA designs. Additionally, it is not irreversible like patellectomy and it is a more nonoperative option for younger patients than TKA.



LITERATURE

[1] AGLIETTI P, INSALL JN, WALKER PS, TRENT P. A new patella prosthesis: Design and application. *Clin Orthop Rel Res 107:175-87, 1975.*

[2] ARCIERO RA, TOOMEY HE: Patellofemoral arthroplasty: A three tonine year follow-up study. *Clin Orthop Relat Res 236:60-71, 1988.*

[3] ARGENSON JN, GUILLAUME JM, AUBANIAC JM. Is there a place for patellofemoral arthroplasty? *Clin Orthop Relat Res 321:162-7,1995.*

[4] ARGENSON JN, FLECHER X, PARRATTE S, AUBANIAC JM. Patellofemoral arthroplasty: an update. *Clin Orthop Relat Res 2005; 440: 50-3.*

[5] BLAZINA ME, FOX JM, DEL PIZZO W, BRONKHIM B, IVEY FM. Patellofemoral replacement. *Clin Orthop Relat Res 144:98-102, 1979.*

[6] CARTIER P, SANOUILLER JL, GRELSAMER R. Patellofemoral arthroplasty. 2-12 year follow-up study. *J Arthroplasty 5:49-54, 1990.*

[7] DE CLOEDT P, LEGAYE J, LOKIETEK W. Femoropatellar prosthesis: A retrospective study of 45 consecutive cases with a followup of 3-12 years. *Acta Orthop Belg 65:170-5, 1999.*

[8] FICAT RP, FICAT C, GEDEON P, TOUSSAINT JB. Spongialization: A view treatment for diseased patellae. *Clin Orthop Relat Res 144:74-83,1979.*

[9] INSALL JN. Intra-articular surgery for degenerative arthritis of the knee: A report of the work of the late K.H. Pridie. *J Bone Joint Surg 49B:211-28, 1967.*

[10] INSALL JN, DORR LD, SCOTT RD, SCOTT WN. Rationale of the Knee Society clinical rating system. *Clin Orthop Relat Res 248:13-14, 1989.*

[11] KOOIJMAN HJ, DRIESSEN APPM, VAN HORN JR. Long-term results of patellofemoral arthroplasty: A report of 56 arthroplasties with 17 years of follow-up. *J Bone Joint Surg 85B:836-40, 2003.*

[12] KRAJCA-RADCLIFFE JB, COKER TP. Patellofemoral arthroplasty. A 2- to 18-year followup study. *Clin Orthop Relat Res 330:143-151, 1996.*

[13] LASKIN RS, VAN STEIJN M. Total knee replacement for patients with patellofemoral arthritis. *Clin Orthop Relat Res 367:89-95, 1999.*

[14] LUBINUS HH. Patella glide bearing total replacement. *Orthopedics 2:119-27, 1979.*

[15] MAQUET P. Advancement of the tibial tuberosity. *Clin Orthop Relat Res 115: 225-30, 1976.*

[16] McKEEVER DC. Patellar prosthesis. *J Bone Joint Surg 37A:1074.*

[17] MERTL P, VAN FT, BONHOMME P, VIVES P. Femoropatellar osteoarthritis treated by prosthesis. Retrospective study of 50 implants. *Rev Chir Orthop Reparatrice Appar Mot 83:712-18, 1997.*

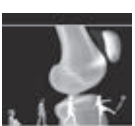
[18] MONT MA, HAAS S, MULLICK T, HUNGERFORD DS. Total knee arthroplasty for patellofemoral arthritis. *J Bone Joint Surg 84A:11: 1977-81, 2002.*

[19] PARVIZI J, STUART MJ, PAGNANO MW, HANSSEN AD. Total knee arthroplasty in patients with isolated patellofemoral arthritis. *Clin Orthop Relat Res 392:147-52, 2001.*

[20] PICKETT JC, STOLL DA. Patellaplasty or patellectomy? *Clin Orthop Relat Res 144: 103-9, 1979.*

[21] TAURO B, ACKROYD CE, NEWMAN JH, SHAH NA. The Lubinus patellofemoral arthroplasty: A five- to ten-year prospective study. *J Bone Surg 83B:696-701, 2001.*

[22] WITVOET J. L'état actuel des prothèses fémoropatellaires. In: Cahiers d'enseignement de la Sofcot. Vol 46. *Paris, Expansion Scientifique Française 79-92, 1994.*



IS PATELLOFEMORAL OSTEOARTHRITIS A PROBLEM FOR UNICOMPARTMENTAL KNEE ARTHROPLASTY?

E. GANCEL, R.A. MAGNUSSEN, F. TROUILLET, S. LUSTIG, E. SERVIEN, P. NEYRET

INTRODUCTION

Osteoarthritis isolated to one compartment of the knee is relatively common. Unicompartmental knee arthroplasty (UKA) is a viable treatment option in these patients, providing durable pain relief and functional improvement [3, 4, 23, 27, 35]. Advantages of UKA over total knee arthroplasty (TKA) include less pain, more rapid functional recovery, and the retention of both cruciate ligaments leading to more normal gait patterns [1, 13, 34, 38].

Because of the advantages noted above, it is desirable to extend the indications of UKA to include patients with disease affecting the patellofemoral joint. Although some authors have described patellofemoral joint involvement as a contraindication to UKA [21, 36] several recent studies have demonstrated no adverse effects of patellofemoral articular cartilage loss on outcomes, particularly when the medial facet is involved [5, 14, 19, 30]. However, some authors have suggested that lateral facet involvement portends worse outcomes, particularly in cases on lateral patellar grooving or bone loss [5, 6].

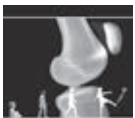
Patellofemoral degenerative change has been shown to be a source of anterior knee pain in patients with normal tibiofemoral joints [11]. Because the lateral patellar facet is the most frequent location of patellofemoral osteoarthritis [18], several authors have reported partial lateral patellar facetectomy as a treatment option. Reported results have generally been good, with improved pain and function at both short- and medium-term follow-up [25, 31, 39]. The majority of treatment failures were related to related to the development and progression of tibiofemoral osteoarthritis [25, 31, 39].

We hypothesize that simultaneous lateral UKA and lateral patellar facetectomy provides durable pain relief and functional improvement in a patient population with degenerative disease of lateral tibiofemoral compartment and the lateral patellofemoral joint.

MATERIALS AND METHODS

Patient Population

Between January 2004 and May 2008, one hundred and thirty-two UKAs were performed at our institution, including 77 medial UKAs and 55 lateral UKAs. Patients with less than 3 years follow-up were excluded. Thirteen UKAs (one medial and twelve lateral) were performed in association with partial lateral patellar facetectomy in thirteen female patients with degenerative changes in one tibiofemoral



compartment and the lateral patellofemoral joint. We interested only by the lateral compartment. The patient who received a medial UKA was also excluded. Another patient received a lateral UKA after a medial UKA on the same knee. In order to obtain the most homogeneous group, this patient was brought of the study. Eleven patients (six right knees and five left knees) form the study group. The average age at the time of the UKA was 66.7 years (range: 49 to 79 years). The mean patient weight was 62.7 kg (range: 49 to 80). The mean body mass index was 23.9 kg/m^2 (range 19.1 to 29.3 kg/m^2).

Surgical Indications

Candidates for UKA demonstrated isolated lateral compartment narrowing with complete or near complete joint space loss. Patients with a coronal plane deformity greater 14 degrees of knee valgus were excluded along with patients in whom a stress radiograph did not demonstrate reductibility of any coronal plane deformity. The integrity of the anterior cruciate and medial collateral ligaments were verified clinically and radiographically. Finally, patients were required to have at least 90 degrees of flexion and an extension deficit of less than 10 degrees. Weight alone was not considered an absolute contraindication.

Partial lateral patellar facetectomy performed concurrently in patient with: **1)** objective evidence of lateral patellofemoral degenerative disease with complete joint space loss, **2)** localized lateral patellar tenderness on physical examination. Patients with severe medial or central patellofemoral degeneration or isolated patellofemoral articular cartilage defects were excluded. Pre-operative radiographs and International Knee Society (IKS) outcome scores were obtained for all patients [17].

Prosthesis

The HLS Uni Evolution (Tornier, Grenoble, France) was utilized in all patients. The femoral implant is symmetric and made from cobalt chrome. This tibial component is polyethylene without a baseplate.

Operative Technique

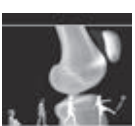
All operations were performed by the senior author. The partial lateral patellar facetectomy was performed first as previously described [39]. With a tourniquet in place and the patient supine, the knee was approached through a lateral parapatellar incision. A lateral retinacular release allowed visualization of the lateral border of the patella without injuring the vastus lateralis. Between 1 and 1.5cm of the lateral border of the patella, including osteophytes and 1 to 2mm of articular cartilage were resected. Any osteophytes on the lateral trochlea were also resected and bone wax was applied to all cut surfaces.

Attention was then turned to the unicompartmental arthroplasty. A tibial tubercle osteotomy was not routinely performed [10].

All patients received peri-operative antibiotics (second generation cephalosprorins) and prophylactic anti-coagulation treatment (low molecular weight heparin). Range of motion and isometric quadriceps exercises were initiated as soon as possible and full weightbearing was allowed the first week postoperatively.

Assessment of Results

Postoperative clinical and radiographic follow-up was performed prospectively at 2 months, 6 months, 1 year, and every 2 years thereafter in all patients. Any subsequent operations on the index knee were recorded. Clinical results were assessed with physical examination and International Knee Society (IKS) scores [17]. Patients were also asked during clinic visits if they were satisfied with their results. Patellofemoral articulation was evaluated with Kujala's score [22]. Radiographic outcomes were assessed by a standardized protocol at follow-up including standing AP, lateral, and



full leg length views, and an axial view in 30 degrees of knee flexion. Overall mechanical axis, patellar tracking, and progression of degenerative disease in the patellofemoral compartment were recorded. We used the classification of Iwano to measure patellofemoral OA [18]. Data were collected and analyzed retrospectively to assess the results of UKA combined with partial lateral patellar facetectomy for treatment of unicompartmental and patellofemoral degenerative disease.

Statistical Analysis

Pre-operative and post-operative IKS scores, range of motion were compared using wilcoxon's test. Statistical significance was defined as $p<0.05$.

RESULTS

The eleven patients were followed clinically and radiographically for a mean of 60.3 months (range: 39 to 91 months). No implant revision was required during the follow-up period.

Functional Results

10 patients (90%) were satisfied with their knee function at final follow-up. The mean IKS knee score improved from 64.9 points (range: 44 to 81 points) pre-operatively to 87.5 points (range: 60 to 100 points) at final follow-up ($p= 0.01$). The mean IKS functional score improved from 65.9 points (range: 15 to 100 points) pre-operatively to a mean of 83.2 points (range: 40 to 100 points) at final follow-up ($p= 0.0117$).

The mean maximum knee flexion was 136 degrees (range 115 to 150 degrees) pre-operatively and 134 degrees (range 120 to 140 degrees) at final follow-up (no statistic difference). The mean extension deficit was 0 degrees (range, 0 to 0 degrees) preoperative and 0.45 degrees (range 0 to 5 degrees) at final follow-up (no statistic difference). The mean Kujala score was 84.3 points (range 63 to 100 points) postoperatively. 4 patients had moderate crepitus.

Radiographic Results

No visible loosening or significant polyethylene wear occurred. On standing full length plain radiographs, the mean alignment was 5.5 degrees of valgus (range: 0 to 12 degrees of valgus) preoperatively and 3.4 degrees of valgus.

Progression of patellofemoral arthritis was observed in 2 cases: one level 1 (3 years follow-up) and one level 2 (7 years follow-up) of the classification of Iwano (fig. 1, 2).

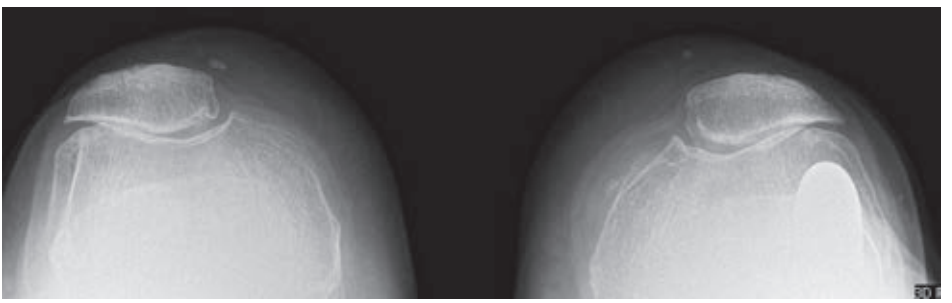
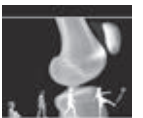


Fig. 1: Postoperative patellofemoral OA grade 2 (7 years follow-up).



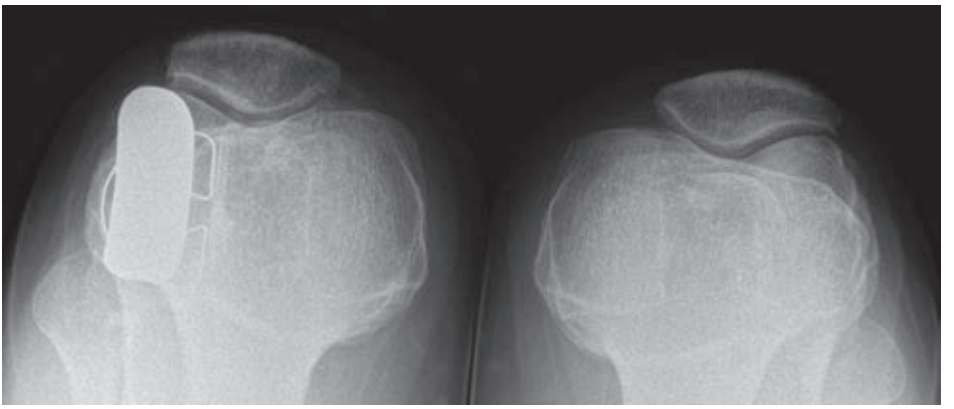


Fig. 2: Patellofemoral articulation without OA (4 years follow-up).

COMPLICATIONS

Any early complication was observed. At last follow-up, any revision was required and any loosening was observed on radiography.

DISCUSSION

This study is a medium-term retrospective analysis of the results of eleven patients in whom simultaneous UKA and partial lateral patellar facetectomy was performed for degenerative disease limited to one tibiofemoral compartment and the lateral patellofemoral joint. Our results indicate that this treatment strategy is viable option for these patients as good pain control and functional outcome were achieved.

Although some UKA femoral component designs have been noted to impinge of the patella in some cases [15], this complication is relatively rare and several authors have demonstrated no significant differences in patellofemoral joint forces and kinematics following UKA [28, 32]. This finding suggests that treatment strategies for patellofemoral joint pathology, such as partial lateral facetectomy, that are successful in patients with isolated patellofemoral involvement will also find success in patients that undergoing UKA.

The only previous results of the combination of a non-arthroplasty procedure to treat patello-femoral arthritis with a UKA were reported by Antoniou *et al* in 1996 [2]. They utilized the patelloplasty technique described by Ficat *et al.* [12] and Marmor [24] to resurface the entire patella with fibrocartilage and noted relief of patellofemoral pain in 90% of patients at six years post-operative [2].

Other options are available for the treatment of associated unicompartmental and patello-femoral degenerative disease. When the patello-femoral disease is asymptomatic, conservative management of the patellofemoral joint has been shown to be effective, with no adverse effects on outcome of UKA noted in this patient population [5, 6, 7, 14]. When patellofemoral disease is symptomatic, some authors have reported good results through the combination of patellofemoral arthroplasty and UKA [16]. More recently, specific bicompartmental arthroplasty systems have been developed and reported [33]. Palumbo reported poor results for the bicompartmental arthroplasty [29]. Both of these techniques have the theoretical advantages of preserving both cruciate ligaments and more normal gait [8, 9, 20]. Finally, TKA remains a standard technique for the management of bicompartmental disease.

Patellofemoral joint osteoarthritis can develop or progress in patients who have had prior UKA

for unicompartmental disease. In patients with normal patellofemoral cartilage at the time of UKA, Berger *et al.* reported a 10% rate of symptomatic patellofemoral degenerative disease at 15 years post-operative [8, 9]. Patellofemoral pain was the cause of both revisions to TKA in their series [8, 9]. Similarly, Kahn *et al.*, Argenson *et al.* and Weale *et al.* reported rates of radiographic evidence of patellofemoral joint degenerative changes 5 to 10 years following UKA to be between 5 and 60% [3, 20, 37]. Argenson *et al.* and Weale *et al.* both noted patellofemoral degenerative change to be the most common reason for revision to TKA [20, 37]. While these patients represent a distinct clinical situation from that explored in the current study, one could consider partial lateral patellar facetectomy for treatment of symptomatic lateral patellar degenerative disease that develops after UKA. Further research is needed in this area.

The strengths of our study include its long follow-up period with no patents lost to follow-up, its use of validated, patient reported outcome measures, and presentation of a successful method of treating a difficult clinical problem. Weaknesses of the study include its relatively small numbers and lack of a control group. Consideration should be given to comparing the treatment method outlines above to TKA or bicompartmental arthroplasty for patients with two-compartment disease.

CONCLUSIONS

Simultaneous UKA and partial lateral patellar facetectomy is a viable treatment option for symptomatic degenerative disease involving one tibiofemoral compartment and the lateral patellofemoral joint. Durable pain control and functional improvement were noted at medium-term follow-up. This treatment approach may be a useful alternative to TKA or bicompartmental arthroplasty in a carefully selected patient population.

LITERATURE

[1] AKIZUKI S, MUELLER JK, HORIUCHI H, MATSUNAGA D, SHIBAKAWA A, KOMISTEK RD. *In vivo* determination of kinematics for subjects having a Zimmer Unicompartmental High Flex Knee System. *J Arthroplasty 24(6): 963, 2009.*

[2] ANTONIOU J, HADJIPAVLOU A, ENKER P, ANTONIOU A. Unicompartmental knee arthroplasty with patelloplasty. *Int Orthop 20(2): 94, 1996.*

[3] ARGENSON JN, CHEVROL-BENKEDDACHE Y, AUBANIAC JM. Modern unicompartmental knee arthroplasty with cement: a three to ten-year follow-up study. *J Bone Joint Surg Am 84-A(12): 2235, 2002.*

[4] ASHRAF T, NEWMAN JH, EVANS RL, ACKROYD CE. Lateral unicompartmental knee replacement survivorship and clinical experience over 21 years. *J Bone Joint Surg Br 84(8): 1126, 2002.*

[5] BEARD DJ, PANDIT H, GILL HS, HOLLINGHURST D, DODD CA, MURRAY DW. The influence of the presence and severity of pre-existing patellofemoral degenerative changes on the outcome of the Oxford medial unicompartmental knee replacement. *J Bone Joint Surg Br 89(12): 1597, 2007.*

[6] BEARD DJ, PANDIT H, OSTLERE S, JENKINS C, DODD CA, MURRAY DW. Pre-operative clinical and radiological assessment of the patellofemoral joint in unicompartmental knee replacement and its influence on outcome. *J Bone Joint Surg Br 89(12): 1602, 2007.*

[7] BEREND KR, LOMBARDI AV Jr, ADAMS JB. Obesity, young age, patellofemoral disease, and anterior knee pain: identifying the unicondylar arthroplasty patient in the United States. *Orthopedics 30(5 Suppl): 19, 2007.*

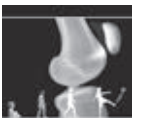
[8] BERGER RA, MENEGHINI RM, JACOBS JJ, SHEINKOP MB, DELLA VALLE CJ, ROSENBERG AG, GALANTE JO. Results of unicompartmental knee arthroplasty at a minimum of ten years of follow-up. *J Bone Joint Surg Am 87(5): 999, 2005.*

[9] BERGER RA, MENEGHINI RM, SHEINKOP MB, DELLA VALLE CJ, JACOBS JJ, ROSENBERG AG, GALANTE JO. The progression of patellofemoral arthrosis after medial unicompartmental replacement: results at 11 to 15 years. *Clin Orthop Relat Res (428): 92, 2004.*

[10] DEJOUR D, NEYRET P, DONELL ST. Tibial tubercle osteotomy for access in lateral unicompartmental knee replacement. *Knee 5(1): 33, 1998.*

[11] DUNCAN R, PEAT G, THOMAS E, WOOD L, HAY E, CROFT P. Does isolated patellofemoral osteoarthritis matter? *Osteoarthritis Cartilage 17(9): 1151, 2009.*

[12] FICAT RP, FICAT C, GEDEON P, TOUSSAINT JB. Spongialization: a new treatment for diseased patellae. *Clin Orthop Relat Res (144): 74, 1979.*



[13] HASSABALLA MA, PORTEOUS AJ, LEARMONTH ID. Functional outcomes after different types of knee arthroplasty: kneeling ability versus descending stairs. *Med Sci Monit 13(2): CR77, 2007.*

[14] HAUPTMANN SM, KREUL U, MAZOOCHIAN F, C VS-P, JANSSON V, MULLER PE. Influence of patellofemoral osteoarthritis on functional outcome after unicondylar knee arthroplasty. *Orthopade 34(11): 1088, 2005.*

[15] HERNIGOU P, DESCHAMPS G. Patellar impingement following unicompartmental arthroplasty. *J Bone Joint Surg Am 84-A(7): 1132, 2002.*

[16] HEYSE TJ, KHEFACHA A, CARTIER P. UKA in combination with PFR at average 12-year follow-up. *Arch Orthop Trauma Surg, 2009.*

[17] INSALL JN, DORR LD, SCOTT RD, SCOTT WN. Rationale of the Knee Society clinical rating system. *Clin Orthop Relat Res (248): 13, 1989.*

[18] IWANO T, KUROSAWA H, TOKUYAMA H, HOSHIKAWA Y. Roentgenographic and clinical findings of patellofemoral osteoarthrosis. With special reference to its relationship to femorotibial osteoarthrosis and etiologic factors. *Clin Orthop Relat Res (252): 190, 1990.*

[19] KANG SN, SMITH TO, SPRENGER DE ROVER WB, WALTON NP. Pre-operative patello-femoral degenerative changes do not affect the outcome after medial Oxford unicompartmental knee replacement. *J Bone joint Surg Br ;93(4) : 476-8. 2011*

[20] KHAN OH, DAVIES H, NEWMAN JH, WEALE AE. Radiological changes ten years after St.-Georg Sled unicompartmental knee replacement. *Knee 11(5): 403, 2004.*

[21] KOZINN SC, SCOTT R. Unicondylar knee arthroplasty. *J Bone Joint Surg Am 71(1): 145, 1989.*

[22] KUJALA UM, JAAKKOLA LH, KOSKINEN SK, TAIMELA S, HURME M, NELIMARKKA O. Scoring of patellofemoral disorders. *Arthroscopy ; 9(2) : 159-63. 1993.*

[23] LUSTIG S, PAILLOT JL, SERVIEN E, HENRY J, AIT SI SELMI T, NEYRET P. Cemented all polyethylene tibial insert unicompartimental knee arthroplasty: a long term follow-up study. *Orthop Traumatol Surg Res 95(1): 12, 2009.*

[24] MARMOR L. Unicompartmental knee arthroplasty. Ten- to 13-year follow-up study. *Clin Orthop Relat Res (226): 14, 1988.*

[25] MARTENS M, DE RYCKE J. Facetectomy of the patella in patellofemoral osteoarthritis. *Acta Orthop Belg 56(3-4): 563, 1990.*

[26] McALLISTER CM. The role of unicompartmental knee arthroplasty versus total knee arthroplasty in providing maximal performance and satisfaction. *J Knee Surg 21(4): 286, 2008.*

[27] MERCIER N, WIMSEY S, SARAGAGLIA D. Long-term clinical results of the Oxford medial unicompartmental knee arthroplasty. *Int Orthop, 2009.*

[28] MILLER RK, GOODFELLOW JW, MURRAY DW, O'CONNOR JJ. *In vitro* measurement of patellofemoral force after three types of knee replacement. *J Bone Joint Surg Br 80(5): 900, 1998.*

[29] PALUMBO BT, HENDERSON ER, EDWARDS PK, BURRIS RB, GUTIERREZ S, RATERMAN SJ. Initial experience of the Journey-Deuce bicompartmental knee prothesis. A review of 36 cases. *J Arthroplasty; 26(6): 40-5. 2011.*

[30] PANDIT H, JENKINS C, GILL HS, SMITH G, PRICE AJ, DODD CAF, MURRAY DW. Unnecessary contraindications for mobile-bearing unicompartmental knee replacement. *J Bone Joint Surg Br ; 93(5): 622-8. 2011.*

[31] PAULOS LE, O'CONNOR DL, KARISTINOS A. Partial lateral patellar facetectomy for treatment of arthritis due to lateral patellar compression syndrome. *Arthroscopy 24(5): 547, 2008.*

[32] PRICE AJ, OPPOLD PT, MURRAY DW, ZAVATSKY AB. Simultaneous in vitro measurement of patellofemoral kinematics and forces following Oxford medial unicompartmental knee replacement. *J Bone Joint Surg Br 88(12): 1591, 2006.*

[33] ROLSTON L, BRESCH J, ENGH G, FRANZ A, KREUZER S, NADAUD M, PURI L, WOOD D. Bicompartmental knee arthroplasty: a bone-sparing, ligament-sparing, and minimally invasive alternative for active patients. *Orthopedics 30(8 Suppl): 70, 2007.*

[34] ROUGRAFF BT, HECK DA, GIBSON AE. A comparison of tricompartmental and unicompartmental arthroplasty for the treatment of gonarthrosis. *Clin Orthop Relat Res (273): 157, 1991.*

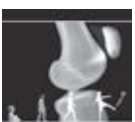
[35] SAH AP, SCOTT RD. Lateral unicompartmental knee arthroplasty through a medial approach. Study with an average five-year follow-up. *J Bone Joint Surg Am 89(9): 1948, 2007.*

[36] STERN SH, BECKER MW, INSALL JN. Unicondylar knee arthroplasty. An evaluation of selection criteria. *Clin Orthop Relat Res (286): 143, 1993*

[37] WEALE AE, MURRAY DW, BAINES J, NEWMAN JH. Radiological changes five years after unicompartmental knee replacement. *J Bone Joint Surg Br 82(7): 996, 2000.*

[38] YANG KY, WANG MC, YEO SJ, LO NN. Minimally invasive unicondylar versus total condylar knee arthroplasty-early results of a matched-pair comparison. *Singapore Med J 44(11): 559, 2003.*

[39] YERCAN HS, AIT SI SELMI T, NEYRET P. The treatment of patellofemoral osteoarthritis with partial lateral facetectomy. *Clin Orthop Relat Res (436): 14, 2005.*



ASSOCIATION OF A MEDIAL UKA AND A PATELLOFEMORAL ARTHROPLASTY: IS IT POSSIBLE?

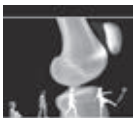
S. PARRATTE, M. OLLIVIER, J.M.AUBANIAC, J.N. ARGENSON

INTRODUCTION

Treatment of limited osteoarthritis of the knee remains a challenging problem. The therapeutic goals are to alleviate pain and restore knee function. Arthroplasty solutions may include unicompartmental knee arthroplasty (UKA) or conventional total knee arthroplasty (TKA). These therapeutic solutions have to be efficient, durable and safe but should preserve the bone stock when possible. TKA may offer durable and satisfying clinical and radiological results when arthritis is affecting the three compartments of the knee, however TKA does not preserve the bone stock and the ligaments. UKA is a bone and ligament sparing technique, which is reliable to restore knee kinematics and function for arthritis limited to one compartment of the knee. The outcomes of UKA improved since its introduction more than 30 years ago due to improvement in designs, indications, materials, and surgical techniques [23, 34]. Reported results of UKA are better when the anterior cruciate ligament is intact. Similarly, outcome and kinematic studies suggest that maintaining the anterior cruciate ligament in bi- and tri-compartmental knee arthroplasty may be advantageous in terms of survivorship [10, 21], stair climbing ability, patient satisfaction and joint kinematics. Bicomparmental arthritis of the knee is not rare and bicompartmental knee arthroplasties have been proposed to bridge the gap between UKA and TKA. There is a renewal interest for bicompartmental knee arthroplasties including association of medial UKA and femoropatellar arthroplasties. A smaller implant size, a reduce operative traumatism, the preservation of both cruciate ligaments and bone stock, and a more "physiologic" knee joint are considered advantageous over total knee replacement. Interesting proprioceptive or kinematic studies, and long-term clinical and radiological studies have been reported. Considering the renewed interest for combined compartmental implants (including association of medial UKA and femoropatellar arthroplasty we aimed to present in this chapter, the surgical technique including tip and tricks of combine UKA and patella-femoral arthroplasties using two separate implants.

SURGICAL TECHNIQUE

The first step of a good surgical technique remains a proper patient selection and a good indication. The second step is related to the implant choice and according to our experience the only way to properly accommodate is to use two separate implants. We recommend the use of a fixed-bearing UKA and patella-femoral implant with an anterior reference including an anterior cut of the femur. Both implant are cemented and this is important factor of



survivorship as in our early experience all the un-cemented femoral implants of the patella-femoral have been revised.

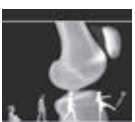
INDICATION

The patient profile analysis for this type of procedure is an important factor. Most of the time, potential candidates are active and want to stay active after the surgery and this is one of their main expectations after the relief of the pain (ref mobile bearing TKA). The first criteria is the location of the pain. In fact, the physical exam should analyse exactly the location of the pain. When the lateral compartment is painful, there is no indication for a combined medial-UKA and patella-femoral arthroplasty. There is also no indication in inflammatory arthritis of the knee and post-traumatic arthritis may be an indication as long as surgeons and patient alike are aware of the higher rate of complication in post-traumatic arthritis. The age, gender or the weight of the patient should be considered but are not represented strict limitations.

The indications for the procedure are: a confirmed diagnosis of painful bicompartmental osteoarthritis (Ahlback Grade 2 or greater) and a preserved status of the lateral compartment joint (based on clinical evaluation and stress-radiographs). A preoperative range of knee flexion greater than 100º associated with a full range of knee extension, and finally a knee clinically stable in the frontal and sagittal planes are also considered as crucial for the indication. A valgus or a varus deformity greater than ten degrees as measured on the long-leg X-rays, or a metaphysal tibial varus greater than 7 degrees are also considered as a contra-indication. Varus and valgus stress radiographs are systematically performed to evaluate the lateral compartment and the correction of the deformities. A full loss of cartilage on the lateral compartment or a fixed deformity observed on the stress radiograph are considered as contra-indications. We do perform an MRI to check the status of the ACL if there is any doubt during the physical exam. Following this analysis, there is two types of potential candidates. The first type is a patient with a varus knee and a bone on bone arthritis of the medial compartment and a significant arthritis of the medial facet of the patella-femoral joint. Most of this type of patients are active male around sixty or younger. The second type of patients has a bone on bone patella-femoral arthritis of the lateral patella-femoral joint with a patellar subluxation associated with a painful arthritis (most of the time albhack 2 or 3) of the medial compartment. Most of the time, this type of patient are female a little less active, around sixty or younger. When the indication is confirmed following the physical and radiological exam, the discussion with the patient is important. We should take the time to explain that the philosophy is to conserve the cruciate ligaments and to replace only the concerned compartment. It's important to note that the progression of the arthritis in the lateral compartment is exceptional but not impossible.

APPROACH

We perform a medial skin incision. The upper limit is around two cm above the patella, the knee in extension. The distal limit of the skin incision is around two cm below the joint line, the knee in flexion, medial to the anterior tibial tubercule. The dissection should then be performed against the superficial part of the vastus medialis as deep as possible against the muscle. The fatty "yellow line" between the muscle and the retinaculum should be found and open using a piker. A Homan retractor is then inserted in the plane to reach the anterior cortex of the femur and the anatomical plane into the muscle fascia is opened proximally. Distally, the electric cautery is used to prolong the incision following the distal border of the vastus medialis until the patella and then the vertical part of the incision is performed until around two cm below the joint line. Like for a standard medial UKA, it's important to not perform any release of the MCL. The knee is then brought in extension, the patella is sub-luxed and the anterior cortex of the femur is cleaned using the cautery. We do mark the Whiteside line at this step using the cautery. The knees in flexion, the osteophytes of the intercondylar notch are removed to avoid any



so-called Marie-Antoinette effect (Guillotine effect of the femur on the ACL when the knee is coming in extension) that may compromise the ACL survival over the time. The osteophytes are also removed from the medial aspect of the femur.

FEMORAL PREPARATION OF THE PATELLA-FEMORAL IMPLANT

The knee still in flexion, after drilling the femur, an intra-medullary road is inserted into the femoral canal. This guide is used to perform the anterior femoral cut. First the rotation should be determined according to the Whiteside line and the posterior-condyle. Second, the thickness of the anterior femoral cut is determinate on the guide, which is then securely fixed on the femur using four screwed pins. The anterior cut is then done. The next step is the determination of the size of the femoral implant. The guide should be positioned in the axis of the cut, slightly lateralized and the two distal parts of the guide in contact with the trochlear groove. It's important to conserve 3 to 5mm of bone on each side of the implant to limit the contact with the soft tissues. The guide is then securely fixed on the femur and a dedicated bur is placed into the milling rails of the guide, starting with the central one and then taking care of each rail on the medial and lateral side of the guide alternatively. After the milling, the finishing holes guide is inserted in the previously milled area and the final holes are drilled for the pegs of the final implant. The femoral trial part of the PFJ is then inserted and leave in place until the final trials.

MEDIAL UKA

The medial UKA is then performed using an extra-medullary technique to perform the proximal tibial and the distal femoral cuts. The sagittal cut of the tibia is then done respecting the medial aspect of the ACL. The femoral size is determinate using the femoral finishing guide. It's important at this step to make sure that there is no contact between the two femoral implants. The femoral preparation is then realized taking a great care in the femoral implant rotation.

Trials are inserted with a minimum polyethylene thickness of 8mm and a targeted thickness of 9mm.

PATELLA

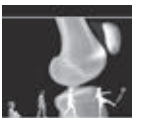
The knee in extension, the patella is then prepared using a standard patellar clamp. The patellar button should be positioned as medial as possible. In case of remaining bone on the lateral aspect of the patella, a lateral facetectomy is performed. In case of history of chronic patellar lateral subluxation, a partial lateral retinaculum release can be performed from inside. Final trials are then done with all implants in place. The patellar tracking is controlled as well as the stability of the knee.

CEMENTING TECHNIQUE

One dose of cement is used and all the components are cemented in the same time. We do cement first the tibial plateau the knee in flexion, then the femoral component of the UKA. Remnants of cement are removed specially at the posterior aspect of the knee on the tibial side and the polyethylene is inserted. The knee is then brought at 30° of extension and the femoral part of the PFJ is cemented and finally, the patellar button, the knee in extension. During the curing of the cement, it is important to maintain a pressure in the axis of the lover limb as well as a pressure on the femoral part of the PFJ, the knee at 30 to 40° and not in full extension to avoid any inversion of the slope related to an excessive pressure on the anterior aspect of the tibia.

POST-OPERATIVE COURSE

Postoperative rehabilitation protocols included immediate weight bearing protected by crutches



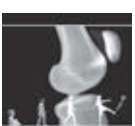
during the first 2 or 3 weeks according to patient tolerance and motivation. Exercises are focusing on immediate active recuperation of flexion and extension.

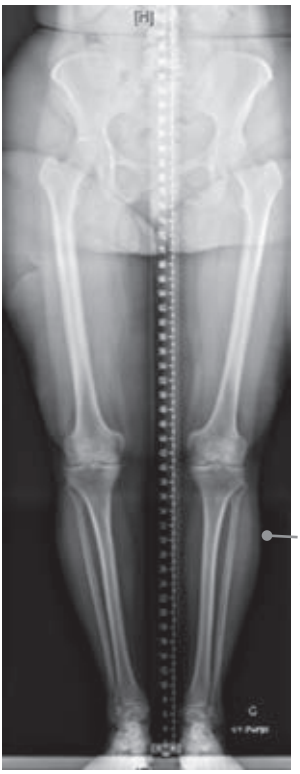
RESULTS

Following the report of our early experience of a bicompartmental arthroplasty and results from 5 to 23 years showing good clinical outcomes, with a long-term survivorship lower than for a TKA, we started a new series of patient with a new patella-femoral arthroplasty. Between 2008 and 2010, 22 patients have been operated for a bicompartmental arthroplasty using modern UKA and PFJ. The indications for the procedure were the same than the indications described early in this chapter. The mean age of the patient was 61 years-old, and 12 patients were women and 10 were men. A minimum follow-up of 2-years was required for this study. Knee Society Function and Knee scores respectively improved from 54 to 91 and from 48 to 94 at a minimum follow-up of two years. Mean pre-operative flexion was 125° and 130° post-operatively. All patients resumed their leisure or even sportive activities 3 to 6 months after surgery. All the items of the KOOS significantly improved post-operatively. According to the results of the QOL KOOS, 12 out of 22 patients considered their knee as a forgotten knee. One hunter patient required a revision for a septic complication following a untreated bite boar of the leg below the operated knee, one year and a half after the procedure. No other complication required revision and at two years follow-up, patient quality of life scores were significantly higher than in TKA patient. The restoration of the lower limb axis was in all the cases in zone C or 2 according to the Kennedy classification.

DISCUSSION AND CONCLUSION

Bicompartmental arthroplasty has been advocated as an alternative to TKA for limited arthritis of the knee to preserve bone stock and restore more normal kinematics. Due to these potential advantages over TKA, there is a renewed interest for combined compartmental implants including association of medial UKA and femoro-patellar arthroplasty. Our previously reported long-term data demonstrated that bicompartmental arthroplasty can provide reasonable function restoration of the knee and adequate mechanical axis restoration of the lower limb for moderate deformities. Survivorship to revision at 17 years was lower than those observed for TKA or UKA. This higher failure rate was probably linked to different factors: first the use of old generation of implant without any reliable instrumentation, second the use of old-generation of resurfacing patello-femoral implants with very limited instrumentation. Finally, a third of the patella-femoral implants used in this series were un-cemented implants and all of them have been removed. In fact, catastrophic failure rate have been observed with the use of cementless implant for the patellofemoral joint. Based on these results and our experience concerning the indications, the implants characteristic, we started in 2008 a new series with clearly defined indications and using modern UKA and PFA cemented implants through a standardized subvastus approach. The short-term follow-up outcomes at two years are encouraging. Function score are comparable to those observed for UKA with high restoration of function, high flexion and high patient satisfaction. Of course longer follow-up is required. Finally, to answer the initial question: association of a medial UKA and a patellofemoral arthroplasty: is it possible? The answer is, yes using a combination of UKA and PFA is possible with good indication, implant and surgical technique, short-terms results are encouraging.





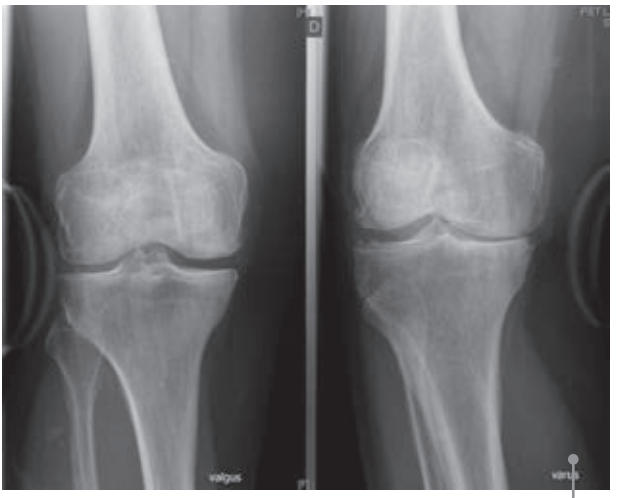


Fig. 1: This full-length X-ray is showing a medial arthritis of the knee on a very-active 61 years-old woman. She presented with a medial and patell-femoral pain without any lateral pain, a stable knee and failure of the medical treatment.

Fig. 2: The varus and valgus stress X-rays are very important to control the reducibility of the deformity and to check the full thickness of the cartilage on the non-resurfaced compartment.

Fig. 3: Patello-femoral sky views are important to assess the wear on the patella-femoral compartment and the position of the patella.

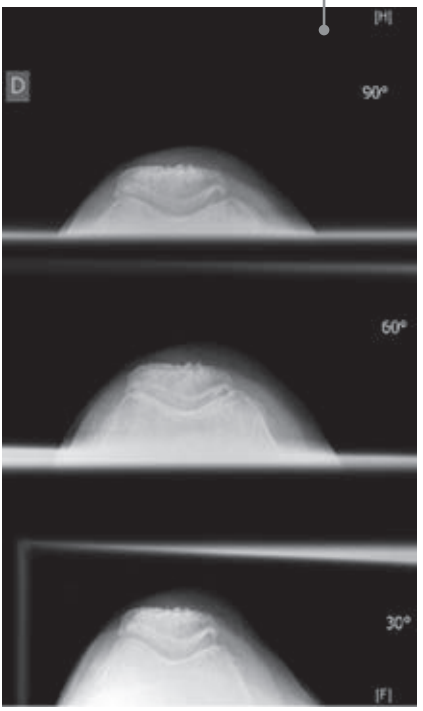
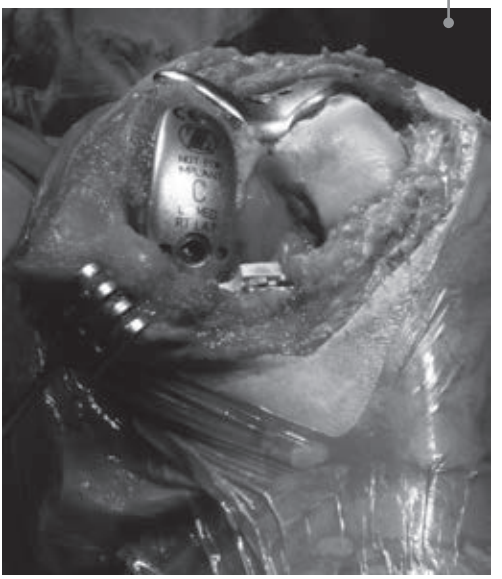
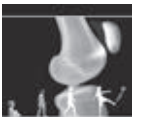


Fig. 4: This intra-operative view of the knee opened through a sub-vastus approach is showing the trial implants in place on the patella-femoral and the medial compartment with preserved cruciate ligament.





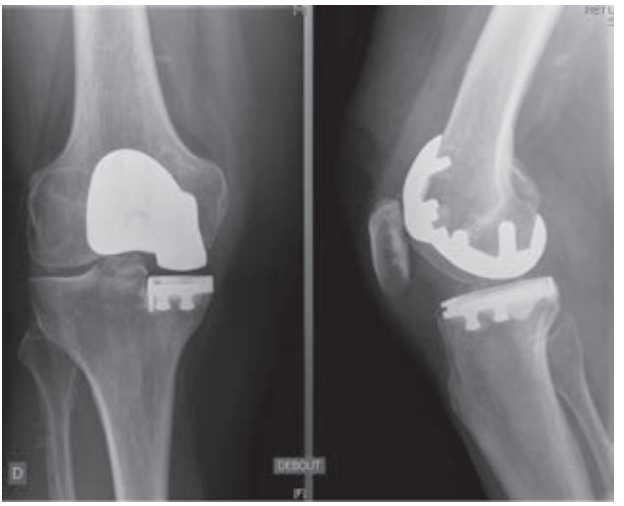


Fig. 5: The 1-year post-operative X-rays are showing a correct implant positioning on the ML and on the AP views.

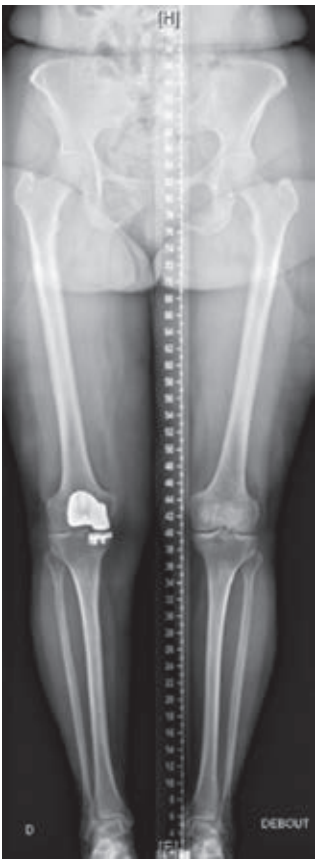


Fig. 6: The postoperative full-length X-ray is showing a proper restoration of axis of the lower limb without any over correction of the deformity.

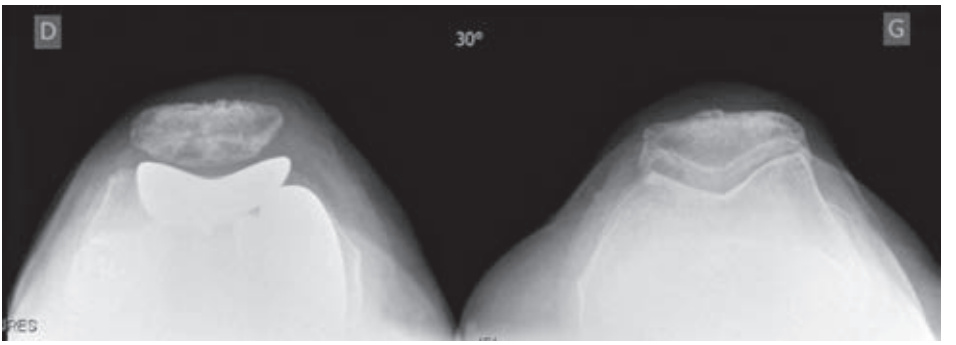


Fig. 7: Patello-femoral sky view at 30° is showing a proper position of the trochlear implant below the patella.

LITERATURE

[1] AHLBACK S. Osteoarthrosis of the knee. A radiographic investigation. *Acta Radiol Diagn (Stockh). 1968; Suppl 277: 7-72.*

[2] ARGENSON JN, CHEVROL-BENKEDDACHE Y, AUBANIAC JM. Modern unicompartmental knee arthroplasty with cement: a three to ten-year follow-up study. *J Bone Joint Surg Am. 2002; 84-A: 2235-39.*

[3] PARRATTE S, PAULY V, AUBANIAC JM, ARGENSON JN. Survival of bicompartmental knee arthroplasty at 5 to 23 years. *Clin Orthop Relat Res 2010; 468: 64-72.*

[4] ARGENSON JN, FLECHER X, PARRATTE S, AUBANIAC JM. Patellofemoral arthroplasty: an update. *Clin Orthop Relat Res. 2005; 440:50-3.*

[5] ARGENSON JN, KOMISTEK RD, AUBANIAC JM, DENNIS DA, NORTHCUT EJ, ANDERSON DT, AGOSTINI S. In vivo determination of knee kinematics for subjects implanted with a unicompartmental arthroplasty. *J Arthroplasty. 2002; 17: 1049-54.*

[6] ARGENSON JN, PARRATTE S, BERTANI A, FLECHER X, AUBANIAC JM. Long-term results with a lateral unicondylar replacement. *Clin Orthop Relat Res. 2008; 466: 2686-93.*

[7] BANKS SA, FREGLY BJ, BONIFORTI F, REINSCHMIDT C, ROMAGNOLI S. Comparing in vivo kinematics of unicondylar and bi-unicondylar knee replacements. *Knee Surg Sports Traumatol Arthrosc. 2005; 13: 551-6.*

[8] FELTS E, PARRATTE S, PAULY V, AUBANIAC JM, ARGENSON JN. Function and quality of life following medial unicompartmental knee arthroplasty in patients 60 years of age or younger. *Orthop Traumatol Surg Res 2010; 96: 861-7.*

[9] BERGER RA, MENEGHINI RM, SHEINKOP MB, DELLA VALLE CJ, JACOBS JJ, ROSENBERG AG, GALANTE JO. The progression of patellofemoral arthrosis after medial unicompartmental replacement: results at 11 to 15 years. *Clin Orthop Relat Res. 2004; 92-9.*

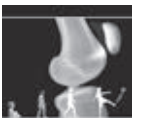
[10] ENGH GA. A bi-compartmental solution: what the Deuce? *Orthopedics. 2007;30: 770-1.*

[11] GIBSON PH, GOODFELLOW JW. Stress radiography in degenerative arthritis of the knee. *J Bone Joint Surg Br. 1986;68: 608-9.*

[12] INSALL JN, DORR LD, SCOTT RD, SCOTT WN. Rationale of the Knee Society clinical rating system. *Clin Orthop Relat Res. 1989; 13-14.*

[13] ORNETTI P, PARRATTE S, GOSSEC L, TAVERNIER C, ARGENSON JN, ROOS EM, GUILLEMIN F, MAILLEFERT JF. Cross-cultural adaptation and validation of the French version of the Knee injury and Osteoarthritis Outcome Score (KOOS) in knee osteoarthritis patients. *Osteoarthritis Cartilage. 2007.16; 423-8.*

[14] ROLSTON L, BRESCH J, ENGH G, FRANZ A, KREUZER S, NADAUD M, PURI L, WOOD D. Bicompartmental knee arthroplasty: a bone-sparing, ligament-sparing, and minimally invasive alternative for active patients. *Orthopedics. 2007;30: 70-3.*



APPROACH AND PATELLA IN TOTAL KNEE ARTHROPLASTY

P. BEAUFILS, M. THAUNAT, D. PASSERON, P. BOISRENOULT, N PUJOL

Patellar complications constitute one of the most common causes of failure of total knee arthroplasty [5] and the leading reason for surgical revision [7, 15]. Depending on the series, the frequency is estimated at 1 to 50% [9, 26]. Patellar complications are usually seen as more or less patent patellar dislocation with or without instability, fracture, patellar loosening, or pain.

Disturbance of patellar blood supply, modifications of patellar thichness or global AP whidth, patellar maltracking are the main origins of these complications.

Medial or lateral approach may have an influence on the patella in terms of blood supply or maltracking.

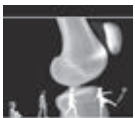
APPROACH AND BLOOD SUPPLY

Vascularization of the patella is given from the Scapinelli's periarticular circle which comes from the genicular arteries, one branch of the femora artery and recurrent branches from the anterior tibialis a. and the posterior tibialis a. [33]. The most important part of the blood flow comes from the medial part which has three practical consequences:

- Medial arthrotomy interrupts the medial blood supply,
- Medial arthrotomy + lateral retinaculum release does interrupt the large majority of the patellar vascularization,
- Iterative surgery should use the same articular approach as the index surgery as often as possible.

APPROACH AND PATELLAR TRACKING

Regarding maltracking, residual patellar malposition, and its radiographic presentation of patellar tilt and/or gliding, contributes to such complications [4, 8, 30]. The proportion of tilted patella varies from 0.8% according to Brick and Scott [10] to 45% reported by Bindelglass and Vince [8]. Lateral tilt is more common [8, 10, 18, 19, 29] and has a worse effect on prognosis. Laughlin [22] demonstrated that external tilt tends to worsen with time, unlike medial tilt which tends to improve. The degree of tilt increases with more pronounced patellar malposition preoperatively [12].



Two different situations have to be considered:

1) Patella is pre operatively well centered

The goal of the procedure is to maintain a well aligned patella. In this occurrence, approach is mainly dictated by other factors such as previous surgery or frontal deformity: medial approaches in varus knees, lateral approach in valgus knees. Respect of several well defined criteria (adapted rotation of femoral and tibial implants, thickness of the surfaced patella, global AP whidth, patellar hight…) should allow to obtain a proper post-operative patellar alignment and thus to avoid a complementary lateral retinacular release. Lateral release in this occurrence is always the witness of a technical error. Patellar tracking is not influenced by the medial or lateral approach. One should however underline the interest of the subvastus approach, first described by Gernez and then by Hofmann [21] or midvastus [16] compared with the standard medial parapatellar approach in terms of quadriceps recovery [13, 14, 17], immediate functional result and patellar tracking. These approaches would limit the risk of associated retinacular release [8, 20, 24]. Matsueda and Gustilo [24] compared the medial parapatellar approach with the medial infra-vastus approach and obtained better centering with the latter (83% versus 63%). But these approaches are not indicated in cases of expected difficulties of exposure: flexum deformity, patella baja, obesity…

2) Patella is laterraly subluxated

The goal is to restore a normal patellar tracking and thus to correct bony or ligamentous abnormalities. As it is, when considering the frontal plane: correction of a varus or valgus deformity to obtain a well aligned knee (table 1). Chan and Gill [12] consider that each 2° of lateral tilt observed preoperatively increases the post-operative tilt 1°. Bindelglass and Vince [8] come to the same conclusions.

Pushing this comparison between axial plane (the patellar alignment) and frontal plane (the frontal knee axis), one can find the same factors of deformity (lateralization) than those of the frontal deformity :

a. Cartilaginous lateral patellar wear which pushes the patella laterally It is the problem of the cartilaginous correction which is probably the easiest factor to correct.

b. Ligamentous abnormalities (lateral retinaculum contraction, medial retinaculum stretchening). It is the problem of the ligament balance

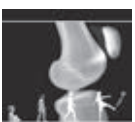
c. Bony ablormalities (patellofemoral dysplasia): it is the problem of the correction of bony deformities in the ligamentous envelope.

In this occurrence of subluxated patella, the choice of the approach may have two interests:

- *Direct influence:* for example on the ligamentous balance. Why to propose a medial approach in case of lateral subluxated patella, that is to say an approach which goes through the "convexity". Do we approach a varus knee with lateral ligamentous stretchening using a lateral approach, or a valgus knee with medial stretchening using a medial approach?
- *Indirect Influence* allowing a better positioning of femoral and tibial implants: in the same way as for a valgus knee: lateral approach allows a better assessment and better correction of lateral bony abnormalities.

For all these reasons we propose the use of the lateral approach with parapatellar lateral arthrotomy when the patella is laterally subluxated, whatever the frontal axis (varus or valgus knee). In this occurrence, the use of a medial approach dramatically increases the risk of lateral retinaculum release. This release considerably increases the risk of patellar fracture [31], even though the effects on patellar blood supply remain a subject of debate [23, 31, 34, 36]. The real effect on patellar tilt remains uncertain [25].

Lateral approach has a bad reputation, with an increased risk of extensor apparatus rupture. In reality, there is no more intra operative or post-operative morbidity compared with standard medial parapatellar approach [27, 35].



The incision is a little bit longer than a medial approach. The approach is passing between the tendons of the rectus femoris and the vastus lateralis. In order to open the patellar retinaculum laterally a few millimeters from the paella, a 1cm incision is made in the prepatellar periosteum along the lateral border of the patella (fig. 1a) followed by medial to lateral removal of the perisoteum, the lateral marginal portion of the patella being exposed and sectioned with an oscillating saw to achieve marginal lateral patellectomy (fig. 1b).

The patella is then medially everted, taking care of the patellar tendon attachment. It is often necessary to release the lateral third of the patellar insertion. Rarely, there is a need for Antertior Tibial Tuberosity (ATT) elevation. Which of course improves the medial exposure? In these cases, ATT osteotomy should be long enough to allow a strong fixation.

The lateral retinaculum is closed at the end of the procedure (fig. 1b). In case of ATT elevation, ATT is fixed with screw and wires (fig. 2), or cerclage alone without medialization.

We conducted a comparative study between medial parapatellear approach and lateral approach [2] with ATT elevation in a selected group of lateral patellar subluxation.

Inclusion criteria were: presence of lateral patellar glide of at least 5mm from the trochlear groove on the preoperative 30° patellofemoral view and a lateral surgical approach with ATT elevation (we systematically used at that time).

Thirteen knees fulfilled the inclusion criteria (lateral "group"). This group was matched with thirteen other knees which also presented lateral patellar glide of at least 5mm preoperatively but which were operated on via a medial approach ("medial" group) during the same period.

The two groups were comparable regarding patient age, gender, body weight, range of motion pre- and postoperative HKA, pre op patellar displacement, patellar height, preoperative patellar thickness.

There were no complication related to lateral approach and specially elevation of the ATT.

Gliding was similarly corrected in both groups, but the residual tilt was in the medial group (lateral tilt), versus in the lateral group (medial tilt) (table 1) (fig. 3).

Our results are similar to those already published. Arnold [3] and Burki [11] demonstrated that the lateral parapatellar approach with elevation of the ATT enables better restitution of good patellar kinematics without patellar resurfacing. For Vielpeau [35] the stability of the patella is one of the advantages of the lateral approach which enables correction of the preoperative lateral tilt if a good ligament balance is achieved in flexion. How does approach influence the patellar tracking?

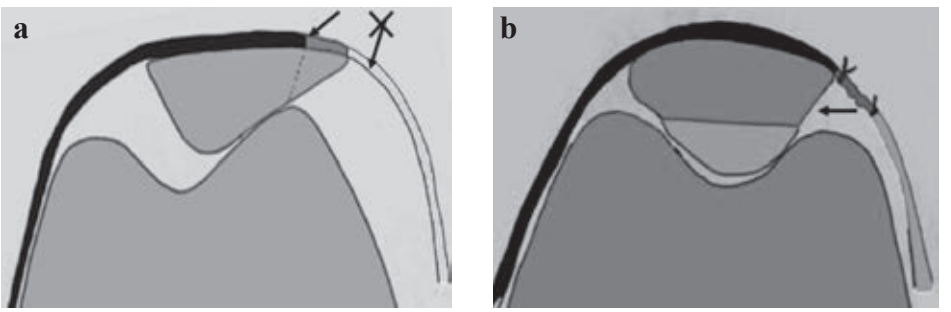
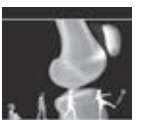


Fig. 1: a) lateral approach. Lateral retinaculum is cutted just at the rim of the patella. Periosteum is then released. - b) allowing a lateral marginal facettectomy which allows to lengthen the lateral retinaculum.



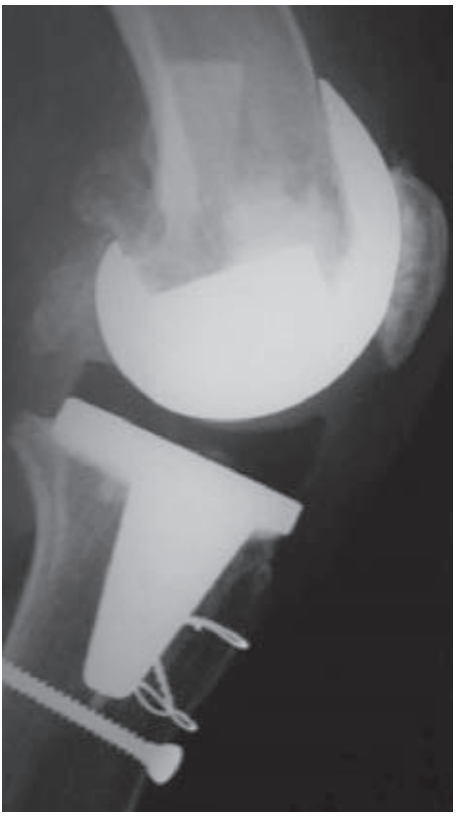


Fig. 2: Lateral approach with ATT elevation. Fixation "in place" with two wires and one screw.

We hypothesized two causes:
- A lengthening effect of the lateral retinaculum due to the lateral marginal patellectomy;
- Better positioning of the tibial implant in external rotation. We know the key role of optimal implants rotation in patellar tracking [1, 6], specially in case of lateral patellar subluxation. Lateral approach would allow a better exposure of the tibial plateau while the mediopatellar approach can induce internal rotation tibial malposition because of constraints imposed by the presence of the patellar ligament.

In order to confirm this last hypothesis, we conducted a computed tomography study, measuring the rotation of the tibial implant according to the type of approach [28].

In a prospective, comparative, non-randomized study, CT scan was undertaken on 50 successive knees in 50 patients at 3 months after primary total knee arthroplasty, for gonarthrosis. Forty-five knees were investigated. Fifteen knees were operated by the lateral approach (8 with osteotomy to elevate the ATT resting on its bed without medialization and 7 without osteotomy of the ATT) and 30 by the medial approach (10 by the subvastus approach and 20 by the medial parapatellar approach). The inclusion

Table 1 : Radiographic measurements in both groups.

	Medial group		Lateral group	
	Preop	Postop	Preop	Postop
Gliding (mm)	7.6 ± 1.3	0.7 ± 1.8	9.7 ± 5.0	0.0 ± 0.0
Tilt (°)		+4.2 ± 3.6		-3.3 ± 5.4

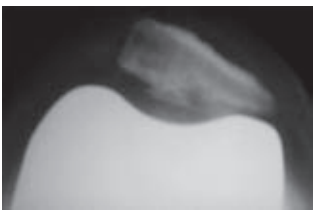


Fig. 3a: residual lateral tilt with a medial approach.

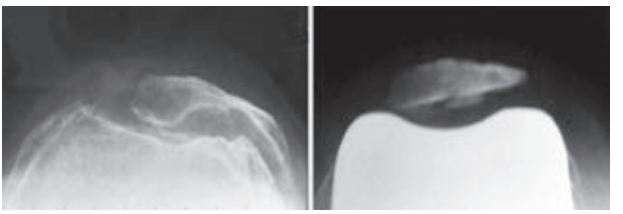


Fig. 3b: residual medial tilt with a lateral approach in a severely laterally subluxated patella.

criteria for the lateral approach were the presence of valgus knee or lateral subluxation of the patella (whatever the frontal axis). The prosthesis used was NexGen LPS Flex (Zimmer) with a fixed, symmetrical tibial baseplate. Rotation of the femoral part was adapted by navigation to measurement of the posterior condylar angle by pre-operative scan. Rotation of the tibial baseplate sought to position the tibial tray in parallel with the femoral implant in complete extension (self-adjustment). Rotation of the tibial implant was not navigated.

The post-op CT scan protocol for the tibia aspect consisted of measuring (fig. 4):

On one hand, the angle formed by a line perpendicular to the transversal axis of the tibial baseplate passing through its center and the line linking this center to the middle of the ATT.

On the other hand, the distance between 2 lines perpendicular to the transversal axis of the baseplate, one passing through the center of the baseplate and the other through the middle of the ATT (SFHG protocol). Two measurements could then be taken. In the medial group, the average distance between the center of the ATT and the center of rotation was +7.3mm (minimum 1, maximum 16mm), and the average angle of rotation was +18.8° (internal tibial baseplate rotation) (minimum +1°, maximum +36°).

In the lateral group, the average distance between the center of the ATT and the center of rotation was +1.4mm (minimum -5, maximum +7mm) and the average angle of rotation was +2.11° (internal rotation) (minimum -14°, maximum +19°). In 6 out of 15 cases, the tibial baseplate was in external hyperrotation: the ATT was medial in comparison to the middle of the baseplate. The lateral approach led to positioning of the tibial baseplate in increased external rotation compared to the medial approach ($p<0.0001$) (fig. 5).

We thus confirm external rotation of the tibial baseplate is more significant using a lateral than a medial approach. The cause is probably better exposure of the tibial plateau. This external rotation favors good patella positioning.

In conclusion, approach may influence patellar tracking. This is partly a direct influence (lengthening of the lateral retinaculum), but mainly an indirect influence: lateral exposure allows an optimal rotation of the tibial implant.

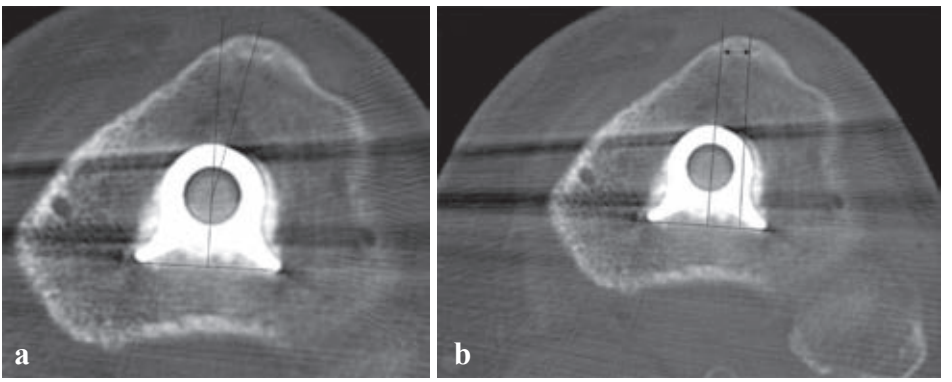
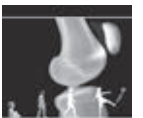


Fig. 4:
a) Measurement of rotation angle of the tibial part between a line passing through the center of the keel perpendicular to the transversal axis of the baseplate and a line between the center of the keel and the middle of the ATT.
b) Measurement of the distance between the line perpendicular to the transversal axis of the baseplate passing through the center of the keel and its parallel passing through the center of the ATT.



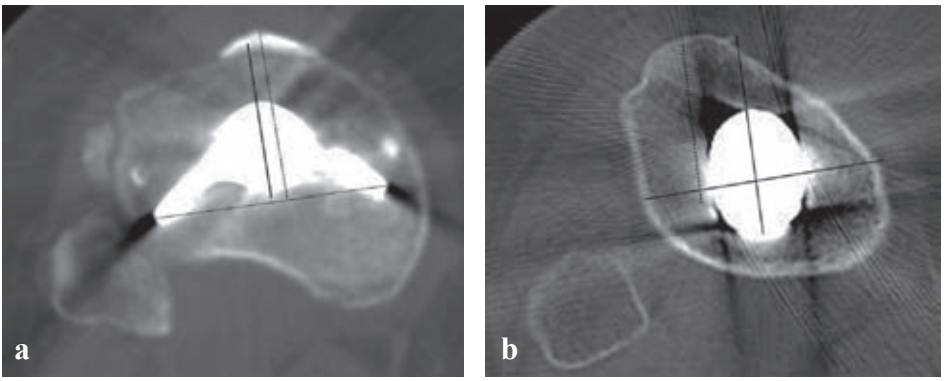


Fig. 5:
a) Example of external rotation by the lateral approach (The ATT is even medialized in relation to the center of the keel).
b) And internal rotation by the medial approach.

LITERATURE

[1] ABADIE P, GALAUD B, MICHAUT M, FALLET L, BOISRENOULT P, BEAUFILS P. Distal femur rotational alignment and patellar subluxation: a CT scan in vivo assessment. *Orthop Traum : Surg Res 2009 ;95: 267-71.*

[2] AMMARI T, ZNIBER B, BOISRENOULT P, CHARROIS O, PERREAU M AND BEAUFILS P. Patellar position and lateral approach for total knee arthroplasty in degenerative knees with lateral femoropatellar arthrosis. *Rev Chir Orthop Reparatrice Appar Mot 2005; 91: 215-21.*

[3] ARNOLD MP, FRIEDERICH NF, WIDMER H, MULLER W. Patellar substitution in total knee prosthesis, important? *Orthopäde, 1998, 27, 637-41.*

[4] BARRACK RL, BERTOT AJ, WOLFE MW. Patellar resurfacing in total knee arthroplasty: a prospective randomised double blind study with five to seven years of follow-up. *J Bone Joint Surg (Am), 2001, 83, 1376-81.*

[5] BEAUFILS P, ABOUCHAYA A. Les reprises pour l'appareil extenseur. In: Les reprises de prothèses totales de genou. Symposium sous la direction de P. Burdin et D. Huten (Réunion annuelle de la SOFCOT 2000). *Rev Chir Orthop, 2001, 87 (suppl au n° 5), 151-6.*

[6] BERGER RA, CROSSETT LS, JACOBS JJ, RUBASH HE. Malrotation causing patellofemoral complications after total knee arthroplasty. *Clin Orthop Relat Res 1998; 356: 144-53.*

[7] BERRY DJ, RAND JA. Isolated patellar component revision of total knee arthroplasty. *Clin Orthop, 1993, 286, 110-5.*

[8] BINDELGLASS DF, VINCE KG. Patellar tilt and subluxation following subvastus and parapatellar approach in total knee arthroplasty. Implication for surgical technique. *J Arthroplasty, 1996, 11, 507-11.*

[9] BOYD AD, EWALD FC, THOMAS WH, POSS R, SLEDGE CB. Long term complications after total knee arthroplasty with or without resurfacing of the patella. *J Bone Joint Surg (Am), 1993, 75, 761-81.*

[10] Brick GW, Scott RD. The patellofemoral component of total knee arthroplasty. *Clin Orthop, 1988, 231, 163-78.*

[11] BURKI H, VON KNOCH M, HEISS C, DROBNY T, MUNZINGER U. Lateral approach with osteotomy of the tibial tubercle in primary total knee arthroplasty. *Clin Orthop, 1999, 362, 156-61.*

[12] CHAN KC, GILL GS. Postoperative patellar tilt in total knee arthroplasty. *J Arthroplast, 1999, 14, 300-4.*

[13] CHANG CH, CHEN KH, YANG RS, LIU TK, Muscle torques in total knee arthroplasty with subvastus and parapatellar approaches. *Clin Orthop, 2002, 398, 189-95.*

[14] CILA E, GUZEL V, OZALAY M, RAN J, SIMSEK SA, KANATH U. Ozturk, Subvastus versus medial parapatellar approach in total knee arthroplasty. *Arch Trauma Surg 265-8002, 122.*

[15] DOOLITTLE KH, TURNER RH. Patellofemoral problems following total knee arthroplasty. *Orthop Rev, 1988, 17, 696-702.*

[16] ENGH GA, HOLT BT, PARKS NL. A midvastus muscle-splitting approach for total knee arthroplasty. *J Arthroplasty 1997, 12 : 322.*

[17] FAURE BT, BENJAMIN JB, LINDSEY B, VOLZ RG, SCHUTTE D. Comparison of the subvastus and paramédian surgical approaches in bilateral knee arthroplasty. *J Arthroplasty 1993, 8, 511-6.*

[18] FIRESTONE TP, TEENY SM, KRACKOW KA. The clinical and roentgenographic results of cementless porous-coated patellar fixation. *Clin Orthop, 1991, 273, 184-9.*

[19] FREEMAN MA, SAMUELSON KM, ELIAS SG, MARIORENZI LJ, G OKCAY EI, TUKE M. The patellofemoral joint in total knee protheses: design considerations. *J Arthroplasty, 1989, 4 (suppl), 69-74.*

[20] GORE DR, SELLINGER DS, GASSNER KJ, GLAESER ST. Subvastus approach for total knee arthroplasty. *Orthopaedics 2003, 26, 33-5.*

[21] HOFMANN AA, PLASTER RL, MURDOCK LE. Subvastus (southern) approach for primary total knee arthroplasty. *Cl Orthop 1991, 269 :70-7.*

[22] LAUGHLIN RT, WERRIES BA, VERHULST SJ, H AYES JM. Patellar tilt in total knee arthroplasty. *Am J Orthop, 1996, 25, 300-4.*

[23] MAC MAHON MS, SCUDERI GR, GLASHOW JL, SCHARF SC, MELTZER LP, SCOTT WN. Scintigraphic determination of patellar viability after excision of infrapatellar fat pad and/or lateral retinacular release in total knee arthroplasty. *Clin Ortho, 1990, 260, 10-16.*

[24] MATSUEDA M, GUSTILO RB. Subvastus and medial parapatellar approaches in total knee arthroplasty. *Clin Orthop, 2000, 371, 161-8.*

[25] MERRILL A, RITTER MA, STEVEN A, HERBST BA, KEATING EM, FARIS PM, MEDING JB. Patellofemoral complications following total knee arthroplasty (Effect of a lateral release and sacrifice of the superior lateral geniculate artery). *J Arhthroplasty, 1996, 11, 368-72.*

[26] MERRILL A, RITTER MA. Patellar complications in total knee arthroplasty. *Clin Orthop, 1999, 367, 149-57.*

[27] MERTL P, JARDE O, BLEJWAS D, VIVES P. L'abord latéral du genou avec relèvement de la tubérosité tibiale pour la chirurgie prothétique. *Rev Chir Orthop, 1992, 78, 264-7.*

[28] PASSERON D, GAUDOT F, BOISRENOULT P, FALLET L, BEAUFILS P. Does lateral versus medial exposure influence total knee tibial component final external rotation? A CT based study. *Orthop Trauma: Surg Res. 2009; 95: 420-4.*

[29] RANAWAT CS. The patellofemoral joint in total condylar knee arthroplasty. *Clin Orthop, 1986, 205, 93-9.*

[30] RAND JA. Current concept review: the patellofemoral joint in total knee arthroplasty. *J Bone Joint Surg (Am), 1994, 76, 612-20.*

[31] RITTER MA, KEATING EM, FARIS PM. Post operative patellar complications with or whithout lateral release during total knee arthroplasty. *Clin Orthop, 1987, 219, 163-8.*

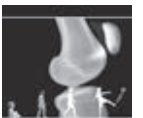
[32] RITTER MA, KEATING EM, FARIS PM. Clinical, roentgenographic, and scintigraphic results after interruption of the superior lateral genicular artery during total knee arthroplasty. *Clin Orthop, 1989, 248, 145-51.*

[33] SCAPINELLI R. Studies on the vasculature of the human knee joint. *Acta Anatom, 1968, 70: 305-31.*

[34] SCUDERI G, SCHARF SH, MELTZER LP, SCOTT WN. The relationship of lateral releases to patella viability in total knee arthroplasty. *J Arthroplasty, 1987, 2, 209-14.*

[35] VIELPEAU C, HULET C, TALLIER E, LOCKER B. La voie d'abord antéro-latérale dans les prothèses totales du genou pour gonarthrose sur genu varum. *Ann Orthop Ouest, 2000, 34, 67-71.*

[36] WETZNER SM, BEZREH JS, SCOTT RD, BIERBAUM BE, NEWBERG AH. Bone scanning in the assessment of patellar viability following knee replacement. *Clin Orthop, 1985, 199, 215-19.*



COMMENT OPTIMISER L'ESPACE ANTÉRIEUR DANS LES PTG ?

M. BONNIN

L'espace antérieur dans les prothèses totales du genou (PTG) peut être défini comme l'espace conditionnant le fonctionnement de l'articulation fémoro-patellaire. Il s'agit d'un espace théorique, prolongeant le concept plus classique "d'espace en extension" et "d'espace en flexion", spécifiques de l'articulation fémoro-tibiale.

L'espace "antérieur" peut également être subdivisé en deux espaces distincts selon que le genou soit en flexion ou en extension. Chacun de ces espaces est conditionné par les coupes osseuses réalisées lors de l'intervention (Table 1). En pratique, une approche dynamique de la fonction fémoro-patellaire, prenant en compte la cinématique articulaire, imposerait plutôt d'analyser l'espace à mi-flexion. D'autre part, le design prothétique, souvent peu anatomique au niveau de la trochlée, conditionne largement la course rotulienne.

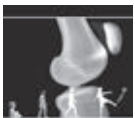
Nous n'aborderons ici que les facteurs dépendant du chirurgien, lors d'un resurfaçage rotulien.

L'ÉPAISSEUR DE LA RÉSECTION ROTULIENNE

L'épaisseur de la résection rotulienne est un point important influençant les contraintes et la cinématique rotulienne. Plusieurs auteurs ont montré qu'une augmentation de l'épaisseur rotulienne après PTG était nocive. Hsu dans une étude *in vitro* a observé que les contraintes fémoro-patellaires étaient augmentées de 10 à 20 % par rapport au genou sain, en cas de stricte reproduction de l'épaisseur rotulienne [6]. En cas d'augmentation d'épaisseur de 2 mm, les forces observées à 90° de flexion étaient augmentées de 31 %. En cas de rotule plus fine

Table 1 : Influence des coupes osseuses sur l'espace fémoro-patellaire (ou "espace antérieur" en flexion et en extension).

Facteurs influençant l'espace antérieur		
	Extension	**Flexion**
1-Epaisseur de coupe rotulienne	✔	✔
2-Orientation de la coupe rotulienne	✔	✔
3-Epaisseur de coupe trochléenne	✔	
4-Rotation de la pièce fémorale	✔	
5-Niveau de coupe fémorale distale		✔



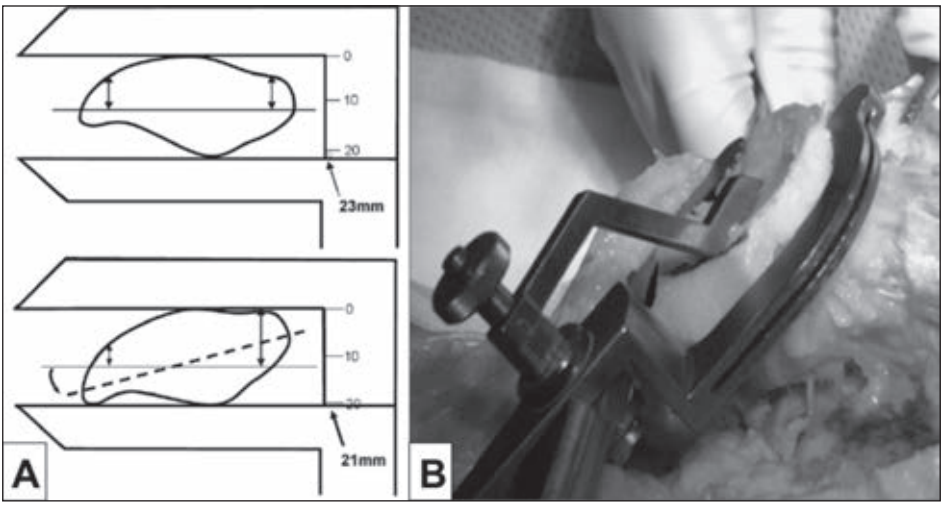


Fig. 1 : La mesure de l'épaisseur rotulienne doit être méticuleuse (A) et réalisée dans plusieurs quadrants de la rotule (B). D'après C. Anglin [2] avec autorisation.

de 2 mm, elles étaient diminuées de 34 % par rapport au genou sain. De même, cet auteur observait que toute augmentation d'épaisseur augmentait le risque de bascule rotulienne. Ce dernier point a été confirmé *in vivo* par Youm qui rapporte une bascule rotulienne significativement augmentée dans un groupe de 35 patients dont l'épaisseur rotulienne était augmentée de 1 mm ou plus, comparés à 111 patients dont l'épaisseur rotulienne était diminuée ou restaurée [13]. De même Laughlin, sur une série de 89 PTG consécutives a observé une épaisseur rotulienne significativement augmentée chez les 46 % ayant une bascule rotulienne externe au dernier recul [9].

De ces observations on peut donc conclure qu'il est important de veiller à ne pas augmenter l'épaisseur rotulienne avec une résection "taille pour taille", voire de diminuer de 1 ou 2 mm l'épaisseur globale. Cette règle doit toutefois être modulée :

- Afin d'éviter une fracture de la rotule resurfacée, il est impératif de conserver une épaisseur d'os minimum, qui pour Reuben est de 15 mm. En cas de rotule très fine, il est donc préférable de sous-couper quitte à augmenter l'épaisseur rotulienne globale [11]. Cette situation est particulièrement fréquente dans les populations asiatiques : l'épaisseur rotulienne moyenne est en effet de 23 à 30 mm dans les populations occidentales, 20 à 23 mm dans la population Malaysienne [12], 21 mm chez les Chinois [7], et les Coréens [13].
- La mesure peropératoire de la rotule peut être soumise à des variations liées à la technique de mesure, source d'erreur de niveau de coupe et/ou de coupes obliques (fig. 1). Afin d'éviter les incertitudes de mesure Pagnano recommande de réaliser la mesure dans les quatre quadrants de la rotule et de reproduire l'épaisseur moyenne.

L'ORIENTATION DE LA COUPE ROTULIENNE

De nombreux auteurs ont montré qu'une résection asymétrique de la rotule entraîne un risque accru de complications fémoro-patellaires, comprenant fractures, instabilité, bascules et conflits. Pagnano et Trousdale ont même rapporté 52 % de complications ou douleurs rotu-

liennes dans une série de 21 PTG avec coupe rotulienne asymétrique [10]. Dans une étude *in vitro*, Anglin a noté que l'asymétrie de coupe rotulienne est un facteur capital, mais qu'elle n'influence vraiment la course rotulienne qu'au-delà de 15° [1, 2]. A l'inverse, Chin, sur une série de 39 instabilités rotuliennes réopérées ne retrouve une asymétrie de coupe rotulienne que dans un cas, alors qu'une coupe insuffisante, conduisant à une rotule trop épaisse est notée chez 8 patients [4].

En pratique, la plupart des auteurs confirment l'importance de la précision de la coupe rotulienne mais certains considèrent qu'il est préférable de réaliser une coupe légèrement asymétrique, laissant plus d'os du côté médial afin de reproduire une asymétrie "physiologique" de 5° à 7.5° [3, 8].

LA RÉSECTION TROCHLÉENNE

L'espace antérieur est largement conditionné par le versant trochléen de l'articulation fémoro-patellaire. Or si le chirurgien garde une marge de manœuvre du côté rotulien, il n'en est pas de même du côté fémoral. L'épaisseur de la trochlée est fixée par le design de l'implant et diminuer la saillie expose au risque de fragiliser la corticale fémorale antérieure tandis que l'augmenter expose au risque de conflits d'engagement de la rotule.

Compte tenu des variations importantes de saillie trochléenne (fig. 2), on comprend qu'il est difficile de restaurer l'espace antérieur natif [5]. Quand à compenser dans la coupe rotulienne, cela peut être risqué car la coupe rotulienne influe également sur l'espace antérieur en flexion.

En pratique, la marge du chirurgien n'est que de quelques millimètres au niveau de la saillie trochléenne compte tenu des limites des implants actuels.

Nous avons étudié l'influence des variations de l'espace antérieur, *in vivo* sur une série de 114 PTG (Noetos fixe, cimentée, Tornier SA). Les variations de saillie trochléenne au niveau latéral influencent le gain de score douleur du score KOOS (r=0,168 et p=0,037) ainsi que la flexion mesurée à un an postopératoire (r=0,149 et p=0,05). Du côté médial, l'influence sur le gain de score douleur était également retrouvée (r=0,152 et p=0,05) mais pas sur la flexion (p=0.,198) (fig. 3). Dans tous ces résultats, une augmentation de saillie trochléenne par rapport à l'état préopératoire diminue le score douleur et la flexion du genou. Nous n'avons pas observé d'effet négatif d'un sous-dimensionnement.

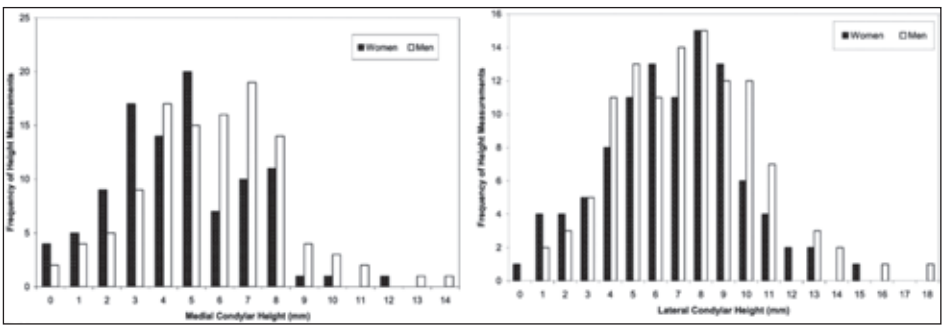
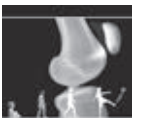


Fig. 2: Distribution de la saillie trochléenne, mesurée sur le compartiment interne (à gauche "medial condylar height") et sur le compartiment externe (a droite "lateral condylar height"), mesurée chez les femmes (colonnes noires) et chez les hommes (colonnes blanches). D'après Frehing [5] avec autorisation.



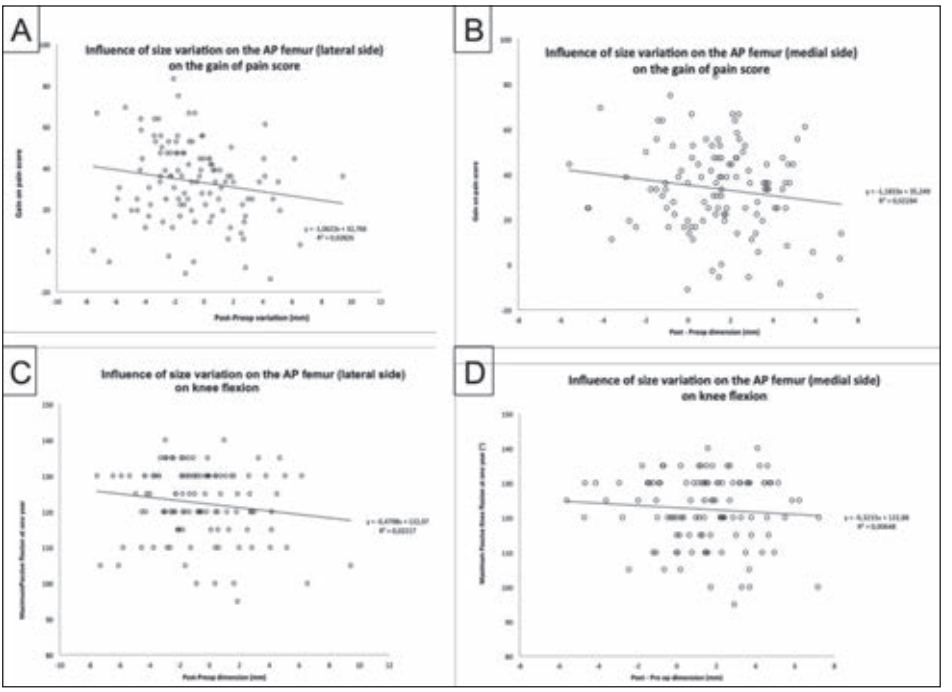


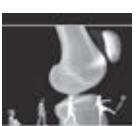
Fig. 3: Influence des variations dimensionnelles antéro-potérieures fémorales (en mm), mesurées sur le compartiment externe (A et C) et le compartiment interne (B et D) sur le score "douleur" du score KOOS (A et B) et sur la flexion du genou (C et D) à un an postopératoire.

LA ROTATION DE LA PIÈCE FÉMORALE

La rotation de la pièce fémorale conditionne la course et la stabilité rotulienne. De nombreuses techniques ont été validées, permettant un alignement sur des repères osseux (ligne de Whiteside, épicondyles ou axe tibial), sur l'espace ligamentaire en flexion, sur des repères radiographiques ou scanographiques. Toutes ces techniques ont pour objet de régler la quantité de rotation externe. En revanche, peu de techniques ou d'instrumentations prennent en compte les conséquences de la rotation fémorale (*i*) sur l'équilibrage ligamentaire en flexion et (*ii*) sur l'espace antérieur.

Pour un angle de rotation donné, si la technique de rotation est basée sur une "sur-coupe" interne (comme dans les cas C de la figure 4), la pièce fémorale sera décalée vers l'avant, ce qui peut entraîner une augmentation involontaire de l'espace antérieur. Si, avec la même technique, on utilise un système prothétique à référence antérieure, l'espace antérieur restera inchangé mais c'est l'espace fémoro-tibial en flexion qui sera modifié.

Si la technique de rotation est basée sur une "sous-coupe" externe (comme en D sur la figure 4), la pièce fémorale sera décalée vers l'arrière, ce qui va diminuer l'espace antérieur.



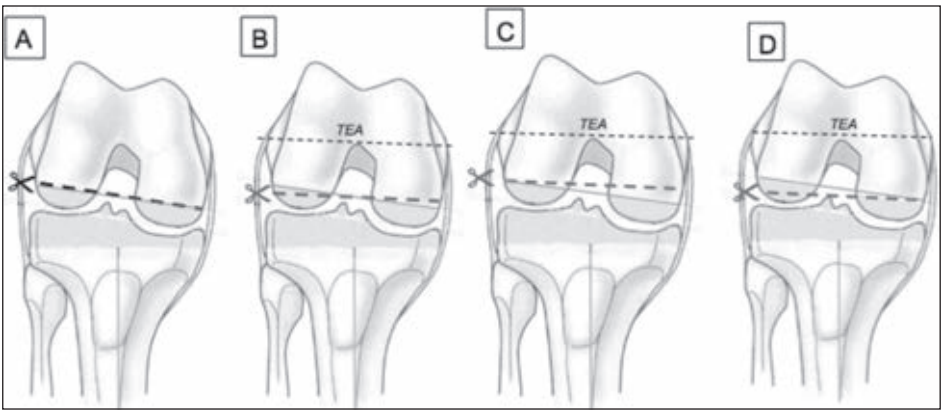


Fig. 4: Les conséquences de la rotation externe fémorale sur l'équilibrage ligamentaire et sur la taille de l'implant prothétique, pour un angle de rotation donné, seront différentes selon la technique utilisée. A) Pas de rotation fémorale, coupe parallèle à la ligne bicondylienne postérieure. B) rotation obtenue par une rotation du guide autour du centre du genou entraînant une sous-coupe externe et une surcoupe interne. C) Rotation obtenue exclusivement par une surcoupe interne. D) Rotation obtenue exclusivement par une sous-coupe externe. D'après : M. Bonnin, A. Amendola, J. Bellemans, S. NacDonald et J. Menetrey, The knee joint: Surgical techniques and Strategy. Springer Paris 2012.

NIVEAU DE COUPE FÉMORALE DISTALE

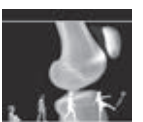
L'espace fémoro-patellaire en flexion dépend de l'épaisseur rotulienne mais également – et surtout – du niveau de la ligne bicondylienne distale. Lors de l'implantation d'une PTG, l'un des objectifs classiques est de tenter de restaurer le niveau de l'interligne articulaire, ce qui doit théoriquement garantir la reproduction des contours fémoraux.

Il est toutefois fréquent d'utiliser le niveau de coupe fémorale distale comme moyen d'ajustement de la stabilité en flexion.

Or toute sur-coupe distale (pour éviter un flexum) raccourci anatomiquement le fémur et donc diminue les contraintes fémoropatellaires en flexion.

A l'inverse, toute sous-coupe distale (pour corriger une laxité en flexion ou un recurvatum), rallonge anatomiquement le fémur, ce qui augmente immédiatement les contraintes fémoro-patellaires (fig. 5).

Par ailleurs, dans un genou de morphotype "classique", l'interligne articulaire est incliné avec deux à trois degrés de varus tibial, compensé par un valgus fémoral équivalent. Lorsque la coupe fémorale distale est réalisée de manière classique à 90°, la résection condylienne distale est asymétrique. La plupart des guides ajustant la coupe sur le condyle interne plus proéminent, la résection condylienne latérale est diminuée, et ne correspond pas à l'épaisseur prothétique implantée (fig. 6). La plupart des prothèses "classiques" implantées rallongent donc le fémur au niveau du compartiment externe, augmentant ainsi les contraintes fémoro-patellaires. Seules les prothèses reproduisant l'asymétrie condylienne distale pourraient donc éviter ce phénomène.



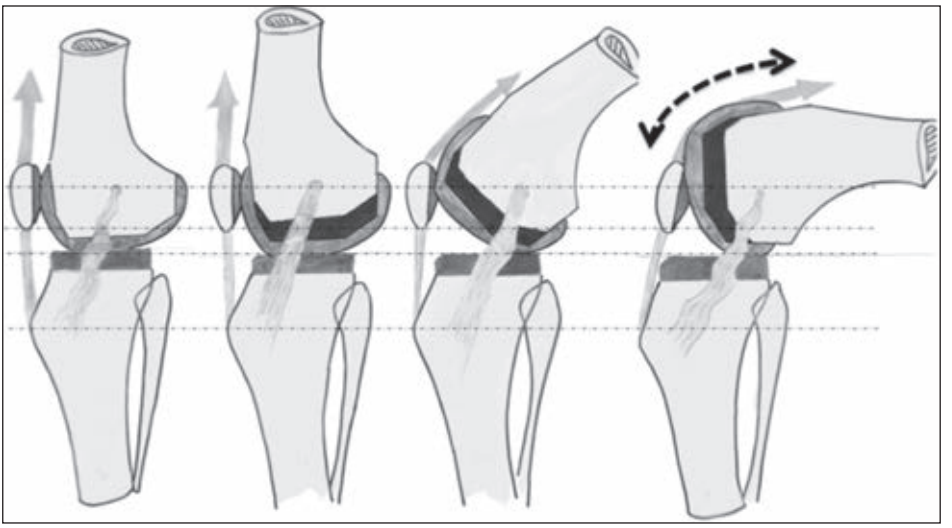


Fig. 5: Toute modification de niveau de coupe fémorale distale modifie la longueur du fémur, ce qui influence l'espace fémoropatellaire en flexion. Toute "distalisation" de la coupe, comme représenté sur le schéma, entraîne un accroissement des contraintes rotuliennes.

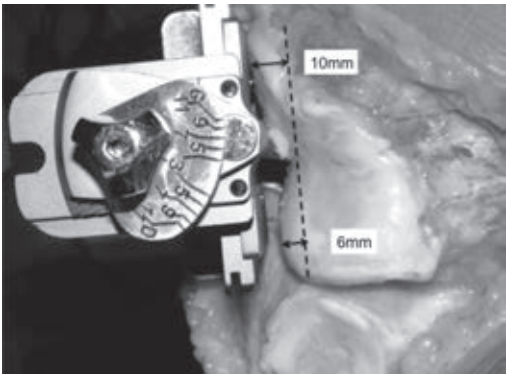
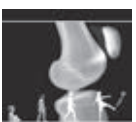


Fig. 6: La coupe fémorale distale (ici avec une instrumentation Tornier HLS-Noetos) est réalisée à 90° par rapport à l'axe fémoral mécanique. Le niveau de coupe – réalisée de manière "standard" à 10 mm – est déterminé par rapport au condyle fémoral le plus saillant, c'est-à-dire le condyle interne. L'épaisseur de la pièce fémorale étant symétrique (10 mm), le condyle externe est "distalisé", ce qui entraîne une augmentation des contraintes patellaires.

CONCLUSION

L'espace antérieur du genou ou "espace fémoro-patellaire" est généralement insuffisamment pris en compte lors de l'implantation d'une PTG primaire. Il est important de comprendre et d'analyser tous les facteurs opératoires qui, de manière indirecte, retentissent sur cet espace. En 2012, la reconstruction prothétique reste encore trop éloignée de l'anatomie et ceci de manière particulièrement marquée au niveau trochléo-patellaire. L'émergence de nouvelles technologies doit nous permettre, dans la prochaine décennie, d'avancer dans la direction d'une adaptation de plus en plus ajustée des implants.



RÉFÉRENCES

[1] ANGLIN C, BRIMACOMBE JM, HODGSON AJ, MASRI BA, GREIDANUS NV, TONETTI J, WILSON DR Determinants of patellar tracking in total knee arthroplasty. *Clin Biomech (Bristol, Avon) 23 (7):900-10 (2008).*

[2] ANGLIN C, FU C, HODGSON AJ, HELMY N, GREIDANUS NV, MASRI BA Finding and defining the ideal patellar resection plane in total knee arthroplasty. *J Biomech 42 (14): 2307-12 (2009).*

[3] CHAN KC, GILL GS Postoperative patellar tilt in total knee arthroplasty. *J Arthroplasty 14 (3): 300-4 (1999).*

[4] CHIN KR, BAE DS, LONNER JH, SCOTT RD Revision surgery for patellar dislocation after primary total knee arthroplasty. *J Arthroplasty 19 (8):956-61 (2004).*

[5] FEHRING TK, ODUM SM, HUGHES J, SPRINGER BD, BEAVER WB Jr Differences between the sexes in the anatomy of the anterior condyle of the knee. *J Bone Joint Surg Am 91 (10):2335-41 (2009).*

[6] HSU HC, LUO ZP, RAND JA, AN KN Influence of patellar thickness on patellar tracking and patellofemoral contact characteristics after total knee arthroplasty. *J Arthroplasty 11 (1):69-80 (1996).*

[7] JIANG CC, YIP KM, LIU DH Patellar thickness in total knee replacement. *J Formos Med Assoc 93 (5):417-20, (1994).*

[8] KAWANO T, MIURA H, NAGAMINE R, URABE K, MATSUDA S, MAWATARI T, MORO-OKA T, IWAMOTO Y Factors affecting patellar tracking after total knee arthroplasty. *J Arthroplasty 17 (7):942-7 (2002).*

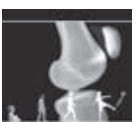
[9] LAUGHLIN RT, WERRIES BA, VERHULST SJ, HAYES JM Patellar tilt in total knee arthroplasty. *Am J Orthop (Belle Mead NJ) 25 (4):300-4, (1996).*

[10] PAGNANO MW, TROUSDALE RT Asymmetric patella resurfacing in total knee arthroplasty. *Am J Knee Surg 13 (4): 228-33, (2000).*

[11] REUBEN JD, MCDONALD CL, WOODARD PL, HENNINGTON LJ. Effect of patella thickness on patella strain following total knee arthroplasty. *J Arthroplasty 6 (3): 251-8* (1991).

[12] SULAIMAN AS, NORDIN S Measurement of patellar thickness in relation to patellar resurfacing. *Med J Malaysia 60 Suppl C:41-4, (2005).*

[13] YOUM YS, CHO WS, WOO JH, KIM BK The effect of patellar thickness changes on patellar tilt in total knee arthroplasty. *Knee Surg Sports Traumatol Arthrosc 18 (7):923-7 (2010).*



WHETHER TO RESURFACE THE PATELLA IN TOTAL KNEE ARTHROPLASTY: contributions from an arthroscopic and radiographic femoropatellar study of a total knee prosthesis - A series of 17 cases

O. COURAGE, L. MALEKPOUR

INTRODUCTION

Nowadays, the indication for patellar resurfacing in prosthetic surgery is controversial. While the decision for resurfacing ought to depend on risk-benefit reports for the patient, this seems more linked to the personal preferences of the surgeon but also depends on training.

A less studied question concerns the notion of patellofemoral congruency between femoral implants and native patellae, which is subject to anatomical variation. Does poor congruency have consequences for the result?

The goal of this study is to observe, by arthroscopy and radiology, the congruency between native patellae of different shapes with a single femoral implant, and to evaluate the differences in clinical results at one year in terms of the congruency observed.

MATERIALS

17 patients, who underwent surgery for a total knee prosthesis, were included between September 2010 and September 2011. Voluntary and informed consent was obtained from each of these patients.

The surgical indication for prosthetic replacement was degenerative arthrosis impinging on daily life, refractory to initial medical treatment.

Exclusion criteria included obesity with BMI > 29, Maldague stage 3 lateral patellar subluxations or an irreducible genu valgum, severe patellofemoral chondropathy.

The prosthetic implant selected for each patient was the tricompartmental KNEETEC prosthesis from Tornier with its inverted trochlear dome possessing a constant radius of curvature over its whole contour, with a raised lateral side (fig. 1).

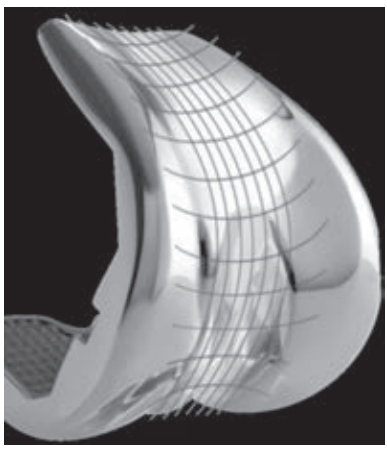
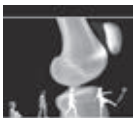


Fig. 1



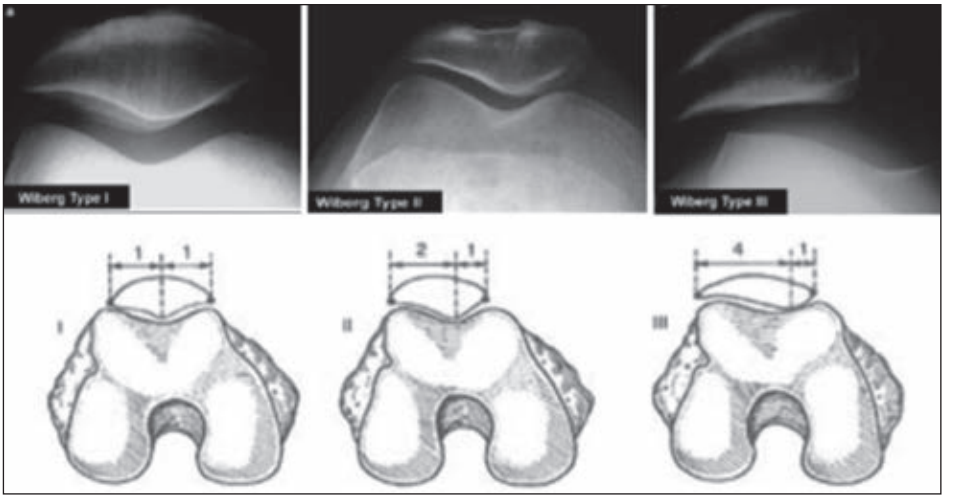


Fig. 2:
Wiberg type 1: medial and lateral surfaces concave and comparable in size
Wiberg type 2: medial surface smaller than the lateral surface with a concave appearance
Wiberg type 3: medial surface largely reduced

METHOD

Each initial patellofemoral contour was analysed in order to rank the patellae according to the Wiberg classification: types 1, 2, 3 (fig. 2).

The access chosen was the same for each patient and performed by the same surgeon: mid-vastus with preservation of the Hoffa (fig. 3).

At the lower part of the incision the patellar tendon is carefully preserved up to its point of termination on the TTA. If necessary for exposure, subperiosteal infracentrimetric displacement of it was performed.

With the leg in extension, the patella is systematically everted and dislocated.
The Hoffa is spared and does not restrict exposure so much.

The arthroplasty is performed according to the original "Lyon" technique. The tibial implant is centred on the TTA. Femoral sectioning is carried out with ligament balancing performed in flexion then extension, taking care to avoid any notching or femoral offset.

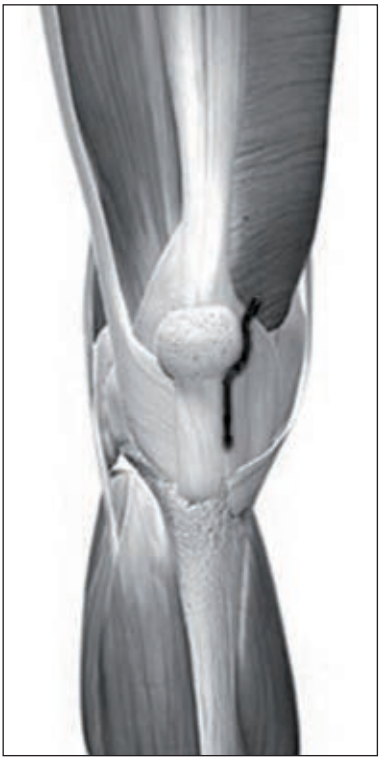
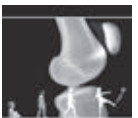


Fig. 2



Photographs under arthroscopy were taken once the femoral and tibial implants were cemented. This arthroscopy is performed without water, in order not to compromise the coupling between the non-resurfaced patella and the femoral plate. The arthroscope was passed via an anteromedial portal with the knee at 45° of flexion.

For characterisation of patellofemoral congruency, both arthroscopic and radiological congruencies were taken into account. The arthroscopic images were analysed according to whether or not the patellar surface was in contact with the entire centre of the trochlear groove in the knee at 45° of flexion, the radiographs according to alignment and the overall contact surface of the patella.

Arthroscopic congruency was defined as a harmonious contact of the patella with the entire trochlear groove, radiographic congruency, as a harmonious contact of the patella with the entire femoral plate.

A patellofemoral pair was only considered congruent if, and only if, congruency was simultaneously found in the arthroscopic images and the radiographic examination.

At the end of this analysis, each patellofemoral pair was then classified into 2 groups: congruent and non-congruent.

The patients were reviewed again at 1 year, with a clinical and radiological evaluation.

The evaluation at 1 year included the Knee Society score, the presence of anterior pain syndrome, the presence of patellofemoral syndrome, a global evaluation of satisfaction ("very satisfied", "satisfied", "not very satisfied", "dissatisfied").

A Fisher test was used for evaluation of significance of the results.

RESULTS

Out of the 17 knees included, 12 presented as congruent patellofemoral pairs and 5 as non-congruent (fig. 4a, b).

Among these 5 non-congruent pairs, 2 patellae were classed as Wiberg 2, and 3 as Wiberg 3 (table 1).

Table 1

	Wiberg 1 =4/17	Wiberg 2 =8/17	Wiberg 3 =5/17
Congruent = 12/17	4	6	2
Non-congruent = 5/17	0	2	3

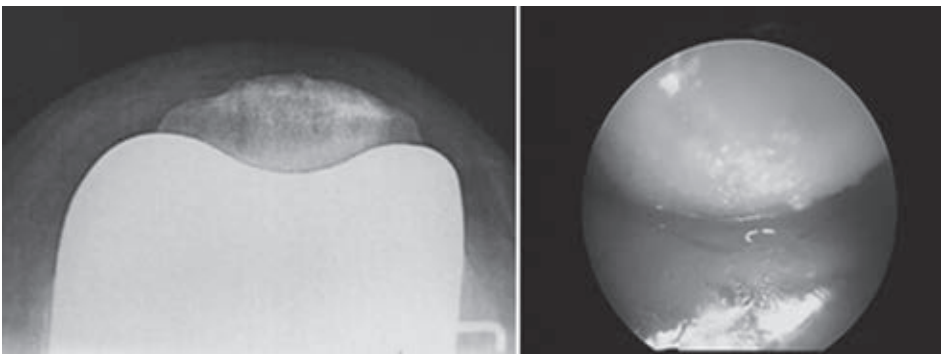
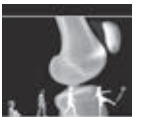


Fig. 4a: Congruent patellofemoral pair: radiographic and arthroscopic.



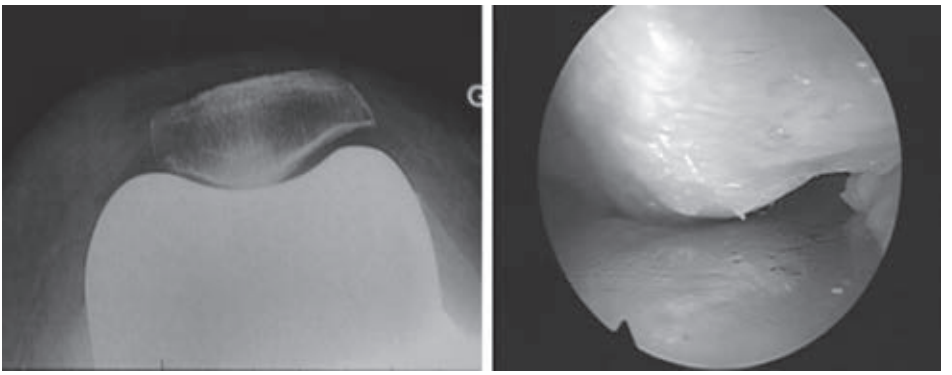


Fig. 4b: Incongruent patellofemoral pair: radiographic and arthroscopic.

The 17 patients initially included could be reviewed at 1 year with a clinical examination and radiographic assessment (front, profile and patellofemoral contour).

Out of these 17 patients, 8 presented with anterior knee pain (table 2a), of which half presented with patellofemoral syndrome (4/17). Out of these 4 patients presenting with patellofemoral syndrome, 3 presented with patellofemoral incongruency (arthroscopic and/or radiographic) (table 2b). The level of satisfaction at one year was 11/17 (or 64%) with a significantly higher level in patients with patellofemoral congruency (10/17 vs 1/17) (p=0.027).

The mean Knee Society score was 69.91 [45-83] for the congruent group versus 46.6 [33-75] for the non-congruent group (table 3).

table 2a

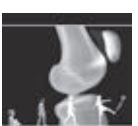
	Anterior pain	Absence of anterior pain
Congruent 12	5	7
Non-congruent 5	3	2

table 2b

	Patellofemoral pain	Absence of patellofemoral pain
Congruent 12	1	11
Non-congruent 5	3	2

table 3

	Knee Society score	Satisfaction
Congruent patellae = 12	45; 58; 78; 81; 70; 83; 78; 64; 76; 67; 68; 71 Mean = 69.91	5 “very satisfied” 5 “satisfied” 2 “not very satisfied”
Non-congruent patellae = 5	33; 38; 75; 42; 45 Mean = 46.6	1 “very satisfied” 2 “not very satisfied” 2 “dissatisfied”



DISCUSSION

Despite numerous publications, still today, opinions differ concerning patellar resurfacing. Even if several prospective randomised studies were able to identify a lack of significant difference [1, 3, 4, 6], numerous other series and authors put forward the contrary opinion [9, 10, 12, 14, 15], each of the techniques being subject to its own risks [5, 7, 8, 11]. Some of these divergences come from the same evaluation of patients in each series [2]. Bonin *et al.* moreover advise using subjective scores rather than the IKS to better pinpoint the patellofemoral origin of the pain.

Few studies have been concerned with the consequences of choice of implant; Waters *et al.* [13] attempted to bring to light a change in the post-operative results dependent on the shape of the trochlea inserted. If this shows that anterior pain appears more relevant in the case of non-resurfacing, it also underlines the fact that this pain would seem more relevant in the case of "inhospitable" trochlear shape, particularly in the case of trochlear crossover and asymmetry.

In our case study, the prosthesis utilised is a prosthesis initially designed to harbour a dome-shaped polyethylene patellar component, with a constant radius of curvature of the femoral plate over the entire contour, allowing us to be assured that the purely static conditions of congruency, for an identical patellar cross-section, remain unchanged at an angle of flexion other than the chosen 45°.

Despite the significant depth of the femoral plate selected, contrary to Waters, there is no evidence for much patellofemoral pain. We appear to encounter this pain primarily in cases of Wiberg 3 patellae.

Patellae with shapes classed as Wiberg 1 and 2 appear to benefit from flexibility of adaptation to the trochlea both arthroscopically and radiographically as well as clinically. They indeed possess a certain degree of rotational and translational freedom on the axial plane, sufficient, taking into account their shape, to obtain a stable patellofemoral pair. Will this "constrained" adaptation be confirmed over the long term?

Our study has already revealed the clinical outcome at one year of these constraints in the case of initial "major incongruency". Thus pre-operative detection of these future "major incongruencies" would be of interest, in order not to promote the occurrence of premature pain.

We suggest pre-operative planning, using drawings of transverse sections of the prosthetic femoral plate. These drawings, applied to a systematic patellofemoral contour, would allow verification of the absence of major incongruency with the native patella, and thus the need to review the decision for resurfacing or patelloplasty (fig. 5).

Good congruency in this case during planning, non-resurfacing method can be suggested. By contrast, in the case of predictable incongruency, it will doubtlessly be wiser to perform resurfacing of the patella or patelloplasty.

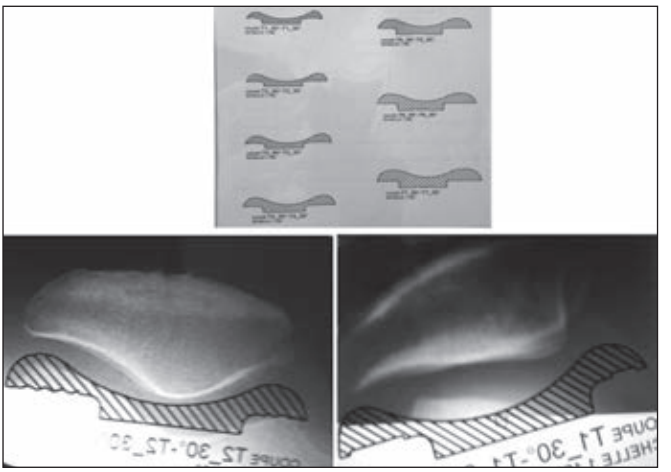


Fig. 5

CONCLUSION

The debate on whether or not to resurface the patella in prosthetic surgery of the knee has to this day not been clear-cut. It is notable that the pain from patellofemoral conflict is difficult to isolate.

Despite the low number of patients, our study being dedicated to only one trochlear shape while focusing on any patellar shape, it was found that pain at 1 year is accompanied by major incompatibility between the native patella and prosthetic plate.

We suggest detecting major patellofemoral incongruencies pre-operatively, particularly through utilisation of drawings to assist in the decision between non-resurfacing, patelloplasty or resurfacing of the patella.

ABSTRACT

Nowadays the indication for patellar resurfacing in prosthetic knee surgery is controversial, the literature results on the subject being contradictory at times. While the decision for resurfacing ought to depend on risk-benefit reports for the patient, this seems rather linked to the personal preferences of the surgeon dependent on his training.

A less studied question concerns the notion of patellofemoral congruency between femoral implants and native patellae, which is subject to anatomical variation. Thus the question may be raised concerning the consequences of congruency defects on the results.

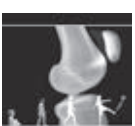
The goal of this study is to observe, by arthroscopy and radiology, the congruency between native patellae of different shapes with a single femoral implant, and to evaluate the differences in clinical results at one year relating to the congruency observed.

The evaluation was carried out at the same centre. The operation was performed by the same surgeon. 17 patients, having given their informed consent, were selected according to predefined inclusion and exclusion criteria.
The access, mid-vastus with preservation of the Hoffa, was the same for each patient.

The 17 patients were separated into three groups depending on the shape of their patella according to the Wiberg classification. The prosthetic implant used was the same for each operation, the patellae were not resurfaced. Patellofemoral congruency was evaluated by arthroscopy at the moment of closure, with the knee at 45° of flexion, without water. Each case was analysed by radiography.

The patients were reviewed again on average at 1 year, with clinical and radiological evaluation.

Patellae of different shapes with the same femoral plate do not systematically result in patellofemoral incongruency. Patients presenting with patellofemoral incongruency appear to present with increased anterior pain at 1 year.



LITERATURE

[1] BARRACK RL, BERTOT AJ, WOLFE MW, *et al.* Patellar resurfacing in total knee arthroplasty. A prospective, randomized, double-blind study with five to seven years of follow-up. *J Bone Joint Surg Am, 2001,83A: 1376-81.*

[2] BONIN N, DESCHAMPS G, DEJOUR D. Intérêt d'un score subjectif dans l'évaluation fémoro-patellaire des Prothèses Totales du genou. Revue de Chirurgie Orthopédique et Traumatologique [Relevance of subjective score in patellofemoral evaluation of total knee arthroplasty. Review of orthopaedic and trauma surgery]. *Volume 93, number S9071 pages 79-80.*

[3] BONIN N, MERCADO J, DESCHAMPS G, *et al.* Lyon Ortho Clinic - Dracy le fort Resurfacing vs Not Resurfacing the Patella in Total Knee Arthroplasty - 4 year results, *13th ESSKA Congress, 21-24 May 2008, Porto (Portugal).*

[4] BURNETT RS, HAYDON CM, RORABECK CH *et al.* Patella resurfacing versus nonresurfacing in total knee arthroplasty: results of a randomized controlled clinical trial at a minimum of 10 years followup. *Clin Orthop Relat Res. 2004; 428: 12-25.*

[5] GOLDBERG VM, FIGGIE HE, III, INGLIS AE, FIGGIE MP, SOBEL M, KELLY M, KRAAY M. Patellar fracture type and prognosis in condylar total knee arthroplasty. *Clin Orthop Relat Res. 1988; 236: 115-22.*

[6] KEBLISH PA, VARMA AK, GREENEALD AS. Patellar resurfacing or retention in total knee arthroplasty. A prospective study of patients with bilateral replacements. *J Bone Joint Surg Br. 1994; 76: 930-7.*

[7] LOMBARDI AV Jr, ENGH GA, VOLTZ RG, ALBRIGO JL, VIRGINIA A, BRAINARD BJ. Fracture/dissociation of the polyethylene in metal-backed patellar components in total knee arthroplasty. *J Bone Joint Surg Am. 1988; 70: 675-9.*

[8] MOCHIZUKI RM, SCHURMAN DJ. Patellar complications following total knee arthroplasty. *J Bone Joint Surg Am. 1979; 61: 879-83.*

[9] NAEDER HELMY MD, CAROLYN ANGLIN PHD, NELSON VAND Al. To Resurface or not to Resurface the Patella in Total Knee Arthroplasty *Clin Orthop Relat Res (2008) 466: 2775-83.*

[10] PAKOS EE, NTZANI EE, TRIKALINOS TA *et al.* Patellar resurfacing in total knee arthroplasty. A meta-analysis. *J Bone Joint Surg Am. 2005 Jul; 87(7): 1438-45.*

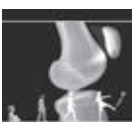
[11] EMERSOn RH, Jr, HEAD WC, MALININ TI. Reconstruction of patellar tendon rupture after total knee arthroplasty with an extensor mechanism allograft. *Clin Orthop Relat Res. 1990; 260: 154-61.*

[12] WATERS TS. Clinical Ant. Knee Pain Score. *J Bone Joint Surg Am, 85-A(2): 212-7, 2003.*

[13] WATERS TS, BENTLEY. The influence of trochlear configuration on anterior knee pain following knee replacement with and without patellar resurfacing – a prospective randomised study. *J Bone Joint Surg British Volume, Vol 84-B, Issue SUPP III, 320-21.*

[14] WATERS TS, BENTLEY G. Patellar resurfacing in total knee arthroplasty: a prospective, randomized study. *J Bone Joint Surg; 85A: 212-217; 2003.*

[15] WATERS TS, BENTLEY G. Anterior knee pain following knee replacement: comparison of patellar resurfacing and non-resurfacing in PFC and PFC Sigma knee replacements. #189. *American Academy of Orthopedic Surgeons 70th Annual Meeting. Feb. 5-9, 2003. New Orleans.*



PRISE EN CHARGE DES RUPTURES CHRONIQUES DE L'APPAREIL EXTENSEUR SUR PROTHÈSE TOTALE DE GENOU (FRACTURE DE ROTULE EXCLUE)

Surgical management of chronic rupture of extensor mechanism after total knee arthroplasty (patellar fracture excluded)

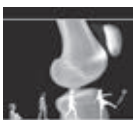
G. DEMEY, S. LUSTIG, E. SERVIEN,
F. TROUILLET, E. GANCEL, P. NEYRET

Les ruptures chroniques de l'appareil extenseur sur prothèse totale de genou (PTG) sont un challenge thérapeutique. En cas de rupture chronique du tendon rotulien, l'existence d'un tissu fibreux en regard du tendon rotulien et la rétraction du tendon quadricipital avec apparition d'une rotule haute empêchent toute suture directe du tendon rotulien avec ou sans renfort. Inversement, en cas de rupture chronique du tendon quadricipital, la présence d'une rotule basse avec rétraction du tendon rotulien ne permet pas cette suture ou du moins la cicatrisation est obtenue au prix d'un résultat fonctionnel très médiocre avec une raideur en flexion importante.

Dans le cadre d'une rupture chronique sur prothèse totale de genou, il convient d'ajouter à ces difficultés l'âge du patient (souvent supérieur à 65 ans), une qualité osseuse médiocre, des antécédents chirurgicaux du même genou, la présence d'un bouton rotulien, la fréquence des pathologies rhumatoïdes, une arthrose ou une pathologie du genou controlatéral…

Ces raisons nous font préférer l'allogreffe de l'appareil extenseur dans cette situation. Les avantages sont une économie de temps chirurgical mais aussi l'absence de lésions iatrogènes sur le genou controlatéral liées au prélèvement. Ceci est aussi utile dans des circonstances particulières comme une maladie du collagène, ou un antécédent sur le genou controlatéral. Si la qualité du tissu peut apparaître parfois moins bonne qu'une autogreffe, il faut souligner la possibilité de prendre des greffons plus larges. La longueur du tendon rotulien et de la rotule doit cependant être adaptée au patient en cas d'allogreffe et des mesures précises dans ce sens doivent être faites (radiographies millimétrées, radiographies du genou controlatéral).

La technique chirurgicale est très semblable à celle décrite par Henri Dejour pour les reconstructions avec autogreffe. La préparation de l'allogreffe est néanmoins de taille supérieure étant donné l'absence de morbidité au site donneur. La greffe utilisée est un appareil extenseur complet avec tubérosité tibiale antérieure, tendon rotulien, rotule et tendon quadricipital. La baguette rotulienne sera volontiers plus large et taillée avec des bords concaves, ce qui permet des prélèvements tendineux de largeur supérieure. La fixation utilise un fil métallique appuyé sur une vis à tête plate associé à des agrafes pour la baguette tibiale, ainsi que des fils métalliques sur la rotule. Une bandelette PDS est agrafée en distal et suturée tout le long de l'appareil extenseur, genou en flexion à 90°. Elle permet de protéger l'allogreffe des contraintes lors de la flexion.



La rééducation est très progressive et le travail des amplitudes articulaires en flexion est particulièrement précautionneux et progressif étant donné le délai de consolidation des baguettes osseuses allongé en cas d'allogreffe. Des radiographies successives permettent de surveiller cette consolidation et d'autoriser une flexion plus importante.

Cette allogreffe est donc la greffe de choix dans notre expérience en cas de rupture chronique de l'appareil extenseur après prothèse totale de genou ; que cette rupture intéresse le tendon rotulien ou le tendon quadricipital.

La rupture du tendon quadricipital avec rotule très basse représente un cas particulier et la technique dans ce cas sera adaptée. En effet, la rétraction du tendon rotulien est telle que la rotule devient un "obstacle" à la chirurgie. Nous conseillons dans ce cas de procéder à une patellectomie totale tout en conservant les fibres de l'appareil extenseur et d'utiliser une allogreffe massive avec un tendon rotulien complet, une rotule entière légèrement désépaissie et un tendon quadricipital avec tendon du droit antérieur complet.

Enfin, une alternative à l'allogreffe de l'appareil extenseur serait la reconstruction ou le renfort avec une bandelette de type Synthetic Mesh. Cette technique est en cours d'évaluation dans notre pratique.

Chronic rupture of the extensor mechanism after total knee arthroplasty (TKA) is a therapeutic challenge. In case of chronic rupture of the patellar tendon, the existence of a fibrous tissue, the quadriceps tendon retraction and the appearance of a patella alta do not allow a direct suture of the patellar tendon with or without reinforcement. Inversely, in case of chronic rupture of the quadriceps tendon, the presence of a patella baja with retraction of the patellar tendon does not allow this suture. The healing would be achieved with a poor functional outcome and an important lack of flexion.

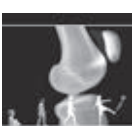
Furthermore, in these patients, should be considered the patient's age (often above 65 years), apoor bone quality, asurgical history of the knee, the presence of a patellar resurfacing, the frequency of rheumatic diseases, osteoarthritis, or pathology of the contralateral knee…

These multiple reasons make the extensor mechanism allograft our first choice. There are benefits as time saving and also absence of iatrogenic lesions in the contralateral knee. This is also useful in special circumstances such as collagen disease, or a history of the contralateral knee. If the quality of tissue may appear worse than autograft, we must emphasize the possibility of taking larger grafts. The length of the patellar tendon and patella of the allograft must be assessed to obtain proper length and specific radiographs are necessary (millimeter radiographs and radiographs of the contralateral knee).

The surgical technique is very similar to that described by Henri Dejour for autograft reconstruction. The preparation of the allograft is larger. The transplant uses complete extensor mechanism with tibial tuberosity, patellar tendon, patella and quadriceps tendon. The patellar bone is wider and cut with concave edges, allowing wider tendon harvest. Fixation uses a wire on a flat head screw associated with staples for the tibial tuberosity and metallic wires for the patella. PDS reinforcement is sutured distally throughout the extensor mechanism, at 90° flexion. It protects the allograft from constraints during flexion.

Rehabilitation is very progressive and range of motion in flexion is particularly cautious and progressive due to the time of consolidation of an allograft. Successive radiographs assess the consolidation and greater flexion will be allowed.

In brief, allograft is the graft of choice in our experience for chronic rupture of the extensor mechanism after TKA, either patellar tendon or quadriceps tendon ruptures.



The rupture of the quadriceps tendon associated with a severe patella baja represents a particular situation. The surgical technique may be slightly modified. The retraction of the patellar tendon is such important that the patella is an «obstacle» to the procedure. We suggest to perform a total patellectomy while retaining the fibers of the extensor mechanism and to use a massive allograft with a complete patellar tendon, a complete patella and a quadriceps tendon.

At last, an alternative to the extensor mechanism allograft reconstruction would be the reinforcement with Synthetic Mesh. This procedure is being evaluated in our practice.

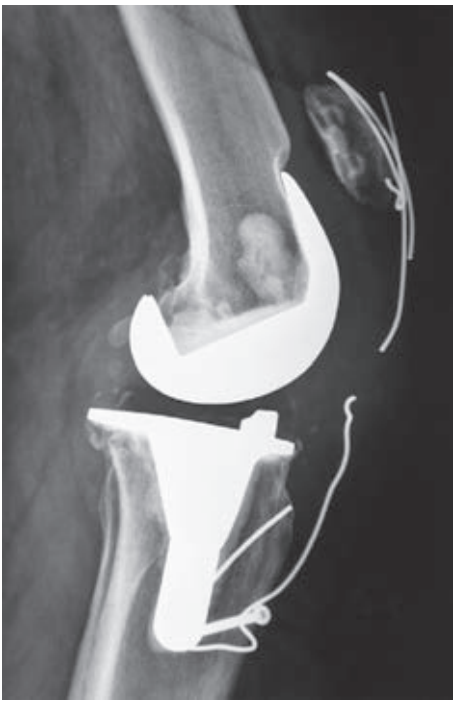


Fig. 1: Chronic rupture of the patellar tendon after TKA, preoperative X-rays (case 1).

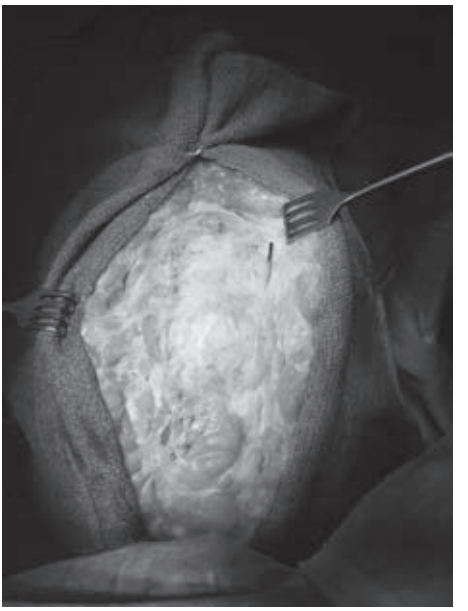


Fig. 2: Peroperative view (case 1)

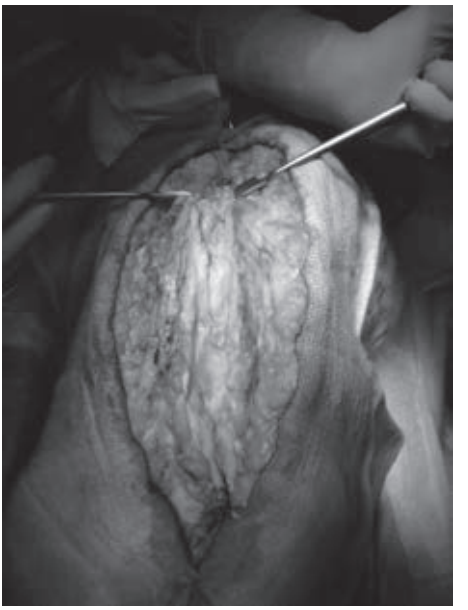
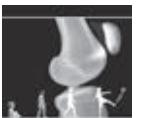


Fig. 3: Preparation before reconstruction (case 1)



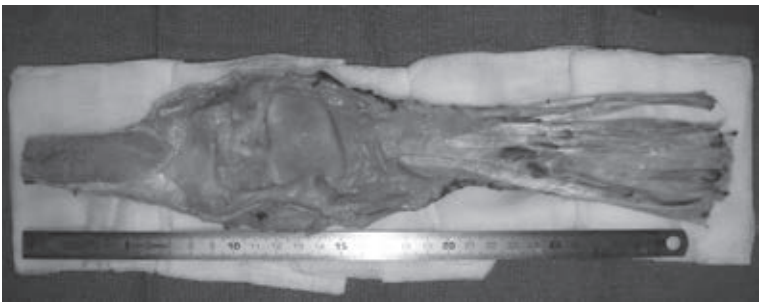


Fig. 4: Allograft of the extensor mechanism before harvesting (case 1)

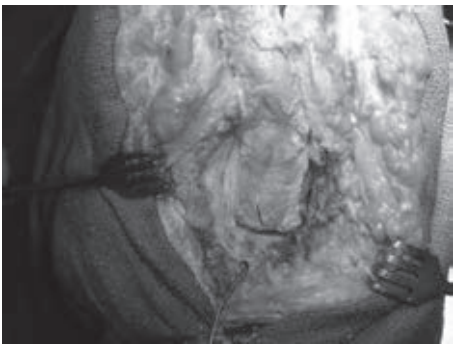


Fig. 5: Tibial fixation (case 1)

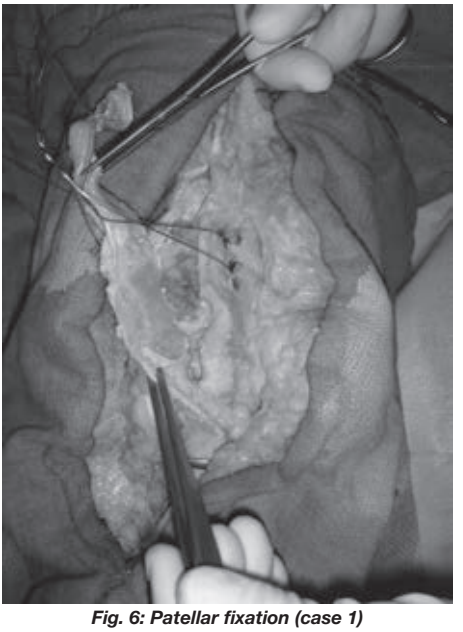


Fig. 6: Patellar fixation (case 1)

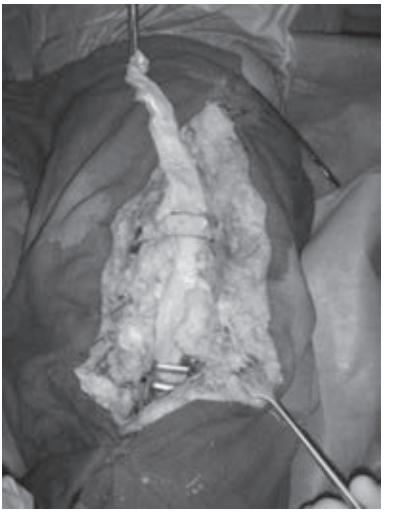


Fig. 7: Proximal fixation (case 1)

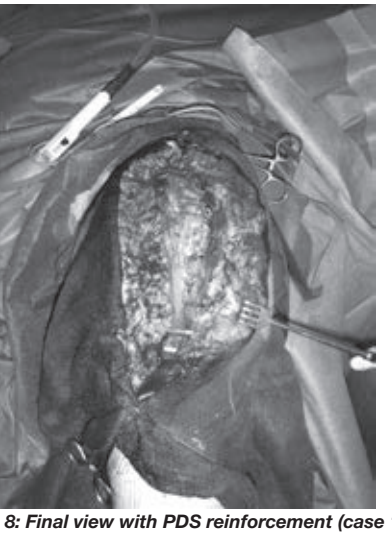


Fig. 8: Final view with PDS reinforcement (case 1)

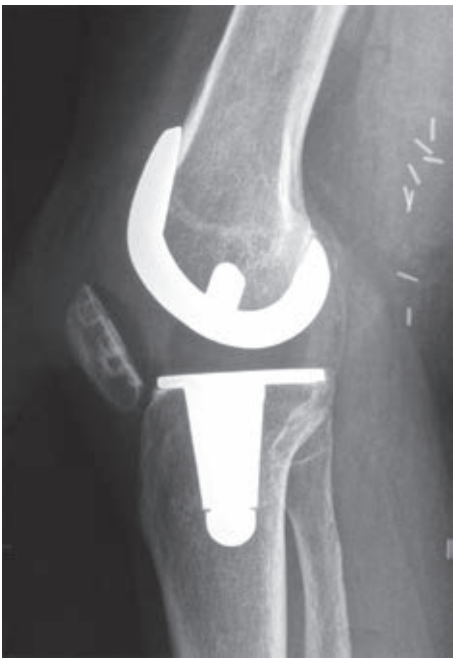


Fig. 9: Quadriceps tendon rupture with severe patella baja, preoperative X-rays (case 2)

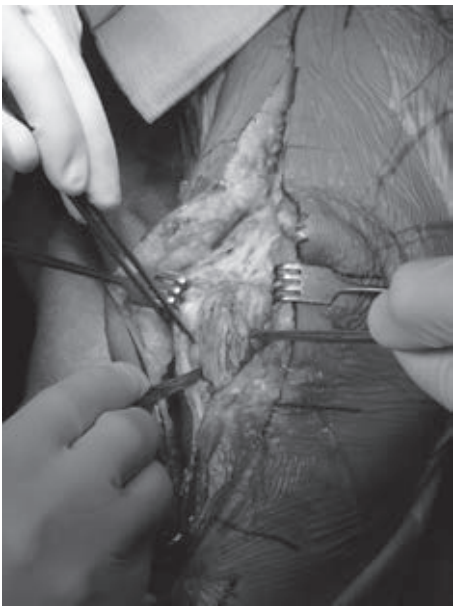


Fig. 10: Total patellectomy (case 2)

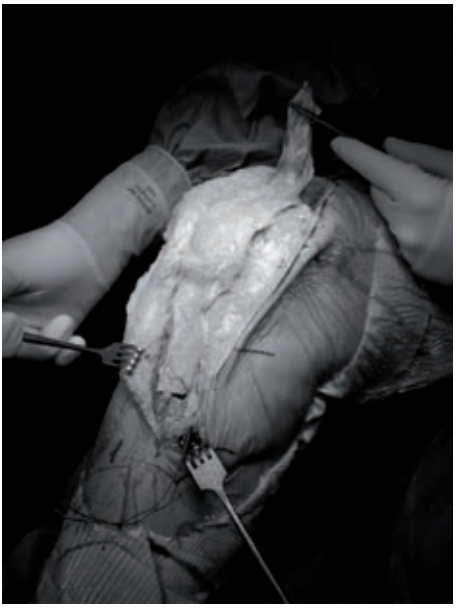


Fig. 11: Allograft reconstruction (case 2)

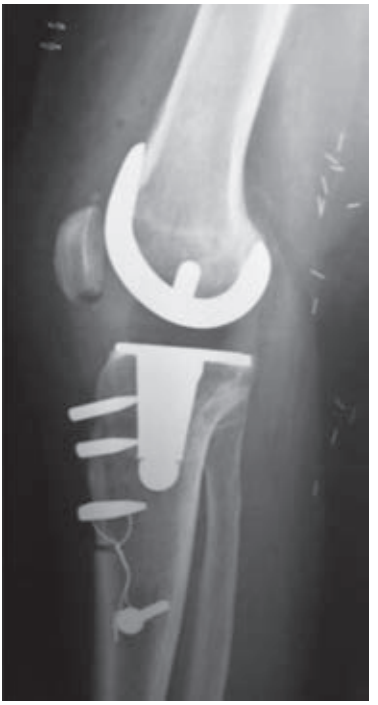
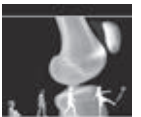


Fig. 12: Postoperative X-rays (case 2)



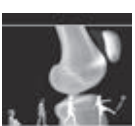
LITERATURE

[1] A EL GUINDY, S LUSTIG, E SERVIEN, C FARY, F WEPPE, G DEMEY, P NEYRET. Treatment of chronic disruption of the patellar tendon in Osteogenesis Imperfecta with allograft reconstruction. *The Knee (2011):18(2): 121-4.*

[2] P NEYRET, G DEMEY. Traité de chirurgie du genou. Rupture chronique de l'appareil extenseur. *Editions Elsevier (2011): 317-32.*

[3] JA BROWNE, AD HANSSEN. Reconstruction of patellar tendon disruption after total knee arthroplasty: results of a new technique utilizing synthetic mesh. *JBJS (Am) (2011):93(12): 1137-43.*

[4] H DEJOUR, S DENJEAN, P NEYRET. Treatment of old or recurrent ruptures of the patellar ligament by contralateral autograft. *RCO (1992): 78(1): 58-62.*



PATELLAR TENDINOSIS : THERAPEUTICS OPTIONS

R. BASTOS-FILHO, V.B. DUTHON, R. BADET

Patellar tendinosis or "jumper's knee" is a common pathology in athletes with an incidence of 13 to 20% in this group [1, 2]. This condition affects athletes in many sports implying jumping and heavy landing, rapid acceleration or deceleration and kicking, such as basketball, volleyball, soccer, tennis, long jump and high jump [1, 2]. A recent epidemiological study showed that the average duration of substantial pain problems and reduced function is nearly three years [3]. So, it has a tendency to be chronic and, the incidence of retiring from sport is as high as 53% for elite athletes [4].

Several studies demonstrate little or no inflammation in tendons exposed to overuse [5, 6, 7] and the patellar tendon is not different [8]. Patellar tendinopathy appears to result from an imbalance between the protective/regenerative changes and the pathologic responses elicited by tendon overuse. The net result is tendon degeneration, weakness, tearing, and pain.

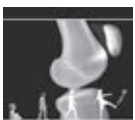
Many different treatment have been described for patellar tendinopathy but no consensus exists regarding the best way to treat this condition:

Eccentric training

Patellar tendinosis is typically managed conservatively in the early stages. Non-operative management includes activity restriction or modification, ice, anti-inflammatory, drugs, eccentric training, massage or taping. Bahr *et al.* [9] performed a randomized controlled trial comparing operative versus conservative management of patellar tendinosis. They concluded that although surgical treatment and eccentric training can produce significant improvement in terms of pain and function scores, only about half of all patients will be able to return to sport within one year after treatment with each option, and fewer still will have relief of all symptoms. No advantage was demonstrated for surgical treatment compared with eccentric training. Eccentric training should be tried for twelve weeks before open tenotomy is considered for treatment of patellar tendinopathy.

Twelve weeks eccentric course of eccentric strengthening exercises is more effective than a traditional concentric strengthening program for treating patellar tendinopathy in recreational athletes [10].

Studies demonstrate statistically significant better results in groups that performed eccentric training compare to groups experiencing pulsed ultra-sound and transverse friction [11], or groups treated by corticosteroid injections [12].



Low intensity pulsed ultrasound (LIPUS)

Warden *et al.*, in a high-quality article showed that LIPUS was not shown to provide any benefit beyong that of the placebo [13] in agreement with another investigation suggesting that LIPUS is questionable in the treatment of tendinopathies [14].

Extra-corporeal shockwave (ECSW) therapy

Wang *et al.*, in a study with a low quality score, compared ECSW with conservative treatment regarding pain reduction and concluded, that the results had an indeterminate basis [15].

Ultrasound-guided sclerosing injections

The etiology for patellar tendinopathy remains uncertain. One explanation would be neovascularization and anticipation of pain by accompanying nerves. So, injection agent such a polidocanol [16, 17] in the area with neovessels should reduce pain. Some studies [18, 19] report reduced pain in patients with patellar tendinopathy after this treatment. But, contradictory results were recently presented in a retrospective study [20] of 48 patients with chronic Achilles tendinopathy revealing less promising results than expected.

Further investigation is required to proove the effectiveness of this treatment.

Open surgery

Multiple open surgical procedures have been described for patellar tendinosis but the most common involves an open excision of the diseased portion of the patellar tendon. Treatment of the paratendon greatly varies, as does post-operative immobilization. In addition, some surgeons have described drilling, debriding or excising the distal pole of the patella [21, 22]. Kaeding *et al.* performed a recent systematic review of the literature concerning surgical treatment [23]. Eight out of nine studies reported >80% success with a variety of principally open techniques. However, success was not always clearly defined and the return to high-level sports varied from 46 to 91%. They also found no difference with respect to whether bony work was performed, whether the paratenon was closed, or whether the patient was immobilized after operation.

The greatest limitation to open surgical procedures appears not to be related to their success rates but to the protracted post-operative course of immobilization and the time needed to return to sport. This can range from six to ten months [24, 25, 26].

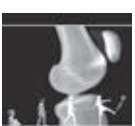
Arthroscopic surgery

Arthroscopic surgical procedures have been described with good results and a faster return to function [26, 27]. At the time of arthroscopic surgery, different authors have described a simple excision of the distal patellar pole, or a tendinosis debridement, or both. All of these studies have reported success rates in terms of symptomatic improvement (>85%). Coleman *and al.* reported that only 46% of their patients returned to sports [26].

Muccioli *et al.*, in a systematic review of the literature, reported that minimally invasive arthroscopically assisted procedures don't have statistically significant better results when compared to open surgery in the treatment of chronic proximal patellar tendinopathy [28].

Platelet-rich plasma injection

Platelet-rich plasma injection for the management of patellar tendinopathy, have shown promising results [29].



Filardo *et al.* [30] investigated the use of autologous growth factors through platelet-rich plasma injections as a treatment of jumper's knee: the clinical results were encouraging and suggested this method could be useful for the treatment of chronic patellar tendinopathy, even in difficult recalcitrant to physiotherapy.

Gosens T. *et al.* showed that there may be a relationship between prior treatments (ethoxysclerol, steroid or surgery) and the benefits of PRP. There were no significant improvement in those who had been treated before compared to those who only received PRP [31].

CONCLUSIONS

Physical training (particularly eccentric training) has appeared to be the treatment of choice for patients suffering from patellar tendinopathy and remains the standard of care. However, exercise type, frequency, load and dosage require further investigations. In cases of failed non-operative treatment, open or arthroscopic debridement of the pathologic tissue has shown improvement in high-level athletes [32].

Ultrasound can be excluded for patellar tendinopathy treatment and shockwave therapy, sclerosing injections and PRP require additional research before their use can be recommended.

LITERATURE

[1] WITVROUW E, BELLEMANS J, LYSENS R *et al.* Intrinsic risk factors for the development of patellar tendinitis in an athletic population; A two-year prospective study. *Am J Sports Med 2001; 29: 190-95.*

[2] JARVINEN M. Epidemiology of tendon injuries in sports. *Clin Sports Med 1992; 11:493-504.*

[3] LIAN OB, ENGEBRETSEN L, BAHR R. Prevalence of jumper's knee among elite athletes from different sports: A cross-sectional study. *Am J Sports Med. 2005; 33: 561-7.*

[4] KETTUNEN JA, KUJALA UM, KAPRIO J. Lower limb function among former elite male athletes. *Am J Sports Med 2001; 29:2-8.*

[5] KHAN KM, COOK JL, BONAR F *et al.* Histopathology of common tendinopathies. Update and implications for clinical management. *Sports Med. 1999;27: 393-408.*

[6] LJUNG BO, FORSGREN S, FRIDEN J. Substance P and calcitonin gene-related peptide expression at the extensor carpi radialis brevis muscle origin: implications for the etiology of tennis elbow. *J Orthop Res. 1999; 17: 554-9.*

[7] SOSLOWSKY LJ, THOMOPOULUS S, FLANAGAN CL *et al.* Neer Award 1999. Overuse activity injures the supraspinatus tendon in an animal model: a histologic and biomechanical study. *J Shoulder Elbow Surg. 2000;9: 79-84.*

[8] COOK JL, FELLER JA, BONAR SF, KHAN KM. Abnormal tenocyte morphology is more prevalent than collagen disruption in asynptomatic athletes' patellar tendons. *J Orthop Res 2004;22: 334-8.*

[9] BAHR R, FOSSAN B, LOKEN S *et al.* Surgical treatment compared with eccentric training for patellar tendinopathy (Jumper's knee). A randomized, controlled trial. *J Bone Joint Surg [Am] 2006; 88-A: 1689-98.*

[10] JONSSON P, ALFREDSON H. Superior results with eccentric compared to concentric quadriceps training in patients with jumper's knee: a prospective randomized study. *Br J Sports Med 2005; 39: 847-50.*

[11] STASINOPOULOS D, STASINOPOULOS I. Comparisson of effects of exercise programme, pulsed ultrasound and transverse friction in the treatment of chronic patellar tendinopathy. *Clin Reabil 2004; 18(4): 347-52.*

[12] KONGSGAARD M, KOVANEN V, AAGAARD P *et al.* Corticosteroid injections, eccentric decline squat training and heavy slow resistance training in patellar tendinopathy. *Scand J Med Sci Sports 2009; 19(6): 790-802.*

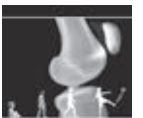
[13] WARDEN SJ, METCALF BR, KISS ZS *et al.* Low-intensity pulsed ultrasound for chronic patellar tendinopathy: a randomized, double-blind, placebo-controled trial. *Rheumatology (Oxford) 2008; 47(4): 467-7.*

[14] KHANNA AN NELMES RT, GOUGOULIAS N *et al.* The effects of LIPUS on soft-tissue healing: a review of literature. *Br Med Bull 2009; 89: 169-82.*

[15] WANG CJ, KO JY, CHAN YS *et al.* Extracorporeal shockwave for chronic patellar tendinopathy. *Am J Sports Med 2007; 35(6): 972-8.*

[16] OHBERG L, ALFREDSON H. Ultrasund guided sclerosis of neovessels in painful chronic Achilles tendinosis: pilot study of a new treatment. *Br J Sports Med 2002; 36(3): 173-5.*

[17] OHBERG L, ALFREDSON H. Sclerosing therapy in chronic Achilles tendon insertional pain-results of a pilot study. *Knee Surg Sports Traumatol Arthrosc 2003; 11(5): 339-43.*



[18] HOKSRUD A, OHBERG L, ALFREDSON H *et al.* Ultrasound guided sclerosis of neovessels in painful chronic patellar tendinopathy: a randomized controlled trial. *Am J Sports Med 2006; 34(11): 1738-46.*

[19] ALFREDSON H, OHBERG L. Neovascularization in chronic painful patellar tendinosis-promising results after sclerosing neovessels outside the tendon challenge the need for surgery. *Knee Surg Sports Traumatol Arthrosc. 2005; 13(2): 74-80.*

[20] VAN STERKENBURG MN, DE JONGE MC, SIEREVELT IN *et al.* Less promising results with sclerosing ethoxysclerol injections for midportion Achilles tendinopathy: a retrospective study. *Am J Sports Med 2010; 38(11): 2226-32.*

[21] AL-DURI ZA, AICHROTH PM. Surgical aspects of patellar tendinitis: Technique and results. *Am J Knee Surg 2001;14: 43-50.*

[22] SMILLIE I. Injuries of the knee joint. *Edimburg: Churchill Livingstone,1962: 264-67.*

[23] KAEDING CC, PEDROZA AD, POWERS BC. Surgical treatment of chronic patellar tendinosis. *Clin Orthop 2006; 455: 102-6.*

[24] JARVINEN M, JOZSA L, KANNUS P *et al.* Histological findings in chronic tendon disorders. *Scand J Med Sci Sports 1997: 7: 86-95.*

[25] PIERETS K, VERDONK R, DE MM *et al.* Jumper's knee: postoperative assessment: a retrospective clinical study. *Knee Surg Sports Traumatol Arthrosc 1999;7: 239-42.*

[26] COLEMAN BD, KHAN KM, KISS ZS *et al.* Open and arthroscopic patella tenotomy for chronic patellar tendinosis. *Am J Sports Med 2000; 28: 183-90.*

[27] LORBACH O, DIAMANTOPOULOS A, PAESSLER HH. Arthroscopic resection of the lower patellar pole im patients with chronic patellar tendinosis. *Arthroscopy 2008;24: 167-73.*

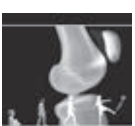
[28] MUCIOLLI GMM, ZAFFAGNINI S, TSAPRALIS K *et al.* Open versus arthroscopic surgical treatment of chronic proximal patellar tendinopathy. A systematic review. *Knee Surg Sports Traumatol Arthrosc 2012 Jun 20.*

[29] TAYLOR DW, PETRERA M, HENDRY *et al.* A systematic review of the use of platelet-rich plasma in sports medicineas a new treatment of tendon and ligament injures. *Clin J Sports Med 2011; 21(4): 344-52.*

[30] FILARDO G, KON E, VILLA SD *et al.* Use of platelet-rich plasma for the treatment of refractory jumper's knee. *International Orthopaedics (SICOT) 2010; 34: 909-15.*

[31] GOSENS T, OUDSTEN BL, FIEVEZ E *et al.* Pain and activity levels before and after platelet-rich plasma injection treatment of patellar tendinopathy: a prospective cohort study and the influence of previous treatments. *International Orthopaedics (SICOT) 2012.*

[32] GEHRON T, JENNIFER AH, MARK DM. What's New in Sports Medicine. *The Journal of Bone and Joint Surg (Am) 2007; 89-A(3): 686.*



ACUTE RUPTURES OF EXTENSOR MECHANISM

V.B. DUTHON, P. NEYRET, E. SERVIEN

INTRODUCTION

The knee extensor mechanism is essential to stand and to walk. It is composed of the quadriceps muscle and its tendon, the patella, and the patellar tendon inserted to the anterior tibial tuberosity (ATT). Those three structures are continuous and form a single biomechanical entity allowing active extension of the knee. Acute quadriceps tendon rupture, transverse and displaced patellar fracture and acute patellar tendon rupture interrupt the continuity of the knee extensor mechanism. A common clinical finding in those three pathologies is an inability to actively extend the knee. Quadriceps and patellar tendon ruptures are rare and 28% are initially undiagnosed [23]. History, clinical exam, radiological exams and treatment of those three pathologies are resumed here.

ACUTE QUADRICEPS TENDON RUPTURES

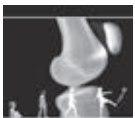
Anamnesis and incidence

Acute quadriceps tendon ruptures are usually found in patients over 40 years-old. The typical history is a knee injury that leads to a period of reduced mobility. Tendon rupture occurs [3-4] weeks later, following sudden eccentric contraction of the quadriceps muscle, while minimal trauma such as descending stairs or stumbling over a pavement. The patient feels a violent pain followed by a functional disability of the lower limb. Systemic diseases which weaken tendons as systemic erythematous lupus must be looked for.

Unilateral ruptures are 15 to 20 times more frequent than bilateral ruptures that are found in patients with a systemic disease as gout, systemic erythematous lupus, rheumatoid arthritis, primary hypoparathyroidism, tuberculosis, syphilis, and acute infection. The male/female ratio is 6/1. Rupture can also be iatrogenous after steroid injection or due to anabolic steroids use in athletes [14].

Diagnosis

The clinical exam is crucial for diagnosis. The patient is unable to achieve a complete active extension of the knee [20]. In supine position, he is unable to rise up the lower limb extended or is unable to hold this position against gravity. While sitting on the border of the exam table (knee flexed at 90°) he is unable to achieve full knee extension. If the retinacula are intact (aponeurosis of vastus medialis and vastus lateralis that insert on the patella), the patient is able to slightly extend the knee, but not



completely. A full incapacity to actively extend the knee is due to a complete rupture of the quadriceps tendon and of the retinacula. By palpation of the quadricipital tendon, a gap is felt, corresponding to an interruption of tendon continuity. 60% of tears occur through the tendon, about 2cm above the patella, and 40% occur at its insertion on the patella. The latter injury is called "osteotendinous tear" and was first described by Albert Trillat. It is due to a periosteal sleeve avulsion at the quadriceps tendon insertion on the patella. Rarely, the tear occurs at the myo-tendinous junction, mainly in patients with a decreased ambulation and muscular hypotrophy. Swelling and hematoma due to the rupture can fill this gap and make its palpation less obvious [7].

The differential diagnosis in a patient unable to fully actively extend the knee is a paralysis of the femoral nerve which can be traumatic or iatrogenic [24].

Diagnosis is first made with anamnesis and clinical exam. Radiological exams can help to confirm and precise the diagnosis. On a standard profile radiograph of the knee, swelling of the soft tissues above the patella can be seen (fig. 1A). Calcifications on the proximal border of the patella are an indirect sign of quadriceps tendinopathy which predisposes the tendon to rupture [6]. The patella is lower than on the controlateral side (patella baja) and may be anteriorely tilted. Echography is a non-invasive, easy and fast diagnostic tool to confirm a partial or complete rupture of the quadriceps tendon. MRI has a high sensitiviy (fig. 1B) and is recommended in cases where a doubt persists after clinical and echographic exam. It also helps to see if the tear is complete (the 4 layers of the tendon are torn) or partial, and if the retinacula are torn [7].

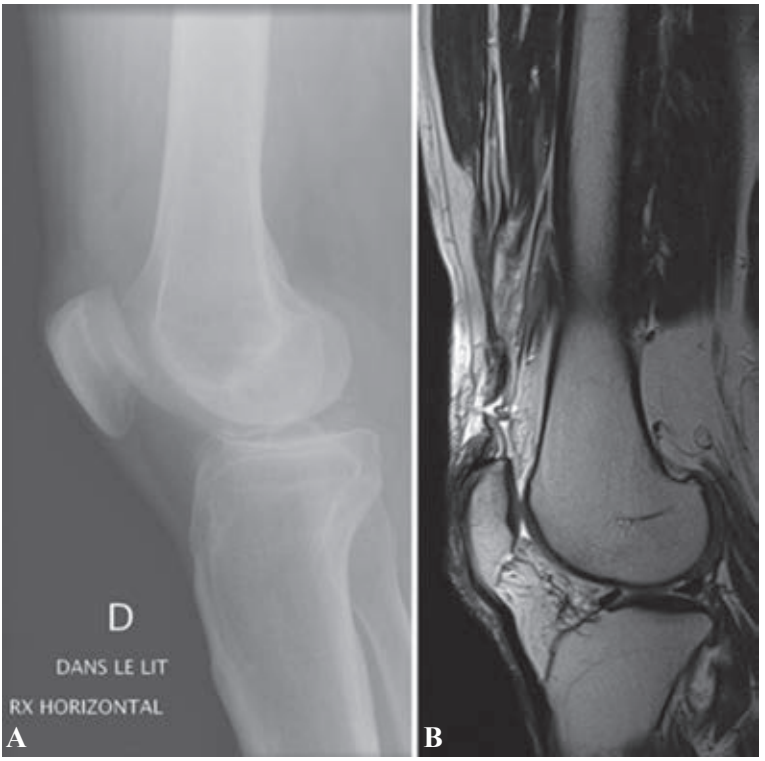


Fig. 1

Traitement

An acute surgical repair gives the best results [7]. Many surgical techniques have been described.

In case of mid-body quadriceps tendon tear, an anatomical end-to-end suture with non-absorbable sutures gives very good results. A median vertical incision is done. The peritenon is incised and the hematoma is cleaned. The tendon stumps are identified. A n° 2 absorbable suture is passed transversally in each stump, 2cm from the tear. Three "U" sutures are done with a nonabsorbable suture (n° 2 Fiberwire). Sutures are tightened with the knee extended and are reinforced by multiple single stitches with absorbable sutures (n° 0 Vicryl) [18].

When the tear occurs at the osteotendinous junction, a median vertical incision is done at the proximal border of the patella to expose the tear. Necrotic or frayed tissues are debrided. Four 2.5 mm transosseous tunnels are done obliquely in the proximal border of the patella. Three "U" nonabsorbable sutures (Fiberwire or Mersuture) are passed through the tunnels and in the quadriceps tendon stump. Knots are done with the knee in extension. Another technique is to baste the proximal tendinous stump with non-absorbable sutures (Krackow-type sutures [12]) which are then passed through vertical transosseous tunnels in the patella. The suture is reinforced by multiple interupted absorbable sutures (n° 0 Vicryl). If torn, the retinacula must also be sutured [18].

After surgery, a 30° knee brace is worn for rest and walking is allowed with a 0° knee brace for 6 weeks. It is mandatory to protect the sutures while the tendon is healing. Passive knee flexion is progressively increased: 0-45° from day 0 to day 15; 0-70° from day 16 to day 30; 0-90° from day 31 to day 45. Full flexion is allowed after 6 months.

Quadriceps amyotrophy is very frequent and difficult to recover in spite of physiotherapy. However, it may not have significant consequences in daily living [23]. Isometric contractions and electrostimulation may prevent this amyotrophy. A secondary rupture can happen but is rare [20].

In case of a delayed diagnosis, repair is more difficult as the quadriceps is retracted and an end-to-end repair is not possible anymore. In such cases, surgical reconstructive techniques have been described: interposition of a tendinous auto- or allograft, tendinous advancement flaps, etc. Results are worse in case of delayed repair or reconstruction and complications rate is higher [13]. Early diagnosis and treatment are mandatory to achieve a good healing and functional recovery.

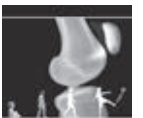
ACUTE PATELLAR FRACTURES

Anamnesis and incidence

Fractures of the patella are more frequent than quadriceps or patellar tendon ruptures. They are due to a direct trauma (fall on the floor with impact of the patella, road accident with direct trauma) or rarely indirect trauma (violent eccentric contraction of the quadriceps while the knee is flexed). In case of car road accident, an associated posterior cruciate ligament tear and ipsilateral hip fracture must be looked for. Many types of patellar fractures exist: longitudinal or transversal, displaced or not. Only the transversal and displaced fractures interrupt the extensor mechanism [16]. In situ transversal fractures and longitudinal fractures (displaced or not) do not interrupt the extensor mechanism.

Diagnosis

Clinical examination of a patient with a transverse displaced fracture of the patella reveals an inability to actively extend the knee, pre-patellar swelling which is the fracture hematoma. The skin overlying the patella has been contused during trauma and may be stretched out by this hematoma, sometimes leading to necrosis. This must absolutely be prevented by early surgical management of the fracture.



Standard knee radiographs (antero-posterior, profile, and axial) are mandatory to evaluate the type and displacement of the fracture. The differential diagnosis is a patella bipartita which is present in 2 to 3% of the population [1]. Computed tomography may help to precise the type and comminution of the fracture. MRI allows evaluation of the patellar cartilage which can be very damaged even in "in situ" fractures.

Treatment

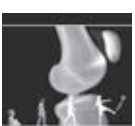
The goal of the treatment is to restore extensor mechanism continuity and the patello-femoral congruency. Treatment depends on the type of fracture : displaced (2-3mm articular step-off or 1-4mm fracture displacement) or not [3]. Non-surgical treatment is only for non-displaced fractures (vertical or transverse) and consists of knee immobilisation with a brace in extension for 4-6 weeks, weight-bearing and isometric quadriceps contractions are allowed. Displaced fractures must be treated surgically (open reduction and internal fixation). Transverse fractures must be fixed adequately to resist quadriceps traction force. Tension band osteosynthesis with two longitudinal parallel K-wires and a fashion-of-eight wire is recommended [17]. A polar cerclage can be added to increase stability [4]. In case of fracture of the patellar nose, the bony fragment can be excised and its patellar tendon fibers reinserted on the patella via transosseous sutures or anchors. In any case, even with very comminutive fracture, patellectomy should be avoided because the quadriceps strength is decreased by more than 49%. Post-operative complications are loss of reduction or fixation (8%), non-union (1%), knee stiffness, patello-femoral arthritis.

ACUTE PATELLAR TENDON RUPTURES

Anamnesis and incidence

Acute patellar tendon ruptures are less frequent than quadriceps tendon ruptures. About 80% of patients with patellar tendon ruptures are less than 40 years-old [23]. Rupture most often occurs at the lower border of the patella and sometimes at its insertion on the anterior tibial tuberosity. As for the quadriceps tendon, mid-body tendon ruptures are rare and often associated to a systemic inflammatory disease, chronic metabolic disorders, anabolic steroid abuse, local steroid injections, and most commonly progressive degenerative processes. In athletes, patellar tendinopathy (jumper's knee) and sequelas of Osgood-Schlatter enthesopathy are risk factors for ruptures [11]. Patellar tendon may tear when a high load is suddently applied to the tendon. The dynamic load during sport is much higher than any static load. In a healthy patient (without any systemic pathology or patellar tendinopathy), the patellar tendon breaking point is 17.5 times the body weight. To compare, climbing stairs loads the patellar tendon of 3.3 times the body weight [19] and jumps 7 to 8 times the body weight. The most frequent mechanism is landing from a jump: deceleration with sudden eccentric contraction of the quadriceps, while the foot is anchored on the ground and the knee is flexed. Patients feel the tear or a painful knee buckling, followed by a functional disability. Direct traumas can also cause patellar tendon tears when the patellar tendon is tight by quadriceps contraction. Bilateral patellar tendon tears are rare and often associated to systemic disease (systemic lupus erythematosus, rheumatoid arthitis, diabetes mellitus, hyperparathyroidy) with chronic inflammation and amyloid deposits in the tendons. Corticosteroids often prescribed in those diseases may alter collagen synthesis and tendons vascularisation [21]. However, a systemic cause of bilateral rupture reported in the literature were found in only 60% of cases [10].

Acute extensor mechanism rupture may be a complication of surgical procedures as total knee arthroplasty, anterior cruciate ligament recontruction with patellar tendon autograft (patellar tendon rupture 0.3%; patellar fracture 0.03%) [2], tibial intramedullary nailing through the patellar tendon.



Diagnosis

Extensor mechanism is interrupted when patellar tendon is torn. The patient is unable to actively extend the knee against gravity, or to hold this position. The patella moves proximally as it is pulled by the quadriceps. A gap is easily palpable where the patellar tendon is torn. Most of the ruptures occur at the inferior pole of the patella, but may also occur in the tendon mid-body or, rarely, at the insertion on the tubercule. The patella is proximally displaced as a result of associated retinacular and capsular disruption caused by the strong pull of the quadriceps mechanism. It is also very mobile when moved medio-laterally.

On standard radiographs, the patella is proximally displaced (patella alta). Axial patello-femoral view shows a "sunrise", the joint line disappears, because of overlapping the patella and the femoral condyles on radiographs. Many radiological indexes have been described to evaluate the height of the patella [22] and to compare it to the contralateral side: the Insall-Salvati index [8], the Blackburne-Peel index, and the Caton-Deschamps index. We prefer using the Caton-Deschamps index as it can be measured on any knee profile radiograph with knee flexion between 10 and 80°, as compared to the Insall-Salvati index which can be measured on 30° of knee flexion radiographs. These indexes are useful for the diagnosis and in post-operative follow-up.

As for the quadriceps tendon, echography and MRI are very useful to confirm and precise the tear (fig. 2) [15]. MRI also allows to diagnose associate lesions as anterior cruciate ligament tear or meniscal tear that can occur during high energy direct trauma.

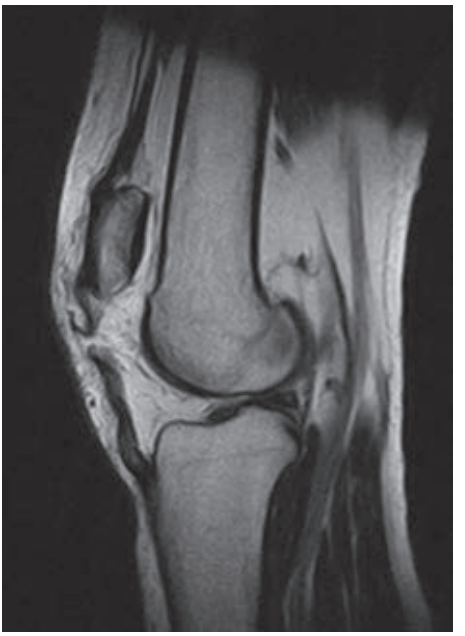


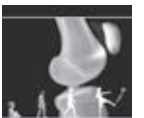
Fig. 2

Treatment

An acute repair of the patellar tendon during the week following the trauma gives the best results [15]. Many surgical techniques can be used [5].

Historically, it was recommended that all immediate repairs of the patellar tendon be reinforced by external devices [23]. Several reports have described reinforcing the repair with various augmentation grafts, including autografts, allografts (fascia lata, semitendinosus, gracilis), and synthetic grafts (Mersilene, Dacron, carbon fiber, and a poly-p-dioxannone cord). However, clinical reports have demonstrated satisfactory results of acute patellar tendon disruption repaired without augmentation [9].

We prefer reinforcing the repair with a semitendinosous autograft and/or with an augment with a PDS band, according to the quality of the suture. We do not recommend a metallic frame because it may sagitally tilt the patella and it implies another surgery to remove it. A straight incision is made on the medial border of the patellar tendon. The patellar tendon paratenon is incised longitudinally and preserved for repair at the time of closure.



Hematoma within the disrupted tendon is evacuated. The stumps are sutured end-to-end with 3 "U" knots with nonabsorbable sutures (n° 2 Fiberwire). This suture is reinforced by single stitches with an absorbable suture (Vicryl). A PDS tape is used to augment and protect the suture: biomechanically it transforms distraction forces into compression forces. It is folded in half and fixed on the tibial tubercule with a staple (Orthomed). The two free ends are sutured in a V-shape on the patellar tendon, on the patellar periosteum, and finally on the quadriceps tendon. This is done at 60° of knee flexion to avoid patellar tendon shortening and patella baja. An intraoperative radiograph is done to check the patellar height [18].

In case of diastasis of the suture, we augment it with a semitendinosous autograft. The semi-tendinosous tendon is harvested. A 4.5mm transosseous tunnel is drilled through the ATT and another through the distal part of the patella. The semitendinosous tendon is passed through these two tunnels and the two free ends are sutured end-to-end while the knee is extended. The graft is also sutured side-to-side with the patellar tendon and the patellar periosteum. This technique creates a box around the patellar tendon. Another option is to harvest an 8cm long – 15mm width quadriceps tendon autograft left inserted on the anterior side of the patella. The graft is flipped down and sutured to the patellar tendon, covering the suture of the tear. Whatever the technique used, the postoperative course is similar to that described earlier [18].

Other techniques are described in the literature [9]. For example, in cases of patellar tendon rupture at the osteotendinous junction, the free end of the tendon is freshened and two n° 5 nonabsorbable sutures are then placed along the medial and lateral halves of the patellar tendon with an interlocking suture (Krackow). The four free ends of suture are left emanating from the proximal portion of the tendon and passed through the patellar transosseous tunnels. An ACL drill guide may be used to place the patellar drill holes precisely. It is important to ensure that the repair has not produced patella baja. At 45° of flexion, the inferior pole of the patella should be above the roof of the intercondylar notch. The medial and lateral retinacula are repaired with n° 0 absorbable sutures.

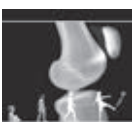
Alternatively, the patellar tendon may be repaired with suture anchors rather than with this transosseous technique. The sutures may still be placed along the medial and lateral halves of the patellar tendon with an interlocking technique (Bunnell or Krackow-Bunnell).

Results of Surgical Repair of Patellar Tendon Disruption

As for quadriceps tendons, the results of acute patellar tendon repair are favorable, regardless of the position of the rupture or the method of repair [15, 23]. However, delayed repairs have worse outcomes. Range of motion approaching that of the contralateral knee is regained and, in athletic individuals, premorbid activity levels and strength can be expected. Complications include rerupture, wound problems, and patellofemoral symptoms. Rerupture is generally related to return to rigorous activity before completion of proper physical therapy. Wound complications are more common than with quadriceps tendon disruption because of a thinner skin over the tibial tubercle; therefore, it is recommended incise skin adjacent to the tubercule but not directly on it. Obtaining an intraoperative radiograph at completion of the patellar tendon repair is useful to ensure patella is not baja.

CONCLUSION

Acute quadriceps and patellar tendon ruptures are rare and must be actively searched. Acute diagnosis and repair are mandatory to achieve an optimal functional recovery.



LITERATURE

[1] ATESOK K, DORAL MN, LOWE J, *et al.* Symptomatic bipartite patella: Treatment alternatives. *J Am Acad Orthop Surg 2008;16: 455-61.*

[2] BUSAM ML, PROVENCHER MT, BACH BR. Complications of anterior cruciate ligament reconstruction with bone-patellar tendon-bone constructs: care and prevention. *Am J Sports Med 2008; 36: 379-94.*

[3] CARPENTER JE, KASMAN R, MATTHEWS LS. Fractures of the patella. *J Bone Joint Surg Am 1993; 75: 1550-61.*

[4] FORTIS AP, MILIS Z, KOSTOPOULOS V, *et al.* Experimental investigation of the tension band in fractures or the patella. *Injury 2002; 33: 489-93.*

[5] GREIS PE, HOLMSTROM MC, LAHAV A. Surgical treatment options for patella tendon rupture, part I: acute. *Orthopedics 2005; 28: 672-79.*

[6] HARDY JRW, CHIMUTENGWENDE-GORDON M, BAKAR I. Rupture of the quadriceps tendon; an association with a patellar spur. *J Bone Joint Surg Br 2005; 87: 1361-63.*

[7] ILAN DI, TEJWANI N, KESCHNER M, *et al.* Quadriceps tendon rupture. *J Am Acad Orthop Surg 2003; 11: 192-200.*

[8] INSALL J, SALVATI E. Patellar position in the normal knee joint. *Radiology 1971; 101: 101-4.*

[9] SCOTT NW. Insall & Scott, Surgery of the Knee. 5th edition. *New York : Elsiever ; 2011.*

[10] KELLERSMAN R, BLATTERT TR, WECKBACH A. Bilateral patellar tendon rupture without predisposing systemic disease or steroid use: a case report and review of the literature. *Arch Orthop Trauma Surg 2005; 125: 127-33.*

[11] KELLY DW, CARTER VS, JOBE FW. Patellar and quadriceps tendon ruptures: jumper's knee. *Am J Sports Med 1984; 12: 975-80.*

[12] KRACKOW KA, THOMAS SC, JONES LC. A new stitch for ligament-tendon fixation: Brief note. *J Bone Joint Surg Am 1996;68 : 764-66.*

[13] LARSEN E, LUND PM. Ruptures of the extensor mechanism of the knee joint. *Clin Orthop 1986; 213: 150-53.*

[14] LEWIS AC, PURUSHOTHAM B, POWER DM. Bilateral simultaneous quadriceps tendon rupture in a bodybuilder. *Orthopedics 2005; 28: 701-2.*

[15] MATAVA MJ. Patellar tendon ruptures. *J Am Acad Orthop Surg 1996; 4: 287-96.*

[16] MELVIN JS, MEHTA S. Patellar fractures in adults. *J Am Acad Orthop Surg 2011; 19: 198-207.*

[17] MULLER ME, ALLGOWER M, SCHNEIDER R,s *et al.* Manual of internal fixation: techniques recommended by the AO group. *Berlin, Germany: Springer, 1979.*

[18] NEYRET P, DEMEY G, SERVIEN E, *et al.* Traité de chirurgie du genou. *Paris: Masson; 2012.*

[19] NORDIN M, FRANKEL VH. Biomechanics of the knee. In: Nordin M, Frankel VH, eds. Basic biomechanics of the musculoskeletal system. *2nd ed. Philadelphia : Lea & Febiger, 1989 :115-34.*

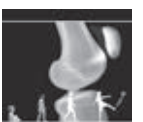
[20] RAMSEY RH, MULLER GE. Quadriceps tendon rupture: a diagnostic trap. *Clin Orthop 1988; 226: 113-17.*

[21] ROSE PS, FRASSICA FJ. Atraumatic bilateral patellar tendon rupture. A case report and review of the literature. *J Bone Joint Surg Am 2001; 83: 1282-86.*

[22] SEIL R, MÜLLER B, GEORG T, *et al.* Reliability and interobserver variability in radiological patellar height ratios. *Knee Surg Sports Traumatol Arthrosc 2000; 8: 231-36.*

[23] SIWEK CW, RAO JP. Ruptures of the extensor mechanism of the knee joint. *J Bone Joint Surg 1981; 63: 932-37.*

[24] THOMS RJ, KONDRASHOV D, SILBER J. Iatrogenic femoral nerve palsy masquerading as knee extensor mechanism rupture. *Am J Orthop 2009; 38: 142-44.*



CHRONIC RUPTURE OF THE EXTENSOR MECHANISM (TKA and patellar fracture excepted)

S. LUSTIG, R.A. MAGNUSSEN, G. DEMEY, E. SERVIEN, P. NEYRET

Chronic ruptures of the extensor mechanism can be classified as chronic patellar tendon ruptures, chronic quadriceps tendon ruptures, and neglected patellar fractures.

Patellar tendon rupture is a rare but devastating injury that frequently results from significant trauma. Primary repair with or without augmentation often yields good outcomes when tears are addressed promptly after injury. Reconstruction of chronic ruptures of the patella tendon is often difficult because of the proximal retraction of the patella, quadriceps muscle contracture and scarring, and poor quality of the remaining patellar tendon tissue. Various methods have been described for addressing chronic tears of the patellar tendon. If sufficient healthy native patellar tendon remains, primary repair followed by augmentation with hamstring tendon [1, 2], cerclage wire [3], or PDS tape [4] can be successful. If the native tendon is completely lost, reconstruction with allograft tissue is an option. Previous authors have described the use of Achilles tendon allograft in this situation [5]. Dejour *et al.* [6, 7] described the use of a contralateral extensor mechanism autograft to reconstruct chronic patellar tendon ruptures. The contralateral autograft consisted of a tibial bone block, the middle third of patellar tendon, a patellar bone block, and the central portion of the quadriceps tendon. Because of the morbidity of contralateral graft harvest, the technique was subsequently modified to use an extensor mechanism allograft of the same shape [8, 9] (fig. 1, 2).

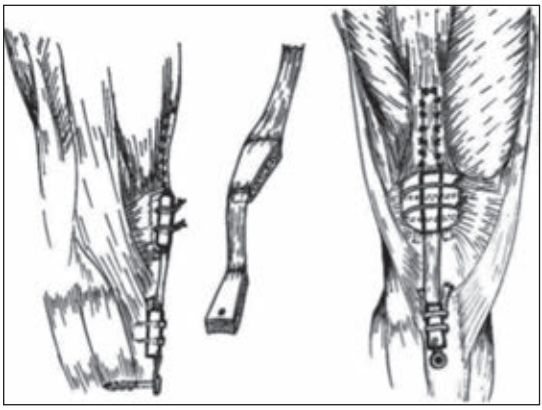
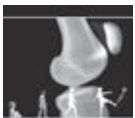


Fig. 1: Reconstruction of Chronic Patellar Tendon Ruptures with partial extensor mechanism allograft



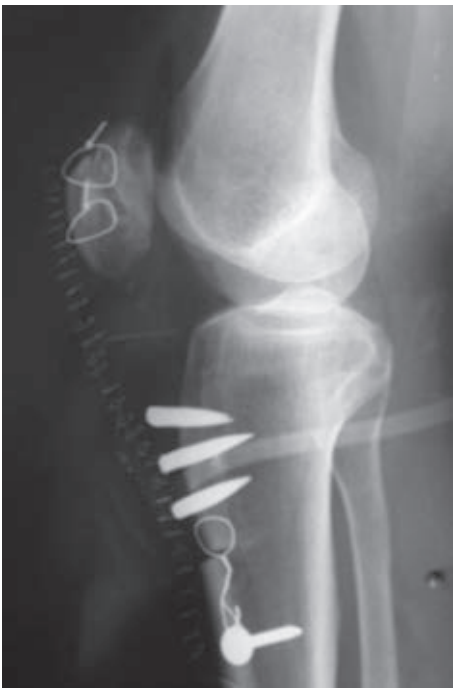


Fig. 2: Post-operative radiograph after a partial extensor mechanism allograft.

Quadriceps tendon ruptures (QTRs) are uncommon injuries that tend to occur in older patients, those with systemic diseases, or patient with significant degenerative change to the tendon prior to injury. Despite relatively obvious findings on physical examination and standard radiographs, delay in the diagnosis of QTRs still occurs [10]. Late repair of QTRs can be technically demanding and the results are less satisfactory than those expected following early repair [11-13]. Over the past two decades, many surgeons have recommended a period of 4 to 6 weeks of cast immobilization and 12 weeks of bracing after repair of QTRs [11-13]. However, several recent studies have advocated various methods of augmenting repair of QTRs to allow early, protected motion and full weight bearing [14-17]. In cases of chronic rupture with a large gap, immobile patella, and patellar tendon scarring, a specific technique has been described by P. Chambat using suture and metallic wire ("sardine can" technique) (fig. 3).

This report summarizes the different techniques available for treatment of chronic extensor mechanism injuries and presents the results of the late repair of patellar and quadriceps tendon ruptures using these different techniques.

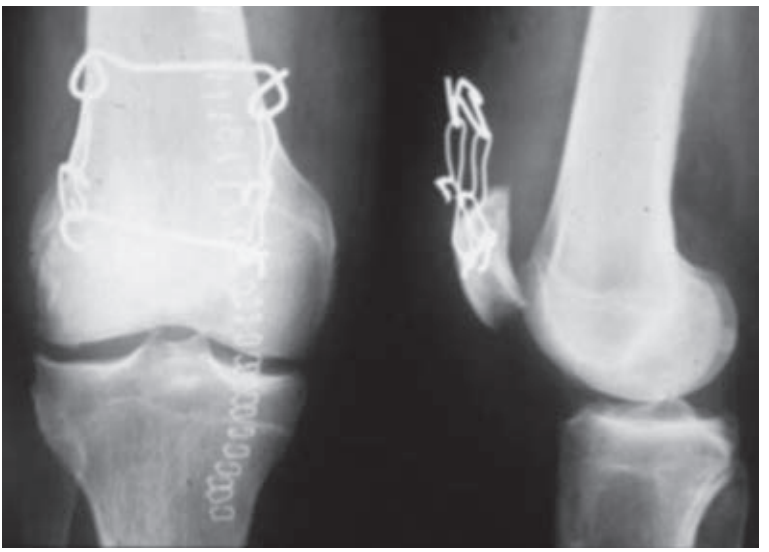
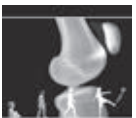


Fig. 3: Post-operative radiograph of the "sardine can" technique for treatment of chronic quadriceps tendon ruptures described by P. Chambat.



LITERATURE

[1] ECKER ML, LOTKE PA, GLAZER RM. Late reconstruction of the patellar tendon. *J Bone Joint Surg Am. 1979; 61: 884-86.*

[2] MANDELBAUM BR, BARTOLOZZI A, CARNEY B. A systematic approach to reconstruction of neglected tears of the patellar tendon. A case report. *Clin Orthop Relat Res. 1988; 268-71.*

[3] McLAUGHLIN HL, FRANCIS KC. Operative repair of injuries to the quadriceps extensor mechanism. *Am J Surg. 1956; 91: 651-3.*

[4] KASTEN P, SCHEWE B, MAURER F, *et al.* Rupture of the patellar tendon: a review of 68 cases and a retrospective study of 29 ruptures comparing two methods of augmentation. *Arch Orthop Trauma Surg. 2001; 121: 578-82.*

[5] FALCONIERO RP, PALLIS MP. Chronic rupture of a patellar tendon: a technique for reconstruction with Achilles allograft. *Arthroscopy. 1996; 12: 623-6.*

[6] DEJOUR H, DENJEAN S, NEYRET P. Treatment of old or recurrent ruptures of the patellar ligament by contralateral autograft. *Rev Chir Orthop Reparatrice Appar Mot 1992; 78: 58-62.*

[7] NEYRET P, DONELL ST, CARRET JP, *et al.* Patellar ligament rupture treated by contralateral patellar ligament autograft and its application in patients with tibial allografts. *Knee 1994;1: 158-60.*

[8] ELGUINDY A, LUSTIG S, SERVIEN E, *et al.* Treatment of chronic disruption of the patellar tendon in osteogenesis imperfecta with allograft reconstruction. *Knee. 2011; 18: 121-4.*

[9] MAGNUSSEN RA, LUSTIG S, ELGUINDY A, *et al.* Reconstruction of chronic patellar tendon ruptures with extensor mechanism allograft. *Tech In Knee Surg, 11(1):34-40, March 2012.*

[10] SIWEK CW, RAO JP. Ruptures of the extensor mechanism of the knee joint. *J Bone Joint Surg Am. 1981;6: 932-7.*

[11] ROUGRAFF BT, REECK CC, ESSENMACHER J. Complete quadriceps tendon ruptures. *Orthopaedics. 1996; 19: 509 -14.*

[12] KATZMAN BM, SILBERBERG S, CALIGIURI DA, *et al.* Delayed repair of quadriceps tendon. *Orthopedics. 1997; 20: 553-4.*

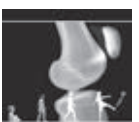
[13] RASUL AT Jr, FISCHER DA. Primary repair of quadriceps tendon ruptures: results of treatment. *Clin Orthop Relat Res. 1993;289: 205-7.*

[14] RAMSEIER LE, WERNER CML, HEINZELMANN M. Quadriceps and patellar tendon rupture. *Injury. 2006; 37: 516 -19.*

[15] LEVY M, GOLDSTEIN J, ROSNER M. A method of repair for quadriceps tendon or patellar tendon ligament (tendon) ruptures without cast immobilization. *Clin Orthop. 1987; 218: 297-301.*

[16] WENZL ME, KIRCHNER R, SEIDE K, *et al.* Quadriceps tendon ruptures: is there a complete functional restitution? *Injury. 2004;35: 922-6.*

[17] WEST JL, KEENE JS, KAPLAN LD. Early motion after quadriceps and patellar tendon repairs: outcomes with single-suture augmentation. *Am J Sports Med. 2008; 36: 316 -23.*



REHABILITATION AND CORE MUSCULATURE IN THE TREATMENT OF PATELLOFEMORAL PAIN

E.A. ARENDT

The "core" is a series of inter-related muscles that provides the force to stabilize and move body segments. In the lower extremity, core could be considered the link between the torso (i.e.) one's CORE, and lower extremities. Core training focuses on pelvic stability. If the pelvis is stable, the lower back and hips (limbs) are stable. In treatment of pain and injury around the knee and kneecap joints, CORE musculature helps to stabilize the limb under the pelvis. Indeed one can think of CORE as base or platform of musculoskeletal strength resulting in control of the trunk (axial skeleton) upon which limb activity is optimally performed.

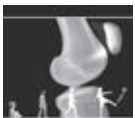
Core muscles (in lower extremity function) are generally considered to be the muscles of your lower spine, abdomen, and hip/buttocks. Core strengthening exercises are most effective when the torso works as a solid unit and both front and back muscles contract at the same time; multi joint movements are performed and stabilization of the spine is monitored. These exercises are most effective when they engage many muscles throughout the torso that cross several joints and work together to coordinate stability.

THE APPROACH

Analyzing limb strength and extremity kinematics are the main components that contribute to the plan of care for the patient with patellofemoral (PF) pain. Focusing this plan of care on improving patient symptoms by improving their lower extremity kinematics has had high success in our clinical practice for decreasing PF pain. This approach focuses on movement-centered therapy with a skilled physical therapist. The patients' response to each intervention will help to guide the next step as well as define the need for further intervention.

Physical therapy should be customized to the patient's level of strength and fitness and whenever possible should be made challenging to the patient. The exercises themselves should be increased in intensity and duration as the patient develops better strength and limb control in their activities.

This chapter will focus on individual clinical strategies used in rehabbing the patient who presents with anterior knee pain [1, 2].



Anterior Knee Pain

As with most pain issues, anterior knee pain often has a root cause that is unknown or unclear, with multiple confounding variables. An analysis of alignment, strength deficit, and potential inciting overuse factors must be elicited by a careful history and physical exam by the treating clinician.

Guiding principles

1. ***Identify and restore muscle imbalances***, esp. proximal hip and pelvic regions.

2. ***Maximize "modifiable" alignment issues.*** Most specifically, this refers to foot position and pelvic position. A neutral foot position is favored over a pronated foot position; a neutral pelvis is favored over positions of excessive pelvic tilt or obliquity. In most patients these are not fixed deformities, but are capable of being repositioned with foot support and/or dynamic muscle strengthening.

There is increasing evidence in the literature linking weak hip musculature and poor pelvic control with anterior knee pain. Rotational control of the limb underneath the pelvis is critical; this is largely achieved through the roles of gluteus medius and maximus musculature as closed kinetic chain (CKC) stabilizers. Strength of these muscle groups is of paramount importance, as a lack of rotational control of the limb can result in a valgus collapse pattern that places the knee at risk for acute and overuse knee injuries (fig. 1a).

3. ***Examine patterns of overuse.*** This can be a repetitive exercise activity such as running, or repetitive work activities that involve lifting, squatting, and/or stair climbing. One must have adequate muscular strength and endurance to perform repetitive activities. If one part of the kinetic chain is weak or injured, the body often finds ways to accomplish an activity by "working around" the injured body part. This often initiates faulty body mechanics that lead to a painful state that centers on the patella and its associated soft tissue structures. One must reduce activities to remain within an envelope of pain-free function. Once one is within an envelope of pain free activities, the patient can begin to expand their envelope concomitant with improvement in strength, coordination, balance, and overall dynamic control of the limb.

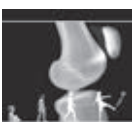
Abnormal motion patterns when performing a Partial Squat

Observation of a patient doing a partial squat can uncover many movement patterns that can relate to PF pain and dysfunction.

1. **Anterior Knee Excursion** (fig. 1b): This refers to the excessive anterior translation of the knee with squatting, thus projecting the torso (center of mass) forward over ones' toes. The patella is thus placed in a position to help keep the torso upright, increasing joint reaction force. It is, in part, the end result of the lack of integrating posterior muscles in this body movement pattern. Excessive anterior knee excursion should be avoided for multiple reasons including excessive PF joint loading, inadequate hip joint utilization/muscular recruitment, and excessive ankle dorsiflexion demands. This is a very common "habit" seen in young teenage girls.

With squat retraining, it is critical to employ a balanced hip and knee strategy in the sagittal plane. Patients often need to be instructed to "sit back" into hip flexion, with associated forward trunk lean, to allow a more centered distribution of the center of mass in the anterior/posterior direction and thus a more balanced load distribution between the joints of the lower extremity (fig. 2 a & b). Cueing the patient to "sit down on a chair" or "lead with their buttocks" can be helpful to begin proper execution of this exercise.

2. **Functional knee valgus** (fig. 1a): The need to control the alignment of the limb in the frontal and transverse planes is of primary importance in the goal of optimal lower



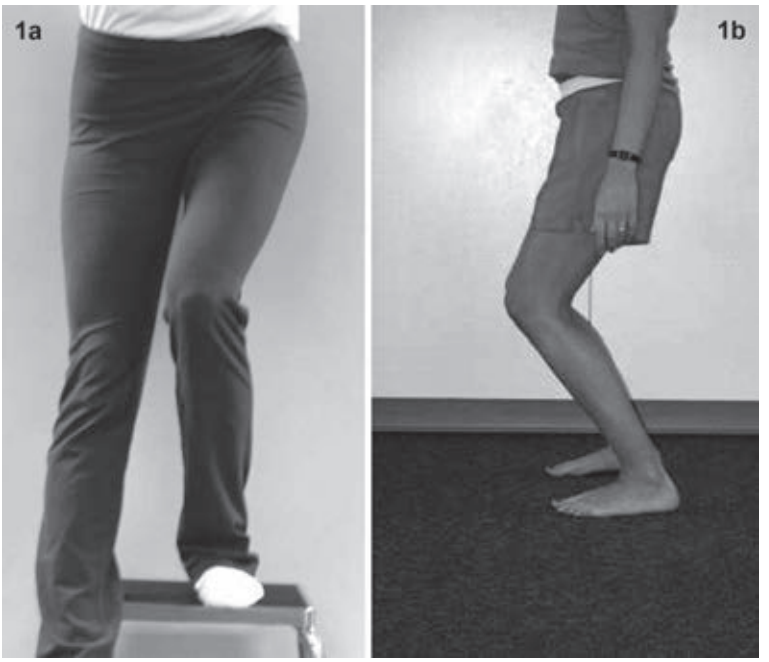


Fig. 1:
a) Example of femoral collapse into adduction and internal rotation ("functional valgus")
b) Example of excessive anterior translation of the center of mass with squatting

Fig. 2:
a) Example of good sagittal plane alignment with squatting
b) Example of good frontal plane alignment with squatting

extremity kinetics. Femoral internal rotation (IR) and adduction (ADD) in the CKC knee flexion can contribute to dysfunctional PF joint loading and tracking [3, 4, 5, 6]. Patients reporting PF pain demonstrate this frontal plane collapse with a wide range of CKC activities [7, 8] (ie) functional or dynamic knee valgus. This collapse pattern is, in part, a result of lack of strength and/or activation of hip external rotators and posterior buttocks musculature.

Patients should be instructed to resist the inward collapse of the knees/thighs toward midline. Often cueing to actively press the knees/thighs outward is advantageous, with the focus of "keeping the knee caps over the toes". The use of a resistance band around the thighs can be useful to physically stimulate this correction with double stance activity. The degree to which frontal plane mechanics can be manipulated depends on the patient's presenting bony alignment. Long limb torsion, in particular excessive external tibial torsion, should be identified by physical exam. The patient should be allowed to have the toes point in the direction of comfort when doing this exercise.

Therapy should progress through the re-training phase from double stance exercises to single leg partial squats to dynamic activities such as jump landing from an elevated height (3 meters) or repeated jumping, with the goal incorporating functional body movement patterns into the patient's sport and daily living activities. Core stability/trunk control should be both assessed and addressed in the open kinetic chain (OKC) and CKC states. Lack of trunk control, particularly in the frontal plane, has been associated with higher risk of lower extremity injury, particularly at the knee joint [9, 10, 11, 12]. Unfortunately there is neither well-established standardization nor validation of clinic-friendly core stability testing measures. Despite that limitation, core/trunk stability training, including plank poses in multiple planes (fig. 3 a,b,c), is broadly viewed as an important element to establish a foundation for neuromuscular control as patients progress into more demanding CKC activities.

"Quadriceps avoidance" movement pattern with gait

Dysfunction of the PF joint is often associated with dysfunction of the quadriceps muscle group, without a clear cause and effect relationship defined [13]. Patients with chronic PF pain and/or instability often demonstrate a gait pattern that avoids deeper knee flexion during the loading response phase of gait. In extreme cases, a hyperextension thrust pattern may be employed, in an attempt to gain knee stability. Pain from impingement of anterior knee structures then feeds back into the pattern of quadriceps avoidance/inhibition [14].

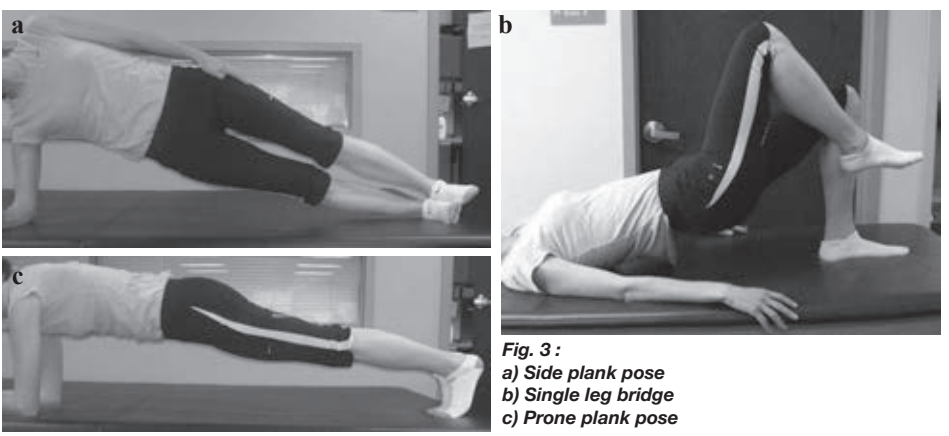
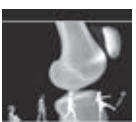


Fig. 3 :
a) Side plank pose
b) Single leg bridge
c) Prone plank pose



Treatment focus

Prior to having the patient engage in closed kinetic chain (CKC) lower extremity strengthening exercises, adequate recruitment of lower extremity musculature in an open kinetic chain (OKC) condition must be demonstrated, specifically strength and endurance of the gluteus maximus and medius musculature. OKC activity of the quadriceps muscle may impose high joint forces across a relatively small contact surface area, aggravating the pain cycle. For this reason, any quadriceps strengthening, in particular OKC exercises, should be approached cautiously with high vigilance to pain.

Some possible exercise suggestions for strengthening individual muscles groups before engaging in strengthening thru co-ordinated body movement patterns are:

- Isometric quadriceps presses in deeper angles of knee flexion, when the patella is fully engaged in the trochlear groove. (Caution: the patient should not have a cartilage lesion that contributes to pain with increased patella on trochlea contact).
- Quadriceps strengthening at a single angle in the knee arc motion has proven to have beneficial effects for quadriceps strength across all knee angles [15]. With more exaggerated quad shutdown and dysfunction, CKC activities often bypass the quad with a kinetic task seemingly performed without appropriate quad activation. Specific isolated quadriceps strengthening may need to be performed to activate the muscle prior to a progression into more complex, coordinated or loaded strengthening activities. To this end, terminal knee extension (TKE) quadriceps activities, often prohibited in PF exercises, may be employed cautiously with the end goal of the patient being able to execute terminal extension without a lag. Resistance is not employed with this activity; only the weight of the limb against gravity is used. With such an activity, heavy consideration must be given for reported pain, observed effusion response, and stress on any surgically influenced structures. Once adequate muscle strength to achieve active TKE is restored, transitioning to CKC quadriceps strengthening is recommended to minimize potentially deleterious loading of the PF joint.
- A good alternative to OKC quadriceps exercises in early rehabilitative phases would be load-reduced CKC activities, such as leg press or leg press simulation into a stabilized exercise ball through the terminal degrees of knee extension/early degrees of knee flexion. Once proficiency with coordinated quadriceps control of this early range of motion is demonstrated, the patient will better tolerate the transition to CKC activities with body weight and/or external resistance.

Physical Performance Tests for PF issues

Physical performance testing of the lower extremity is a method of assessing knee function & movement coordination impairments. These tests typically establish the "norm" as the patient's contra lateral limb and thus compute a side-to-side difference in lower extremity function (Limb Symmetry Index – LSI). Such testing is common in the recovery of function after a ligamentous knee injury and surgery, most commonly employed in the ACL-deficient and ACL-reconstructed population [16, 17]. In dealing with the patellofemoral joint, this method can be challenging or less useful as often these patients demonstrate bilateral abnormalities and/or symptoms; using the opposite limb as "normal" has to be viewed with some skepticism.

Strength tests around the knee involve:

- Testing of individual muscle groups. Isokinetic testing of knee flexion and extension is a common tool to establish objective strength parameters about the knee. Proximal lower extremity muscle testing may be performed through standardized manual muscle testing methods or with the use of hand-held or isokinetic dynamometry when such equipment is available.
- Testing of functional (co-ordinated) knee activity: (e.g.) hop tests, balance reach tests.
- CORE testing (proximal limb control afforded

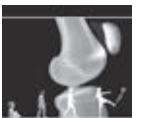


Fig. 4 : Strength test: Single limb maximum depth squat: maximum knee flexion angle reached at depth of squat is measured and compared to the opposite side.

by hip and core musculature): Core/trunk stability is harder to test, as it is not limited to an individual muscle group, and measurement of muscle strength becomes more challenging the more one progresses proximally on the limb. Tests that can be performed in clinic include timed holds of plank poses in positions of progressively greater challenge (fig. 3).

Lower extremity physical performance tests that have found utility in our clinical practice for patients with PF problems are itemized below (Table 1). The traditional hop tests, employed with many lower extremity patients who intend to return to cutting and pivoting sports, require a higher level of functional activity, and may not always be appropriate with the PF population. Many PF pain patients have experienced physical limitations from a young age due to their joint dysfunction. To this end, simpler, lower impact functional test activities may prove more appropriate measures.

Many lower level physical performance test activities have not been validated in the literature, but still find utility in the clinical setting. Our testing activities commence with basic anthropometric measures at the limb including range of motion, joint line circumference (effusion) and proximal thigh circumference (return of thigh muscle girth). We then proceed with progressively more challenging activities, ranging from mat-based core stability poses to static and dynamic CKC balance testing to dynamic and propulsive lower extremity strength, endurance and power activities. Limb to limb differences, when appropriate, are recorded and reported through a LSI %. We also utilize qualitative observations on body form and movement pattern. Though these are difficult to quantify, we have found utility in "grading" the form of the body movement pattern and as way to convert a qualitative observation into a quantitative number (eg.) excellent form: grade 3, some body form collapse: grade 2, poor form: grade 1, cannot perform test: grade 0. Additionally we ask the patient to report a perceived exertion level with all of the CORE endurance tests.

CONCLUSION

Though insights on the suspected causative factors in patellofemoral dysfunction continue to be developed, the day to day care of an individual patient mandates customization of physical therapy regiment coupled with a trained physical therapist for maximum success. Training CORE muscles and optimizing body movement patterns has great utility in a clinical practice to reduce anterior knee pain and improve body function.

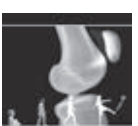
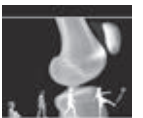


Table 1: Physical performance testing elements used at our facility

Domain Tested	Test Activity	Recorded Value
Anthropometric Data	Knee ROM	Degrees of motion
	Joint line circumference	Centimeters around the joint line
	Thigh circumference	Centimeters around the thigh (15 cm proximal to suprapatellar border)
Core Stability	Prone plank timed hold (fig. 3c)	Seconds held, maintaining ideal alignment (out of maximum of 60 seconds)
	Side plank timed hold (fig. 3a)	Seconds held, maintaining ideal alignment (out of maximum of 60 seconds)
	Single leg bridge repetitions to fatigue (fig. 3b)	Maximum repetitions to muscle fatigue
Balance	Single limb balance with eyes closed	Seconds held (out of maximum of 60 seconds)
	Single limb stand and reach	Centimeters reached with opposite arm of stance limb
	Star excursion balance test	Centimeters reached with opposite toe from stance limb
Lower Extremity Muscle Strength	Single limb maximum depth squat (fig. 4)	Maximum knee flexion angle reached at depth of squat
	Retro step-up/down	Maximum step height successfully completed (inches)
Lower Extremity Muscle Endurance	2 Minute Single Leg Repeated Squat Test	Maximum number of squats completed to 60° KF at 60 bpm tempo x 2 minutes, preserving ideal trunk and limb alignment (max value = 60 squats)
Lower Extremity Power	Single limb hop for distance	Maximum distance hopped in centimeters/ meters
	6M Timed hop	Maximum speed recorded in seconds
	Triple cross-over hop for distance	Maximum distance hopped in centimeters/ meters



LITERATURE

[1] FAGAN V, DELAHUNT E. Patellofemoral pain syndrome: a review on the associated neuromuscular deficits and current treatment options. *Br J Sports Med. 2008; 42: 789-95.*

[2] HEINTJES E, BERGER MY, BIERMA-ZEINSTRA SM, BERNSEN RM, VERHAAR JA, KOES BW. Exercise therapy for patellofemoral pain syndrome. *Cochrane Database Syst Rev. 2003:CD003472.*

[3] POWERS CM. The influence of altered lower-extremity kinematics on patellofemoral joint dysfunction: a theoretical perspective. *J Orthop Sports Phys Ther. 2003; 33: 639-46.*

[4] POWERS CM, WARD SR, FREDERICSON M, GUILLET M, SHELLOCK FG. Patellofemoral kinematics during weight-bearing and non-weight-bearing knee extension in persons with lateral subluxation of the patella: a preliminary study. *J Orthop Sports Phys Ther. 2003; 33: 677-85.*

[5] LI G, DEFRATE LE, ZAYONTZ S, PARK SE, GILL TJ. The effect of tibiofemoral joint kinematics on patellofemoral contact pressures under simulated muscle loads. *J Orthop Res. 2004; 22: 801-6.*

[6] LEE TQ, MORRIS G, CSINTALAN RP. The influence of tibial and femoral rotation on patellofemoral contact area and pressure. *J Orthop Sports Phys Ther. 2003; 33: 686-93.*

[7] WILLSON JD, DAVIS IS. Lower extremity mechanics of females with and without patellofemoral pain across activities with progressively greater task demands. *Clinical biomechanics (Bristol, Avon). 2008; 23: 203-11.*

[8] SOUZA RB, POWERS CM. Differences in hip kinematics, muscle strength, and muscle activation between subjects with and without patellofemoral pain. *J Orthop Sports Phys Ther. 2009;39: 12-19.*

[9] ZAZULAK BT, HEWETT TE, REEVES NP, GOLDBERG B, CHOLEWICKI J. Deficits in neuromuscular control of the trunk predict knee injury risk: a prospective biomechanical-epidemiologic study. *Am J Sports Med. 2007; 35: 1123-30.*

[10] ZAZULAK BT, HEWETT TE, REEVES NP, GOLDBERG B, CHOLEWICKI J. The effects of core proprioception on knee injury: a prospective biomechanical-epidemiological study. *Am J Sports Med. 2007;35: 368-73.*

[11] LEETUN DT, IRELAND ML, WILLSON JD, BALLANTYNE BT, DAVIS IM. Core stability measures as risk factors for lower extremity injury in athletes. *Med Sci Sports Exerc. 2004;36: 926-34.*

[12] WILLSON JD, IRELAND ML, DAVIS I. Core strength and lower extremity alignment during single leg squats. *Med Sci Sports Exer. 2006; 38: 945-52.*

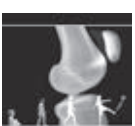
[13] POWERS CM. Patellar kinematics, part I: the influence of vastus muscle activity in subjects with and without patellofemoral pain. *Phys Ther 2000;80: 956-64.*

[14] HODGES PW, MELLOR R, CROSSLEY K, BENNELL K. Pain induced by injection of hypertonic saline into the infrapatellar fat pad and effect on coordination of the quadriceps muscles. *Arthritis Rheum. 2009;61: 70-7.*

[15] BARAK Y, AYALON M, DVIR Z. Transferability of strength gains from limited to full range of motion. *Med Sci Sports Exerc. 2004; 36: 1413-20.*

[16] NOYES FR, BARBER SD, MANGINE RE. Abnormal lower limb symmetry determined by function hop tests after anterior cruciate ligament rupture. *Am J Sports Med. 1991;19: 513-8.*

[17] FITZGERALD GK, AXE MJ, SYNDER-MACKLER L. A decision-making scheme for returning patients to high-level activity with nonoperative treatment after anterior cruciate ligament rupture. *Knee Surg Sports Traumatol Arthrosc. 2000;8: 76-82.*



ÉVALUATION D'UN PROTOCOLE DE RÉÉDUCATION ISOCINÉTIQUE DANS LA PRISE EN CHARGE DES DOULEURS ANTÉRIEURES DU GENOU APRÈS LIGAMENTOPLASTIE DU LCA

F. GADEA, B. QUELARD, D. MONNOT, R. MORTATI, J.M. FAYARD, M. THAUNAT, P. CHAMBAT, B. SONNERY-COTTET

Introduction

L'utilisation de la rééducation isocinétique dans la prise en charge des douleurs antérieures après reconstruction du LCA n'a jamais été évaluée. En cas de stagnation ou d'aggravation des douleurs antérieures malgré une rééducation bien conduite, il n'existe à l'heure actuelle aucune alternative validée.

Matériel et méthode

La population est composée de 48 patients pris en charge par un protocole identique de rééducation isocinétique dans le cadre de douleurs antérieures après ligamentoplastie du LCA. Les résultats fonctionnels ont été évalués à 3 reprises – avant de débuter l'isocinétisme, à la fin de l'isocinétisme et au dernier recul – au moyen du score IKDC subjectif, et pour la douleur à l'aide de l'échelle EVA.

Résultats

Le recul moyen après la chirurgie est de 27,7 mois. On note une amélioration significative du score IKDC entre la première et la dernière séance d'isocinétisme (+ 28 points) ; et entre la dernière séance d'isocinétisme et le dernier recul (+ 9 points). Le seul facteur prédictif de bons résultats est le délai entre la chirurgie et le dernier recul (p=0,037).

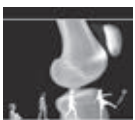
Discussion

Si l'amélioration des résultats fonctionnels était attendue, l'objectif principal de l'isocinétisme comme outil de rééducation dans le contexte de ligamentoplastie du LCA est de sortir du cercle vicieux "déficit musculaire-douleur" et de permettre la reprise de la rééducation classique. L'autre intérêt de l'isocinétisme est le dépistage précoce dans les douleurs antérieures d'origine organique d'un déficit musculaire débutant afin de prévenir le risque de décompensation d'un syndrome rotulien fonctionnel.

Conclusions

La rééducation isocinétique doit faire partie intégrante de la stratégie de prise en charge des douleurs antérieures après reconstruction du LCA. Son utilisation, en revanche, ne se conçoit qu'en complément de la rééducation classique car l'isocinétisme ne permet pas, à lui seul, la régression des douleurs antérieures.

Niveau de preuve.- IV étude rétrospective.



INTRODUCTION

Dans la méta-analyse de Freedman *et coll.*, il est rapporté 22 % de douleurs antérieures après reconstruction du ligament croisé antérieur (LCA) par tendon rotulien et 11,5 % avec les ischiojambiers [1]. Les principaux facteurs incriminés dans le syndrome rotulien fonctionnel après reconstruction du LCA sont la faiblesse du quadriceps et dans une moindre mesure la contracture des ischiojambiers, sans que l'on ait réellement montré si la dysfonction musculaire en était la cause ou la conséquence [2, 3]. S'il existe une corrélation positive entre le déficit musculaire postopératoire et les douleurs antérieures [4, 5], il a été montré que la douleur antérieure retardait la récupération musculaire [6] et que la récupération musculaire était corrélée à la diminution des douleurs antérieures [7].

Quel que soit le type de greffe et malgré les protocoles classiques de rééducation, les déficits musculaires, 6 mois après la reconstruction d'un LCA, sont inévitables avec des différentiels moyens de 10 à 30 % [8]. De plus, à long terme, le déficit, de l'ordre de 5 à 10 %, est très souvent asymptomatique [6, 9-11].

L'utilisation de l'isocinétisme dans la rééducation du LCA apporte 2 nouveautés par rapport aux techniques conventionnelles. Sur le plan diagnostique, l'isocinétisme est capable d'évaluer avec précision la symétrie de chaque muscle par rapport au côté controlatéral ainsi que le ratio agoniste/antagoniste sur chaque membre [12]. Sur le plan thérapeutique, la maîtrise de la vitesse et des amplitudes permet de travailler efficacement la contraction musculaire en zone infradouloureuse, ce qui lui donne un avantage considérable par rapport aux techniques classiques y compris celles utilisant la chaîne cinétique fermée [13].

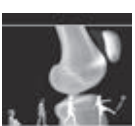
L'isocinétisme a largement été utilisé dans l'évaluation des déficits musculaires après reconstruction du LCA [4-6, 8-11, 14-17]. Mais à notre connaissance, aucune étude n'a évalué, à ce jour, l'utilisation de l'isocinétisme comme outil de rééducation dans les syndromes rotuliens fonctionnels après ligamentoplastie du LCA. Même dans le cadre du syndrome rotulien primitif, la rééducation isocinétique reste assez peu étudiée avec seulement 2 séries retrouvées dans la littérature, qui font état de résultats très satisfaisants pour la diminution des douleurs [18, 19].

Notre objectif est d'évaluer l'efficacité de la rééducation isocinétique au moyen de l'EVA et du score IKDC subjectif dans la prise en charge des douleurs antérieures après ligamentoplastie du LCA. Notre hypothèse est de montrer qu'en cas de douleurs antérieures résistantes aux techniques de rééducation classiques, notre protocole de rééducation isocinétique permettait une diminution des douleurs et une reprise des activités quotidiennes et sportives.

MATÉRIEL ET MÉTHODES

Il s'agit d'une étude monocentrique, rétrospective portant sur l'ensemble des patients présentant une douleur antérieure invalidante dans les suites d'une reconstruction du LCA, pris en charge par un protocole de renforcement musculaire isocinétique. Pour être inclus, la première séance de rééducation isocinétique doit avoir été réalisée dans les 3 ans suivant la ligamentoplastie. Afin de minimiser les facteurs de confusion, les patients ayant présenté une réintervention pour rupture itérative précoce – arbitrairement fixée à 3 ans suivant la première ligamentoplastie, ainsi que ceux dont le déficit musculaire du quadriceps était inférieur à 15 % par rapport au côté non opéré ont été exclus. En revanche les patients réopérés pour une cure de cyclope ont été inclus.

La population est composée de 48 patients. Il y a 27 femmes pour 21 hommes. L'âge moyen au moment de la chirurgie est de 35 ans (16-55). Six 6 patients sont repris pour cure de cyclope. Les greffes utilisées se répartissent en 18 transplants libres de tendon rotulien (TR), 26 Droit-Interne-Demi-Tendineux (DIDT), 3 Demi-Tendineux (DT) et 1 transplant libre de tendon quadricipital (TQ). Afin d'avoir le maximum de puissance, les greffes sont réparties en 2 groupes : appareil extenseur et ischiojambiers. Le délai moyen entre la ligamentoplastie et la première séance d'isocinétisme est de 11 mois (4-35 mois).



Les séances d'isocinétisme sont toutes réalisées sur un dynamomètre Con-Trex™ en pratiquant un protocole rigoureusement identique. Avant la première séance, un bilan isocinétique initial est réalisé sur le membre opéré et sur le membre controlatéral afin d'évaluer le moment de force maximale pour le quadriceps et pour les ischiojambiers. Le tableau 1 décrit les différentes mesures réalisées pour chaque muscle lors de ce bilan isocinétique. Après un repos de 48 heures minimum, la rééducation est débutée au rythme de 2 séances par semaine. Le tableau 2 décrit le protocole de rééducation isocinétique. Chaque séance dure en moyenne 25 minutes, débute toujours par 5 minutes d'échauffement sur bicyclette ergométrique et se termine par un auto-étirement des différentes chaînes musculaires sous-pelviennes.

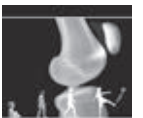
Tableau 1 : Mesures réalisées lors du bilan isocinétique initial.

Essai Test	Concentrique 180°/s - 4 répétitions Concentrique 180°/s - 6 répétitions
Essai Test	Concentrique 90°/s - 3 répétitions Concentrique 90°/s - 5 répétitions
Essai Test	Concentrique 240°/s - 4 répétitions Concentrique 240°/s - 20 répétitions
Essai Test	Concentrique 30°/s - 3 répétitions Concentrique 30°/s - 5 répétitions

Tableau 2 : Description du protocole de rééducation isocinétique

	Mode concentrique	Mode excentrique
Première séance	• 6 X 70% Mfmax - 450°/s r = 15 s 6 X 70% Mfmax - 400°/s r = 15 s 6 X 70% Mfmax - 350°/s r = 15 s • 4 X 70% Mfmax - 300°/s r = 15 s 4 X 70% Mfmax - 300°/s r = 15 s 4 X 70% Mfmax - 270°/s r = 15 s 4 X 70% Mfmax - 270°/s	4 X 70% Mfmax - 15°/s r = 15 s 4 X 70% Mfmax - 15°/s r = 15 s 4 X 70% Mfmax - 15°/s
Deuxième séance	• 6 X 70% Mfmax - 400°/s r = 15 s 6 X 70% Mfmax - 350°/s r = 15 s 6 X 70% Mfmax- 350°/s r = 15 s • 4 X 70% Mfmax - 300°/s r = 15 s 4 X 70% Mfmax - 270°/s r = 15 s 4 X 70% Mfmax - 240°/s r = 15 s 4 X 70% Mfmax - 240°/s	4 X 70% Mfmax - 15°/s r = 15 s 4 X 70% Mfmax - 30°/s
Séances suivantes	4 X 70% Mfmax - 240°/s 4 X 70% Mfmax - 210°/s 4 X 70% Mfmax - 180°/s 4 X 70% Mfmax - 150°/s	4 X 70% Mfmax - 30°/s 4 X 70% Mfmax - 30°/s 4 X 70% Mfmax - 30°/s 4 X 70% Mfmax - 30°/s

MFmax : Moment de force maximal déterminé lors du bilan isocinétique initial, **r :** temps de repos en secondes



Les patients ont été revus au cours de l'année 2012. L'évaluation fonctionnelle est réalisée en utilisant le score IKDC subjectif et l'EVA : avant la première séance de rééducation isocinétique (Iso T1), à la fin de la dernière séance isocinétique (Iso T2) et au dernier recul (DR). Au total, 45 patients ont répondu à notre enquête selon 2 modalités, soit en remplissant le questionnaire lors d'un entretien téléphonique, soit en renvoyant par courrier électronique ou postal les questionnaires.

L'analyse statistique est réalisée par l'intermédiaire du test *t* de Student pour les données non appariées. Les scores IKDC sont comparés par le test de Wilcoxon. La distribution des variables quantitatives est évaluée par le test de corrélation de Pearson. Les différences sont considérées statistiquement significatives lorsque le risque d'une erreur de type I est inférieur à 5 %. Les statistiques sont réalisées à l'aide du logiciel de statistique *R*.

RÉSULTATS

Au total, 45 des 48 patients inclus sont revus. Les 3 patients manquants sont perdus de vue. La répartition des scores IKDC en fonction du sexe et du type de greffe est reproduite dans le tableau 1. Les reculs moyens sont respectivement de 10 mois ± 5 pour le délai Chirurgie-Iso T1, de 28 mois ± 11 pour le délai Chirurgie-DR et de 17 mois ± 9 pour le délai IsoT1-DR. Les scores IKDC à la fin de l'isocinétisme et au dernier recul se révèlent indépendants de l'âge au moment de la chirurgie, du sexe et du type de greffe. Les scores IKDC et EVA sont améliorés de manière très significative entre la première (Iso T1) et la dernière séance d'isocinétisme (Iso T2), ainsi qu'entre la dernière séance d'isocinétisme et le dernier recul (DR) avec respectivement un gain moyen de 28 et de 9 points pour l'IKDC ($p<0,0001$), et une diminution moyenne de 2,3 et de 1,4 points pour l'EVA ($p < 0,0001$).

L'IKDC au dernier recul est corrélé positivement de manière significative avec le temps, aussi bien pour le délai après chirurgie ($p=0,047$, $r=0,297$), que pour le délai après la première séance d'isocinétisme ($p=0,019$, $r=0,347$). La figure 1 présente la courbe de corrélation entre l'IKDC au dernier recul et le délai après la chirurgie et la figure 2 la courbe de corrélation entre l'IKDC au dernier recul et le délai après la première séance l'isocinétisme. La corrélation entre les résultats fonctionnels et le délai avant la première séance d'isocinétisme n'est pas significative, mais le lien s'est révélé très faible, aussi bien pour l'IKDC à la fin de l'isocinétisme ($p=0,26$, $r=0,171$), que pour l'IKDC au dernier recul ($p=0,86$, $r=0,027$).

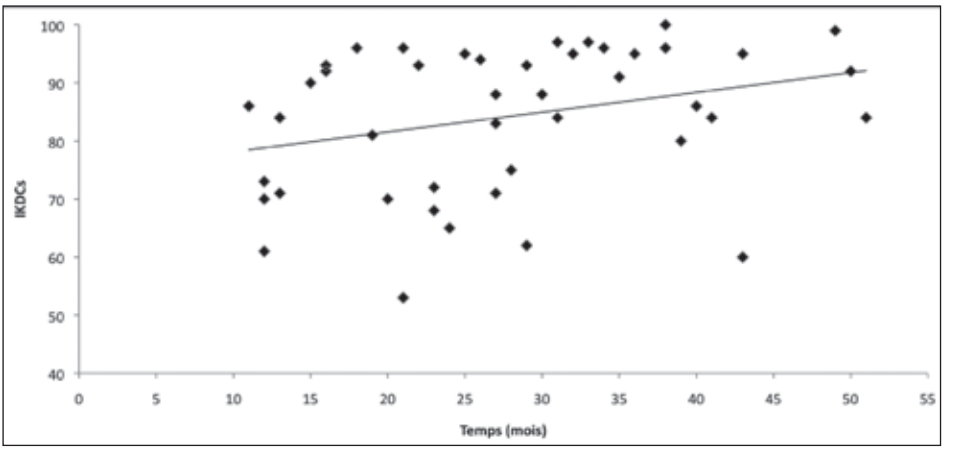


Fig. 1 : Corrélation entre L'IKDC au dernier recul et le délai entre la chirurgie et le dernier recul.

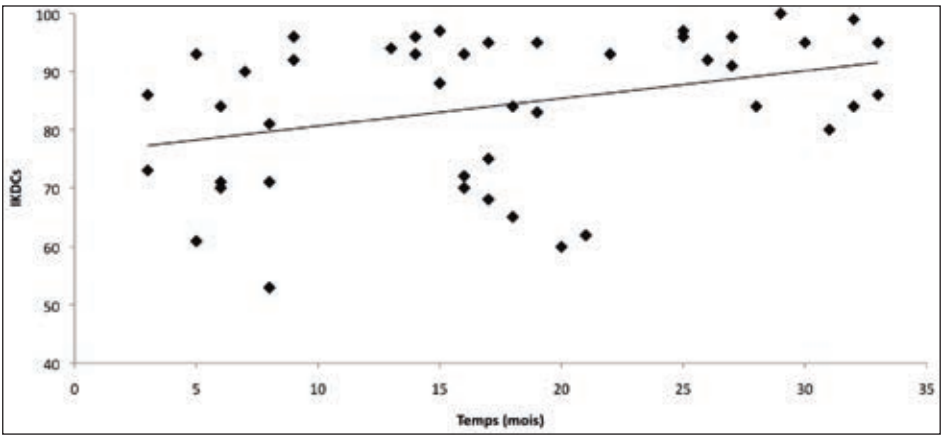


Fig. 2 : Corrélation entre L'IKDC au dernier recul et le délai entre la première séance d'isocinétisme et le dernier recul.

Tableau 3 : Moyenne des résultats fonctionnels

		Iso T1		Iso T2		DR	
		IKDC	EVA	IKDC	EVA	IKDC	EVA
Greffe	Extenseur	52	6,15	75	3,2	84	1,8
	Ischiojambiers	43	4,9	75	3,3	84	2,1
Sexe	Femme	44	5,8	75	3,2	85	1,6
	Homme	51	5,5	75	3,4	83	2,2
Total		47	5,6	75	3,3	84	1,9

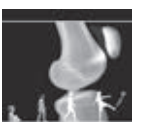
Iso T1 : avant la première séance d'isocinétisme, **Iso T2 :** après la dernière séance d'isocinétisme, **DR :** lors de la révision au dernier recul.

On dénombre 6 reprises pour cure d'un cyclope. Le délai médian était respectivement de 8 mois (7-10) entre la reprise chirurgicale et la première séance d'isocinétisme et de 31,5 mois (7-39) entre la reprise chirurgicale et le dernier recul. Avec respectivement un IKDC médian de 75,5 (60-84) et 84 (60-97) points et une EVA médiane de 2,5 (2-8) et de 2,5 (1-8) points à la fin de l'isocinétisme et au dernier recul, les cyclopes ne semblaient pas associés à une diminution des résultats fonctionnels (ns).

DISCUSSION

Notre étude démontre que la rééducation isocinétique est efficace dans les douleurs antérieures après reconstruction du LCA notamment lorsque celles-ci résistent aux techniques usuelles de rééducation. Les résultats montrent une amélioration incontestable du score IKDC subjectif et une diminution de l'EVA dès la fin de l'isocinétisme. Par analogie avec le traitement du syndrome rotulien idiopathique, ces résultats ne sont pas surprenants [18, 19]. En revanche, la poursuite assez nette de l'amélioration entre la fin de l'isocinétisme et le dernier recul ainsi que la corrélation entre la durée du recul et l'amélioration de l'IKDC indépendamment de la mise en place de l'isocinétisme sont des paramètres beaucoup plus originaux.

Dans notre série, un des résultats surprenant est l'amélioration en 2 temps, avec un premier palier après l'isocinétisme puis un deuxième qui survient plus progressivement avec la reprise

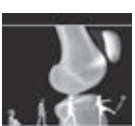


des activités. Tout se passe comme si l'isocinétisme était un catalyseur de la rééducation permettant de relancer le processus de renforcement musculaire. Par sa capacité à travailler en zone infra-douloureuse, l'isocinétisme casse le cercle vicieux *"douleur-déficit musculaire"* et restaure un environnement musculaire compatible avec la réalisation d'exercices plus classiques. En revanche, il ressort clairement de notre étude que l'isocinétisme ne permet pas à lui seul de traiter un syndrome rotulien douloureux fonctionnel. Cela tient probablement au caractère imprévisible de la récupération musculaire après rupture puis reconstruction du LCA faisant intervenir des paramètres aussi divers que des processus neurophysiologiques réflexes d'inhibition musculaire [20], de cicatrisation des sites de prélèvement [21], ou encore de cocontraction quadriceps-ischiojambiers [22]. Ces résultats nous conduisent à émettre une deuxième hypothèse sur le rôle délétère joué par les douleurs antérieures dans l'entretien du déficit musculaire. L'apparition d'un syndrome rotulien fonctionnel entraîne non seulement une stagnation dans la récupération musculaire mais aussi, du fait de la douleur, une sous-utilisation du membre responsable qui aggrave le déficit. Dans cette hypothèse, le recours rapide à l'isocinétisme dans les douleurs antérieures invalidantes résistantes, ou s'aggravant avec la rééducation classique, nous apparaît comme une priorité.

Même si le coefficient de corrélation est assez faible, l'analyse des courbes entre l'IKDC au dernier recul et le temps montre une corrélation positive significative entre les deux paramètres. Une analyse monofactorielle simpliste pourrait conduire à relativiser les effets de la rééducation isocinétique car, en corrélant le niveau de récupération fonctionnelle à la durée du recul, nos résultats laissent à penser que l'introduction de l'isocinétisme n'accélère pas la régression des douleurs antérieures après reconstruction du LCA. Cette analyse nous semble erronée car il faut toujours garder à l'esprit que l'origine des douleurs antérieures après ligamentoplastie est mixte et très hétérogène. On dénombre notamment des douleurs tendineuses et osseuses secondaires au prélèvement, des douleurs sur le tunnel tibial, des douleurs neuropathiques sur des lésions des branches du nerf saphène, des douleurs dans le cadre du "cyclop syndrome" passés inaperçus et un syndrome douloureux régional complexe le plus souvent associé à une patella baja [23]. Chacune de ces causes a sa propre évolution et interfère de manière directe sur la chronologie du syndrome douloureux. L'absence de prise en charge spécifique ou tout simplement l'évolution naturelle de ces étiologies sont probablement des biais qui conduisent à une sous-évaluation de l'efficacité de l'isocinétisme. Dans l'avenir, la comparaison au sein d'une population identique – des délais de récupération entre 2 protocoles de rééducation, classique versus isocinétisme, devrait nous permettre de confirmer cette hypothèse.

A l'heure actuelle, il n'existe pas de certitude sur le délai minimum à partir duquel la ligamentisation et l'évolution histologique des ancrages autorisent la pratique d'exercices isocinétiques. L'isocinétisme est un modèle de rééducation en chaîne cinétique ouverte. Or l'utilisation du principe de la chaîne ouverte après reconstruction du LCA est discutée. Le risque apparaît double, avec sur le plan mécanique la faillite de la greffe par défaut de maturation qui présente un risque de laxité antéropostérieure [24, 25], et sur le plan fonctionnel le risque de décompensation d'un syndrome rotulien par mise en charge de la fémoro-patellaire [26, 27]. En se basant sur les travaux d'Amiel [28], nous restons prudents et ne préconisons pas l'utilisation de l'isocinétisme avant 4,5 mois révolus. En ce qui concerne la surcharge fémoropatellaire, la maîtrise de la vitesse et l'asservissement de la résistance confèrent à l'isocinétisme un avantage considérable par rapport aux protocoles de rééducation classiques en permettant un renforcement dans des conditions infra douloureuses [13].

Une des données majeures de notre étude est le taux de cyclop syndrome opéré qui avec 6 cas soit 12,5 % de la série, est bien supérieur à celui retrouvé dans la littérature (3 %) [29]. Par définition, le cyclop syndrome est responsable d'un flessum irréductible ce qui en fait un mo-



dèle *in vivo* de déficit d'extension. Ces résultats font penser que le déficit d'extension en post-opératoire est un facteur péjoratif du résultat de la ligamentoplastie. Ceci rejoint les résultats de Natri qui avait montré, dans une population de LCA reconstruits, le lien entre le déficit d'extension et le déficit musculaire [5]. Nous manquons à ce jour d'arguments scientifiques formels, mais ce phénomène s'explique probablement par la sous-utilisation du Vastus Médialis secondaire à la limitation des activités contractiles du quadriceps dans des secteurs proches de l'extension. Ce processus peut entraîner une fonte musculaire rapide avec une désynchronisation de l'unité musculo-articulaire Vastus Latéralis/Vastus Médialis Oblique [30]. Tout en contre-indiquant l'hyperextension [31], ces résultats vont dans le sens de nos protocoles de rééducation qui font de la lutte contre le flessum une priorité. Nous insistons particulièrement sur la lutte contre la co-contraction quadriceps-ischiojambiers avec l'apprentissage dès le préopératoire d'exercices d'auto-contractions isométriques du quadriceps. Nous avons de plus introduit dans nos protocoles la réalisation précoce d'étirements doux en chaîne cinétique fermée des ischiojambiers au moyen d'une planche à roulette. Comme le propose Isberg, une des pistes de réflexion dans l'avenir pourrait être l'introduction précoce, uniquement en rééducation, de certains exercices en chaîne cinétique ouverte [32].

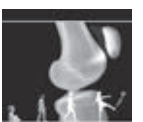
En termes de renforcement musculaire, l'apport des appareils isocinétiques comme outil de rééducation nous apparaît considérable car il permet un renforcement spécifique du quadriceps et des ischio-jambiers aussi bien en mode concentrique qu'excentrique. Le quadriceps est à l'heure actuelle considéré comme le muscle responsable du syndrome rotulien fonctionnel. Or, après reconstruction du LCA, quel que soit le type de greffe et malgré des rééducations bien conduites, c'est le quadriceps qui met le plus de temps à récupérer [6]. Il nous paraît important de rappeler dans cet article quelques principes biomécaniques du rôle joué par le quadriceps sur la rotule [33]. La contraction concentrique du quadriceps est essentielle pour le verrouillage de la rotule, principalement proche de l'extension où le vastus médialis joue un rôle capital dans le centrage de la rotule. La contraction excentrique du quadriceps est essentielle dans les situations de surcharge de l'articulation fémoropatellaire comme lors de la descente des escaliers car il absorbe comme un amortisseur la majeure partie du poids du corps. Si le rôle du déficit des ischiojambiers, dans les syndromes rotuliens après reconstruction du LCA, est plus discuté que celui du quadriceps [22, 34, 35], nous pensons qu'il ne faut pas le négliger. Dans l'avenir, les objectifs de la rééducation isocinétique pourraient à terme s'étendre à la récupération du ratio agoniste/antagoniste, mais à l'heure actuelle nos protocoles ne prennent pas en compte ce paramètre dans les objectifs de rééducation.

Les limites de notre travail sont le caractère rétrospectif, la faiblesse de l'effectif, le caractère subjectif de l'évaluation et l'absence de comparaison avec les techniques de rééducation classiques. Notre méthodologie est critiquable, car la population inclue n'est pas représentative de toutes les douleurs antérieures après reconstruction du LCA mais seulement des syndromes rotuliens fonctionnels pris en charge par isocinétisme.

CONCLUSION

Notre étude montre que l'isocinétisme après reconstruction du LCA est une technique qui permet une amélioration fonctionnelle considérable dans les syndromes rotuliens fonctionnels résistants aux techniques usuelles de rééducation.

A notre connaissance, il s'agit de la première étude basée sur l'évaluation de l'isocinétisme comme outil de rééducation dans les syndromes rotuliens fonctionnels après reconstruction du LCA. L'isocinétisme ne permet pas, à lui seul, la régression des douleurs antérieures, en revanche son apport comme catalyseur de la rééducation en fait un outil indispensable en complément des techniques classiques de rééducation pour débloquer les situations d'échec de ces dernières.



LITTERATURE

[1] FREEDMAN KB, D'AMATO MJ, NEDEFF DD, KAZ A, BACH BR Jr Arthroscopic anterior cruciate ligament reconstruction: a metaanalysis comparing patellar tendon and hamstring tendon autografts. *Am J Sports Med. 2003 févr;31(1): 2-11.*

[2] SACHS RA, DANIEL DM, STONE ML, GARFEIN RF. Patellofemoral problems after anterior cruciate ligament reconstruction. *Am J Sports Med. 1989 déc; 17(6): 760-5.*

[3] DAUTY M, TORTELIER L, HUGUET D, POTIRON-JOSSE M, DUBOIS C. Consequences of pain on isokinetic performance after anterior cruciate ligament reconstruction using a semitendinosus and gracilis autograft. *Rev Chir Orthop Reparatrice Appar Mot. 2006 sept; 92(5): 455-63.*

[4] CONDOURET J, COHN J, FERRET J-M, LEMONSU A, VASCONCELOS W, DEJOUR D. *et al.* Isokinetic assessment with two years follow-up of anterior cruciate ligament reconstruction with patellar tendon or hamstring tendons. *Rev Chir Orthop Reparatrice Appar Mot. 2008 déc; 94(8 Suppl): 375-82.*

[5] NATRI A, JÄRVINEN M, LATVALA K, KANNUS P. Isokinetic muscle performance after anterior cruciate ligament surgery. Long-term results and outcome predicting factors after primary surgery and late-phase reconstruction. *Int J Sports Med. 1996 avr; 17(3): 223-8.*

[6] KOBAYASHI A, HIGUCHI H, TERAUCHI M, KOBAYASHI F, KIMURA M, TAKAGISHI K. Muscle performance after anterior cruciate ligament reconstruction. *Int Orthop. 2004 févr; 28(1): 48-51.*

[7] SETO JL, OROFINO AS, MORRISSEY MC, MEDEIROS JM, MASON WJ. Assessment of quadriceps/hamstring strength, knee ligament stability, functional and sports activity levels five years after anterior cruciate ligament reconstruction. *Am J Sports Med. 1988 avr; 16(2): 170-80.*

[8] CARTER TR, EDINGER S. Isokinetic evaluation of anterior cruciate ligament reconstruction: hamstring versus patellar tendon. *Arthroscopy. 1999 mars; 15(2): 169-72.*

[9] DAUTY M, TORTELLIER L, ROCHCONGAR P. Isokinetic and anterior cruciate ligament reconstruction with hamstrings or patella tendon graft: analysis of literature. *Int J Sports Med. 2005 sept; 26(7): 599-606.*

[10] LAUTAMIES R, HARILAINEN A, KETTUNEN J, SANDELIN J, KUJALA UM. Isokinetic quadriceps and hamstring muscle strength and knee function 5 years after anterior cruciate ligament reconstruction: comparison between bone-patellar tendon-bone and hamstring tendon autografts. *Knee Surg Sports Traumatol Arthrosc. 2008 nov; 16(11): 1009-16.*

[11] AGEBERG E, ROOS HP, SILBERNAGEL KG, THOMÉE R, ROOS EM. Knee extension and flexion muscle power after anterior cruciate ligament reconstruction with patellar tendon graft or hamstring tendons graft: a cross-sectional comparison 3 years post surgery. *Knee Surg Sports Traumatol Arthrosc. 2009 févr; 17(2): 162-9.*

[12] ROCHCONGAR P. Isokinetic thigh muscle strength in sports: a review. *Ann Readapt Med Phys. 2004 août; 47(6): 274-81.*

[13] FOSSIER E, DANIEL F. Renforcement musculaire isocinétique. Méthodologie, intérêt et limites. Muscle et rééducation. *Paris: Masson; 1988. p. 180-8.*

[14] FELLER JA, WEBSTER KE, GAVIN B. Early post-operative morbidity following anterior cruciate ligament reconstruction: patellar tendon versus hamstring graft. *Knee Surg Sports Traumatol Arthrosc. 2001 sept; 9(5): 260-6.*

[15] JÄRVELÄ T, KANNUS P, LATVALA K, JÄRVINEN M. Simple measurements in assessing muscle performance after an ACL reconstruction. *Int J Sports Med. 2002 avr; 23(3): 196-201.*

[16] NAKAMURA N, HORIBE S, SASAKI S, KITAGUCHI T, TAGAMI M, MITSUOKA T, *et al.* Evaluation of active knee flexion and hamstring strength after anterior cruciate ligament reconstruction using hamstring tendons. *Arthroscopy. 2002 août; 18(6): 598-602.*

[17] SHELBOURNE KD, GRAY T. Anterior cruciate ligament reconstruction with autogenous patellar tendon graft followed by accelerated rehabilitation. A two- to nine-year followup. *Am J Sports Med. 1997 déc; 25(6): 786-95.*

[18] ALACA R, YILMAZ B, GOKTEPE AS, MOHUR H, KALYON TA. Efficacy of isokinetic exercise on functional capacity and pain in patellofemoral pain syndrome. *Am J Phys Med Rehabil. 2002 nov; 81(11): 807-13.*

[19] HAZNECI B, YILDIZ Y, SEKIR U, AYDIN T, KALYON TA. Efficacy of isokinetic exercise on joint position sense and muscle strength in patellofemoral pain syndrome. *Am J Phys Med Rehabil. 2005 juil; 84(7): 521-7.*

[20] JOHANSSON H, SJÖLANDER P, SOJKA P. Activity in receptor afferents from the anterior cruciate ligament evokes reflex effects on fusimotor neurones. *Neurosci Res 1990 avr; 8(1): 54-9.*

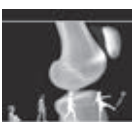
[21] KARTUS J, MOVIN T, KARLSSON J. Donor-site morbidity and anterior knee problems after anterior cruciate ligament reconstruction using autografts. *Arthroscopy 2001 déc; 17(9): 971-80.*

[22] MORE RC, KARRAS BT, NEIMAN R, FRITSCHY D, WOO SL, DANIEL DM. Hamstrings--an anterior cruciate ligament protagonist. An *in vitro* study. *Am J Sports Med. 1993 avr; 21(2): 231-7.*

[23] GAUDOT F, CHALENCON F, NOURISSAT G, DEJOUR D, POTEL J.F, FRISCHTY D. *et al.* Impact of anterior knee pain on mid term outcome after anterior cruciate ligament reconstruction. *Rev Chir Orthop Reparatrice Appar Mot. 2008 déc; 94(8 Suppl): 372-4.*

[24] HENNING CE, LYNCH MA, GLICK KR Jr. An *in vivo* strain gage study of elongation of the anterior cruciate ligament. *Am J Sports Med. 1985 févr; 13(1): 22-6.*

[25] HEIJNE A, WERNER S. Early versus late start of open kinetic chain quadriceps exercises after ACL reconstruction



with patellar tendon or hamstring grafts: a prospective randomized outcome study. *Knee Surg Sports Traumatol Arthrosc. 2007 avr; 15(4): 402-14.*

[26] STEINKAMP LA, DILLINGHAM MF, MARKEL MD, HILL JA, KAUFMAN KR. Biomechanical considerations in patellofemoral joint rehabilitation. *Am J Sports Med. 1993 juin; 21(3): 438-44.*

[27] ESCAMILLA RF, FLEISIG GS, ZHENG N, BARRENTINE SW, WILK KE, ANDREWS JR. Biomechanics of the knee during closed kinetic chain and open kinetic chain exercises. *Med Sci Sports Exerc. 1998 avr; 30(4): 556-69.*

[28] AMIEL D, KLEINER JB, ROUX RD, HARWOOD FL, AKESON WH. The phenomenon of "ligamentization": anterior cruciate ligament reconstruction with autogenous patellar tendon. *J Orthop Res 1986; 4(2): 162-72.*

[29] JACKSON DW, SCHAEFER RK. Cyclops syndrome: loss of extension following intra-articular anterior cruciate ligament reconstruction. *Arthroscopy. 1990; 6(3): 171-8.*

[30] VOIGHT ML, WIEDER DL. Comparative reflex response times of vastus medialis obliquus and vastus lateralis in normal subjects and subjects with extensor mechanism dysfunction. An electromyographic study. *Am J Sports Med 1991 avr; 19(2): 131-7.*

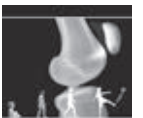
[31] WASCHER DC, MARKOLF KL, SHAPIRO MS, FINERMAN GA. Direct *in vitro* measurement of forces in the cruciate ligaments. Part I: The effect of multiplane loading in the intact knee. *J Bone Joint Surg Am 1993 mars; 75(3): 377-86.*

[32] ISBERG J, FAXEN E, BRANDSSON S, ERIKSSON BI, KÄRRHOLM J, KARLSSON J. Early active extension after anterior cruciate ligament reconstruction does not result in increased laxity of the knee. *Knee Surg Sports Traumatol Arthrosc. 2006 nov; 14(11): 1108-15.*

[33] BOUCHER JP, KING MA, LEFEBVRE R, PÉPIN A. Quadriceps femoris muscle activity in patellofemoral pain syndrome. *Am J Sports Med. 1992 oct; 20(5): 527-32.*

[34] MARTINEZ-MORENO JL. Idiopathic painful patella syndrome: an etiopathogenic hypothesis. *Rev Chir Orthop Reparatrice Appar Mot 1994; 80(3): 239-45.*

[35] DRAGANICH LF, JAEGER RJ, KRALJ AR. Coactivation of the hamstrings and quadriceps during extension of the knee. *J Bone Joint Surg Am. 1989 août; 71(7): 1075-81.*



ANTERIOR KNEE PAIN 3 MONTHS AFTER ACL RECONSTRUCTION: AN INTERNATIONAL SURVEY OF PRACTICE

J. BARTH, J.C. PANISSET, F. MAURIS, N. BONIN, B. SONNERY-COTTET, D. DEJOUR and THE ALRM Team

The morbidity of anterior knee pain occurring after ACL reconstruction may be moderate to severe and if symptoms persists and resist to medical care, the patient may not be able to resume sports practice on time or with lower performances. The origin of symptoms is sometimes obvious, such as Cyclops syndrome with an associated lack of extension (fig. 1). In this case, the solution is arthroscopic arthrolysis, which gives satisfactory results for the patient. But most of the time, the pain is not clearly identified and is frequently called "tendinitis" without any evidence in terms of imaging!

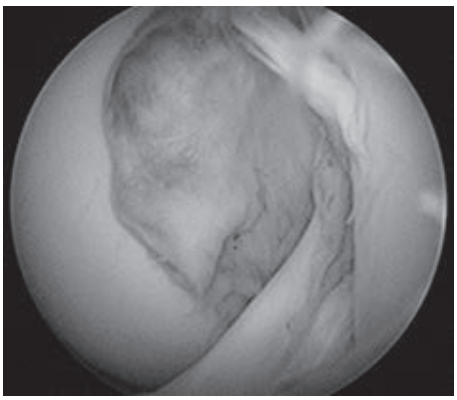


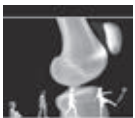
Fig. 1: Anterior knee pain related to a Cyclops syndrome hanging at the roof of the notch. Note that the patient had a very slight lack of extension.

Actually, this type of pain could be explained by various pathologies, not always easy to diagnose or to treat successfully, such as cartilage edema, stiffness, neuroma, muscle insufficiency, adhesive wounds, bone bruise, graft harvesting sequels, tendinitis, complex regional pain syndrome, or reflex sympathetic dystrophy syndrome [1].

The aim of our study was to analyze how physicians from all over the world diagnose and treat these problems. We also wanted to design a simple general algorithm to facilitate our approach to this difficult issue.

MATERIAL AND METHODS

A survey was designed and emails were sent to a mailing list delivered by the MCO Congress. Orthopedic surgeons, sports medicine physicians, and rehabilitation physicians were the target of our survey. The survey was built on a free web access service delivered by *sondageonline.com* in two possible languages (French or English). A simple questionnaire was sent to all participants via a link on an E-card at *http://www.sondageonline.com/live.php?code=b7d520a*. Answers were collected and processed on excel spreadsheets.



RESULTS

On August 26, 2012, 94 volunteers had participated in the survey. The mean age of physicians was 47.5 years (30 to 65). Most of them were French (45%) and Orthopedic surgeons (83%). The details of answers are listed in the following figures and tables.

The number of participants (NP) for each question is given at the beginning to understand the tables. For each line, the participants could choose one answer in the various columns (C1, C2, …, Cn). The number on each column (NC1, NC2, …, NCn) is the number of answers. Therefore NC1+NC2+….+NCn = NP. Column ∅ shows where most of the responses are located. For example, if there are 4 columns and if C1 is "never", C2 is "sometimes"; C3 is "often", C4 is "always", and ∅ = 2.5, this means that most of the answers are located between "sometimes" and "often". Column G is the mean score of subjective confidence for each item. For example, 0% means that the item is not relevant, whereas 100% means that the item is highly relevant.

DISCUSSION AND CONCLUSION

This was not a literature review but a general practice analysis on an international sample of physicians. We will give you our opinion later, without any scientific evidence but based on our local experience (Level 5: Expert Opinion).

The preferred imaging for early postoperative care (3-6 months) was plain X-rays whereas MRI was the gold standard after 6 months. On plain X-rays, one might consider tunnel placement, especially tibial or femoral tunnels paced too anteriorly, fractures due to graft harvesting such as for patellar tendon or quadriceps tendon, but also patellofemoral dysplasia. MRI scans are useful to screen for Cyclops syndrome, bone bruising around tunnels, edema in the fat pad, arthrofibrosis around the graft, tendinitis (fig. 2), or meniscal lesions. We agree that ultrasound is not always contributive to understand anterior pain, especially for intra-articular problems, even though some peripheral disorders may be identified with this inexpensive exam. Examinations with an injected contrast medium may to required in doubtful cases, such as cartilage lesions, meniscal bucket handle lesions, or on the physician's special requestor associate an injection of corticoids. Arthro-MRI is probably the best option to avoid radiations and it allows better evaluating soft tissues.

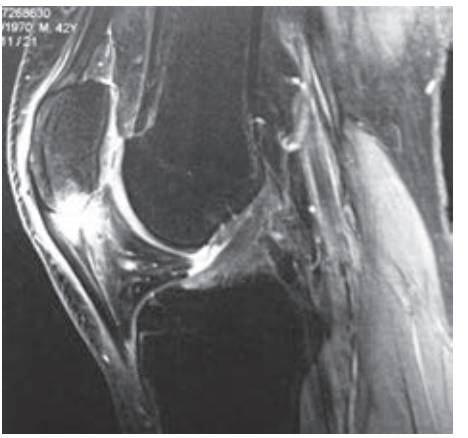
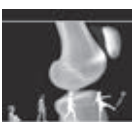


Fig. 2: Anterior knee pain related to patellar tendinitis.

Isokinetic evaluation seems to become a routine tool since only 20.5% of participants had no access to the machine. Indeed, it is the only way to have an objective evaluation of the functional recovery of muscle chains (extensors and flexors), with precise and reproducible measures. Moreover some conditions such as the patellofemoral syndrome may be diagnosed by analyzing the shape of the curves.

Our physicians embraced the self-rehabilitation protocol to treat anterior knee pain, using cryotherapy, stretching, and cycling (67 to 79% of reliability). These standard techniques were also commonly prescribed by physicians, with supervised rehabilitation by a physiotherapist. Scar massage, deep transverse massage, and shockwaves seem to be more controversial (46 to 50% of reliability). However, in severe



cases of tendinitis after a failed standard treatment and within a reasonable delay (after one year), shockwaves might be helpful but we are currently lacking evidence.

Isokinetic rehabilitation proved to be of real interest since it was considered reliable for 67% of our participants. We believe that this technique improves the standard technique of cycling by providing better feedback on the patient's reactions and it offers the possibility to use eccentric strengthening programs.

Analgesics and NSAIDs were considered as standard for symptomatic treatment (respectively 65 and 63% of reliability) whereas corticoids, PRP, mesotherapy, conservative podiatric management, local NSAID were rarely believed to be effective to relieve anterior knee pain (oscillating between 19 and 38% of reliability). The use of cortisone injection and visco-supplementation is more controversial but should find a place in cases of cartilage lesions in the patellofemoral or tibiofemoral compartment.

We believe that the "tendinitis" should no longer be used for all disorders related to anterior knee pain. The physician needs to differentiate structural or anatomical lesions from functional problems. To do so, the first step is to obtain objective findings such as postoperative plain X-ray pictures to look for patellofemoral dysplasia (N. Bonin reported 15% of dysplasia in the population of ruptured ACL compared to 3% in the general population), fractures, osteopenia (reflex sympathetic dystrophy), patella bipartita, tunnel mal-positioning, type 2 Patella in the Grelsamer classification [1] (the "big nose" that could be a cause of tendinitis by impingement). Furthermore, MRI should be performed to identify the precise origin of the pain that could be inside the patellofemoral compartment or be an anterior projection of a femorotibial compartment disorder. In the patellofemoral compartment, one might look for cartilage edema, tendinitis, Hoffa syndrome, Cyclops syndrome, synovitis, arthrofibrosis, or bone bruising (condyles or tibial tunnel) [2]. The femorotibial compartments need to be screened as well for cartilage or meniscal lesions. If no structural lesion can be seen on the MRI and if a neuropathic or neurological problem (such as neuroma of infrapatellar branches of the saphenous nerve) is excluded by the DNS4 score, functional disorders might be investigated with an isokinetic evaluation (fig. 3). After 3 postoperative months, flexor insufficiency often jeopardizes the jogging phase. After 6 postoperative months, it is possible to assess ratios between extensor and flexor muscles, which need to be well balanced, with deficits lower than 15% to avoid pain or a new injury. Condouret showed that there was a relationship between the level of extensor and flexor recovery and the quality of functional results with minimal muscle deficits close to 5% if the IKDC score was over 90 and deficits falling to 15% in the group with IKDC score less than 90 [3].

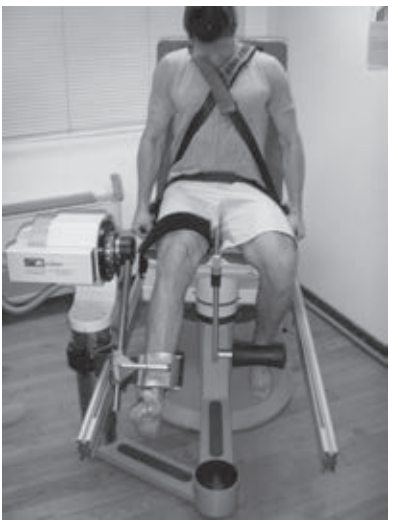
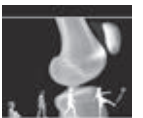


Fig. 3: Anterior knee pain due to extensor insufficiency. Isokinetic assessment.

Once the diagnosis is accurately made, an adapted treatment is possible in addition to cryotherapy, NSAIDs, and painkillers. If cycling and strengthening programs remain the gold standard for patellofemoral pain with or without signs of dysplasia, we learned from E. Arendt, that core strengthening was equally



important. According to a recent non-published study by Gadea, isokinetic rehabilitation (at 240°/sec), by acting as catalyst, seemed to boost the healing phase [4].

Corticoid injections are effective for cartilage edema, Hoffa syndrome, synovitis, arthrofibrosis, or degenerative meniscal lesions. In case of severe cartilage lesions (arthritis), visco-supplementation is a good option.

If a true tendinitis is identified with MRI, one might consider eccentric stretching and strengthening. We believe that isokinetic rehabilitation permits to better control all parameters to work under the threshold of pain.

Finally, an arthroscopic evaluation should never be considered as a simple diagnostic arthroscopy but only to treat a Cyclops syndrome or a meniscal lesion previously analyzed by imaging.

RESULTS OF THE SURVEY

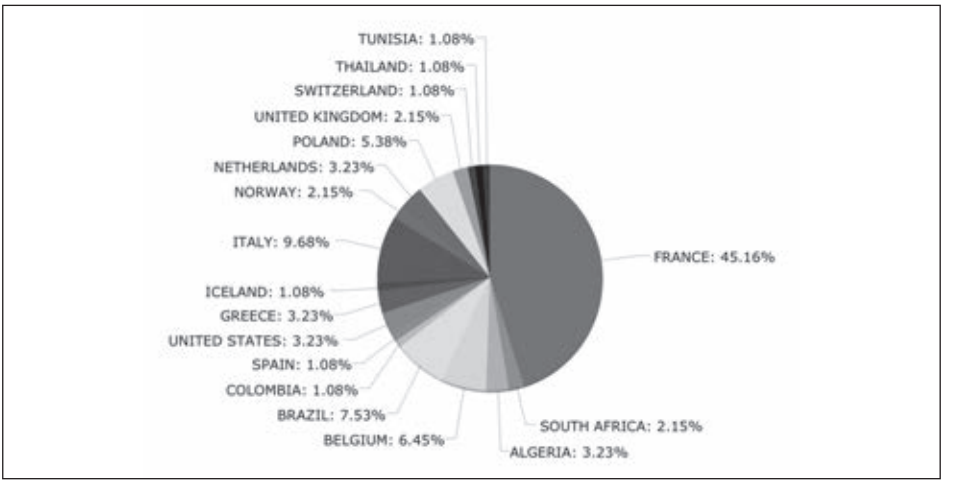


Fig. 1: Country of physicians having participated in the survey (n=93).

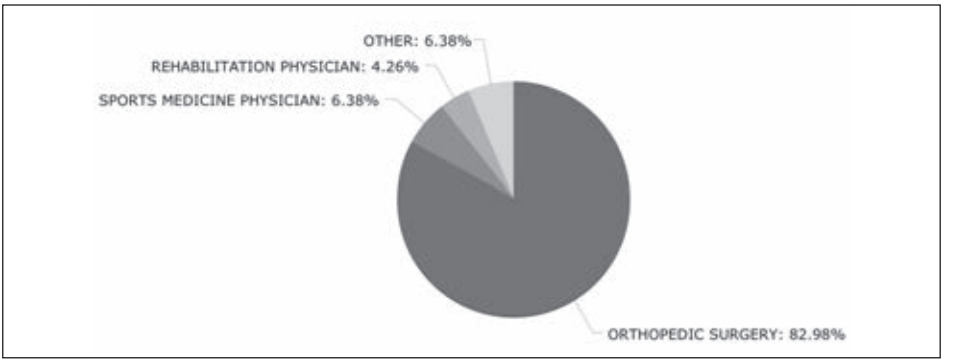


Fig. 2: Specialty of physicians having participated in the survey (n=94).

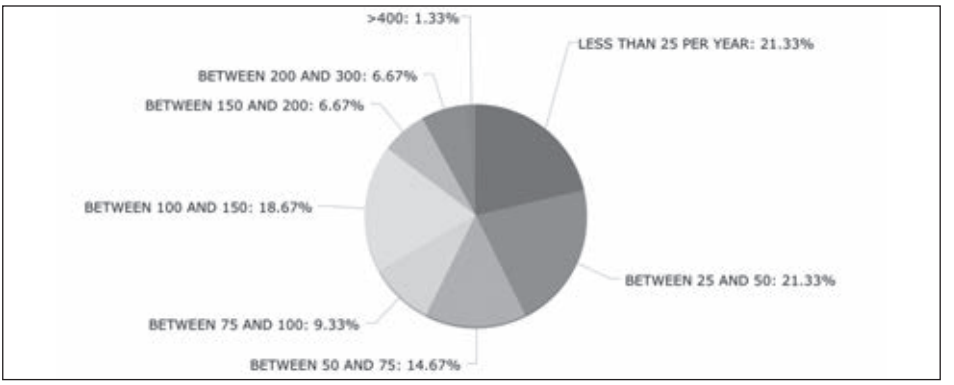


Fig. 3: Number of ACL reconstructions performed per year by orthopedic surgeons (n=75).

Nombre de réponses : 76

	NEVER (1)	SOMETIMES (2)	OFTEN (3)	ALWAYS (4)	Ø
PATELLAR TENDON	5x	37x	26x	8x	2.49
Hamstring	5x	12x	38x	19x	2.96
QUADRICIPITAL TENDON	43x	28x	2x	-	1.44
FASCIA LATA	68x	4x	1x	-	1.08
SYNTHETIC GRAFT	67x	5x	1x	-	1.10
ALLOGRAFT	46x	26x	1x	-	1.38
OTHER	72x	1x	-	-	1.01

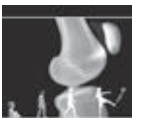
Table 1: Type(s) of ACL reconstruction(s) performed by orthopedic surgeons (n=76).

Nombre de participants: 78

	3-6 MONTHS (1)	6-12 MONTHS (2)	> 12 MONTHS (3)	NOT prescribed (0)	Ø	G
ULTRASOUND	23x	8x	1x	46x	1.31	36%
Plain radiographs	56x	14x	-	8x	1.20	65%
MRI	22x	40x	9x	7x	1.82	82%
ARTHRO-MRI	1x	3x	3x	71x	2.29	32%
ARTHRO-CT-SCAN	6x	16x	7x	49x	2.03	53%
OTHER	4x	4x	4x	66x	2.00	22%

Ø = Répartition moyenne de points par ligne
G = Pondération de l'importance par ligne en % (0% insignifiant / 100% primordial)

Table 2: Answers to the question "What type of images do you routinely ask to identify the cause of anterior knee pain and when?" (n=78).



Nombre de participants: 78

	NEVER (1)	SOMETIMES (2)	OFTEN (3)	ALWAYS (4)	NO ACCESS FOR isokinetic (0)	Ø	G
YOU ASK FOR ISOKINETIC EVALUATION	18x	22x	10x	12x	16x	2.26	57%

Ø = Répartition moyenne de points par ligne
G = Pondération de l'importance par ligne en % (0% insignifiant / 100% primordial)

Table 3: Answers to the question "Do you use isokinetic evaluation for anterior knee pain after ACL reconstruction?" (n=78).

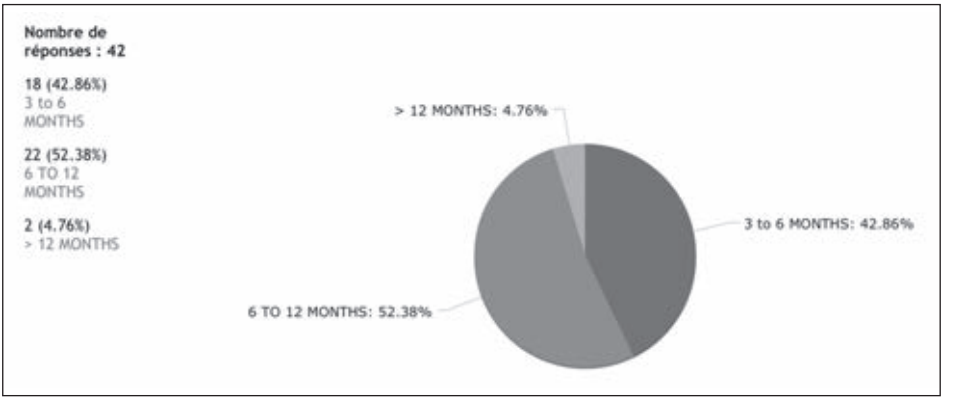


Fig. 4: Answers to the question: "When do you use isokinetic evaluation for anterior knee pain after ACL reconstruction?" (n=42).

Nombre de participants: 67

	NEVER (1)	SOMETIMES (2)	OFTEN (3)	ALWAYS (4)	Ø	G
CRYOTHERAPY	6x	12x	28x	21x	2.96	67%
STRETCHING	2x	5x	21x	39x	3.45	79%
CYCLING	6x	11x	23x	27x	3.06	70%
OTHER	34x	9x	13x	11x	2.01	37%

Ø = Répartition moyenne de points par ligne
G = Pondération de l'importance par ligne en % (0% insignifiant / 100% primordial)

Table 4: Answers to the question "Do you use a self-rehabilitation protocol to treat pain after ACL reconstruction?" (n=67).

Nombre de participants: 67

	NEVER (1)	SOMETIMES (2)	OFTEN (3)	ALWAYS (4)	Ø	G
SCAR MASSAGE	15x	20x	19x	13x	2.45	50%
DEEP TRANSVERSE MASSAGE	21x	20x	19x	7x	2.18	47%
STRETCHING	3x	5x	25x	34x	3.34	78%
CYCLING	7x	8x	25x	27x	3.07	68%
STRENGTHENING	5x	8x	23x	31x	3.19	72%
ISOKINETIC REHABILITATION	10x	16x	28x	13x	2.66	67%
SHOCKWAVES	25x	30x	9x	3x	1.85	46%
CRYOTHERAPY	5x	14x	28x	20x	2.94	66%
OTHER	39x	12x	8x	8x	1.78	31%

Ø = Répartition moyenne de points par ligne
G = Pondération de l'importance par ligne en % (0% insignifiant / 100% primordial)

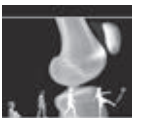
Table 5: Answers to the question "Do you ask for supervised rehabilitation by a physiotherapist for anterior knee pain after ACL reconstruction?" (n=67).

Nombre de participants: 67

	AFTER 3 MONTHS, AT ANY TIME (1)	3-6 MONTHS (2)	6-12 MONTHS (3)	> 12 MONTHS (4)	NOT prescribed (0)	Ø	G
ANALGESIC	48x	5x	-	-	14x	1.09	65%
NON STEROID ANTI-INFLAMMATORY PILS	42x	14x	1x	-	10x	1.28	63%
CORTICOSTEROIDS PILS	2x	2x	2x	1x	60x	2.29	19%
CORTISON INJECTION	3x	7x	11x	5x	41x	2.69	40%
VISCO-SUPPLEMENTATION	2x	13x	11x	8x	33x	2.74	43%
PRP INJECTIONS	2x	6x	12x	4x	43x	2.75	38%
LOCAL NSAI	18x	6x	3x	1x	39x	1.54	34%
MESOTHERAPY	7x	8x	7x	2x	43x	2.17	31%
CONSERVATIVE PODIATRIC MANAGEMENT	4x	2x	12x	4x	45x	2.73	32%
OTHER	11x	2x	1x	-	53x	1.29	18%

Ø = Répartition moyenne de points par ligne
G = Pondération de l'importance par ligne en % (0% insignifiant / 100% primordial)

Table 6: Answers to the question "What kind of medical treatment do you use for anterior knee pain after ACL reconstruction?" (n=67).



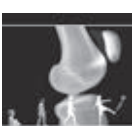
LITERATURE

[1] S ZAFFAGNINI, D DEJOUR, E ARENDT. Editors. The Book entitled Patellofemoral Pain, Instability, and Arthritis. Clinical presentation, Imaging, and treatment. *Springer-Verlag Berlin Heidelberg 2010*.

[2] F GAUDOT, F CHALENCON, G NOURISSAT, D DEJOUR, JF POTEL D FRITSCHY, P. BEAUFILS. La société française d'arthroscopie. Impact of anterior knee pain on mid term outcome after anterior cruciate ligament reconstruction. *Rev Chir Orthop 2008; 94S: S372-S374*.

[3] J CONDOURET, J COHN, JM FERRET, A LEMONSU, W VASCONCELOS, D DEJOUR, JF POTEL, ET LA SOCIÉTÉ FRANÇAISE D'ARTHROSCOPIE. Isokinetikc assessment with two years follow-up of anterior cruciate ligament reconstruction with patellar tendon or hamstring tendons. *Rev Chir Orthop 2008; 94S: S375-S382*.

[4] M. GADEA FRANÇOIS SOUS LA DIRECTION DE B. SONNERY-COTTET. Evaluation d'un protocole de rééducation isocinétique dans la prise en charge des douleurs antérieures du genou après ligamentoplastie du LCA. *Mémoire DIU d'arthroscopie*. *Année universitaire 2011-12*.



L'INTRODUCTION DE L'EXAMEN TOMODENSITOMÉTRIQUE DANS L'ÉTUDE DES INSTABILITÉS ROTULIENNES

P. CHAMBAT, J.M. FAYARD

La pathologie rotulienne a toujours fait partie des préoccupations de l'école lyonnaise du genou dont le précurseur a été A. Trillat, avec à la fin des années 50, début des années 60 la mise au point de l'intervention de transfert médial de la tubérosité tibiale antérieure. Ce geste chirurgical, dont la technique n'avait pas été publiée préalablement, faisait suite à une discussion entre A. Trillat et un chirurgien anglais du nom d'Elmslie. C'est sous ce nom que va être connue cette technique caractérisée par la persistance d'une "charnière" distale lors du transfert de l'insertion du ligament rotulien diminuant les contraintes au niveau du bloc osseux et permettant sans problème la fixation par une seule vis en compression.

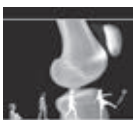
C. Roux [1] de Lausanne avait préalablement décrit une technique de transfert médial de la TTA mais le bloc osseux correspondant à l'insertion du tendon rotulien était totalement détaché pour être médialisé comme l'a également décrit J. Goldthwait [2].

Cette expérience concernant l'opération d'Elmslie a été publiée en 64 par A. Trillat et H. Dejour [3] qui était alors chef de clinique. Dans cet article, nous retrouvons pour le diagnostic le signe de l'appréhension appelé signe de Smillie [4] préalablement décrit par Fairbank [5], la description de l'intervention du transfert de la tubérosité antérieure et les résultats cliniques obtenus.

Cette orientation va se perpétuer, et dans tous les programmes des *"Journées Lyonnaises du Genou"* depuis 1971 sous la direction d'Albert Trillat, d'Henri Dejour puis des plus jeunes, nous retrouvons un chapitre consacré à la rotule.

Une nouvelle impulsion quant à cette pathologie a été donnée pour la préparation des 4e Journées du Genou de 1982 grâce au travail d'un chef de clinique du service de H. Dejour, P. Brugère qui a conduit une étude sur l'analyse clinique, radiologique et tomodensitométrique de 3 groupes d'individus : 1 groupe témoin, 1 groupe de patients présentant un syndrome rotulien et 1 groupe souffrant d'une instabilité rotulienne objective avec luxation vraie de la rotule.

Il notait cliniquement, outre le signe de l'appréhension positif, plus de valgus, de rotations externes du genou, d'antéversions de hanche dans le groupe des instabilités. Radiologiquement, il retrouvait une augmentation de l'angle trochléen, un rapport, longueur de la facette latérale de la trochlée sur longueur de la facette médiale augmentée et une rotule plus haute dans ce même groupe.



Depuis la fin des années 79, l'examen tomodensitométrique (scanner) des membres inférieurs avait fait progressivement parti de l'arsenal mis à la disposition des chirurgiens orthopédiques pour affiner les diagnostics cliniques. JL. Lerat en 1982 [6] avait publié une étude sur l'usage de cet examen pour l'étude morphologique du membre inférieur.

Il devenait alors intéressant de déterminer de façon plus précise avec cet examen le morphotype des patients présentant une instabilité rotulienne. L'étude des torsions fémorales et tibiales ou des rotations dans le genou déjà décrites ne posait pas de problème particulier. Mais dans cette pathologie, le scanner pouvait apporter plus en permettant de mieux mesurer l'angle trochléen, la bascule rotulienne. Il a permis aussi de simplifier l'évaluation d'une mesure publiée en 1978 par D. Goutallier [7] correspondant à "l'écart tubérosité antérieure - gorge de la trochlée TA-GT".

Toutes ces mesures sur le scanner ont, dans un premier temps, été effectuées de façon artisanale en utilisant un rétroprojecteur projetant les images obtenues de façon à avoir sur l'écran un agrandissement de 100 %. Les images étaient alors décalquées rassemblées sur une même feuille de papier ce qui permettait de mesurer les différents angles et la TAGT.

Le travail de P. Brugère jamais publié avait montré dans le groupe "instabilité rotulienne" par comparaison avec le groupe "syndrome rotulien", un angle d'antéversion fémorale plus élevé, une mesure de la TA-GT plus importante et une plus grande bascule rotulienne.

A partir de cette période, l'examen tomodensitométrique a fait partie des moyens diagnostiques obligatoires lorsqu'il s'agissait d'évaluer une pathologie rotulienne. La technique des radiologues s'était améliorée avec des mesures d'angle ou de TA-GT réalisées sur leur table numérique.

C'est à G. Walch dans le service de H. Dejour, aidé par J. Tuneu que revient le mérite d'avoir repris l'idée du travail de P. Brugère et d'avoir travaillé entre autres, sur le scanner pour définir le niveau des coupes et les mesures à effectuer. Un protocole strict a alors été mis au point avec les pieds fixés en rotation externe de 15°, l'examen se déroulant avec un temps statique et un temps dynamique.

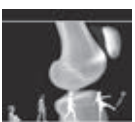
Pour l'exploration statique, il était préconisé :
- Une coupe au niveau des hanches passant par le sommet de la fossette digitale. JL. Lerat préconisait une coupe à ce niveau plus une coupe passant par la base du col fémoral en cas de coxa valga.
- Une coupe passant par le milieu de la rotule et une autre au niveau de la trochlée. Le repère pour cette coupe étant l'échancrure intercondylienne au niveau où elle réalise un arc "roman".
- Une coupe passant par l'épiphyse tibiale supérieure située entre la partie supérieure de l'articulation péronéo tibiale supérieure et le bord postérieur du plateau tibial externe.
- Une coupe passant par la tubérosité tibiale antérieure.
- Une coupe au niveau de la cheville passant par les malléoles.

L'exploration dynamique était réalisée avec une coupe passant par le milieu de la rotule, quadriceps contracté, plus une coupe à 15° de flexion sans contraction.

Ces coupes permettaient de définir :

Les angles de torsion
- *Antéversion fémorale :* angle formé par l'axe du col et l'axe bi-condylien postérieur.
- *La rotation dans le genou :* angle entre l'axe bi-condylien postérieur et l'axe d'orientation postérieure de l'épiphyse tibiale.
- *La torsion tibiale externe :* angle formé entre l'axe d'orientation postérieure de l'épiphyse tibiale et l'axe bi-malléolaire.

La TA-GT
- Réalisé par la superposition de deux coupes passant par la tubérosité antérieure et la trochlée. Deux points définissant le milieu de la



Tubérosité antérieure et le fond de la trochlée sont abaissés perpendiculairement sur la ligne bi-condylienne postérieure où la mesure TA-GT est effectuée. Cette mesure prend en compte la latéralisation de la Tubérosité antérieure plus une éventuelle rotation dans le genou.

L'articulation femoro patellaire

- L'angle trochléen.
- La pente du versant externe de la trochlée.
- La subluxation rotulienne mesurée par la distance en millimètre entre deux lignes perpendiculaires à la ligne bi-condylienne postérieure passant par le fond de la trochlée et la crête médiane de la rotule.
- L'inclinaison trochléenne : angle formé par une ligne joignant les sommets des deux berges de la trochlée et la ligne bi-condylienne postérieure.
- La bascule rotulienne : angle formé entre l'axe transversal de la rotule et la ligne bi-condylienne postérieure.

L'étude tomodensitométrique a porté sur quatre groupes de patients avec :

- *16 scanners* pour ceux présentant des instabilités rotuliennes majeures (IRM) (luxation permanente ou habituelle).
- *217 scanners* pour ceux présentant des instabilités rotuliennes objectives (IRO) (au minimum une luxation vraie).
- *53 scanners* pour ceux présentant des instabilités rotuliennes potentielles (IRP) (définies par l'examen clinique).
- *60 scanners* pour des sujets témoins.

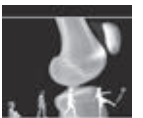
Les résultats publiés ultérieurement par H. Dejour [8, 9, 10] montraient :

- Une anté-version significativement plus importante dans le groupe IRO par comparaison avec le groupe témoin.
- Pas de différence significative au niveau de l'angle condyle-malléole (somme de la torsion tibiale et de la rotation dans le genou).
- Une rotation dans le genou significativement plus importante dans le groupe IRM comparativement aux 3 autres groupes (IRO, IRP, Témoins) et le groupe IRO comparativement aux 2 autres groupes (IRP et témoins).
- Une TA-GT significativement plus importante dans les groupes IRM et IRO par comparaison avec le groupe témoin avec une corrélation statistique positive entre la rotation dans le genou et la TA-GT.

Toutes ces évaluations restent d'actualité avec une importance plus particulière pour la mesure de la TA-GT, l'angle de la trochlée et la bascule rotulienne. En dehors du scanner, l'étude radiologique faite sur les mêmes groupes de malades va permettre de confirmer l'étude faite préalablement par P. Brugère et de définir les dysplasies de trochlée, ce qui va être débattu ultérieurement.

CONCLUSIONS

L'examen tomodensitométrique a représenté un progrès certain dans l'évaluation de la pathologie rotulienne et reste 24 ans après d'actualité. Après 1987, année où les *6e Journées du Genou* ont été consacrées à la rotule, des améliorations ont été apportées. Certaines écoles ont adopté les images par résonance magnétique qui intègrent le cartilage et qui permettent aussi de calculer plus exactement la longueur du tendon rotulien. Mais ces deux examens ont un point commun, la connaissance dans le plan horizontal, très mal exploré par la radiologie conventionnelle.



RÉFÉRENCES

[1] ROUX D. Luxation habituelle de la rotule. *Rev Chir Paris 1888; 8: 682-9.*

[2] GOLDTHWAIT JE. Dislocation of the patella. *Trans Am Orthop Assoc 8: 237, 1895.*

[3] TRILLAT A, DEJOUR H, COUETTE A. Diagnostic et traitement des subluxations récidivantes de la rotule. *Rev Chir Orthop, 1964 50: 813-24.*

[4] SMILLIE IS. Injuries of the knee joint. *The Williams & Wilkins Cº 1962.*

[5] FAIRBANK HA. Internal derangement of the knee in children. *Proc R Spc London 1937; 3: 11.*

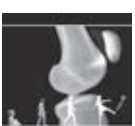
[6] LERAT JL, MOYEN B, BOCHU M. Clinical examination of bone axes in the adult. Tomodensitometry. *Rev Chir Orthop Reparatrice Appar Mot. 1982; 68 (1) 37-43.*

[7] GOUTALLIER D, BERNAGEAU J, LECUDONNEC B. Mesure de l'écart tubérosité tibiale antérieure gorge de la trochlée (TA-GT) technique. Résultat, Intérêt. *Rev Chir Orthop 64: 423-8, 1978.*

[8] DEJOUR H, WALCH G, NOVE-JOSSERAND L, GUIER C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc. 1994; 2: 19-26.*

[9] DEJOUR H, WALCH G, NEYRET P, ADELEINE P. Dysplasia of the femoral trochlea *Rev Chir Orthop Reparatrice Appar Mot. 1990; 76: 45-54.*

[10] WALCH G, DEJOUR H. Radiology in femoro-patellar pathology. *Acta Orthop Belg. 1989; 55(3): 371-80.*



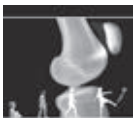
MAGNETIC RESONANCE IMAGING IN PATELLOFEMORAL INSTABILITY

M. CHARLES, R. AFRA, D.C. FITHIAN

INTRODUCTION

Diagnosis of patellofemoral instability can be difficult because patellar instability, patellofemoral pain, meniscal and cruciate ligament insufficiencies can produce a similar presentation of nonspecific pain and knee instability. Careful examination and imaging are helpful in making the correct diagnosis. In addition to the history, evaluation should include a search for abnormal limb alignment, patellar tilt, crepitus, patellar tracking, tenderness, apprehension, and laxity [1]. Imaging is another component to the diagnosis of patellofemoral instability. Fithian *et al.* demonstrated that recurrent dislocators had specific factors such as smaller angles of Laurin. A smaller angle of Laurin demonstrates lateral rotation of the patella along in its cephalad-caudal axis and larger lateral patellar overhang on imaging at the time of their first dislocation [2]. Historically, plain radiographic imaging and subsequently CT have dominated the clinician's attention regarding the evaluation of the patellofemoral joint. More recently, MRI has gained a more prominent role in the imaging armamentarium for patellofemoral instability because it gives the clinician the ability to visualize the cartilaginous articular surfaces and ligaments in addition to the bony alignment observed on X-ray and CT.

Patellofemoral instability is a problem orthopedic surgeons have tried to address as early as Albee in 1915 [3]. Merchant helped visualize the patellofemoral joint in 1974 with patient's knee flexed at 45° and the camera angled at 30° [4]. Dejour *et al*. utilized lateral radiographs to analyze osseous femoral morphology (i.e. trochlear dysplasia) to classify trochlear dysplasia into 4 types (I-IV) based on radiographic findings of the crossing sign, trochlear bump >3mm, and of trochlear depth <4mm [5, 6]. Radiographic and CT based standard values for the patellofemoral joint are well established. Measurements of trochlear morphology like lateral trochlear inclinination <11°, sulcus angle >145°, and trochlear groove depth <4mm have been well documented cutoffs [1, 5-7]. Limb geometry evaluation with measurements like Tibial Tuberosity-Trochlear Groove (TTTG) distance, which was first established by Judet *et al.*, have proven useful for evaluating patients with instability. A TTTG value greater than 20mm is considered pathologic [5, 8]. Standards for Caton-Deschamps and Insall-Salvati ratios indicative of *patella alta*, are based in either plain films or CT imaging [5, 9, 10]. Even the analysis of dynamic stabilizers, as measured by their effects on patellar tilt, also have their roots in CT and plain films [5, 11-14].



More recently, MR imaging has been applied to the analysis of patellofemoral instability. The ability of MR images to clearly represent the articular cartilage has improved the clinician's understanding of severe cartilaginous dysmorphology that was not brought to light with previous radiographic studies [15-17]. Recent articles have compared the reliability of MRI and CT in evaluating the patellofemoral joint [9, 17-23]. Some studies have demonstrated the accuracy of MRI to evaluate the patellofemoral joint. However, these articles have also highlighted discrepancies between previously established CT and radiographic cutoffs and the MRI based evaluation of those same measures.

Currently, there is a limited amount of research applying MRI imaging to patellofemoral instability. We have investigated some of the key morphological differences between normal knees and those with recurrent patellofemoral instability [24].

OBSERVATIONS

Patellar Tilt (fig. 1)

As with all the measurements of patellar tilt, these angles reflect that patients with patellofemoral instability had an increase in the lateral rotation of the patella around its superior to inferior pole. All patellar tilt measurements were found to be significant between the two groups. Angle of Laurin (Controls 10.10°±0.48; PFJDs -5.23°±2.96; p<.001) and Angle of Fulkerson (Controls 18.18°±0.56; PFJDs -3.5°±2.62; p<.001) are examples of classic measures that were found to be significant. The lateral displacement of the patella (Controls 3.28mm±0.24; PFJDs 6.59±0.69; p<.001) was also significant.

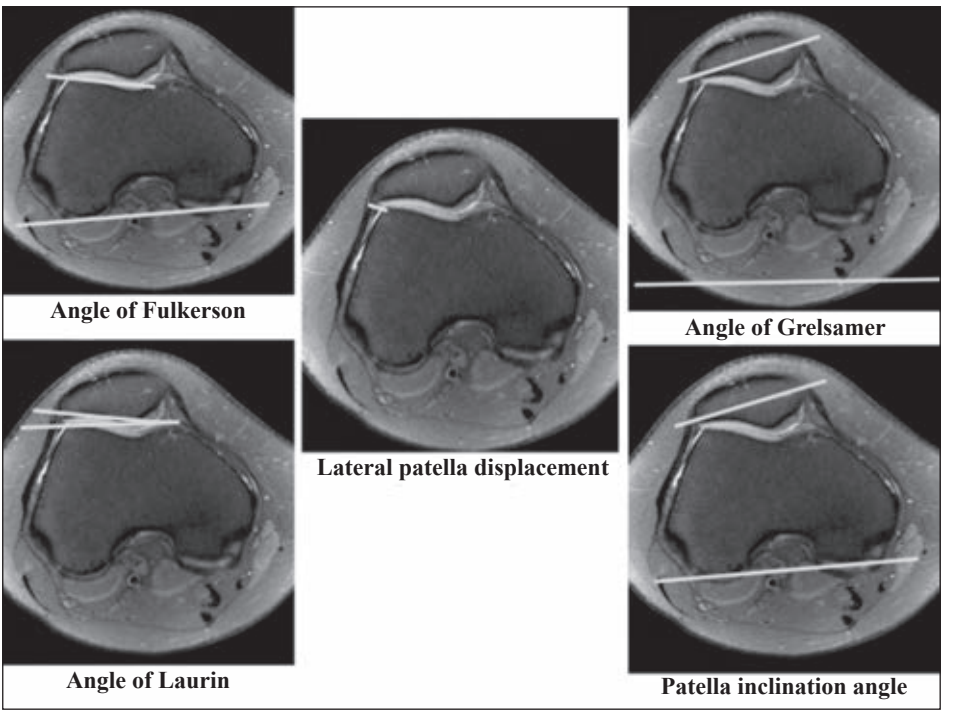


Fig. 1: Patellar Alignment

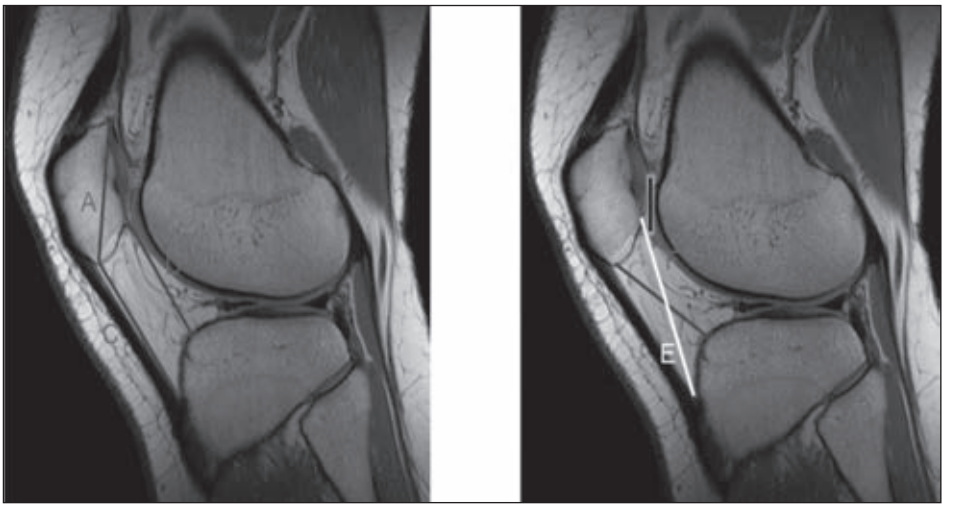
Patellar Station (fig. 2)

Patellar station has been researched extensively [1, 5, 8, 9, 20, 25-28], yet our data demonstrated significance only with the Insall-Salvati Ratio (Controls 1.08±0.02; PFJDs 1.26±0.03; p<.001) and the Caton-Deschamps Ratio (Controls 1.13±0.02; PFJDs 1.29±0.03; p<.001). The remaining measurements lacked both non-overlapping confidence intervals and p values <0.05.

Trochlear Morphology (fig. 3)

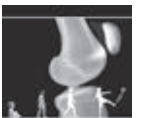
Numerous measurements of trochlear morphology were significant at the proximal and distal trochlea. At the proximal trochlea (1st Cut) significant measurement included classic measurements like Sulcus Angle (Controls 148.48°±0.94; PFJDs 165.57°±2.65; p<.001) and Lateral Trochlear Inclination (Controls 21.27°±0.66; PFJDs 13.31°±1.36; p<.001). But lesser known measurements like ETIT (Controls 1.51±0.05; PFJDs 2.11±0.17; p<.001), which is a measurement of facet asymmetry, were also found to be significant. Lateral Condylar Height (LCH), Central Condylar Height as percentage of epicondylar width (% CCH), Medial Condylar Height (MCH) and Medial Condylar Height as percentage of epicondylar width (% MCH) were found to be significant at the proximal trochlea. The medial condylar height was significant in the distal trochlea.

Measurements at the distal trochlea were more likely to demonstrate significance, though the difference between the means was often larger at the proximal trochlea. Interestingly, Lateral Condylar Height lost significance at the distal trochlea (p=0.643). Also worth noting is that while all initial condylar height measurements lacked significance at the distal trochlea, when adjusted for epicondylar width they all proved significant. For example, LCH as a percent of epicondylar width had a mean of 81%±1 for Controls and 88%±1 for PFJDs. Transepicondylar width demonstrates significant difference between groups (Controls 71.00mm±0.76; PFJDs 75.23mm±0.95; p<.001). The Trochlear Groove Thickness was not significant at either point along the trochlea (fig. 4).



Insall-Salvati = C/A Modified Insall = E/B Articular Overlap Canton-Deschamps = D/B
Morphology Ratio = B/A PF Contact Surface Ratio = B/Articular Overlap

Fig. 2: Patellar Station



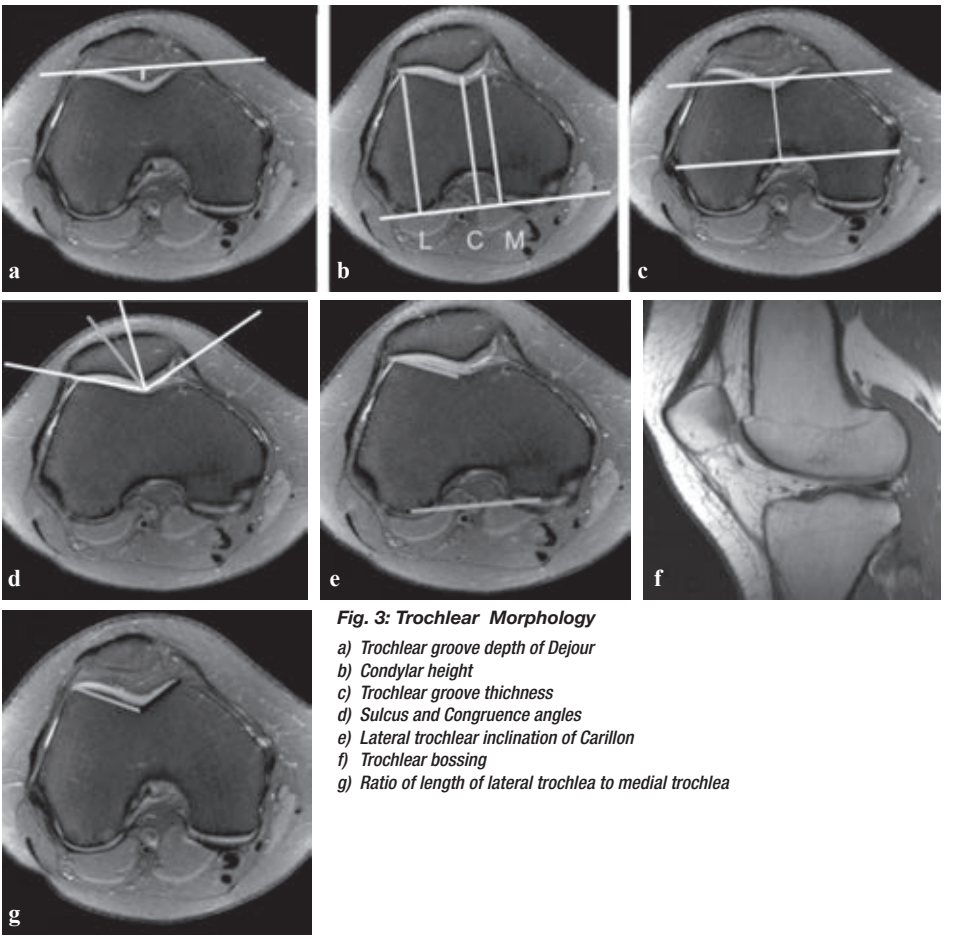


Fig. 3: Trochlear Morphology

a) Trochlear groove depth of Dejour
b) Condylar height
c) Trochlear groove thichness
d) Sulcus and Congruence angles
e) Lateral trochlear inclination of Carillon
f) Trochlear bossing
g) Ratio of length of lateral trochlea to medial trochlea

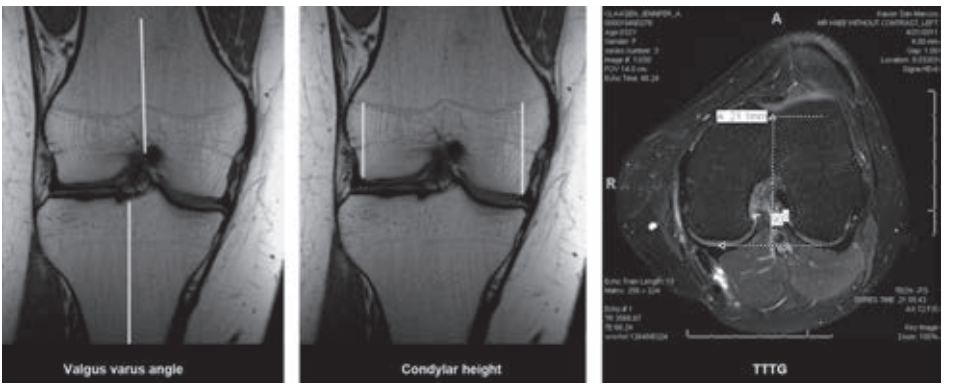


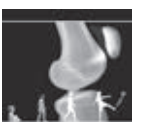
Fig. 4: Limb Geometry and Other measurements

DISCUSSION

Previous authors have utilized MRI imaging to perform measurements of patellar tilt [9, 23]. Patellar tilt proved to be an excellent group of measurements for delineating between Controls and those with instability. The confidence intervals between the two groups were separated by at least 5° for each measurement. For the Angle of Fulkerson the means of 18.18°±0.56 and -3.5°±2.62 for Controls and PFJDs respectively support the established cutoff of <8° for pathology established by Schutzer *et al.* [29]. Patellar Inclination Angle is another well-studied measurement. Our means of 8.10°±0.55 for Controls and 24.03°±2.42 for PFJDs differed from Dejour *et al.* (10°±4.3 vs. 16°±3.3) CT based study [5]. Our results were very similar, however, to those by Escala *et al.* whose MRI based study found means of 9.2 and 21.7 for controls and PFJDs, respectively [9]. This is an interesting result, as patellar inclination angle would not be predicted to change between CT and MRI imaging as articular cartilage is not involved in its measurements. For the Angle of Laurin our results (Controls 10.10°±0.48; PFJDs -5.23°±2.96) again differed from Biedert's CT based study with means of -0.1° and -3.1° [7]. The Angle of Laurin relies on the articular cartilage both on the trochlea and the patella; thus this difference is to be expected. As mentioned earlier, all our measurements reflected an increase in the lateral tilt of the patella. The increase in lateral tilt can be attributed to laxity or even rupture of medial soft tissue structures, specifically the medial patellofemoral ligament (MPFL) [8]. Biedert *et al.* indirectly showed that even a weak vastus medialis could contribute to an increase in lateral tilt by demonstrating a difference in patellar angles with and without active quadriceps contraction [7].

The significance of *patella alta* is that an elevated patella will not engage the bony architecture of the proximal trochlea that is necessary to prevent lateralization of the patella. Patellar station, demonstrated limited success in terms of finding significant differences. One could assume that MRI inclusion of articular cartilage would make some of these measurements unreliable, however Miller *et al.* demonstrated that patellar height measurements could be reliably recorded on MRI [20]. The Insall-Salvati Ratio has a well-documented pathological value of >1.2 for *patella alta* based on XR measures [30]. Our means of 1.08±0.02 for Controls and 1.26±0.03 for PFJDs follows this initial cutoff. Our findings are also similar to what Escala *et al.* found in their MRI based study (1.11 and 1.35) [9]. Caton-Deschamps Ratio was the only other patellar height ratio found to be significant. Our means of 1.13±0.02 and 1.29±0.03 for Controls and PFJDs again reflect the standard cutoff of >1.2 significant for *patella alta* [5, 10].

Trochlear morphology was the main focus of our research and yielded some very interesting results. Our Sulcus Angle means 148.48°±0.94 (Controls) and 165.67°±2.65 (PFJD) reflect a difference from the classic cutoff of 145° [1, 5-7]. While they do reflect an increase in the angle, likely due to inclusion of articular cartilage, our patellofemoral instability patients did not have quite as large sulcus angle as found by Van Huyssteen *et al.* and Ali *et al.* Van Huyssten's group showed cartilage based mean sulcus angles of 186.5° [17], while Ali *et al.* published angles of 173° [18]. These differences could be attributed to a difference in the location where the measurement was made: Van Huuyssteen *et al.* made their measurements within 3mm of the start of articular cartilage and Ali *et al.* based their location on the portion with the greatest ventral prominence. It should be noted that the mean sulcus angles at our distal trochlea were closer to the classic cutoff. But since the initial cutoff was established on a Merchant view of the knee, one can see how the values may vary. Trochlear Groove depth is another classic measurement with a cutoff of <4mm being pathological for patellofemoral instability [5]. Other authors have reported that MRI based trochlear depth measures have not varied much from the initial CT or X-ray values [9, 18, 21]. Our means of 6.47mm±0.24 for Controls and 4.00mm±0.43 for PFJDs at the proximal trochlea differ from the classic cutoff but prove similar to the results of Escala *et al.*

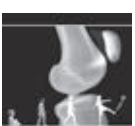


[9]. This could be a result of better visualization of the articular cartilage but other MRI studies have actually had means less than 3mm for patients with patellofemoral instability [18, 21]. The means of trochlear groove depth at the distal trochlea of our patients were nearly equal to those at the proximal trochlea.

Lateral trochlear inclination is another measurement of trochlear morphology prevalent in patellofemoral literature [16, 18, 31-33]. These studies have primarily been performed in MRI based studies, with Carrillon *et al.* establishing the cutoff between Controls and those with patellar instability at 11° [31]. This value is lower than our means of 21.27°±0.66 and 13.31°±1.36 at the proximal trochlea and 21.74°±0.52 and 15.95°±0.85 at the distal trochlea. Salzmann *et al.* had values closer to ours, but their research was based on patients selected on a radiographic criteria of trochlear dysplasia [16]. Carrillon's research was similar to our own in that patients were selected on basis of a history and physical examination consistent with patellofemoral dislocations. Carillon *et al.* also performed the measurements at the most proximal slice with complete articular cartilage. Our data still reflects the trend that a decrease in LTI results in less resistance to the lateralization of the patella, which increases chances of patellar instability.

Facet asymmetry is an aspect of trochlear dysplasia that has had very limited research until recently. This has mostly been studied in MRI based studies [16, 21]. Pfirrmann *et al.* first showed that a facet ratio greater than 5:2 (lateral to medial). Note that Pfirrmann *et al.* reported a facet ratio of less than 2:5 (medial to lateral). We inverted this ratio to correspond to our lateral to medial ratio of ETIT. Using ETIT, Pfirrmann identified those with trochlear dysplasia with sensitivity and specificity of 100% and 96%, respectively [21]. We found our means of 1.51 and 2.11 at the 1st cut and 1.40 and 1.97 at the 2nd cut for Controls and PFJDs respectively to be lower than the 2.5 cutoff Pfirrmann documented. This difference may be due to difference in patient selection in that ours were selected on criterion of patellar dislocation, while Pfirrmann *et al.* divided patients based on radiographic evidence of trochlear dysplasia. Salzmann *et al.* also researched facet asymmetry in comparing axial radiographs to MRI. Their research did not find significance for the measurement, but their means for MRI saw the facet ratio increase from 1.6 to 1.9 as the degree of dysplasia increased [16]. This makes our research unique in that it was the first application of facet asymmetry to patellar instability.

Condylar height has thus far had limited research and it has lacked any clear answers. Escala *et al.* evaluated lateral, medial, and central condylar height measurements but none were found to be significant between Controls and those with patellar instability [9]. Our data demonstrated that several measures of trochlear morphology (LCH, % CCH, MCH and % MCH) proved significantly different at the proximal trochlea. LCH presumably has a mechanical role in resisting lateral patellar displacement, while MCH may simply reflect overall hypoplasia of the trochlea or condyles. Interestingly, we also evaluated condylar heights as a proportion of epicondylar width as Biedert *et al.* published in 2009 [34]. At the distal trochlea, despite having no significance with our standard condylar heights, all three condylar heights expressed as a percent of epicondylar width proved significant. Biedert's published means for LCH (Controls 81%; PFJDs 82%), Central Condylar Height (CCH) (Controls 73%; PFJDs 77%), and Medial Condylar Height (MCH) (Controls 76%; PFJDs 79%) [34]. Biedert's results proved similar to our own at the lateral condyle of the distal trochlea, but our groups differed at the central and medial condyle. Biedert *et al.* and our group, however, did find a larger difference between the Normal versus PFJD groups with the medial and central condylar heights than at the lateral condyle. The discussion of which



condylar measurement is ideal for evaluating patellar instability is still muddled. While our standard measurement of the lateral condylar supports its biomechanical role in resisting lateral displacement of the patella, the significance of the central and medial condyles may reflect a lack of condylar development or failure to form trochlear groove.

TTTG has been well researched in patients with patellar instability since Judet *et al.* first established the measurement [1, 5, 8, 19, 22]. Investigators have had mixed results regarding the validity or reliability of MRI-based TTTG measurements compared to CT [19, 22]. Our mean values (Controls 10.96mm±0.39; PFJDs 18.69mm±0.81) reflect the cutoff of 20mm as recommended by Dejour, *et al.* [5]. TTTG is a measurement that is classically based in axial radiographs and CT scans, but as Schoettle *et al.* demonstrated, this measurement can reliably be applied to MRI [22] with similar cutoffs. Our research further corroborates this.

Axial radiographs and CT imaging of the patellofemoral joint is well documented and is the basis for many of our current diagnostic measurements. MRI imaging has the distinct advantage of revealing chondral morphology, which more accurately depicts the patellar-trochlear relationship in comparison to subchondral bone morphology (as would be seen by CT scan and radiography) [15-17]. Recent MR-based studies have begun to show that although MRI is accurate (in that the measured values are reproducible), the preciseness between CT and MRI varies with respect to previously established pathological patellofemoral measures, such as sulcus angle for example [17, 18]. In contrast, other measures, such as Trochlear Groove Depth, have remained similar to CT based measures [9, 18, 21]. Given these mixed results, one could question the practice of applying cut-off values obtained from CT based studies to MRI measurements. Our research shows that some of these classic CT based measurements change while others remain the same when working with MRI imaging.

CONCLUSIONS AND FUTURE DIRECTIONS

By detecting significant morphological differences between the two groups, our findings justify the use of MRI to obtain many of the measures of patellofemoral instability historically obtained with CT scan and plain radiographs. In fact, measures of all the four recognized factors of patellar instability were found significant. This demonstrates that patellofemoral instability is a result of multiple factors, with instances of some small changes over many measurements or a large change in a few key measurements. Patellar tilt measures, such as Angle of Fulkerson, proved to be an excellent group of measurements for delineating between Controls and those with instability. Patella alta ratios, such as Insall-Salvati and Caton-Deschamps, demonstrated statistically significant difference between controls and recurrent dislocators that coincided with established CT cutoff. Trochlear morphology measures such as Sulcus Angle, trochlear groove depth, and lateral trochlear inclination demonstrated statistical significance, though Sulcus Angle and Lateral Trochlear Inclination did differ from established values. The next logical step for research is to pursue statistical analysis of our data to create established cutoffs for MRI as previous groups have with CT and XR and apply them to prospective trials in order to establish which measurements remain a reliable delineators between normal knees and those with patellofemoral instability. By replacing the need for CT, the recurrent patellofemoral patient is exposed to significantly less radiation. As such, MRI is an appropriate tool to aid the clinician in obtaining the radiographic information that would have been obtained by CT scan in patients with recurrent patellofemoral instability.

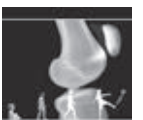


Table 1

Measurement	
Transepicondylar Width	Length of a straight line parallel to the horizontal axis from the most medial to most lateral aspect of the trochlea at the 2nd cut.
Patellar Angle	
Angle of Fulkerson	Line 1 is drawn across the posterior margins of the medial and lateral condyles, and line 2 is drawn along the lateral facet of the patella. The angle between the two lines is PTF.
Angle of Grelsamer	Line 1 is drawn to be parallel to the horizontal axis. Line 2 is drawn through the transverse the axis of the patella. The angle between the two lines is PTG. A positive angle is designated as one that opens up to the medial
Angle of Laurin	Line 1 is drawn to be across the anterior margin of the medial and lateral trochlea. Line 2 is drawn tangential to the slope of the lateral facet of the patella. A positive angle is one that opens up to the laterally
Patellar Inclination Angle	Line 1 is drawn across the posterior margins of the medial and lateral condyles, and line 2 is drawn through the transverse the axis of the patella. A positive angle is designated as one that opens up to the medially.
Lateral Patellar Displacement	This is the shortest distance between the lateral margin of the trochlea and the lateral margin of the patella facet.
Trochlear Morphology	
Sulcus Angle	The angle between the slopes of the medial and lateral trochlea
Congruence Angle	Line 1 is drawn to bisect the sulcus angle of the trochlear groove. Line 2 is drawn from the center of the trochlear groove to the patella apex. A positive angle is designated as one that is towards the laterally. Also known as Merchant Angle.
Trochlear Groove Depth	Line 1 is drawn across the anterior margins of the medial and lateral condyles. TGD is the distance from the center and deepest portion of the trochlear groove to Line 1.
Roman Arch	Line 1 is drawn across the posterior margins of the medial and lateral condyles. RA is the distance from the center of the posterior femoral condylar groove to line 1.
Trochlear Groove Thickness	Line 1 is drawn across the posterior margins of the medial and lateral condyles. Two lines parallel to line 1 are drawn crossing the centers of the trochlear groove and the posterior femoral condylar groove respectively. TGT is the distance between these two lines.
% of Epicondylar Width TGT	Is TGT distance expressed as a percentage of the transepicondylar width.
ETIT	Measures the distance of the lines drawn from the lateral and medial margins of the trochlea along the trochlear surface to the center of the trochlear groove. ETIT is the ratio of ET to IT. A ratio > 1.0 equates to having a lateral facet larger than the medial facet.
Lateral Condylar Height	Line 1 is drawn across the posterior margins of the medial and lateral condyles. LCH is the distance from the most anterior margin of the lateral condyle to Line 1 that is perpendicular to line 1.
% of Epicondyalr Width LCH	LCH expressed as a percentage of the transepicondylar width.
Biedert Central Condylar Height	Line 1 is drawn across the posterior margins of the medial and lateral condyles. CCH is the distance from line 1 to the deepest part of the trochlear groove.
% of Epicondylar Width CCH	CCH expressed as a percentage of the transepicondylar width.

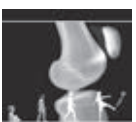


Table 1 (suite)

Medial Condylar Height	Line 1 is drawn across the posterior margins of the medial and lateral condyles. MCH is the distance from the most anterior margin of the medial condyle to Line 1 that is perpendicular to line 1.
% of Epicondylar Width MCH	MCH expressed as a percentage of the transepicondylar width.
Lateral Trochlear Inclination	Line 1 is drawn across the posterior margins of the medial and lateral condyles. Line 2 is drawn along the lateral slope of the trochlear groove. LTI is the angle between the two lines. A positive angle opens laterally.
Limb Geometry	
TTTG	TT-TG offset is defined as the lateral distance of the tibial tuberosity with respect to the center of the trochlear groove, measured parallel to a line connecting the posterior condyles. One way to measure the offset is as follows: **1.** Connect posterior condyles of femur **2.** Draw perpendicular reaching apex of femoral groove **3.** Scroll to apex of tibial tuberosity and leave cursor there **4.** Scroll back to trochlear slice **5.** Draw a line to the 1st perpendicular and measure. This line is parallel to the posterior femoral condylar line
Coronal Measurements	
Varus-Valgus	Line 1 is drawn through the axis of the femur, and line 2 is drawn through the axis of the tibia. CA is the angle between these two lines.
Lateral Condylar Height	LCH is the distance from the physeal scar to the lateral margin of the lateral femoral condyle articular surface.
Medial Condylar Height	MCH is the distance from the physeal scar to the medial margin of the medial femoral condyle articular surface.
Patellar Station	**Using the sagittal image through the mid patella facet as determined on axial image, the following measurements are made.** **Patella Station A:** Measured from the most proximal articular margin of the patella to the most distal (nonarticular) aspect of the patella. **Patella Station B:** Measured from most proximal to most distal articular margin of the patella. **Patella Station C:** Measured from the most distal aspect of the patella to the distal insertion of the patellar tendon at the tibial tuberosity (patella tendon length). **Patella Station D:** Measured from the most distal articular surface of the patella to the anterior margin of the articulating surface of the tibia plateau. **Patella Station E:** Measured from the most distal aspect of the articular surface of the patella to the distal insertion of the patellar tendon at the tibial tuberosity. **Patella Station F:** Measured from the most distal aspect of the patella to the anterior margin of the articulating surface of the tibia plateau
Patellar Trochlear Overlap	Measures the length of the overlap between patellar and trochlear articulating cartilage.
Trochlear Bossing (AP)	Anterior posterior dimensions are estimated on sagittal image.
Insall-Salvati Ratio	Station C divided by Station A
Modified Insall-Salvati Ratio	Station E divided by station B
Caton-Deschamps Ratio	Stations D divided by station B
Morphology Ratio	Station A divided by station B
Femoral Contacting Surface Ratio	Station B divided by the trochlear overlap.

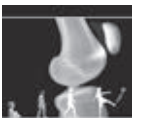


Table 2

Measurement	Control Means + SE	PFJD means + SE	CI 95% Normal PFJDs	Significance
Transepicondylar Width	71.00 ± 0.76	75.23 ± 0.95	80.14 - 83.16 73.30 - 77.16	< .001
Patellar Angle				
Angle of Fulkerson	18.18° + 0.56	-3.5° + 2.62	17.08 - 19.29 -8.79 - 1.79	< .001
Angle of Grelsamer	14.00° + 0.73	24.82° + 2.03	12.55 – 15.45 20.72 – 28.92	< .001
Angle of Laurin	10.10° + 0.48	-5.23° + 2.96	9.14 – 11.05 -11.23 – 0.76	< .001
Patellar Inclination Angle	8.10° + 0.55	24.03° + 2.42	7.02 – 9.19 19.14 – 28.92	< .001
Lateral Patellar Displacement	3.28 + 0.24 mm	6.59 + 0.69 mm	2.79 – 3.77 5.19 – 7.97	< .001
Trochlear Morphology (1st cut)				
Sulcus Angle	148.48° + 0.94	165.57° + 2.65	146.61 – 150.34 160.76 – 171.44	< .001
Congruence Angle	13.95° + 2.00	40.14° + 4.57	9.97 – 17.92 30.89 – 49.38	< .001
Trochlear Groove Depth	6.47 + 0.24 mm	4.00 + 0.43 mm	5.99 – 6.94 3.11 – 4.87	< .001
Roman Arch	17.13 + 0.45 mm	15.39 + 0.73 mm	16.23 – 18.02 13.91 – 16.87	.036
Trochlear Groove Thickness	42.90 + 0.48 mm	42.73 + 0.95 mm	41.95 – 43.86 40.81 – 44.64	.853
% of Epicondylar Width TGT	0.53 ± 0.00	0.56 ± 0.01	0.52 – 0.54 0.55 – 0.58	< .001
ETIT	1.51+ 0.05	2.11 + 0.17	1.42 – 1.61 1.77 - 2.46	< .001
Lateral Condylar Height	63.94 + 0.61 mm	59.69 + 0.89 mm	62.73 – 65.16 57.89 – 61.01	< .001
% of Epicondyalr Width LCH	0.78 ± 0.01	0.80 ± 0.01	0.77 – 0.79 0.78 – 0.82	.061
Biedert Central Condylar Height	60.03 ± 0.63	58.12 ± 1.43	58.78 – 61.29 55.22 – 61.01	.157
% of Epicondylar Width CCH	0.73 ± 0.01	0.76 ± 0.01	0.72 – 0.75 0.74 – 0.79	.018
Medial Condylar Height	58.77 + 0.63 mm	55.53 + 0.99 mm	57.52 -60.03 53.52 – 57.53	.005
% of Epicondylar Width MCH	0.72 ± 0.01	0.74 ± 0.01	0.70 – 0.73 0.72 – 0.77	.036
Lateral Trochlear Inclination	21.27° + 0.66	13.31° + 1.36	19.95 – 22.58 10.56 – 16.07	< .001
TTTG	10.96 ± 0.39	18.69 ± 0.81	10.19 – 11.73 17.06 – 20.32	< .001

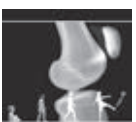
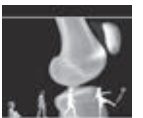


Table 2 (suite)

Trochlear Morphology (2nd Cut)				
Trochlear Groove Depth	5.87 + 0.15 mm	4.06 + 0.37 mm	5.57 – 6.18 3.31 – 4.81	< .001
Roman Arch	24.25 + 0.42 mm	21.28 + 0.48 mm	23.42 – 25.08 20.30 – 22.25	< .001
Trochlear Groove Thickness	37.75 + 0.65 mm	41.35 + 0.93 mm	36.46 – 39.04 39.46 – 43.24	.002
% of Epicondylar Width	0.47 ± 0.01	0.57 ± 0.01	0.46 – 0.49 0.56 – 0.58	< .001
ETIT	1.40 + 0.02	1.97 + 0.07	1.36 – 1.44 1.83 – 2.12	< .001
Sulcus Angle	137.57° + 0.93	155.33° + 1.98	135.72 – 139.41 151.32 – 159.34	< .001
Lateral Condylar Height	65.63 + 0.52 mm	65.21 + 0.72 mm	64.59 - 66.67 63.76 – 66.66	.643
% of Epicondylar Width LCH	0.81 ± 0.01	0.88 ± 0.01	0.80 – 0.82 0.86 – 0.89	< .001
Biedert's Central Condylar Height	62.00 ± 0.55	62.63 ± 0.74	60.90 – 63.10 61.13 – 64.12	.508
% of Epicondylar Width CCH	0.77 ± 0.00	0.84 ± 0.01	0.76 – 0.78 0.83 – 0.86	< .001
Medial Condylar Height	63.83 + 0.52 mm	62.02 + 0.73 mm	62.79 – 64.87 60.54 – 63.50	.048
% of Epicondylar Width MCH	0.78 ± 0.00	0.83 ± 0.01	0.77 – 0.79 0.82 – 0.84	< .001
Lateral Trochlear Inclination	21.74° + 0.52	15.95° + 0.85	20.71 – 22.77 14.24 – 17.66	< .001
Coronal Measurements				
Varus-Valgus	5.45° ± 0.29	10.02° ± 0.53	4.87 – 6.03 8.94 – 11.10	< .001
Lateral Condylar Height	27.94 ± 0.36	26.48 ± 0.50	27.22 – 28.67 25.47 – 27.49	.022
Medial Condylar Height	34.23 ± 0.44	33.08 ± 0.55	33.36 – 35.10 31.97 – 34.19	.122
Patellar Station				
Patellar Trochlear Overlap	14.49 ± 1.52	11.91 ± 1.00	11.89 – 14.29 9.88 – 13.94	.297
Trochlear Bossing (AP)	3.64 ± 0.13	3.97 ± 0.31	3.38 – 3.91 3.35 – 4.60	.245
Insall-Salvati Ratio	1.08 + 0.02	1.26 + 0.03	1.04 – 1.12 1.20 – 1.33	< .001
Modified Insall-Salvati Ratio	1.64 + 0.03	1.72 + 0.04	1.58 – 1.69 1.64 – 1.80	.085
Caton-Deschamps Ratio	1.13 + 0.02	1.29 + 0.03	1.09 – 1.17 1.22 – 1.35	< .001
Morphology Ratio	0.76 + 0.01	0.79 + 0.01	0.74 – 0.78 0.76 – 0.83	.029
Patellofemoral Contacting Surface Ratio	0.45 + 0.05	0.37 + 0.03	0.36 – 0.54 0.30 – 0.43	.240



LITERATURE

[1] BOLLIER M, JP FULKERSON. The role of trochlear dysplasia in patellofemoral instability. *The Journal of the American Academy of Orthopaedic Surgeons, 2011. 19(1): p. 8-16.*

[2] FITHIAN DC *et al.* Epidemiology and natural history of acute patellar dislocation. *Am J Sports Med, 2004. 32(5): p. 1114-21.*

[3] ALBEE RH. The bone graft wedge in the treatment of habitual dislocation of the patella. *Medical Record, 1915. 88: p. 257-259.*

[4] MERCHANT AC *et al.* Roentgenographic analysis of patellofemoral congruence. *J Bone Joint Surg [Am], 1974. 56(7): p. 1391-6.*

[5] DEJOUR H *et al.* Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc, 1994. 2(1): p. 19-26.*

[6] COLVIN AC, RV WEST. Patellar instability. *J Bone Joint Surg Am, 2008. 90(12): p. 2751-62.*

[7] BIEDERT RM, C GRUHL. Axial computed tomography of the patellofemoral joint with and without quadriceps contraction. *Arch Orthop Trauma Surg, 1997. 116(1-2): p. 77-82.*

[8] DIEDERICHS G, AS ISSEVER, S SCHEFFLER, MR imaging of patellar instability: injury patterns and assessment of risk factors. *Radiographics: a review publication of the Radiological Society of North America, Inc, 2010. 30(4): p. 961-81.*

[9] ESCALA JS *et al.* Objective patellar instability: MR-based quantitative assessment of potentially associated anatomical features. *Knee Surg Sports Traumatol Arthrosc, 2006. 14(3): p. 264-72.*

[10] HINTON RY, KM SHARMA. Acute and recurrent patellar instability in the young athlete. *Orthop Clin North Am, 2003. 34(3): p. 385-96.*

[11] ARENDT E. Anatomy and malalignment of the patellofemoral joint: its relation to patellofemoral arthrosis. *Clinical orthopaedics and related research, 2005. (436) (436): p. 71-75.*

[12] FUCENTESE SF *et al.* CT changes after trochleoplasty for symptomatic trochlear dysplasia. Knee surgery, sports traumatology, arthroscopy: *official journal of the ESSKA, 2007. 15(2): p. 168-74.*

[13] HAIM A *et al.* Patellofemoral pain syndrome: validity of clinical and radiological features. *Clin Orthop Relat Res, 2006. 451: p. 223-8.*

[14] LAPRADE J, E CULHAM. Radiographic measures in subjects who are asymptomatic and subjects with patello-femoral pain syndrome. *Clin Orthop, 2003(414): p. 172-82.*

[15] SHIH YF, AM BULL, AA AMIS. The cartilaginous and osseous geometry of the femoral trochlear groove. *Knee surgery, sports traumatology, arthroscopy: official journal of the ESSKA, 2004. 12(4): p. 300-6.*

[16] SALZMANN GM *et al.* Comparison of native axial radiographs with axial MR imaging for determination of the trochlear morphology in patients with trochlear dysplasia. *Archives of orthopaedic and trauma surgery, 2010. 130(3): p. 335-40.*

[17] VAN HUYSSTEEN AL *et al.* Cartilage-bone mismatch in the dysplastic trochlea. An MRI study. *J Bone Joint Surg Br, 2006. 88(5): p. 688-91.*

[18] ALI SA, R HELMER, MR TERK. Analysis of the patellofemoral region on MRI: association of abnormal trochlear morphology with severe cartilage defects. *AJR. American journal of roentgenology, 2010. 194(3): p. 721-7.*

[19] JONES RB *et al.* CT determination of tibial tubercle lateralization in patients presenting with anterior knee pain. *Skeletal Radiol, 1995. 24(7): p. 505-9.*

[20] MILLER TT, RB STARON, F FELDMAN. Patellar height on sagittal MR imaging of the knee. *AJR. American journal of roentgenology, 1996. 167(2): p. 339-41.*

[21] PFIRRMANN CW *et al.* Femoral trochlear dysplasia: MR findings. *Radiology, 2000. 216(3): p. 858-64.*

[22] SCHOETTLE PB *et al.* The tibial tuberosity-trochlear groove distance; a comparative study between CT and MRI scanning. *Knee, 2006. 13(1): p. 26-31.*

[23] SHELLOCK FG *et al.* Patellar tracking abnormalities: clinical experience with kinematic MR imaging in 130 patients. *Radiology, 1989. 172(3): p. 799-804.*

[24] CHARLES M *et al.* MRI Based Topographical Differences between Normal and Recurrent Patellofemoral Instability Patients, in *American Academy of Orthopaedic Surgeons 2012: San Francisco.*

[25] WARD SR, MR TERK, CM POWERS. Patella alta: association with patellofemoral alignment and changes in contact area during weight-bearing. *J Bone Joint Surg Am, 2007. 89(8): p. 1749-55.*

[26] ROGERS BA *et al.* Interobserver variation in the measurement of patellar height after total knee arthroplasty. The Journal of bone and joint surgery. *British volume, 2006. 88(4): p. 484-8.*

[27] GRELSAMER RP, D DEJOUR, J GOULD. The pathophysiology of patellofemoral arthritis. *The Orthopedic clinics of North America, 2008. 39(3): p. 269-74, v.*

[28] ARENDT EA. Dimorphism and patellofemoral disorders. *The Orthopedic clinics of North America, 2006. 37(4): p. 593-9.*

[29] SCHUTZER SF, GR RAMSBY, JP FULKERSON. Computed tomographic classification of patellofemoral pain patients. *Orthop Clin North Am, 1986. 17(2): p. 235-48.*

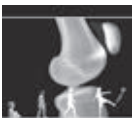
[30] INSALL J, E SALVATI. Patella position in the normal knee joint. *Radiology, 1971. 101(1): p. 101-4.*

[31] CARRILLON Y *et al.* Patellar instability: assessment on MR images by measuring the lateral trochlear inclination-initial experience. *Radiology, 2000. 216(2): p. 582-5.*

[32] KESER S *et al.* Is there a relationship between anterior knee pain and femoral trochlear dysplasia? Assessment of lateral trochlear inclination by magnetic resonance imaging. *Knee surgery, sports traumatology, arthroscopy: official journal of the ESSKA, 2008. 16(10): p. 911-15.*

[33] WONG YM, GY NG. The relationships between the geometrical features of the patellofemoral joint and patellar mobility in able-bodied subjects. *Am J Phys Med Rehabil, 2008. 87(2): p. 134-8.*

[34] BIEDERT RM, M BACHMANN. Anterior-posterior trochlear measurements of normal and dysplastic trochlea by axial magnetic resonance imaging. *Knee Surg Sports Traumatol Arthrosc, 2009.*



LA PATHOLOGIE FEMORO-PATELLAIRE

6èmes JOURNEES LYONNAISES DE CHIRURGIE DU GENOU

LYON 1987

organisées par

H. DEJOUR
G.WALCH

TERMINOLOGIE - CLASSIFICATION DES AFFECTIONS FEMORO-PATELLAIRES

H. DEJOUR

La terminologie des différentes affections rotuliennes est confuse et souvent trompeuse. Parfois c'est l'état du cartilage qui résume la pathologie : chondromalacie, chondrite et, plus moderne, chondropathie. L'état du cartilage est évidemment une donnée essentielle mais elle est insuffisante. Ces termes, plus ou moins consciemment, veulent exprimer essentiellement un état douloureux. Or nous constatons tous les jours des sujets qui souffrent de leur rotule et qui n'ont aucune lésion cartilagineuse, au moins macroscopique, et fait encore plus important, des sujets qui présentent des lésions cartilagineuses évoluées sans aucune symptômatologie rotulienne. En outre, dans l'optique thérapeutique qui est la nôtre, nous ne pouvons nous contenter de cette constatation sauf si nous pensons que les lésions chondrales sont primitives.

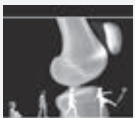
Les vraies questions sont : pourquoi y-a-t-il une lésion cartilagineuse ? pourquoi y-a-t-il une souffrance rotulienne ?

1/ Place des lésions cartilagineuses

La réduction de la pathologie à ce terme est donc dangereuse car elle conduit implicitement à "traiter" uniquement la lésion cartilagineuse. Traitement médical, traitement chirurgical (chondrectomie, Pridie) qui sont pour nous aujourd'hui, et considéré isolemment, des utopies ou au mieux des palliatifs.

Il existe une pathologie où l'on pourrait, avec juste raison, évoquer en premier la pathologie du cartilage, ce sont les troubles rotuliens douloureux survenant après un choc direct. L'étiologie et les symptômes cadrent bien avec l'existence d'une lésion chondrale de type tassement. Or l'exploration arthroscopique de la rotule est souvent négative montrant que les lésions ne sont pas visibles macroscopiquement. Le terme consacré de chondrite post-traumatique n'est peut-être pas très exact, il a au moins l'intérêt d'écarter l'idée de fracture, de fissure du cartilage incitant ainsi à la prudence thérapeutique, dans ces cas où l'aspect médico-légal est important.

La notion de chondropathie doit rester, dans notre raisonnement, hiérarchiquement secondaire. La première question ne doit pas être y-a-t-il ou non une lésion cartilagineuse ? mais bien, s'agit-il d'une désaxation du système extenseur, d'une surcharge liée au morphotype, au myotype, à l'activité physique ? ce n'est que secondairement que l'on pourra poser la question. Toute pathologie rotulienne peut s'accompagner ou non de lésions cartilagineuses. La classification des lésions cartilagineuses est simple. Elle est basée sur le type d'atteinte du cartilage et sur sa localisation. Nous parlons de chondromalacie fermée devant une lésion de simple ramollissement voire avec des fibrillations superficielles, de chondromalacie ouverte devant l'existence de fissures plus ou moins profondes, d'arthrose lorsque l'abrasion du cartilage met à nu l'os sous chondral et qu'il existe des lésions de même type en miroir sur la trochlée. La localisation des lésions cartilagineuses est capitale. Nous n'attachons que peu d'importance aux lésions limitées au versant interne surtout dans sa partie inférieure. Leur banalité leurs enlève presque toute signification pathologique. Les vraies lésions de chondromalacie ouverte siègent toujours au niveau



de la crête médiane débordant plus ou moins sur le versant externe, c'est d'ailleurs là que l'on observe en règle les abrasions caractéristiques de l'arthrose.
Les lésions de la trochlée sont rares mais il faut toujours les rechercher.

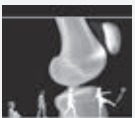
2/ Relation entre lésions chondrales et arthrose

Chondropathie, arthrose, nous devons évoquer le lien qui, pour le clinicien, existe en tre ces deux lésions. Ce lien existe, et d'autant plus qu'on le juge sur une longue période. Mais ce lien n'est pas direct et il ne semble pas obligatoire. Les chondropathies qui conduisent à l'arthrose sont toujours des chondropathies secondaires à un trouble cinématique de l'articulation. L'exemple le plus typique est celui des désaxations externes pathologiques du système extenseur qui fournissent les lésions chondrales les plus importantes et les plus fréquentes chez le jeune. Les lésions chondrales sont ici des fractures traumatiques du cartilage, entraînées par les luxations de rotule. La symptômatologie est essentiellement liée à la désaxation du système extenseur et non à la lésion cartilagineuse. L'arthrose qui va survenir quelques décades plus tard doit beaucoup plus à la persistance de l'hyper-pression externe qu'aux lésions cartilagineuses proprement dites. A l'inverse, on peut dire qu'une lésion cartilagineuse sans aucun trouble cinématique n'a aucune chance d'évoluer vers une arthrose évolutive et invalidante. Ceci est particulièrement net dans les fractures de rotule ou, en l'absence de cal vicieux, de désaxation ou d'anomalie de hauteur, 40 ans après la fracture la radiographie permet bien de trouver des ostéophytes mais jamais de pincement véritable même si le patient à gardé, ce qui est rare, un syndrôme rotulien douloureux.

3/ Instabilités subjective et objective

Une autre source de confusion est le glissement du raisonnement du fait de termes qui peuvent tout aussi bien s'appliquer à des symptômes qu'à une véritable pathologie. Le terme d'instabilité est ici particulièrement dangereux. L'instabilité est avant tout un symptôme subjectif, c'est la sensation que le genou n'est pas solide, qu'il lâche ou qu'il peut lâcher. Les causes de ce symptôme sont évidemment très diverses : quadricipitale (paralysie, inhibition, atrophie), ligamentaire, rotulienne (luxation ou subluxation de rotule). Mais on peut également utiliser ce terme pour exprimer par exemple un déplacement de la rotule, ce n'est plus le genou qui subjectivement est instable mais la rotule qui, objectivement, se désaxe, n'est plus stable sur la trochlée fémorale. Depuis une dizaine d'années ce terme est de plus en plus employé dans ce sens vraisemblablement sous l'influence anglo-saxone, surtout pour l'épaule et la rotule. Il est pratique parce qu'il est imprécis et qu'il ne préjuge pas de l'importance et de l'origine de la déstabilisation. Il permet ainsi de regrouper dans un même cadre pathologique des faits aussi différents cliniquement que les luxations (terme précis) et les subluxations (terme imprécis quant à l'importance ou à la réalité du mouvement). Ce terme employé seul n'est donc pas satisfaisant car toutes les instabilités - (symptôme clinique, subjectif et d'origine rotulienne), ne sont pas des instabilités objectives de la rotule (défaut de stabilité de la rotule).

Le terme de subluxation est lui aussi ambigü puisque dans la pratique il traduit une impression clinique par un terme anatomique préjugeant un déplacement qui n'existe pas toujours. D'autant plus que le terme



subluxation récidivante, employé largement par A. TRILLAT, s'est progressivement transformé en subluxation de rotule, ce qui accentue le caractère anatomique qui dans ce cas précis n'est souvent pas la réalité, soit par excès (la rotule apparaît subluxée en extension elle n'est pas instable), soit par défaut (la rotule n'est pas subluxée mais elle est cependant instable).

4/ La souffrance rotulienne

Mais l'aspect le plus difficile à cerner de la pathologie rotulienne reste ce qui à trait aux symptômes douloureux. Nous manquons ici, cruellement, de fil conducteur. D'où part le reflexe douloureux ? où chemine-t-il ? nous n'avons pratiquement que des connaissances négatives, nous savons que le cartilage est dépourvu de récepteur, nous savons que les tentatives de "dénervation" de la rotule n'ont guère eu plus de succès que les dénervations de la hanche préconisées il y a 50 ans, en fait ce n'est pas la rotule qui souffre mais l'articulation fémoro-patellaire et ici comme ailleurs nous savons qu'une articulation souffre soit parce qu'elle est le siège d'une hyper-pression, soit parce qu'elle est soumise à des sollicitations excessives de traction, soit enfin qu'il existe un trouble de la cinématique qui perturbe le jeu articulaire normal. Quant aux troubles vasculaires, parfois évoqués, ils semblent bien hypothétiques dans la majeure partie des cas.

I - CLASSIFICATION

La classification est une étape essentielle car elle définie les concepts qui seront la base de notre diagnostic et de notre action thérapeutique. Classer est toujours un peu arbitraire et réducteur. La classification doit être à la fois étiologique, clinique, anatomique et physiopathologique, elle traduit obligatoirement l'importance relative que l'on donne à chacun de ces termes et aussi la précision de nos connaissances. Les tableaux cliniques sont, pour nous thérapeutes, l'idéal. Les syndrômes cliniques aux causes mal connues nous permettent de créer un cadre dont la valeur doit être au contraire l'imprécision (c'est-à-dire l'absence d'apriori) obligeant dans ces cas à la recherche des causes probables.

1) Les lésions traumatiques

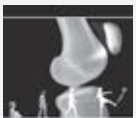
a) Les fractures de la rotule : nous n'aborderons pas ce châpitre qui est simple, bien connu, seules seront évoquées certaines séquelles des fractures.

b) La rupture du tendon rotulien est un accident assez spécifique du saut en hauteur, il pose des problèmes de diagnostic et de traitement assez intéressants. L'arrachement de la tubérosité tibiale est une forme particulière de la rupture du tendon rotulien chez l'enfant et l'adolescent.

c) La rupture du tendon quadricipital est un accident d'origine dégénérative observé chez l'adulte âgé.

d) Le décollement ostéopériosté du quadriceps est une lésion très spécifique de l'adolescent particulièrement intéressant car il montre combien un traumatisme rotulien banal, traité par plâtre peut entraîner rapidement une raideur du quadriceps et une ostéoporose rotulienne.

e) La luxation traumatique est un indiscutable tableau clinique, avec en particulier chez l'adolescent, des lésions anatomiques sévères : rupture de l'aileron interne, fracture ostéochondrale,



hémarthrose. Par contre il n'est pas rare d'observer une luxation, pratiquement sans traumatisme ni sans lésion anatomique, il s'agit là plutôt d'une luxation première que d'une véritable luxation traumatique et même lorsque les lésions traumatiques sont très marquées, il est très rare, de ne pas constater dans ces cas, des anomalies fémoro-patellaires pré-existantes. Nous retrouverons plus loin cette pathologie.

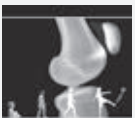
II - LA DYSPLASIE LUXANTE DE LA ROTULE

C'est le cadre pathologique le mieux défini dans ces caractéristiques anatomiques et physiopathologiques. Nous montrerons qu'un examen radiologique et tomo- densitométrique précis, permet de définir une véritable dysplasie fémoro-patellaire qui permet de regrouper des entités cliniques très variées et dont l'absence permet d'éliminer des tableaux cliniques voisins mais fort éloignés dans leur physiopathologie. Comme toute dysplasie, la dysplasie luxante de la rotule revêt des dégrés de gravités variables, les dysplasies sévères se révèlant chez l'enfant, les dysplasies moyennes chez l'adolescent et les formes discrètes pouvant rester asymptomatiques ou presque très longtemps. Parfois ce n'est que la constatation d'une arthrose chez l'adulte âgé qui la révèle. En effet, comme toutes dysplasies, la dysplasie fémoro-patellaire qui traduit un trouble cinématique de la rotule conduit irrémédiablement à l'arthrose. La caractére bilatéral des anomalies est également caractéristique, la dysplasie anatomique est presque toujours symétrique à peu de chose près, par contre la traduction clinique est souvent asymétrique. Plus la dysplasie est grave et plus il y a de chance pour que les troubles soient bilatéraux et souvent même équivalents, par contre il est fréquent dans les formes moyennes et discrètes d'observer des formes asymétriques cliniquement et des formes asymptomatiques ou presque. Le caractère génétique est peu marqué mais il existe et il n'est pas rare de voir des instabilités rotuliennes objectives chez la mère et la fille.

Cette dysplasie luxante définie anatomiquement, exprime sur le plan physio- pathologique la prédominance des forces de subluxation externe sur les forces de stabilisation interne. Il s'agit donc d'une désaxation externe du système extenseur soit anatomique (luxation), soit dynamique (hyperpression externe).

1/ Luxations permanentes et habituelles

C'est la forme majeure de la désaxation du système extenseur. Notons qu'il est incorrect de parler de luxation en extension. Dans cette position, et surtout si le quadriceps est contracté, la rotule est au-dessus de la trochlée. Il n'y a donc pas de véritable articulation fémoro-patellaire, que la rotule soit en position très externe, qu'elle soit basculée est certes très important, mais ce que nous devons apprécier sur le plan clinique, c'est ce que fait la rotule lorsque le genou se met en flexion. La luxation est par définition, le déboitement de la rotule qui enjambe le versant externe de la trochlée voire du condyle externe lors de la flexion.
Les luxations permanentes et habituelles sont la forme majeure de la désaxation du système extenseur. La rotule ne s'engage même pas dans la trochlée ou bascule en dehors à chaque flexion. Ces formes se constituent toujours chez l'enfant, parfois très jeune, voire l'adolescent.



2/ Les instabilités rotuliennes objectives

Nous regroupons sous ce terme plusieurs formes cliniques

a) Les luxations récidivantes

C'est un tableau très précis. Les luxations sont indiscutables, elles sont plus ou moins fréquentes suivant l'activité physique, fréquemment il existe une course anormale de la rotule qui, en position très externe en extension, se recentre dans les premiers degrés de flexion. L'examen trouve une rotule luxable avec un signe de Smillie très positif.

b) Les luxations traumatique suivies de troubles

Ce tableau clinique nécessite déjà une analyse plus poussée car le phénomène de luxation n'est plus au premier plan. Il y a eu une luxation mais celle-ci n'a pas toujours été reconnue car on a pu parler d'entorse interne, de contusion du genou.

Le maître symptôme reste l'instabilité, mais cette impression de genou qui lâche, qui n'est pas sûr, cette appréhension n'est pas immédiatement rapportée à un déboîtement, ni à une subluxation rotulienne. Par ailleurs, il existe souvent d'autres signes qui sont liés aux lésions chondrales secondaires avec douleurs à la station assise prolongée, aux escaliers, voire hydarthrose récidivante, plus rarement blocages rotuliens.

Le caractère objectif de cette instabilité rotulienne est donné par :

- les antécédents précis et indiscutables de luxation reconnue.
- Les stigmates radiologiques pathognomoniques des luxations : fracture ostéochondrale du versant externe de la rotule, plus rarement du condyle externe.
- L'existence clinique d'une rotule luxable (signe de Smillie très positif).

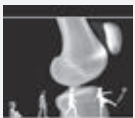
La mise en évidence de la dysplasie radiologique vient confirmer le tableau clinique.

3/ Les instabilités rotuliennes potentielles

Elles sont un groupe particulièrement important à étudier. Ici il n'y a pas eu de luxation, la rotule n'est pas luxable (signe de Smillie négatif parfois douteux) les symptômes allégués mettent au premier plan la douleur, les blocages, l'instabilité subjective est discrète, elle n'est souvent pas même évoquée d'emblée. Ici ce n'est ni le tableau clinique, ni l'examen clinique qui permettent de rattacher ces cas à la dysplasie luxante de la rotule mais uniquement la mise en évidence de la dysplasie fémoro-patellaire.

4/ L'arthrose fémoro-patellaire externe

L'arthrose fémoro-patellaire avec subluxation externe que l'on découvre entre 50 et 60 ans à elle aussi les stigmates radiologiques qui caractérisent la dysplasie. Si dans 45% ont retrouve des antécédents d'instabilité rotulienne vraie, tous les autres n'ont eu aucun symptôme rotulien jusqu'à l'apparition de l'arthrose. Cette évolution exprime bien les formes d'hyper-pression externe que l'on retrouve dans les instabilités rotuliennes potentielles. Lorsque l'arthrose survient après des antécédents d'instabilités rotuliennes de l'adolescence, la dysplasie est toujours importante. Lorsque l'arthrose apparaît comme primitive vers la cinquantaine, la dysplasie est discrète de même type que celle observée dans les instabilités rotuliennes potentielles.



III - LES SYNDROMES ROTULIENS DOULOUREUX

Nous regroupons ici tous les cas où la symptômatologie ne relève pas d'une dysplasie luxante de la rotule. Il s'agit donc d'une entité clinique caractérisée d'abord par ses symptômes où la douleur domine toujours, même si l'on peut retrouver des blocages voire une instabilité subjective discrète. Ces syndrômes rotuliens douloureux relèvent de causes très diverses qu'il faut rechercher et analyser. Nous distinguerons d'abord : les syndrômes rotuliens secondaires et les syndrômes rotuliens primitifs.

1/ Les syndrômes rotuliens secondaires

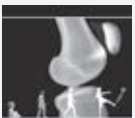
a) La chondrite post-traumatique : c'est le plus typique dans son histoire clinique et le plus mystérieux dans sa cause réelle. Il n'est pas rare de trouver, surtout dans les cas rebelles des éléments que nous analyserons dans les syndrômes rotuliens primitifs. Le traumatisme peut donc jouer un rôle de révélateur à des facteurs pré-existants.

b) Le syndrôme rotulien post-fracturaire : Il n'est guère étonnant d'observer un syndrôme rotulien après une fracture de la rotule, mais il faut bien distinguer les troubles liés à un cal vicieux, ou hypertrophique et ceux dus à une anomalie de hauteur de la rotule (rotule basse) et ceux, plus rares, où apparemment on ne peut invoquer à leur origine que la lésion chondrale.

c) Les syndrômes rotuliens douloureux post-opératoires que l'on peut voir dans les suites de n'importe quelle intervention, fût-elle particulièrement bénigne (arthroscopie) parfois même après une simple immobilisation plâtrée. Ils ont comme caractéristique de survenir toujours après des suites opératoires difficiles (douleurs, raideur) avec une rééducation pénible, souvent d'ailleurs mal adaptée.

Nous distinguerons deux types de syndrômes rotuliens post-opératoires :

- *La rotule basse* : c'est la complication majeure qu'il faut toujours soupçonner dans les suites douloureuses. D'installation immédiate elle peut régresser si elle est reconnue et traitée dans les semaines qui suivent l'intervention, sinon il s'agit d'une séquelle indélébile nécessitant des gestes chirurgicaux importants lorsqu'elle est très génante.
- *Les chondropathies post-opératoires* : elles se voient surtout après des interventions importantes où des immobilisations plâtrées longues (6 à 8 semaines) et elles sont pratiquement toujours liées à une raideur articulaire. Arthrotomie, hémarthrose post-opératoire, plâtre sont certainement des facteurs qui fragilisent le cartilage rotulien. Si celui-ci subit une agression supplémentaire sous la forme d'une mobilisation sous anesthésie, d'une rééducation excessive et brutale, il va se produire des lésions chondrales généralisées et irréversibles. Les arthrolyses pratiquées pour raideurs post-opératoires montrent très souvent des lésions particulièrement graves avec même parfois une symphyse, non seulement du cul de sac quadricipital et des joues condyliennes, mais de l'articulation fémoro-patellaire elle-même, le cartilage s'étant transformé en fibro-cartilage soudant la rotule à la trochlée.
- *La rupture du ligament croisé postérieur* : l'existence d'un important tiroir postérieur entraîne un abaissement relatif de la rotule et une surcharge fémoro-patellaire, ce syndrôme douloureux est souvent le seul symptôme des laxités chroniques postérieures. Lorsqu'une intervention chirurgicale toujours complexe, une immobilisation plâtrée longue n'ont pas réussi à supprimer le tiroir postérieur, la



chondropathie post-opératoire, toujours possible, va majorer les troubles induits par la perturbation de la cinématique.

- *La plica médio-patellaire* : bien que souvent accusé à tort, la plica peut entraîner une gêne et une chondromalacie du versant interne de la rotule. Il est intéressant de noter que l'ablation de la plica fait disparaître le syndrôme rotulien malgré la persistance des lésions chondrales.

2/ Les syndrômes rotuliens primitifs

Ils sont bien évidemment les plus nombreux, les plus intéressants et les plus difficiles à démembrer.

a) Instabilité rotulienne potentielle : ces cas, très importants à étudier et à isoler, ont été placés dans les dysplasies luxantes de la rotule car il existe une anomalie morphologique caractéristique de la trochlée, mais le tableau clinique est en tous points un syndrôme rotulien douloureux, ce cadre mérite donc d'être rappelé ici ne serait-ce que pour l'opposer aux autres syndrômes rotuliens douloureux.

b) Les troubles de torsion fémoro-tibiale : l'aspect le plus typique est représenté par le strabisme convergent de la rotule traduisant une antéversion fémorale et/ou une torsion tibiale externe exagérée. L'angle du pas est très variable traduisant l'importance de la détorsion sous malléolaire : externe lorsqu'elle est faible, nulle lorsqu'elle est importante.

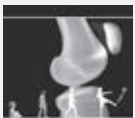
c) L'hyper-pression interne de la rotule : y-a-t-il une dysplasie inverse de la dysplasie luxante et responsable d'un conflit fémoro-patellaire interne ? Certains faits vont dans ce sens mais ils sont encore disparates. La constatation d'arthrose fémoro-tibiale interne, voire d'arthrose centrée isolée qui devrait amener la preuve de cette dysplasie est rare, bien que non exceptionnelle. Nous verrons cependant que l'on peut retenir à l'origine du syndrôme douloureux deux anomalies : la trochlée creuse et la rotule basse constitutionnelle.

d) L'hyperpression externe

L'hyperpression externe telle que l'a décrite FICAT trouve son cadre le plus typique dans les instabilités rotuliennes potentielles. Y-a-t-il des hyperpressions externes sans dysplasie de la trochlée ? Nous ne pouvons à l'heure actuelle trancher ce problème important. Il n'est pas exclu que certaines trochlées de type B avec un aspect de subluxation de la rotule en flexion s'accompagnent d'une hyperpression externe. Cependant, l'évolution vers l'arthrose semble rare dans de tels cas et il n'est pas certain que ce syndrôme existe véritablement.

e) La raideur des ischio-jambiers : c'est l'un des cadres les plus précis et les plus importants du syndrôme rotulien douloureux. Cette raideur peut-être constitutionnelle et s'observer chez l'enfant, elle peut-être acquise, liée le plus souvent à un déséquilibre des activités physiques par la pratique exclusive de sports qui favorisent le travail du genou en flexion ou d'un entraînement physique incorrect. Les sports les plus concernés sont la montagne, le cyclisme et de plus en plus la course sur longue distance et sur terrain dur.

Le tableau clinique le plus caractérisitique est celui de la maladie d'Osgood-Schlatter où les troubles observés au niveau de la tubérosité tibiale antérieure doivent être considérés comme l'analogue des troubles rotuliens proprement dits. Ici on retrouve les deux aspects que nous avons retenus dans la raideur des ischio-jambiers : une raideur constitutionnelle souvent très importante même chez le jeune garçon de 13 ans (profil type de cette pathologie) et une activité sportive intense et non contrôlée, le plus souvent "football sauvage".

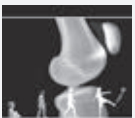


Nous retiendrons un deuxième tableau clinique que nous appelerons le "genou forcé". Il s'agit en général d'adultes ayant dépassé la trentaine, présentant une raideur constitutionnelle non reconnue et non traitée, qui voient apparaître brutalement un syndrôme rotulien après un effort sportif dépassant leur performance habituelle (course en montagne, marathon). Le syndrôme douloureux devient alors particulièrement rebelle et résistant aux thérapeutiques comme si des fractures de fatigue du cartilage apparaissaient créant des lésions difficilement réversibles.

f) L'hyperlaxité constitutionnelle : il s'agit d'un cadre purement clinique, bien difficile à définir d'une façon précise mais que l'on invoque souvent chez des adolescentes qui présentent un syndrôme rotulien douloureux. Au niveau du genou, l'hyperlaxité peut être définie par la constatation d'un certain degré de laxité antéro- postérieure (Lachman arrêt dur retardé, tiroir antéro-postérieur en flexion) et surtout d'un recurvatum excessif (supérieur à 10°) qui s'accompagne toujours d'une hyper- rotation externe, genou fléchi à 90°. Nous avons cependant quelques réticences à inclure ce tableau clinique dans les syndrômes rotuliens. D'abord parce que l'origine de la douleur n'est pas claire, le terme de pseudo patella alta est un terme facile qui ne veut rien dire, l'étude de la marche et de la position au repos en station bipodale et içi capitale. On ne peut retenir l'hyperlaxité et le recurvatum comme étant à l'origine des douleurs que dans les cas (rares) où le genou bascule à la marche en recurvatum et où la position de repos naturel est le blocage en recurvatum. Mais nous pensons que dans ces cas les douleurs sont beaucoup plus fémoro-tibiales que patellaires, fémoro-tibiales externes du fait de la traction sur le point d'angle postéro-externe, fémoro-tibiales internes du fait de la bascule du genou en varus.

g) La tendinite rotulienne ou genou du sauteur : le Jumping knee est une forme très particulière du syndrôme rotulien où la lésion anatomique essentielle ne semble pas résider au niveau du cartilage rotulien mais au sein même du tendon. La palpation trouve une douleur sur le tendon rotulien en dessous de la pointe de la rotule, l'échotomographie peut montrer des lésions intéressantes en particulier pseudo-kystes traduisant la dégénérescence des fibres.

h) La chondromalacie primitive : ce cadre est pour nous très réduit et il traduit vraisemblablement souvent notre défaut d'analyse. Il arrive cependant d'observer des cas, toujours chez des sujets jeunes, où malgré l'absence de tout antécédent et de facteurs mécaniques prédisposants l'on constate des chondromalacies ouvertes, en général sévères, atteignant non seulement la rotule mais la trochlée, s'accompagnant d'une réaction inflammatoire synoviale avec hydarthrose chronique. Il n'est par rare d'observer dans ces cas une atteinte cartilagineuse des condyles posant le problème d'une arthropathie d'origine inconnue.



LA RADIOLOGIE DANS LA PATHOLOGIE FEMORO-PATELLAIRE

G. WALCH - H. DEJOUR

Le bilan systématique des affections rotuliennes comprend un cliché de face du genou un cliché de profil STRICT à 30° de flexion sans appui et une vue axiale des rotules à 30° de flexion en rotation neutre et en rotation externe des pieds. Cet examen est FONDAMENTAL et à lui seul il permet une approche diagnostic très précise.

I - LA RADIOGRAPHIE SIMPLE DU GENOU DE FACE

La radiographie simple du genou de face en appui monopodal n'est pas d'un grand intérêt si ce n'est d'éliminer une pathologie associée.

La pratique de grands clichés d'axe de face est nécessaire pour quantifier avec précision le valgus et le varus chaque fois que le morphotype clinique laissera apparaître une anomalie majeure et surtout chaque fois qu'une ostéotomie sera envisagée. En pratique courante, nous ne les demandons pas systématiquement.

II - PROFIL STRICT DU GENOU A 30° DE FLEXION

Toutes les mesures nécessitent un profil strict, c'est-à-dire que les deux condyles doivent être confondus en arrière et au niveau de l'interligne. Il doit être effectué si nécessaire sous contrôle scopique.

1/ La hauteur rotulienne (figure 0)

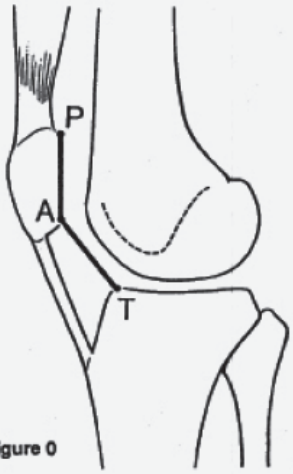


Figure 0

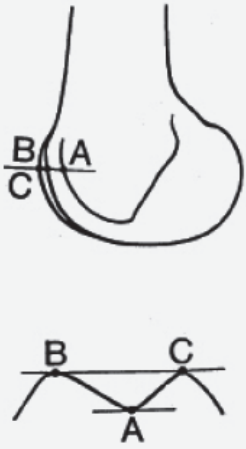
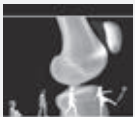


Figure 1



Elle est mesurée par l'indice de hauteur rotulienne (I.H.R.) (CATON-DESCHAMPS). C'est le rapport entre :
* A.T. : distance entre le bord inférieur de la surface articulaire de la rotule et le rebord antéro-supérieur du tibia.
* et A.P. : qui représente la longueur de la surface articulaire de la rotule.
Cette méthode est fiable quelque soit le degrés de flexion du genou entre 30 et 60°. Les points de repère sont fixes, facilement identifiables. Elle prend en compte la longueur de la surface articulaire, ce qui élimine les erreurs dues aux modifications de la pointe de la rotule. Lorsque la hauteur rotulienne est supérieure ou égale à 1,2 on parle de "rotule haute", lorsqu'elle est inférieure à 0,8 on parle de "rotule basse".

2/ La trochlée

Depuis BRATTSTROM la dysplasie de la trochlée a toujours été reconnue comme un facteur d'instabilité rotulienne. Elle est classiquement étudiée sur la vue axiale à 30°, mais récemment, MALDAGUE a souligné l'intérêt du cliché de profil dans sont analyse. Il détermine la profondeur de la trochlée mesurée à 1 cm de son pôle supérieur et décrit des insuffisances de creusement qui peuvent être globale ou focale. Nous avons entrepris une étude analytique de la trochlée fémorale sur un cliché de profil strict du genou sur plus de 1800 genoux pour savoir si l'on pouvait caractériser avec plus de précision la dysplasie trochléenne.
Sur un cliché de profil strict trois courbes sont individualisables à la partie antérieure de la trochlée (figure1).

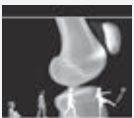
Les deux lignes les plus antérieures correspondent aux contours des condyles qui peuvent être confondus ou dédoublés, la courbe immediatement en arrière correspond au ***fond de la trochlée fémorale*** qui s'articule avec la rotule lors des mouvements de flexion-extension. (Elle prolonge vers l'avant l'échancrure intercondylienne bien décrite par BLUMENSTATT). Elle est concave vers l'arrière et à la partie supérieure elle a deux modes de terminaison possibles (figure 2) :

Figure 2A

Figure 2B

Figure 2C

* soit elle disparaît insensiblement sans jamais atteindre les lignes condyliennes en se recourbant vers l'arrière ou en devenant verticale (figure 2A).



* soit elle croise les lignes condyliennes antérieures ; il peut s'agir d'un croisement avec un seul condyle (figure 2B) ou avec les deux condyles (figure 2C).

Pour réaliser une bonne analyse de la trochlée nous traçons une ligne droite, tangentielle à la corticale antérieure du fémur sur les 10 derniers centimètres (figure3).

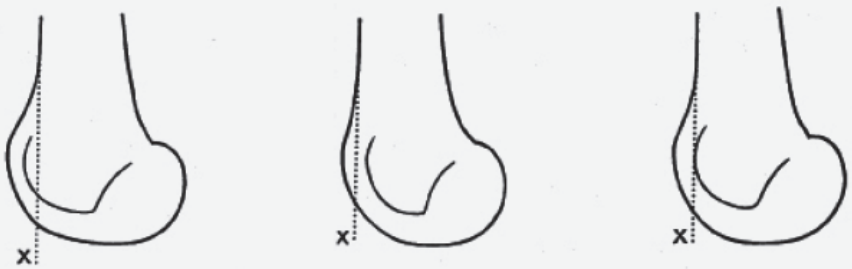


Figure 3

Par rapport à cette ligne droite on se rend compte que le fond de la trochlée peut être en avant, en arrière ou à l'aplomb de la corticale antérieure. On détermine ainsi la "Saillie" de la trochlée qui peut être positive, négative ou nulle. Si l'on veut analyser le rôle de cette Saillie en pathologie il faut la quantifier (figure 4).

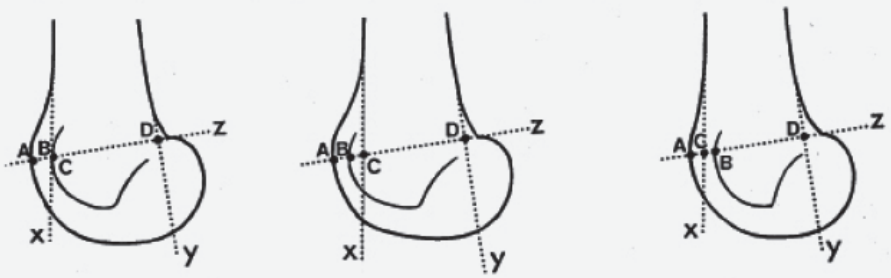


Figure 4

Nous traçons alors deux autres lignes droites. Une parallèle aux 10 derniers centimètres de la corticale postérieure du fémur (ligne Y), une autre joignant en avant le point le plus antérieur de la ligne de fond de trochlée (point B) et en arrière le sommet des condyles fémoraux (ligne Z).

Par définition la Saillie est mesurée au point la plus antérieur de la ligne de fond de trochlée. Elle est quantifiée soit en millimètres, soit en rapport pour tenir compte des données morphométriques de l'individu. Selon que la mesure s'effectue en avant ou en arrière de la corticale antérieure elle sera affectée du signe + ou -.

Sur le schéma 4A la Saillie est nulle, sur les schémas 4B et 4C elle est définie par BC en millimètres ou BC x 100 / AB. Sur le schéma 4D elle est définie par -BC ou

-BC x 100 / AD.

Après la Saillie, une deuxième notion résultant de l'analyse du cliché de profil du genou est la profondeur

de la trochlée, dont avait déjà parlé MALDAGUE. Nous repérons le point de plus faible profondeur de la trochlée qui correspond au point le plus antérieur de la ligne de fond de trochlée précedemment défini. Sur la même droite, la profondeur correspond à AB. Elle peut être quantifiée en mm ou en rapport pour tenir compte des données morphométriques de l'individu. Elle est alors égale à AB x 100 / AD (figure 4).

Ces notions de "Saillie" et "profondeur" de la trochlée mesurées sur le profil strict du genou revêtent une IMPORTANCE CAPITALE dans la détermination des "Instabilités Rotuliennes". Nous nous sommes en effet rendu compte que dans les instabilités rotuliennes, la profondeur était toujours nulle, c'est-à-dire que la ligne de fond de trochlée croisait toujours les 2 lignes condyliennes en avant. Ce "SIGNE DU CROISEMENT" est donc un signe PATHOGNOMONIQUE de dysplasie trochléenne traduisant à partir d'un point précis, facilement déterminable, une trochlée totalement plate.

La répétition de ces mesures sur certaines radiographies de profil nous a montré que ce croisement pouvait se faire selon des modalités différentes et nous a conduit à déterminer et à décrire des TYPES DE TROCHLEE.

a) La trochlée normale

Par définition il n'y a jamais de croisement de la ligne de fond avec le condyle externe. La morphologie du condyle interne permet de définir deux types de trochlées normales A et B (figure 5).

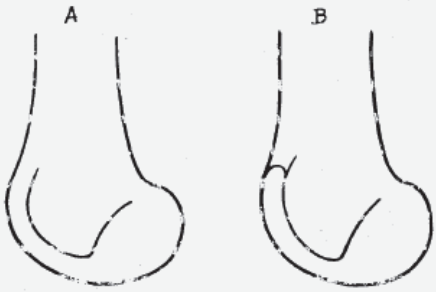


Figure 5

- Dans le type A, les deux condyles sont symétriques, la ligne de fond de trochlée ne croise jamais les condyles.

- Dans le type B, le condyle interne croise de manière isolée la ligne de fond après s'être recourbé en arrière.

b) La trochlée dysplasique

Les types I, II, III sont les trochlées dysplasiques (figure 6) où l'on observe un croisement de la ligne de fond de trochlée avec les deux condyles. La différence entre ces 3 types vient du niveau de croisement, et de la symétrie ou non des deux condyles.

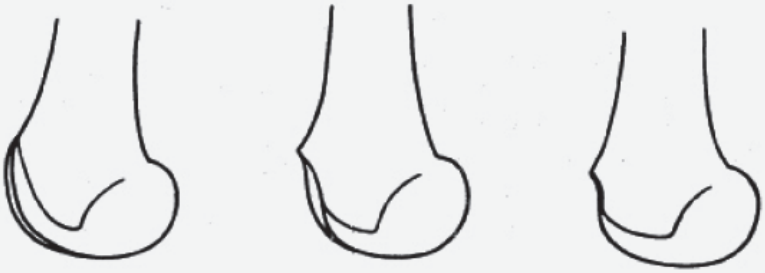
- Dans le type I les deux condyles sont symétriques et la ligne de fond les croise en même temps à la partie haute de la trochlée, c'est la dysplasie la moins marquée.

- Dans le type II, les deux condyles sont asymétriques. Le condyle interne est dysplasique, il croise le fond de la trochlée avant le condyle externe à un niveau qui peut-être variable. Puis la ligne de fond se poursuit en haut et en avant jusqu'à croiser le condyle externe. Le type II est donc caractérisé par un

croisement séparé des deux condyles et une dysplasie plus ou moins importante du condyle interne. Plus le croisement avec le condyle interne se fait bas, plus la dysplasie est importante. Plus le croisement avec le condyle interne se fait haut, plus on se rapproche du type I avec croisement commun condyles externe et interne.

- Dans le type III, les deux condyles sont symétriques, mais le croisement se fait bas traduisant un comblement précoce de la trochlée qui est totalement plate sur une étendue variable, c'est la dysplasie majeure.

Figure 6



Type I Type II Type III

c) Les formes intermédiaires

La classification que nous avons exprimé correspond à 95% des cas. Dans 5% on observe des formes que nous appellons intermédiaires. Fondamentalement il ne s'agit pas de formes dysplasiques car il n'y a pas de croisement entre la ligne de fond de trochlée et le condyle externe. Elles se rapprochent cependant des trochlées dysplasiques et nous avons noté des troubles cliniques qui nous incitent à les isoler malgré leur rareté.

- Le type AI (figure 7) : est un type A qui est très proche du type I. Il n'y a pas de croisement mais à la partie haute, la ligne de fond de trochlée est très proche des condyles traduisant une profondeur très faible.
- Le tye BII (figure 8): la ligne de fond ne croise pas le condyle externe mais le condyle interne est très dysplasique, se terminant plus tôt que dans le type B et surtout cette terminaison se fait sur un mode très effilé avec un angle très aigu. Nous avons observé quelques cas de luxation récidivante de la rotule dans ce type de trochlée.

Il est possible que ce type BII dans un certain nombre de cas soit en fait un type II et que notre distinction traduise des constantes radiologiques différentes.

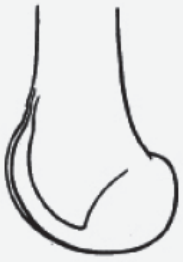
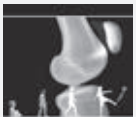


Figure 7 : type A I

Figure 8 : type B II



d) La hauteur de la trochlée

La hauteur de la rotule jouant un rôle important dans l'instabilité rotulienne, il est logique de penser, comme RAGUET, que la hauteur de la trochlée influe également.

La quantification de la hauteur trochléenne n'est cependant pas aussi simple. Nous avons essayé de la mesurer comme RAGUET, c'est-à-dire en prenant comme point de référence le point le plus antérieur des condyles fémoraux. Cette mesure est apparue statistiquement non significative.

Nous avons alors pris comme point de référence le croisement de la ligne de fond de trochlée avec le condyle interne. On peut admettre qu'à partir de ce point, la trochlée n'est plus véritablement creuse puisqu'elle n'est représentée que par le versant externe. Ce point peut être facilement déterminé dans les trochlées dysplasiques et dans les trochlées de type B. Dans le type A, nous avons pris par convention le point le plus antérieur de la ligne de fond (qui sert à mesurer la Saillie et la profondeur) car dans les types B c'est à ce niveau que croise le condyle interne.

L'indice de hauteur de trochlée est défini (figure 9) par le complémentaire d'un angle formé d'une part par la corticale postérieure du fémur, d'autre part par une ligne joignant le sommet des condyles en arrière et en avant le point précédemment défini.

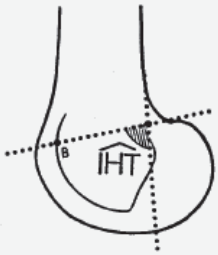


Figure 9

Nous avons choisi l'angle complémentaire pour que le chiffre soit bas dans les trochées dites basses et haut dans les trochlées dites hautes. Cette mesure ne s'est pas révélée très significative dans la distinction des différents types de pathologie. Ce qui tendrait à prouver que la hauteur de la trochlée n'est pas d'une importance primordiale. Sauf cependant dans les trochlées de type III (figure10) où manifestement plus la trochlée est basse, plus la dysplasie est grave. C'est pour cette raison que nous avons gardé cette mesure dans nos études.

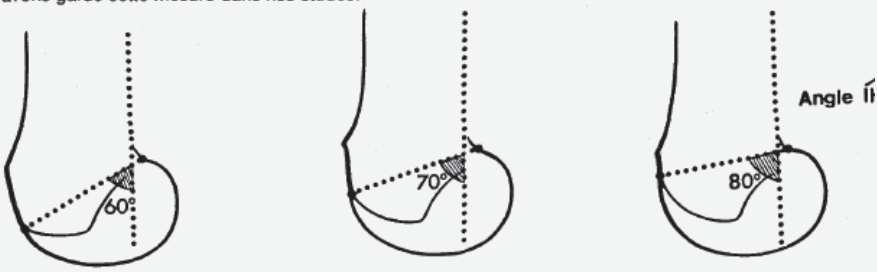


Figure 10 : les différentes valeurs de IHT dans le type III

III - LES VUES AXIALES

De routine, nous demandons 2 clichés à 30° de flexion. Un pied parallèle, l'autre en rotation externe des deux pieds. Les clichés à 60° et 90° ne sont demandés que dans les cas spéciaux. Il faut être certain que le cliché a bien été pris à 30° de flexion et il existe un moyen simple de le vérifier : à cette angulation le sommet du condyle interne a une forme caractéristique pointue (figure11).

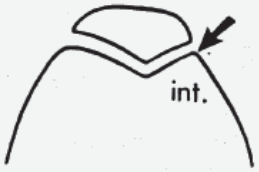


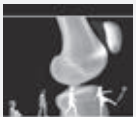
Figure 11

Ce cliché fournit des données qualitatives et quantitatives. La constatation d'une fracture ostéochondrale du versant interne de la rotule ou du condyle externe, l'existence d'ossifications type PELLIGRINI-STIEDA sur l'aileron rotulien interne ont une valeur essentielle car elles permettent d'affirmer à postériori une luxation de rotule.
Les données quantitatives sont représentées par les mesures de l'angle trochléen, de l'angle rotulien, du rapport TE/TI, du rapport VE/VI, de la bascule rotulienne, de la subluxation rotulienne.

***1/ L'angle trochléen** (figure12)*

Figure 12 : calcul de l'angle trochléen

Une ligne passant par le sommet des deux condyles est tracée. Trois points sont repérés : les points les plus antérieurs sur les deux condyles et le point le plus profond de la gorge de la trochlée. Deux lignes rejoignent ces points, elles se croisent au fond de la trochlée en réalisant un angle ouvert en avant : l'angle trochléen.
Nous considérons que cet angle est normal lorsqu'il est > 145° et < 124°.



***2/ L'angle rotulien** (figure13)*

Figure 13 : mesure de l'angle trochléen

Il est formé par le croisement des deux lignes tangentes aux versants rotuliens interne et externe. Souvent malaisé à déterminer, il semble plus logique de tenir compte de la classification de WIBERG et décrire deux types de rotules dysplasiques : le premier type est constitué par une rotule plutôt plate avec un angle rotulien > 140°, le deuxième type a un angle rotulien très fermé < 100° avec un versant extrerne large presque seul articulaire contrastant avec un versant interne court et abrupt. Une rotule "normale" a un angle de 120 à 140°, mais compte tenu de la différence pour effectuer des mesures précises et répétitives, nous accordons à cet angle une valeur très relative.

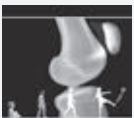
***3/ le rapport trochlée externe / trochlée interne** (figure14)*

Figure 14 : rapport TE/TI

Rapport des mesures des versants articulaires de la trochlée en conservant les mêmes repères que pour la mesure de l'angle trochléen. Cette mesure traduit la dysplasie du versant interne de la trochlée par rapport au versant externe. On peut retenir qu'une trochlée est dysplasique lorsque le rapport est supérieur à 1,7.

4/ La mesure du rapport VI / VE

La mesure du rapport VI / VE des deux versants rotuliens externe et interne rencontre les mêmes objections que l'angle rotulien. Dans la pratique nous ne l'utilisons plus.



5/ La bascule rotulienne (figure15)

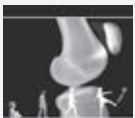
Figure 15 : la bascule rotulienne selon LAURIN

Etudiée par LAURIN qui trace deux droites, une joignant les sommets des deux condyles, l'autre tangente au versant externe de la rotule. Les droites forment un angle qui est positif lorsqu'il est ouvert en dehors. Les deux lignes peuvent être parallèles et l'angle de LAURIN est alors dit nul, ou former un angle ouvert en dedans, l'angle de LAURIN étant dit négatif.
Un angle de LAURIN négatif traduit une bascule externe et est alors synonyme d'instabilité rotulienne. Cette bascule peut être augmentée par la mise en rotation externe des pieds lors de la réalisation du cliché (JUILLARD). La bascule est alors augmentée de manière significative en cas d'instabilité rotulienne.

6/ La subluxation rotulienne (figure16)

Figure 16 : appréciation de la subluxation rotulienne par l'angle de MERCHANT

Elle peut être étudiée par l'angle de congruence de MERCHANT. On trace la bissectrice de l'angle trochléen tel qu'il a été décrit plus haut. Une deuxième ligne est ensuite tracée entre le fond de la trochlée et le point le plus bas de la surface articulaire de la rotule. L'angle mesuré entre ces deux lignes est l'angle de congruence. Si le point rotulien est situé à l'extérieur de la bissectrice, l'angle est positif (+) et il traduit plutôt une subluxation externe. Si le point rotulien est situé à l'intérieur de la bissectrice, l'angle mesuré est négatif (-) et cela traduit plutôt une subluxation interne.



FACTEURS MORPHOLOGIQUES DE L'INSTABILITE DE LA ROTULE
DONNEES DE LA CLINIQUE, DE LA RADIOLOGIE ET DU SCANNER

G. WALCH

L'instabilité de la rotule peut avoir trois traductions cliniques qui ont été bien individualisées et étudiées par P. BRUGERE lors des 4èmes journées de chirurgie du genou en 1982 :

- L'instabilité rotulienne majeure (IRM) : la rotule est luxée en permanence ou se luxe de manière habituelle lors de chaque flexion.
- L'instabilité rotulienne objective (IRO) où l'on retrouve au moins une luxation indiscutable prouvée cliniquement ou radiologiquement. Ce terme englobe également les luxations récidivantes.
- L'instabilité rotulienne potentielle (IRP) : il n'y a jamais eu de luxation de la rotule. La rotule n'est pas luxable, les symptômes sont de type syndrome rotulien avec douleur et instabilité subjective mais il existe une dysplasie de la trochlée et nous verrons que l'existence de certains facteurs luxants en font l'intermédiaire entre les instabilités rotuliennes objectives et les syndromes rotuliens douloureux.

Le but de notre étude est de dégager les anomalies morphologiques qui conduisent à ces différentes pathologies pour en faire une distinction aussi large que possible et peser des indications chirurgicales sur des bases étiologiques précises.

Ces anomalies morphologiques sont connues depuis longtemps mais leurs importances relatives et leurs implications thérapeutiques prêtent encore à discussions :

- Le sexe (prédominance féminine classique)
- Le morphotype : influence défavorable du valgus.
- La hauteur de la rotule : la "patella alta" ou rotule haute pour certains (DEJOUR, INSALL) est source d'instabilité de la rotule.
- Les troubles de torsion des membres inférieurs surtout étudiés par LERAT sont d'interprétation difficile. Leur influence est encore imprécise.
- La TA.GT de BERNAGEAU et GOUTALLIER mesurée radiologiquement ou sur des coupes de scanner joue un rôle indiscutable.
- La dysplasie de la trochlée. Etudiée la première fois par BRATTSTROM elle a depuis été constatée opératoirement et analysée de nombreuses fois sur les vues axiales (FICAT), la radio de profil (MALDAGUE, RAGUET) ou le scanner (SCHUTZER). Nous étudierons cette dysplasie trochléenne par le mesure de :

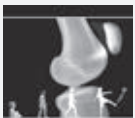
. La profondeur et la hauteur de la trochlée qui aboutissent à la description de différents types trochléens.

. La saillie de la trochlée, situation plus ou moins proéminente de la gorge par rapport à la corticale antérieure du fémur.

. L'angle trochléen sur une vue axiale à 30°.

. Le rapport TE/TI des versants externe et interne sur une vue axiale à 30°.

. La mesure de l'angle trochléen au scanner et de la pente extene de la trochlée fournissent des résultats analogues à ceux des autres facteurs trochléens. Nous les avons analysés mais nous ne les



détaillerons pas car en fonction du niveau des coupes de la trochlée que nous fournissent les radiologues, ils sont susceptibles de variations importantes.

Pour dégager l'importance relative et l'implication thérapeutique de ces différents facteurs, nous avons étudié sur le plan clinique, radiologique et scannographique (tableau I) :

- 22 genoux présentant une luxation permanente ou habituelle de la rotule (IRM : Instabilité Rotulienne Majeure).
- 413 genoux ayant présenté au moins une luxation de la rotule (98 genoux) ou une luxation récidivante (315 genoux). Ce sont les IRO : Instabilité Rotulienne Objective).
- 87 genoux présentant une Instabilité Rotulienne Potentielle (IRP).

L'étude informatique a été réalisée par le Docteur ADELEINE au Laboratoire d'Informatique Médical des Hospices Civils de Lyon. Tous les résultats ont été soumis à l'analyse statistique. Pour l'exposition des résultats, nous utiliserons le signe (*)qui traduit l'existence d'une corrélation statistique très significative (P<0,01).

BILAN RADIO ET SCANNER

	Radio de profil	Vue axiale à 30°	Scanner
I.R.M.	22 genoux	22 genoux	16 genoux
I.R.O.	413 genoux	413 genoux	217 genoux
I.R.P.	87 genoux	87 genoux	53 genoux
Témoins	194 genoux	194 genoux	60 genoux

1/ Le sexe

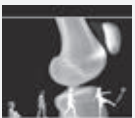
	I.R.M.	I.R.O.	I.R.P.	Témoins
Homme	50%	31%	24%	57%
Femme	50%	69%	76%	43%

Le pourcentage d'hommes est équivalent à celui des femmes dans les formes majeures et la prédominance féminine apparaît puis s'affine avec les IRO et les IRP.

2/ Le morphotype

	I.R.M.	I.R.O.	I.R.P.	Témoins
Normo-axé	33%	34%	39%	31%
Varus	17%	20%	27%	46%
Valgus	50%	46%	34%	23%

La valgus se détache de manière significative dans les instabilités rotuliennes majeures et objectives.



3/ Le recurvatum

	I.R.M.	I.R.O.	I.R.P.	Témoins
Pas de recurvatum	27%	40%	18%	65%
Recurvatum = 0	73%	60%	82%	35%
Moy de recurvatum	5°	4°	6,8°	1,7°

4/ La hauteur de la rotule

	I.R.M.	I.R.O.	I.R.P.	Témoins
Hauteur rot. Moy	1,09	1,12	1	0,95

	I.R.M.	I.R.O.	I.R.P.	Témoins
hauteur rot >1,20	33%	38%	18%	7%

La rotule haute est incontestablement plus fréquente dans les instabilités rotuliennes mais elle n'est présente que dans 1/3 des cas.

5/ L'antéversion fémorale

	I.R.M.	I.R.O.	I.R.P.	Témoins
Antéversion fem moy	22°	21°	19°	15°

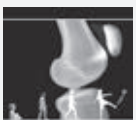
	I.R.M.	I.R.O.	I.R.P.	Témoins
AVF > 24°	50%	38%	41%	17%

Comme la rotule haute, l'antéversion fémorale importante est plus fréquente dans les instabilités rotuliennes.

6/ L'angle condyle-malléole
(torsion tibiale externe + rotation dans le genou)

	I.R.M.	I.R.O.	I.R.P.	Témoins
Angle C.M.	38°	41°	35°	39°

Aucune différence significative.



7/ La rotation dans le genou

	I.R.M.	I.R.O.	I.R.P.	Témoins
Rotation moyenne	13°	8°	4°	4°

La rotation dans le genou est incontestablement plus importante dans les instabilités de la rotule et elle augmente significativement avec les degrés de cette instabilité.
Il est difficile de dire s'il s'agit d'une cause ou d'une conséquence.
Dans les luxations permanentes il est logique de penser qu'il s'agit d'une conséquence, dans les IRO et IRP aucun facteur ne nous permet de trancher.
Il existe dans les instabilités une corrélation statistique positive avec la bascule rotulienne et la TAGT nous en reparlerons ultérieurement.

8/ LERAT a proposé de considérer la ***différence des torsions fémorale et tibiale*** (index fémoro-tibial) et la ***somme des torsions fémoral et tibial***

	I.R.M.	I.R.O.	I.R.P.	Témoins
Index fémoro-tibial	16°	20°	16°	24°
somme des torsions	60°	62°	54°	54°

Ces chiffres reflètent les variations de l'antéversion fémorale.
L'hypothèse pathogénique de la "rotule pris entre deux systèmes de torsions de sens inverse" ne se vérifie pas dans l'instabilité rotulienne puisque la différence moyenne est au contraire plus élevée chez les sujets témoins.

9/ La TA.GT

	I.R.M.	I.R.O.	I.R.P.	Témoins
Moyenne de la TAGT	20 mm	18 mm	15 mm	11 mm

La TA.GT permet indiscutablement de différencier les témoins de l'instabilité rotulienne. En revanche, la différence n'est pas significative entre les différents degrés d'instabilité.

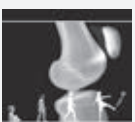
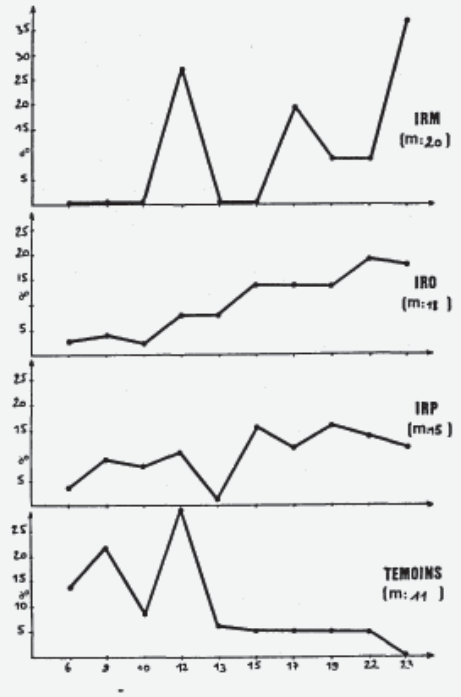


Tableau I

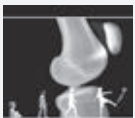


TA.GT en mm - Etude des populations

L'étude des populations montre la prédominance très nette des TAGT hautes dans les IRO et leur rareté chez les sujets témoins. La valeur seuil se situe à 22 mm, on ne retrouve pas de TAGT supérieure à 22 chez les témoins. On peut donc admettre comme pathologique toute valeur supérieure.

Dans les IRM et les IRP on peut distinguer 2 populations, une à TA.GT faible < 13 mm, une à TA.GT élevée > 15 mm. On confirme donc qu'une TA.GT elevée est un facteur indiscutable d'instabilité rotulienne. Mais il ne s'agit pas d'un facteur indispensable.

On retrouve une corrélation statistique positive entre la rotation dans le genou et la TAGT. Cela signifie que plus la rotation dans le genou augmente, plus la TAGT augmente. Compte tenu des variations de la rotation dans le genou que l'on observe au sein des instabilités, on peut se demander si ce n'est pas la rotation qui conditionne la TAGT. Il s'agit d'une question lourde de conséquence car elle aboutirait à une



conception radicalement différente du traitement des instabilités rotuliennes. A l'heure actuelle nos études informatiques ne nous ont pas encore permis de trancher définitivement.

En revanche il n'existe pas de variation statistique de la TAGT avec les torsions osseuses fémorale et tibiale ou le morphotype frontal ou saggital. Le recurvatum et le valgus sont des facteurs indépendants.

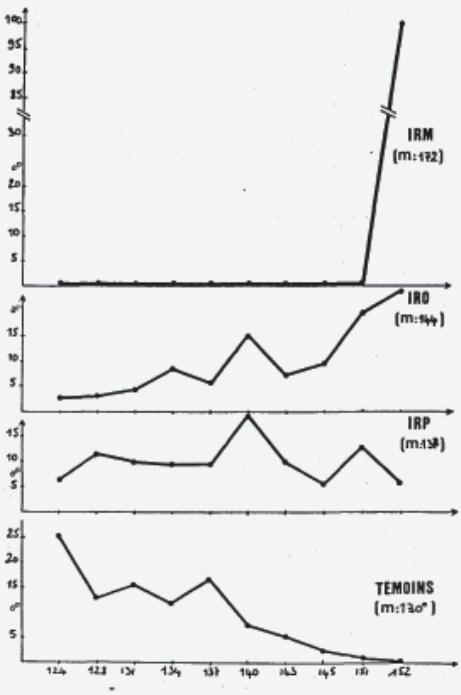
10/ La dysplasie de la trochlée

a) L'angle trochléen à 30° sur une vue axiale

	I.R.M.	I.R.O.	I.R.P.	Témoins
moy angle trochléen	172°	144°	134°	130°

La valeur diagnostique bien connue de cet angle se confirme. On remarque qu'il n'existe pas d'angle trochléen supérieur à 145° chez les sujets témoins. Tout angle trochléen, sur une vue axiale à 30°, supérieur à 145° est pathologique (tableau 2).

Tableau 2



ANGLE TROCHLEEN à 30° - Etude des populations

b) Le rapport TE/TI

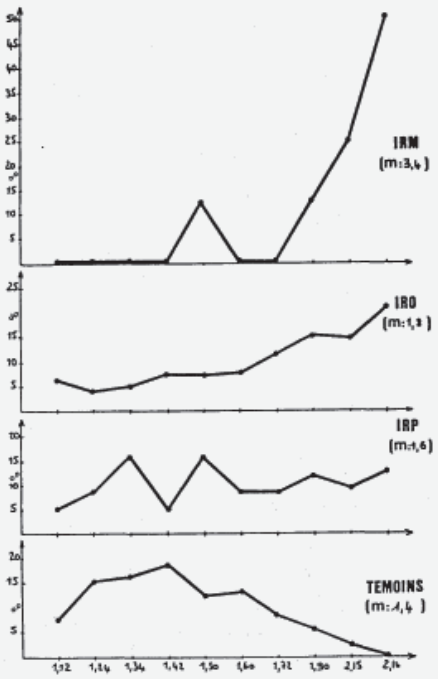
	I.R.M.	I.R.O.	I.R.P.	Témoins
Moyenne TE/TI	3,4	1,84	1,61	1,40

Les chiffres montrent encore une différence plus nette entre les différents degrés d'instabilité et entre les instabilités et les témoins mais il n'y a pas de différence significative entre IRO et IRP (tableau 3).

Dans les instabilités de la rotule, l'angle trochléen s'ouvre et le versant interne de la trochlée diminue par rapport au versant externe.

- Soit il se produit une atrophie du versant interne de la trochlée,
- soit la gorge de la trochlée se déplace en dedans et c'est alors le versant externe de la trochlée qui devient hypertrophique par rapport au versant interne. Nous essayerons de trancher plus tard entre ces deux propositions.

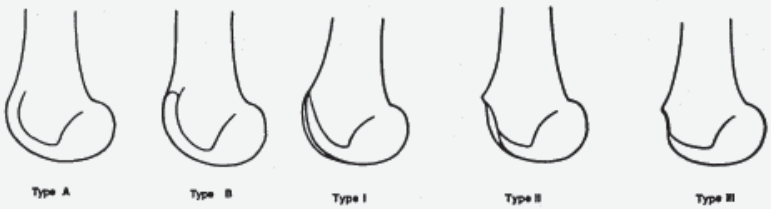
Tableau 3



TE/TI - Etude des populations

c) Les types de trochlée

	I.R.M.	I.R.O.	I.R.P.	Témoins
Type A		2%	0%	50%
Type B		3%	0%	48%
Type I		26%	61%	2%
Type II		58%	33%	0%
Type III	100%	11%	6%	0%

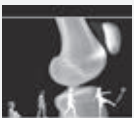


Les types A et B sont les types non dysplasiques. Les types I, II et III traduisent une dysplasie. Le critère commun aux 3 types de dysplasie est le "croisement" entre la ligne de fond de trochlée et le condyle externe.

Ce "croisement" signifie qu'en un point précis le fond de la trochlée et le versant externe sont au même niveau et donc que la trochlée est plate à ce niveau. Ce signe a une valeur diagnostique fondamentale, on retrouve seulement 2% de dysplasie trochléenne ches les sujets témoins et 5% des instabilités rotuliennes objectives n'ont pas de dysplasie. Dans ces 5% il nous est chaque fois apparu à l'analyse que la dysplasie existait mais que le fond de la trochlée ne croisait pas de manière indiscutable le condyle externe. la dysplasie se traduisait soit par une profondeur extrèmement faible (type A I) à la partie supérieure de la trochlée, soit par une dysplasie nette du condyle interne (type B II). Pour ne pas créer de trop nombreux types de trochlée nous avons préféré assimilé ces 5% aux types A et B mais il est possible d'émettre à quelques "pour cents" près le dogme qu' "il n'existe pas d'instabilité rotulienne objective sans dysplasie de la trochlée". Le signe du croisement a pour nous la même valeur que le ressaut en rotation interne dans la rupture du LCA.

Dans les IRP, on retrouve 100% de trochlées dysplasiques puisque c'est la définition même de ce groupe qui présente un syndrome rotulien douloureux. Proportionnellement le type I est beaucoup plus fréquent dans les IRO, il s'agit de dysplasie discrète localisée à la partie supérieure de la trochlée.

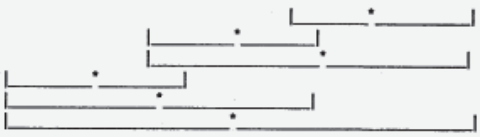
Au sein des IRM on retrouve 100% de type III.



Par rapport à l'angle trochléen à 30° de flexion et au rapport TE/TI le cliché de profil strict du genou se distingue donc surtout par l'analyse qualitative qu'il permet. En effet, certains types I et II peuvent avoir des profondeurs normales à 30° de flexion, la dysplasie étant tout à fait supérieure et indiscutable sur le cliché de profil. Ce qui explique que l'angle trochléen sur la vue axiale à 30° soit peu différente chez les IRP et les témoins. De même sur le cliché de profil on peut se rendre compte que certaines trochlées plates à la partie supérieure se creusent rapidement, ce qui est de bon pronostic pour le traitement alors que d'autres trochlées se creusent peu ou très tard. Les solutions thérapeutiques sont alors très pauvres. Si l'on utilise les techniques chirurgicales classiques notre action ne sera que palliative.

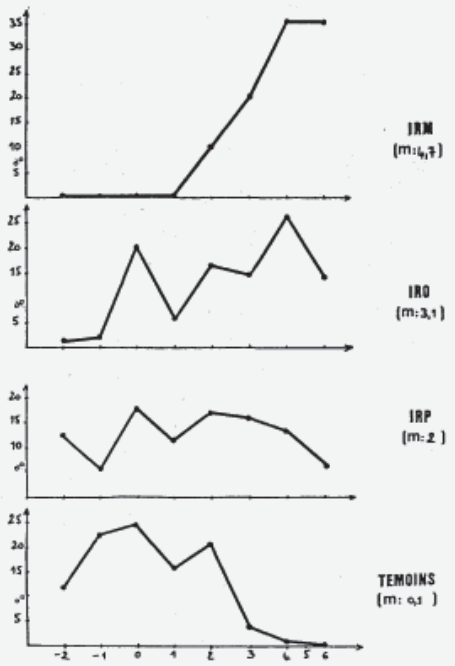
d) La saillie de la trochlée

	I.R.M.	I.R.O.	I.R.P..	Témoins
Moy. de la saillie	4,7 mm	3,1 mm	2 mm	0,1 mm



La saillie moyenne apparaît statistiquement comme le facteur le plus discriminant non seulement entre Témoins et Instabilité mais entre les différents types d'instabilité. On ne retrouve pas de saillie supérieure à 5 mm chez les sujets témoins (tableau 4).

Tableau 4



SAILLIE en mm
Etude des populations

Toute valeur supérieure est un argument indiscutable d'instabilité de la rotule. Cette saillie est interprétable de deux manières différentes :

- Soit elle correspond seulement au comblement de la trochlée et le comblement augmente progressivement des IRP aux IRM. En faveur de ce raisonnement, il existe une corrélation statistique négative forte entre la saillie et la profondeur. C'est-à-dire que plus la saillie augmente plus la profondeur de la trochlée est faible. Cette forte corrélation statistique se vérifie sur l'ensemble des genoux pathologiques que nous avons étudiés sur ordinateur (Instabilité rotulienne, syndrome rotulien, arthrose). En revanche elle n'existe absolument pas chez les sujets témoins.
- Deuxième interprétation possible, la saillie est "en soi" un facteur d'instabilité. La saillie maximum est toujours située au point de soudure antérieur du cartilage de croissance de l'extrémité inférieure du fémur et pourrait correspondre à une hypertrophie anormale par trouble de croissance. Il ne s'agit pas d'une épiphysiodèse prématurée, tout se passe comme s'il y avait un "emballement" très localisé de la croissance osseuse. La saillie jouerait alors, en plus de l'absence de creusement de la trochlée un rôle de "tremplin" pour la rotule qui serait d'autant plus destabilisée dans les premiers degrés de flexion que la saillie serait importante. En faveur de cette deuxième interprétation on retrouve une différence de Saillie statistiquement significative entre les IRM et les IRO. Or, dans les IRO la profondeur est déjà nulle, la Saillie devrait donc être à son maximum si l'hypothèse du comblement était la seule explication. Or, dans les IRM la Saillie croît encore ce qui va en faveur d'un "emballement" très localisé de la croissance. L'absence de corrélation statistique avec la hauteur de rotule élimine la possibilité d'un défaut d'appui dans sa génèse. On retrouve une corrélation statistique avec l'antéversion fémorale et surtout avec la bascule rotulienne en extension. Il est donc possible que ce trouble de croissance soit sous la dépendance d'une bascule ou d'une latéralisation externe de la rotule et que la saillie résulte de la loi de DELPECHE. Nous y reviendrons dans l'histoire naturelle.

SYNTHESE SUR L'ETUDE DE LA TROCHLEE

Les types de trochlée, la Saillie, le rapport TE/TI permettent de dégager des notions qui peuvent être utile au plan thérapeutique.

La dysplasie se traduit par :

a) Un comblement de la gorge de la trochlée

b) Une dysplasie du versant interne de la trochlée par rapport au versant externe visible sur la vue de profil et sur la vue axiale à 30° de flexion.

c) Une hypertrophie "relative" et "absolue" du versant externe de la trochlée qui devient peu à peu seul articulaire. Plus la saillie est importante plus cette hypertrophie est marquée et le contact articulaire en extension (et au maximum en flexion à 30°) ne sera qu'externe, il n'y a plus de versant interne cartilagineux. On assiste à un déplacement en dehors du cartilage articulaire de la trochlée. Les difficultés chirurgicales sont maximum et même avec une trochléoplastie de MASSE on ne peut recréer une trochlée puisqu'il n'y a pas de cartilage interne (schéma 5).

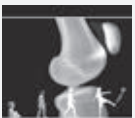
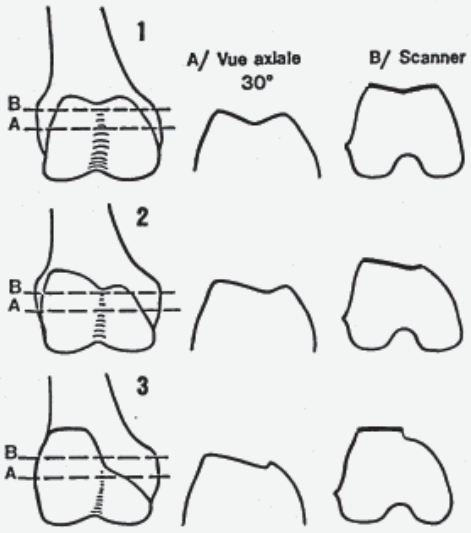


Schéma 5



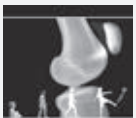
Au terme de cette étude **morphologique**, il apparaît :

- un facteur fondamental de l'instabilité de la rotule, c'est la dysplasie de la trochlée qui a deux composantes, le comblement de la gorge et la saillie antérieure
- 2 facteurs majeurs présents à des degrés divers et avec une fréquence plus faible
  - . la rotule haute
  - . une TAGT élevée

4 facteur mineurs :

- . une antéversion fémorale haute
- . une rotation exagérée dans le genou
- . le genu-valgum
- . le sexe féminin

Il est important de remarquer qu'il existe très peu de corrélation statistique entre ces différents facteurs "majeurs" et mineurs". Les seules corrélations qui soient intéressantes concernent la TAGT. Elle est statistiquement corrélée à la rotation dans le genou mais il n'y a pas de corrélation avec la torsion tibiale externe ou l'antéversion fémorale.



LE SCANNER DANS LA PATHOLOGIE FEMORO-PATELLAIRE

J. TUNEU - G. WALCH

Il est demandé de manière presque systématique dans le service depuis 1979 pour toute pathologie rotulienne opérée.

I - LE PROTOCOLE

Le patient est placé en décubitus dorsal, les pieds fixés en rotation externe de 15° sur une planchette perpendiculaire à la table (figure 1) de façon à ce qu'il n'y est pas de mouvement pendant l'examen. LERAT préconise de placer les pieds selon l'angle du pas ce qui n'est pas toujours respecté par les radiologues. Nous avons réalisé des coupes comparatives en rotation neutre, rotation interne et rotation externe des pieds pour savoir si la position des pieds pouvaient modifier les mesures. Il est apparu que les mesures n'étaient pas modifiées par les différentes rotations pourvu qu'elles ne s'effectuent pas, bien sûr, entre deux coupes. Néanmoins le protocole que nous utilisons recommande de mettre les pieds à 15° de rotation externe, ce qui est un repère facile pour les radiologues. L'appui plantaire est indispensable pour éviter la contraction du quadriceps.

L'examen se déroule en deux temps : statique puis dynamique.

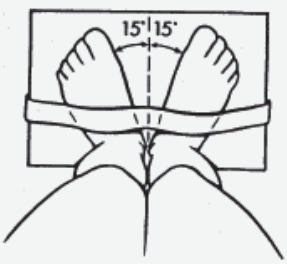


Figure 1

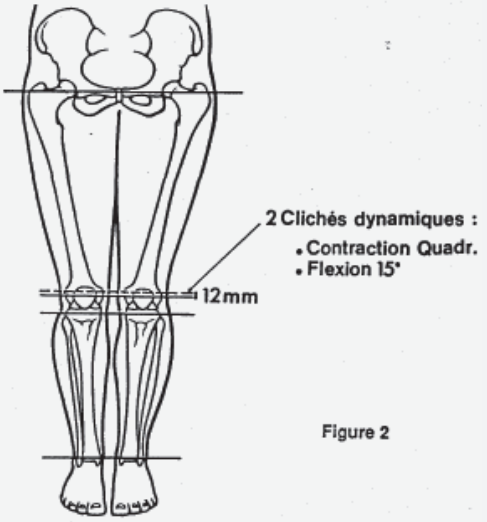


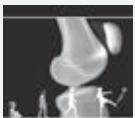
Figure 2

1/ Exploration statique

Elle doit toujours être réalisée en premier de manière complète pour ne pas que les manoeuvres dynamiques modifient la position du membre inférieur.

Nous demandons (figure 2) :

a) Une coupe au niveau des hanches passant par le sommet de la fossette digitale. LERAT recommande de pratiquer deux coupes, une passant par les têtes fémorales, l'autre passant par la base du col. Avec une seule coupe il est apparu que l'erreur possible dans la mesure de l'axe du col est minime.



b) Une coupe passant par le milieu de la rotule (figure 3) qui permet d'analyser la TROCHLEE. Si la rotule est haute, comme c'est fréquemment le cas dans les instabilités, un autre cliché doit être réalisé passant par le sommet de la trochlée. L'étude des coupes étagées au scanner et les coupes d'os secs nous ont permis de déterminer avec grande précision l'aspect que revêt la coupe devant être retenue pour les mesures de la trochlée : Le meilleur repère est l'échancrure intercondylienne qui revêt différents aspects selon le niveau.

Figure 3A

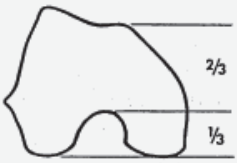


Figure 3B

Figure 3C

- A la partie la plus distale (figure3A), la paroi externe de l'échancrure intercondylienne forme une ligne verticale et la paroi interne forme une courbe en forme d'arc Gothique. L'angle trochléen à ce niveau est de l'ordre de 90° à 100° et la pente externe est largement supérieure à 40°. On ne peut donc effectuer les mesures à ce niveau.
- Lorsque le niveau de la coupe remonte vers la partie proximale de l'échancrure, petit à petit cette dernière prend une forme de plus en plus arrondie et l'angle trochléen varie très rapidement dans le sens d'une diminution, ainsi que la pente du versant externe.
- A la partie la plus haute de la trochlée, l'échancrure forme un "arc Roman" arrondie (figure 3B). C'est ce repère que l'on doit s'attacher à retrouver. A ce niveau la largeur antéro-postérieure de l'échancrure intercondylienne forme le tiers de la largeur de l'ensemble de l'épiphyse, allant de la ligne bicondylienne en arrière à la gorge de la trochlée en avant. Sur cette coupe, à la partie externe, l'épicondyle est dessiné, il a une forme pointue, et on voit encore parfaitement la forme des deux condyles postérieurs.
- Dès que la coupe est trop haute (figure 3C), l'échancrure prend une forme en arc de cercle représentant le quart d'une sphère et les condyles disparaissent. A la partie externe, la surface d'insertion du tendon poplité a disparu. L'angle trochléen se modifie de nouveau et les lignes qui forment cet angle ne sont plus des droites mais des courbes, ce qui correspond à la fossette sus trochléenne.

c) Une coupe passant par l'épiphyse tibiale supérieure juste en dessous de l'interligne articulaire. Cette coupe permet de tracer l'axe d'orientation postérieure de l'épiphyse tibiale.
La plus grande partie de la rotation externe tibiale se produit dans les premiers centimètres de l'épiphyse supérieure. La mesure doit donc être effectuée le plus haut possible sur le tibia (figure 4). Les plateaux tibiaux intern et externe ont une forme bien différente. Le plateau interne est concave vers le haut et se termine brusquement à la partie postérieure. Son repère est facile à trouver. En revanche, le plateau externe est convexe et sa paroi postérieure décalée de 4 mm vers le bas par rapport au plateau tibial interne. C'est la raison pour laquelle l'axe de la métaphyse tibiale postérieure est difficile à tracer. La zone idéale de coupe a pour limites en bas l'articulation tibio-péronière qui est déjà située trop bas (la rotation externe tibiale a déjà déburée) et en haut le début de la paroi postérieure verticale du plateau tibial externe. Cet espace très réduit explique que les erreurs de mesure de l'axe tibial postérieur soient

fréquentes. Sa détermination souffre souvent d'une imprécision de quelques degrés.

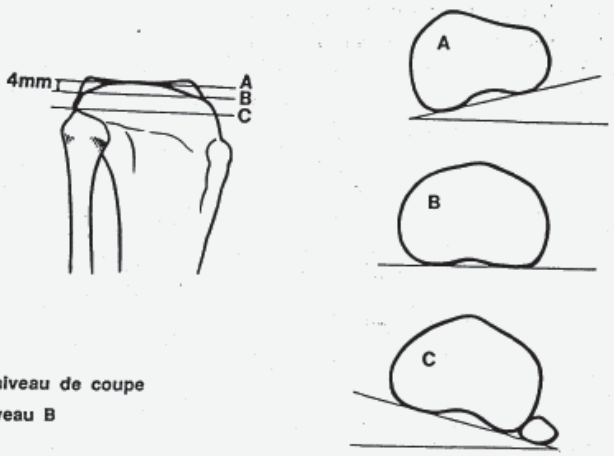


Figure 4 **Le bon niveau de coupe est le niveau B**

d) Une coupe passant par la tubérosité tibiale antérieure à sa partie haute.

e) Une coupe au niveau de la cheville passant par la base des malléoles.

f) Une coupe passant 2 cm au dessus de la plante du pied.

2/ Exploration" dynamique"

Des coupes dites "dynamiques" sont réalisées, passant par le milieu de la rotule.

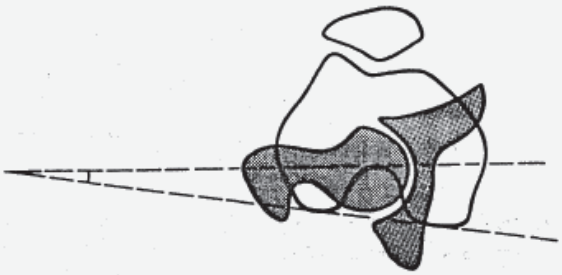
* un cliché en extension, quadriceps contracté
* un cliché à 15° de flexion sans contraction du quadriceps

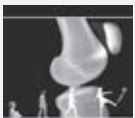
II - LES MESURES

1/ Les angles de torsion

Ils sont obtenus en superposant sur un calque les différents clichés ou mieux actuellement par reconstruction sur ordinateur.

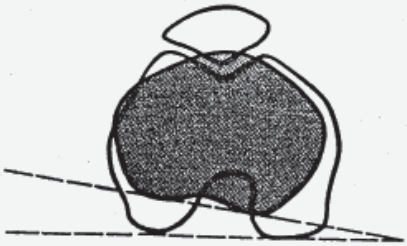
a) L'antéversion fémorale : angle formé par l'axe du col du fémur et l'axe bicondylien postérieur (figure 5).





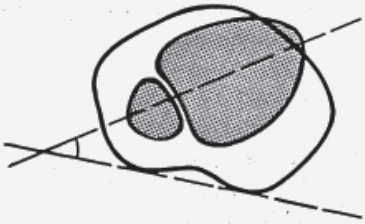
b) La rotation dans le genou : angle entre l'axe bicondylien postérieur et l'axe d'orientation postérieur de l'épiphyse tibiale. Cette mesure est rendue très imprécise par la forme du plateau tibial externe (figure 6).

Figure 6



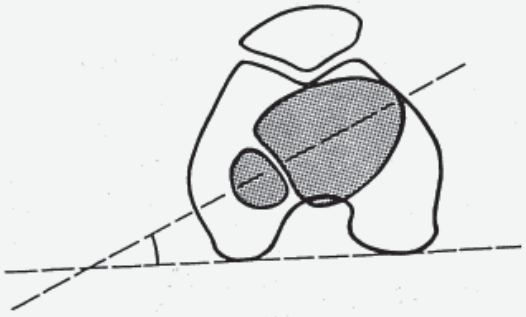
c) La torsion tibiale externe : angle formé par l'axe d'orientation postérieure de l'épiphyse tibiale et l'axe bimalléolaire (schéma 7).

Figure 7



d) L'angle condyle-malléole (schéma 8). Dans la pratique, il tend à remplacer la torsion tibiale externe. Il occulte la rotation dans le genou mais sa précision est supérieure puisqu'il il n'y a pas l'incertitude de l'axe d'orientation postérieure de l'épiphyse tibiale.

Figure 8

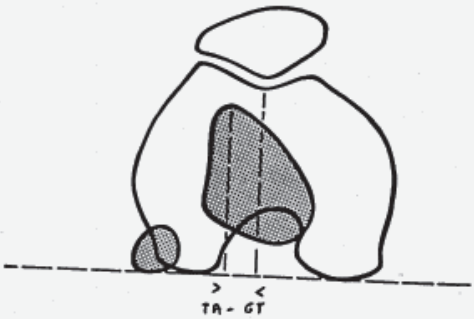


e) La détorsion sous-malléolaire : angle formé par l'axe bimalléolaire et l'axe du 3ème métatarsien. Nous avons abandonné sa mesure systématique.

2/ La TA-GT

Obtenue par superposition (figure 9) de la coupe passant par la partie haute de la trochlée et la coupe passant par la Tubérosité Tibiale Antérieure. Deux points fixant le fond de la gorge trochléenne et le milieu de la TTA sont abaissés perpendiculairement sur la ligne bicondylienne postérieure où le TA-GT peut être mesurée en mm en tenant compte du facteur d'agrandissement. C'est un des grands apports du scanner qui rend cette mesure beaucoup plus facile qu'avec la radiographie. Cette mesure demande des grands clichés ou une projection par épidiascope pour les petits clichés. L'étude des scanners pré et post-opératoires nous a montré que la mesure avait une marge d'erreur de ± 4 mm. Cette mesure exprime la somme de l'implantation externe de la TTA sur le tibia et de la rotation externe du genou qui lorsqu'elle existe, la majore.

Figure 9



3/ L'articulation fémoro-patellaire

a) L'angle trochléen : est mesuré de la même manière que sur une vue axiale à 30° (cf châpitre radio) (figure 10).

Figure 10

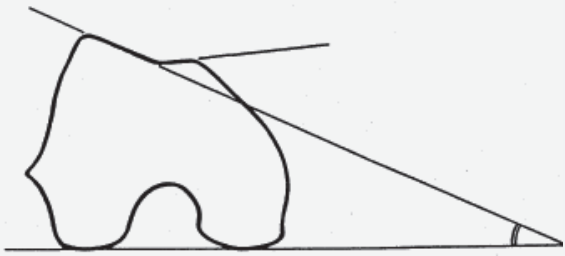
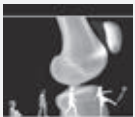


Figure 11

b) La pente du versant externe de la trochlée : angle formé par une ligne tangentielle au versant externe de la trochlée et la ligne bicondylienne postérieure sur la coupe passant à la partie haute de la trochlée (figure11).



e) La subluxation rotulienne est mesurée par l'angle de congruence (figure14A) tel qu'il est mesuré sur la vue axiale à 30°. Là encore, sa détermination est mal aisée sur une coupe scannographique car les points de référence sont difficiles à déterminer. La subluxation peut également être mesurée en mm (figure 14B) en prenant comme point de repère la crête médiane de la rotule et le fond de la gorge de la trochlée ; ces deux points sont abaissés perpendiculairement sur la ligne bicondylienne postérieure où la distance est mesurée en mm. Elle est positive si elle est externe par rapport à la gorge de la trochlée, elle est négative lorsqu'elle est interne. Sa valeur normale moyenne est de + 2,5 mm.

Ces deux mesures "bascule" et "subluxation" doivent être faites sur le cliché du genou en extension quadriceps décontracté et sur les coupes dynamiques. Il s'agit de mesures difficiles à réaliser mais qui nous paraissent fondamentales. En particulier la bascule rotulienne est pour nous représentative de la dysplasie du vaste interne.

Figure 14A

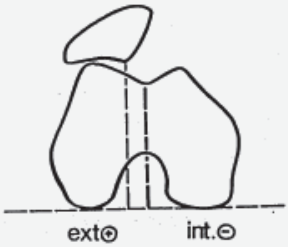
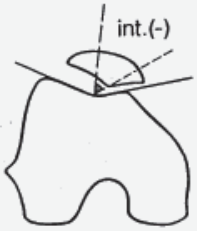


Figure 14B



c) L'inclinaison trochléenne : angle formé par une ligne joignant les sommets des deux condyles et la ligne bicondylienne postérieure (figure 12).

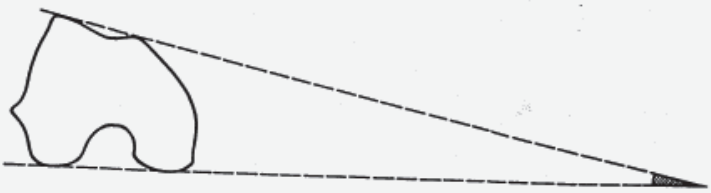


Figure 12

Ces mesures sont représentatives de la forme de la trochlée dans les vingts premiers degrés de flexion. Elle ont pour inconvénient majeur d'être très dépendantes du niveau de coupe. Il vaut mieux ne pas les effectuer si les coupes dont on dispose ne sont pas adéquates. La pente du versant externe est plus facile à mesurer que l'angle trochléen dans les dysplasies car, dans ce cas, le point le plus profond de la trochlée est mal aisé à déterminer. La pente du versant externe et l'angle trochléen traduisent le degré de creusement ou de comblement de la trochlée et sont donc une mesure indirecte de sa profondeur. Malheureusement on ne dispose pas toujours de la bonne coupe et l'on peut méconnaître certaines dysplasies à la partie toute supérieure de la trochlée. Il apparaît indiscutable que la radiographie simple de profil strict fournie plus de renseignements sur les anomalies de profondeur de la trochlée. De plus le scanner méconnaît totalement la saillie qui nous paraît capitale dans l'analyse de la dysplasie trochléenne. La mesure de l'inclinaison trochléenne est moins intéressante bien qu'elle permette de mettre en évidence la dysplasie du condyle interne. Cette anomalie est fréquente dans les instabilités rotuliennes, sa génèse, sa quantification et son rôle pathogène restent encore mystérieux et imprécis.

d) La bascule rotulienne mesurée sur une coupe passant par le milieu de la rotule (figure13), c'est l'angle formé par l'axe transversal de la rotule (plus facile à déterminer que la facette articulaire externe) et la ligne bicondylienne postérieure. Pour faciliter la mesure on peut tracer une ligne parallèle à la ligne bicondylienne passant par le fond de la trochlée.

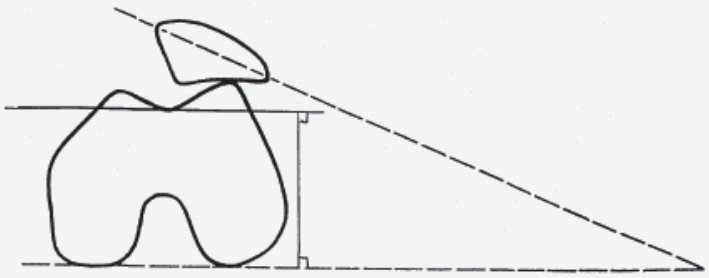
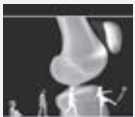


Figure 13



ARTHROSE FEMORO-PATELLAIRE EXTERNE

G. DESCHAMPS, G. PY

I - INTRODUCTION

L'étude est complémentaire de celle réalisée pour la détermination des facteurs radiologiques et scannographiques qui, d'emblée, portait sur les arthroses fémoro-patellaires (A.F.P.) isolées : en excluant les cas d'AFP associés à une arthrose fémoro-tibiale où les gestes réalisés ne s'adressaient pas spécifiquement à la rotule.

Nous avons ensuite exclu les cas d'arthroses fémoro-patellaires :

- non opérées
- globales et internes (rares)
- externes (AFPE) ayant un dossier clinique trop incomplet ou n'ayant pas un recul d'au moins deux ans.

La série porte donc finalement sur 106 sujets représentant 132 genoux opérés d'arthrose fémoro-patellaire externe (AFPE) répartis en trois groupes :

- AFPE "essentielles" : secondaires à une dysplasie luxante de l'enfance ou apparemment primitive,
- AFPE liées à une chondrocalcinose,
- AFPE post-traumatiques.

II - ETUDE DES AFPE ESSENTIELLES

1/ Matériel d'étude et classification étiologique

La série regroupe 89 patients représentant 114 genoux opérés. L'étude a un double objectif :

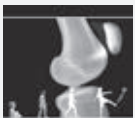
- classer ces AFPE en fonction de critères étiologiques afin de préciser leur genèse,
- juger les résultats des interventions réalisées habituellement dans le service : essentiellement ELMSLIE-MAQUET.

Les données anamnestiques et radiographiques ont permis de classer ces patients en trois groupes d'importance inégale :

. avec antécédents de luxation(s) représentant les instabilités objectives (IRO) : 40 patients, dont 14 bilatéraux soit 54 genoux.

Les critères d'IRO sont : antécédents de luxation de rotule retrouvés à l'interrogatoire ou attestés dans les cas douteux par un Pellegrini-Stieda du versant interne de la rotule ou d'une fracture ostéochondrale ou à l'examen clinique d'un SMILLIE non discutable. L'histoire clinique est dans ces cas souvent caractéristique avec des accidents d'instabilité jusqu'à 25 à 30 ans, suivis d'un relatif silence jusqu'à la cinquantaine puis d'une récidive douloureuse avec constatation d'une AFP. Deux cas sont typiques à cet égard opérés par tansposition de la tubérosité tibiale antérieure (TTA) pour luxation récidivante d'un côté et revus 20 ans plus tard avec une AFP unilatérale du côté opposé malgré une instabilité objective.

. avec antécédent de "syndrome rotulien" isolé (SR) qui est en général séparé de la période



douloureuse en relation avec l'AFPE par un intervalle libre de plusieurs années : 17 patients, dont 6 bilatéraux soit 23 genoux. Les critères de SR correspondent à toute douleur fémoro-patellaire ne répondant pas aux critères d'IRO (genoux faibles , syndrome douloureux isolé dans l'adolescence, entorse atypique, etc...).

- sans aucun antécédent rotulien (SA) : 32 cas, dont 5 bilatéraux soit 37 genoux.

a) Sexe : très nette majorité de femmes, supérieure à celle rencontrée dans les autres types de pathologie fémoro-patellaire : 92 %.

b) Age : moyenne

à l'intervention : 53 ans (30-72 ans),

à la révision : 59 ans (34-79 ans).

L'analyse en fonction de l'étiologie revèle que le groupe des IRO est plus jeune à l'intervention (50 ans en moyenne) que celui des SR (53 ans) et des SA (56 ans).

c) Côté opéré :

- 41 % du genou droit.

- 27,3 % des sujets ont été opérés des 2 côtés dont 5,3 % ne rentrent pas dans l'étude car leur deuxième genou a été opéré avant l'apparition de l'AFPE ou pour AFP avec un recul inférieur à 2 ans.

2/ Analyse clinique

Les patients ont été classés dans le système CLAS et étudiés avec la cotation ARPEGE.

a) CLAS avant l'intervention

- En pré-opératoire

- 6 % sont classés "Loisir" (L)
- 71 % sont "actifs" (A)
- 23 % sont "sédentaires" (S)

Cette classification n'est pas en rapport avec l'âge et montre que l'AFP est assez bien tolérée avec une majorité d'actifs et même quelques sportifs de loisir (cyclisme).

- En fonction de l'étiologie, il y a une différence nette de niveau avec :

pour les IRO :

- 85 % d'actifs ou loisirs,
- 15 % de sédentaires

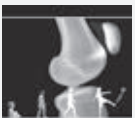
pour les SR :

- 75 % d'actifs,
- 25 % de sédentaires

pour les SA :

- 65 % d'actifs
- 35 % de sédentaires

Ceci est peut être en rapport avec l'âge plus élevé des SA



b) Gêne fonctionnelle pré-opératoire (Stabilité, Douleur, Mobilité)

- Douleurs dans la vie quotidienne :

Nous avons choisi d'isoler ce facteur de la fiche ARPEGE, car il représente une des plaintes essentielles du patient. La majorité déclare des douleurs classées "importantes, discontinues" avec un score moyen de 1,3/3.

- Douleurs et résistance à la fatigue :

Le score moyen pour l'ensemble est de 4,82/9 sans différence significative en fonction de l'étiologie.

- Stabilité :

La gêne des AFP dans ce domaine est moindre avec des scores moyens à 5,82/9. Les plus gênés sont les IRO : 5,31, ce qui est logique.

- Escaliers :

Presque tous les patients, 85 %, sont gênés à la descente des escaliers, 41 % le sont également à la montée.

- Périmètre de marche (PM)

72% ont un PM ≥ 1500 m et 13 % disent avoir un PM illimité.

En résumé, c'est essentiellement la douleur qui est la gêne principale de ces patients, ce qui est à garder en mémoire pour l'analyse des résultats.

c) Morphotype global

On note une nette prédominance de genu valgum : 55 % contre 31 % de normo-axés et 15 % de genu varum. Cette proportion de genu valgum est plus importante que dans la population témoin ainsi que dans celle des dysplasies. En fonction de l'étiologie il n'y a pas de différence significative dans les 3 groupes.

d) Hauteur de rotule

- L'index rotulien moyen est de 0,96.
- Il est plus élévé pour les IRO (1,05) que pour les deux autres groupes (0,88).

e) Morphologie trochléenne

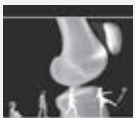
* Vues axiales à 30° :

L'angle trochléen moyen est de 142° pour les IRO alors qu'il n'est que de 134° pour les SR et 131° pour les SA. Cet angle trochléen moyen est, pour les IRO, identique à celui retrouvé dans l'étude générale, alors qu'il est pour les SR et les SA, proche de celui des IRP et des syndromes rotuliens douloureux. La subluxation jugée par l'angle de congruence de Merchant, en moyenne de + 33° en pré-opératoire, n'a pas de différence de moyenne nette entre les étiologies mais surtout a une trop grande dispersion pour une analyse convenable. L'importance du pincement, qui n'est pas réellement quantifiable, est sans signification.

* Le type de trochlée :

- 34 % ne sont pas classables du fait de l'usure arthrosique,
- 25 % sont de type I,
- 35 % de type II,
- 0,8 % de type III,
- 7 cas seulement sont de type A (1,7 %) ou B (3,5 %).

Il est notable que 93,5 % des trochlées identifiables sont dysplasiques, comme pour les instabilités. La répartition est par contre très différente avec un pourcentage très faible de type III, c'est certainement dans cette catégorie que les données radiographiques deviennent plus précaires en cas d'arthrose. Il faut



souligner aussi que les types III correspondent à des formes sévères et il est bien rare quelles ne soient pas opérées plus jeunes.

* La saillie :

C'est une des constantes particulièrement intéressante car elle ne peut guère être modifiée par l'arthrose. Cette saillie est presque toujours positive avec une moyenne plus élevée dans les IRO (2,76 mm) que dans les autres groupes (SA : 1,79 mm, SR : 1,72 mm).

* L'Indice de Hauteur Trochléenne (IHT)

Il n'y a pas de différence nette entre les groupes étiologiques sauf en ce qui concerne les types II où l'indice trochléen est plus bas (70°) dans les IRO.

III - TYPES D'INTERVENTIONS REALISEES

Il y a eu

- 106 tanspositions de la TTA de 5 à 15 mm (moyenne 12,6 mm) associées à 96 "effet MAQUET" de 5 à 15 mm (en moyenne 10 mm).
- 1 seul MAQUET isolé
- 5 patellectomies
- 2 prothèses fémoro-patellaires (type BOUSQUET).

Les gestes associés ont été :

* des gestes rotuliens :
  - 75 patellectomies externes (70 %)
  - 55 PRIDIE de rotule (52 %)
  - 25 carbonisations (24 %)
* d'autres gestes :
  - 10 méniscectomies internes
  - 3 méniscectomies externes
  - 2 doubles méniscectomies
  - 3 SLOCUM
  - 1 plastie du vaste interne
  - 4 ablations de corps étrangers

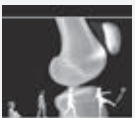
IV - COMPLICATIONS

Sur 114 interventions :

- 15 phlébites dont 3 avec embolies pulmonaires,
- 8 mobilisations sous anesthésie générale,
- 1 fracture du 1/3 supérieur du tibia au bas de la baguette d'ELMSLIE après une chute.

V - RESULTATS

Le recul moyen est de 7 ans (2 à 22 ans).



1/ Résultats subjectifs

Très satisfaits (TS) :	43 %
Satisfaits (S) :	42 %
Déçus (D) :	13 %
Mécontents (M) :	2 %

Soit 85 % de patients satisfaits ou très satisfaits. Ce taux varie légèrement avec l'étiologie : IRO : 86 %, SR:91 %, SA : 81 %.

2/ Résultat fonctionnel global

Evalué à partir de la fiche ARPEGE, ont été notés :

EXCELLENTS : les patients satisfaits ou très satisfaits avec un niveau d'activité au moins égal et surtout un score SDM (stabilité, douleur, mobilité) de la fiche ARPEGE à 989 au moins.

BONS : les patients au moins satisfaits avec un niveau d'activité égal et un score à 878 au moins.

MOYENS : les patients au moins satisfaits avec un niveau d'activité égal ou immédiatement inférieur et un score de 767 au moins.

MAUVAIS : les patients déçus, mécontents, satisfaits ou très satisfaits mais avec un niveau d'activité égal ou inférieur et surtout un score à partir de 656.

La plus mauvaise des 5 données (appréciation subjective, niveau d'activité, stabilité, douleur, mobilité) donne le classement.

A noter :

- que nous n'avons pas tenu compte de l'appréciation des 2 sujets déçus qui avaient augmenté leur niveau de CLAS.
- que nous avons systématiquement côtés les patients de moins de 65 ans avec le critères "d'Actifs" et de plus de 65 ans avec ceux de "Sédentaires".
- que nous avons considéré la mobilité selon l'ancienne classification ARPEGE ne considérant pas le caractère purement fonctionnel mais uniquement physique.

Ces résultats montrant l'état fonctionnel objectif du genou post-opératoire sont :

* Globalement :

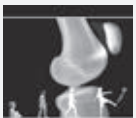
- Excellents à	11 % (E)
- Bons à	30 % (B)
- Moyens à	22 % (My)
- Mauvais à	37 % (Mv)

Soit seulement 41 % de résultats satisfaisants (excellents ou bons) à l'analyse objective.

* En fonction de l'étiologie :

- IRO :	46 % }
- SR	41,5 % }de résultats excellents ou bons...
- SA :	30 % }

Il nous paraît essentiel d'expliquer autant que faire se peut cette discordance entre résultats objectifs et subjectifs.



L'analyse des chiffres permet en partie de répondre car elle montre que beaucoup de patients sont pénalisés par de mauvais scores en stabilté. Si ce facteur est essentiel à juger pour le chirurgien et justifie le maintien de ces résultats décevants, il n'en est pas de même pour les patients qui jugent en fait beaucoup plus le résultat sur la douleur.

En effet, la comparaison résultat subjectif-score-(douleur-stabilité) montre :

a) Pour la douleur

- 80 % d'améliorés chez les très satisfaits (19 genoux),
- 51 % d'améliorés chez les satisfaits (46 genoux),
- 19 % d'améliorés seulement chez les déçus ou mécontents (16 genoux).

b) Pour la stabilité

- 65 % d'améliorés chez les très satisfaits,
- 55 % d'améliorés chez les satisfaits,
- 31 % d'améliorés chez les déçus et mécontents.

Le fait qu'un tiers des patients TS ne soit pas amélioré pour la stabilité témoigne que ce facteur est moins sévèrement jugé par le patient. Les autres éléments tels que l'épreuve des escaliers, la mobilité ou le périmètre de marche n'influent pas de façon significative sur les résultats.

3) Etude analytique

Elle confirme si besoin est ce que nous venons de dire.

a) L'évolution des scores DRF

Elle montre une nette progression des scores de 4,82 à 6,44, bien que les chiffres restent modestes en valeur absolue.

Il n'y a pas de différence significative entre les 3 groupes étiologiques sauf peut être pour les SR qui passent de 4,43 à 6,41 gagnant presque 2 points. Nous avons isolé de la fiche ARPEGE la "douleur dans la vie quotidienne" qui confirme ces résultats en montrant que les patients passent en majorité du score 1 "douleurs importantes discontinues" au score 2 " douleurs modérés occasionnelles".

b) Evolution du score stabilité

Nous avons vu que la gêne fonctionnelle était moindre et ce facteur est également moins bien amélioré passant de 5,82 à 6,78.

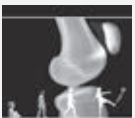
En fonction de l'étiologie, les IRO sont les plus améliorées de 5,31 à 6,9 ce qui est assez logique. Les patients sans antécédent (SA) régressent même de 6,65 à 6,57. Nous verrons que c'est dans ce groupe que la proportion de bons résultats est la plus faible de même que le résultat subjectif.

c) Mobilité

Elle n'est ni améliorée ni aggravée par l'intervention (de134° en pré-opératoire à 133° à la révison) bien que quelques patients aient ponctuellement perdu de la mobilité sans jamais être inférieure à 90° sauf dans un cas déjà raide auparavant.

d) Epreuve des escaliers

Il n'y a pratiquement aucune amélioration en ce domaine, 85 % sont gênés avant et 77 % le restent. Nous avons vu que ce facteur n'influençait pas le résultat subjecif.



e) Périmètre de marche

Il est peu perturbé en pré-opératoire avec 72 % ≥ 1500 mètres ; à la révision 82 % sont capables de marcher plus de 1500 mètres. Cependant 37 % ont un périmètre de marche illimité contre 13 % avant. Ceci traduit l'effet antalgique de l'intervention.

f) Facteurs influençant le résultat

Cette analyse est a priori difficile compte-tenu de la forte proportion des résultats décevants. Cependant un certain nombre de facteurs semble jouer un rôle non négligeable.

* CLAS avant l'intervention :

L'analyse montre une différence significative avec

- 86 % d'excellents et bons résultats chez les sujets classés "Loisirs"
- 40 % chez les actifs
- 30 % seulement chez les "Sédentaires".

* Age à l'intervention :

Les excellents et bons résultats passent de 57 % avant 50 ans à 12 % seulement si l'intervention à lieu après 60 ans. C'est un élément fondamental de l'analyse factorielle en ce qui concerne l'indication thérapeutique.

* Age à la révision :

Il joue également un rôle important puisque les résultats excellents ou bons passent de 62 % avant 50 ans à 29 % après 60 ans et même à 15 % après 70 ans.

* Recul par rapport à l'intervention :

Il explique peut être en partie ces deux dernières données puisque si jusqu'à 9 ans de recul, le pourcentage d'excellents et bons résultats reste autour de 42,5 %, il tombe à 30 % au-delà de 10 ans de recul.

* Etiologie :

Nous l'avons déjà vue jouer un rôle plus modéré mais significatif avec :

- dans l'IRO	excellents + bons	46 %
	mauvais	32 %
- dans les SR	excellents + bons	41,5 %
	mauvais	46 %
- dans les SA	excellents + bons	30 %
	mauvais	43 %

* Influence du morphotype :

L'analyse montre que les meilleurs résultats sont obtenus chez les sujets normo-axés avec 50 % d'excellents ou bons résultats.

Par contre aux deux extrêmes :

- les valgus forts ≥ 5 cm comptent 70 % de mauvais résultats
- les varus forts ≥ 3 cm comptent 71 % de mauvais résultats (avec 54 AFTI de stade II sur 76).

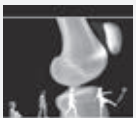
* Influence de la hauteur de la rotule :

Tous les cas où l'index rotulien était < 0,75 ont un résultat mauvais ou moyen.

* Influence de la morphologie trochléenne :

. Sur les vues axiales

L'angle de congruence de Merchant est en moyenne amélioré surtout dans le cas des IRO (où il passe de



32 à 19 contre 32 à 25 dans les SA).

Par contre, le pincement est en général peu amélioré

. Sur les vues de profil

- le type de trochlée, l'indice de hauteur rotulienne n'ont aucune influence sur le résultat.
- la saillie semble influencer légèrement le résultat en dehors des IRO (où elle est d'ailleurs initialement plus élevée) ; plus la saillie est faible, meilleur serait le résultat (mais la dispersion des chiffres ne permet pas d'en faire une règle).

* Influence du type d'intervention :

Les 5 patellectomies ont toutes donné un mauvais résultat bien que 3 patients contents du résultat. Ceci confirme l'analyse qu'avait faite LECLERC-CHALVET dans sa thèse.

Les 2 prothèses fémoro-patellaires ont donné un résultat objectif mauvais et moyen, les patientes étant cependant satisfaites.

Les 106 transpositions TTA ont donné :

- 42 % d'excellents et bons résultats
- 23 % de moyens résultats
- 35 % de mauvais résultats.

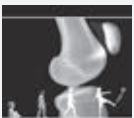
Nous manquons de données numériques dans un nombre non négligeable de dossiers anciens pour analyser de façon formelle l'influence de certains gestes. Ceci explique peut-être que :

- L'importance de la transposition, supérieure ou inférieure à 13 mm, ne semble pas influencer le résultat. Sauf peut-être pour les IRO où le pourcentage d'excellents et bons résultats passe de 55 % si la médialisation est inférieure 13 mm à 65 % si la médialisation est supérieure à 13 mm.
- Par ailleurs, il n'y a pas de concordance entre l'importance de la médialisation et le recentrage de la rotule obtenu à la révision, et ce recentrage lui-même ne semble pas influencer le résultat puisque l'amélioration du pincement ne modifie pas la proportion de bons ou mauvais résultats.
- Enfin on aurait pu penser qu'une forte transposition ≥ 13 mm favorise un remodelé interne ou une AFTI. En fait, l'AFTI n'est pas la conséquence de la transposition mais celle du morphotype initial.
- Parmi les autres gestes associés sur la rotule, patellectomie externe, PRIDIE et carbonisation n'influencent pas non plus sur la qualité du résultat. On peut tout juste noter une plus forte proportion d'échecs dans les PRIDIE : 38 % contre 31 % d'excellents et bons résultats.

VI - ETUDE DES AFPE AVEC CHONDROCALCINOSE

Ce type d'AFPE doit être séparé car il s'agit d'une maladie évolutive où ni la dysplasie ni le syndrôme d'hyperpression externe ne représentent la cause initiale de la pathologie. Elles doivent être detéctées avec soins. En effet, si l'étude de 11 cas ne montre pas de facteur étiologique particulier (une seule aurait été considérée comme une IRO au départ, 1 seule est bilatérale, la moyenne d'âge est de 54 ans (45-73 ans), il y a deux hommes, par contre, le résultat fonctionnel des interventions est, selon les critères objectifs utilisés précedemment, toujours mauvais, que l'intervention ait été une patellectomie (2 cas) ou un ELSMLIE-MAQUET (7 cas). Quatre patients sont d'ailleurs déçus dans cette dernière intervention ce qui est particulièrement elevé. Les autres s'estiment cependant satisfaits ou très satisfaits. Deux patients ont eu une prothèse totale du genou (un initialement, un autre en reprise).

A noter qu'un cas d'arthrose fémoro-patellaire interne isolée sur chondrocalcinose a été retrouvé.



VII - ETUDE DES ARTHROSES POST-TRAUMATIQUES

Elle ne concerne que celles présentant une AFPE.

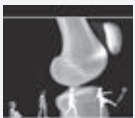
Au point de vue étiologique, l'AFPE s'est développée sur deux fractures de rotule et cinq chocs directs sans fracture (entre 4 et 29 ans avant) concernant 4 femmes et 3 hommes sans cas bilatéral.

L'âge de l'intervention est très variable **entre 28 et 74 ans ???**

Le geste est dans ces cas un ELMSLIE-MAQUET six fois et un ELMSLIE isolé une fois avec patellectomie externe 5 fois.

Le résultat est sur le plan fonctionnel réservé (un bon, un moyen, cinq mauvais) mais trois résultats mauvais améliorent leur niveau d'activité ce qui explique un bon taux de satisfaction (un seul étant déçu).

Le résultat anatomique n'est pas probant sur les vues axiales (angle de congruence légèrement amélioré dans six cas mais pincement maintenu).



La Patella

La patella 25 ans plus tard…
Ce livre regroupe toutes les conférences faites au cours du congrès des 15èmes Journées Lyonnaises de Chirurgie du Genou intitulé « la Patella ». Ces articles font le point sur l'évolution du « menu à la carte » 25 ans plus tard à Lyon mais aussi à l'international. Il regroupe aussi une sélection d'articles « originaux » publiés en 1987 qui ont fait la base du démembrement de la pathologie fémoro-patellaire sur la scène internationale.

The Patella 25 years later…
This textbook contains all the conferences presented during the 15th Journées Lyonnaises de Chirurgie du Genou entitled « la Patella ». These articles state the position of the Lyon's team and the international faculty 25 years after the first publication of the "menu à la carte". There is also a collection of the "original publications" done in 1987, which have led to the basis and the identification of the main factors of patellofemoral disorders on the international scene.

L'ALRM and his president David DEJOUR.

ALRM's Team:

AIT SI SELMI Tarik
BADET Roger
BARTH Johannes
BONIN Nicolas
BONNIN Michel
BUISSON Laurent
BUSSIERE Christophe
CHAMBAT Pierre
CHOL Christophe
DEMEY Guillaume
DEROCHE Philippe
DEJOUR David
DESCHAMPS Gérard
FAYARD Jean Marie
LUSTIG Sébastien
NEYRET Philippe
PANISSET Jean Claude
PERNIN Jérome
PINAROLI Alban
SERVIEN Elvire
SONNERY-COTTET Bertrand
THAUNAT Mathieu
TROUILLET Franck

www.livres-medicaux.com