

# JOURNÉES de la SOFOP



SoFOP  
SOCIÉTÉ  
FRANÇAISE  
D'ORTHOPÉDIE  
PÉDIATRIQUE

*Séminaires médical et recherche*



## Enfant & Sport

[www.sofop-les-seminaires.org](http://www.sofop-les-seminaires.org)

**MARCH 25-26, 2021**

**VIRTUAL  
MEETING**

## CHILDREN & SPORT

## ENGLISH VERSION



Organisation générale : MCO Congrès - Villa Gaby - 285 Corniche JF. Kennedy - 13007 Marseille  
Tél. : (+33)4 95 09 38 00

# **CHILDREN & SPORT**

# CHILDREN & SPORT

Directed by:  
Franck Launay and Jean-Luc Jouve





## **SOFOP BUREAU**

<b>Président</b>	Jérôme SALES DE GAUZY
<b>1<sup>er</sup> Vice-Président</b>	Bruno DOHIN
<b>2<sup>e</sup> Vice-Président</b>	Pierre JOURNEAU
<b>Ancien Président</b>	Jean-Luc JOUVE
<b>Secrétaire Général</b>	Franck LAUNAY
<b>Trésorier</b>	Franck ACCADBLED
<b>Membres du bureau</b>	Vincent CUNIN
	Aurélien COURVOISIER
	Pierre-Louis DOCQUIER
	Yan LEFEVRE
	Alice FASSIER
	Sébastien PESENTI
<b>Webmaster</b>	Aurélien COURVOISIER

# List of authors

## **F. Accadbled**

Service d'orthopédie pédiatrique de l'hôpital des Enfants du CHU de Bordeaux  
Service d'orthopédie pédiatrique  
de l'Hôpital Purpan-CHU de Toulouse

## **F. Alkar**

Service de chirurgie infantile - CHU de Montpellier  
Service de MPR - Institut Saint-Pierre - Palavas les flots

## **E. Alladot**

Centre Universitaire de Médecine du Sport  
et Activité Physique Adaptée du C.H.R.U. de Nancy  
Allée du Morvan. 54511 Vandœuvre-lès-Nancy  
EA DevAH - Département de Physiologie  
Faculté de Médecine de Nancy - Université de Lorraine  
9, avenue de la Forêt de Haye. 54505 Vandœuvre-lès-Nancy

## **M. Bolzinger**

Service d'Orthopédie Pédiatrique, Hôpital des Enfants,  
CHU de Toulouse - 330 avenue de Grande-Bretagne  
31026 Toulouse cedex

## **A. Chalopin**

Nantes, France

## **K. Chaumoitre**

Service d'Imagerie Médicale - Hôpital Nord - Pôle  
d'Imagerie Assistance Publique - Hôpitaux de Marseille  
Aix Marseille Univ, CNRS, EFS, ADES, Marseille, France

## **B. Chenuel**

Centre Universitaire de Médecine du Sport  
et Activité Physique Adaptée du C.H.R.U. de Nancy  
Allée du Morvan. 54511 Vandœuvre-lès-Nancy  
EA DevAH - Département de Physiologie  
Faculté de Médecine de Nancy - Université de Lorraine  
9 avenue de la Forêt de Haye  
54505 Vandœuvre-lès-Nancy

## **F. Chotel**

Orthopédie Pédiatrique, Cliniques Universitaires  
UCL Saint-Luc, 10 avenue Hippocrate, 1200 Bruxelles  
- Grand Hôpital de Charleroi, 3 Grand'Rue, 6000  
Charleroi, Belgique  
Département de Chirurgie Orthopédique Pédiatrique,  
Hôpital Femme Mère Enfant  
59 boulevard Pinel - 69699 Bron

## **E. Choufani**

AP-HM, Service d'Orthopédie Pédiatrique, Hôpital  
Timone Enfants, 264 rue St Pierre, 13005 Marseille,  
France  
Aix-Marseille Université, Faculté de Médecine,  
27 boulevard Jean Moulin - 13005 Marseille - France

## **R. Compagnon**

Service d'Orthopédie Pédiatrique, Hôpital des Enfants,  
CHU de Toulouse - 330 avenue de Grande-Bretagne  
31026 Toulouse cedex

## **A. Courvoisier**

Service d'orthopédie pédiatrique  
Hôpital Couple-Enfant  
CHU Grenoble Alpes

## **J. Cottalorda**

Service de chirurgie infantile - CHU de Montpellier  
Service de MPR - Institut Saint-Pierre - Palavas les flots

## **B. De Courtivron**

Centre de pédiatrie G de Clocheville, CHRU Tours

## **C. Decante**

Nantes, France

## **M. Delpont**

Service de chirurgie infantile - CHU de Montpellier  
Service de MPR - Institut Saint-Pierre - Palavas les flots

## **C. Desvignes**

Service d'Imagerie Médicale - Hôpital Nord - Pôle  
d'Imagerie Assistance Publique - Hôpitaux de Marseille  
Service de Radiologie Pédiatrique - Hôpital d'Enfants  
Timone - Pôle d'Imagerie

## **H. Ducou Le Pointe**

Service de Radiologie Pédiatrique, Hôpital Armand-  
Trousseau, 26 avenue du Dr A. Netter - 75012 PARIS

## **C. Duran Joya**

Centre Pédiatrique de Médecine Physique et de  
Réadaptation Roquetaillade. 32550 Montegut  
2 Hôpital des Enfants CHU de Toulouse

## **E. Ebermeyer**

Unité rachis - CHU BELLEVUE  
Bd Pasteur 42030 St Etienne cedex 2

# List of authors

## **B. Fraisse**

Service de chirurgie pédiatrique  
CHRU Rennes - Hôpital Sud  
16, Boulevard de Bulgarie - 35203 Rennes Cedex

## **R. Gouron**

Service d'Orthopédie Pédiatrique  
CHU Amiens - Groupe Hospitalier Sud  
F-80054 AMIENS Cedex 1 - France

## **S. Guillard**

Nantes, France

## **P. Guillemot**

Service de médecine du sport  
Centre Hospitalier Universitaire De Rennes  
2 Rue Henri Le Guilloux - 35033 Rennes Cedex

## **A. Hamel**

Nantes, France

## **A. Hays**

HAYS Arnaud Institut des Sciences du Mouvement  
Aix-Marseille Université - France

## **C. Jeandel**

Service de chirurgie infantile - CHU de Montpellier  
Service de MPR - Institut Saint-Pierre - Palavas les flots

## **R-E. Jeantet**

Service de Chirurgie Pédiatrique, CHU de Rennes,  
Hôpital Sud, Boulevard de Bulgarie 35203 Rennes

## **P. Joly Monrigal**

Service de chirurgie infantile - CHU de Montpellier  
Service de MPR - Institut Saint-Pierre - Palavas les flots

## **A. Josse**

Service de chirurgie pédiatrique,  
Centre Hospitalier Universitaire De Rennes - Hôpital Sud  
16, Boulevard de Bulgarie - 35203 Rennes Cedex

## **J-L. Jouve**

AP-HM, Service d'Orthopédie Pédiatrique  
Hôpital Timone-Enfants  
264 rue St Pierre, 13005 Marseille - France  
Aix-Marseille Université, Faculté de Médecine  
27 boulevard Jean Moulin - 13005 Marseille, France

## **S. Krissian**

Centre de pédiatrie G de Clocheville - CHRU Tours

## **A. Lalioui**

Service d'orthopédie pédiatrique de l'hôpital  
des Enfants du CHU de Bordeaux  
Service d'orthopédie pédiatrique de l'Hôpital Purpan-  
CHU de Toulouse

## **F-X. Lambert**

Nantes, France

## **K. Lambot**

Cabinets de radiologie  
17 cours Joseph Thierry - 13001 Marseille  
97 avenue William Booth - 13012 Marseille  
Service de Radiologie - Hôpital Nord  
chemin des Bourrely - 13015 Marseille

## **F. Launay**

AP-HM, Service d'Orthopédie Pédiatrique  
Hôpital Timone-Enfants  
264 rue St Pierre - 13005 Marseille - France  
Aix-Marseille Université - Faculté de Médecine  
27 boulevard Jean Moulin - 13005 Marseille - France

## **C. Le Gall**

Service de Chirurgie Pédiatrique - CHU de Rennes -  
Hôpital Sud Boulevard de Bulgarie - 35203 Rennes

## **Y. Lefèvre**

Service d'orthopédie pédiatrique de l'hôpital  
des Enfants du CHU de Bordeaux  
Service d'orthopédie pédiatrique de l'Hôpital Purpan  
CHU de Toulouse

## **D. Louahem**

Service de chirurgie infantile - CHU de Montpellier  
Service de MPR - Institut Saint-Pierre - Palavas les flots

## **G. Lucas**

Service de chirurgie pédiatrique  
Centre Hospitalier Universitaire De Rennes  
Hôpital Sud - 16 Boulevard de Bulgarie - 35203 Rennes

## **S. Marleix**

Service de chirurgie pédiatrique  
Centre Hospitalier Universitaire De Rennes  
Hôpital Sud - 16 Boulevard de Bulgarie - 35203 Rennes

# List of authors

## **E. Mayrargue**

Nantes, France

## **P. Neagoe**

Service de chirurgie infantile - CHU de Montpellier  
Service de MPR - Institut Saint-Pierre - Palavas les flots

## **Th. Odent**

Centre de pédiatrie G de Clocheville - CHRU Tours

## **M. Panuel**

Service d'Imagerie Médicale - Hôpital Nord  
Pôle d'Imagerie Assistance Publique  
Hôpitaux de Marseille  
Aix Marseille Univ, CNRS, EFS, ADES, Marseille, France

## **K. Patte**

Service de chirurgie infantile - CHU de Montpellier  
Service de MPR - Institut Saint-Pierre - Palavas les flots

## **S. Pesenti**

AP-HM, Service d'Orthopédie Pédiatrique  
Hôpital Timone-Enfants  
264 rue St Pierre - 13005 Marseille - France  
Aix-Marseille Université - Faculté de Médecine  
27 boulevard Jean Moulin - 13005 Marseille - France

## **P. Petit**

Service de Radiologie Pédiatrique - Hôpital d'Enfants  
Timone - Pôle d'Imagerie  
Aix Marseille Université - EA 3279 - Marseille - France

## **J. Porterie**

Centre Pédiatrique de Médecine Physique et de  
Réadaptation Roquetaillade. 32550 Montegut  
Hôpital des Enfants CHU de Toulouse

## **V. Rampal**

Service d'orthopédie infantile  
Hôpitaux Pédiatriques de Nice CHU Lenval  
06000 Nice - France

## **S. Raux**

Orthopédie Pédiatrique - Cliniques Universitaires  
UCL Saint-Luc - 10 avenue Hippocrate, 1200 Bruxelles  
Grand Hôpital de Charleroi  
3 Grand'Rue - 6000 Charleroi - Belgique  
Département de Chirurgie Orthopédique Pédiatrique  
Hôpital Femme Mère Enfant  
59 boulevard Pinel - 69699 Bron

## **A. Safi**

Centre Pédiatrique de Médecine Physique et de  
Réadaptation Roquetaillade. 32550 Montegut  
2 Hôpital des Enfants CHU de Toulouse

## **J. Sales De Gauzy**

Service d'Orthopédie Pédiatrique - Hôpital des Enfants  
CHU de Toulouse. 330 avenue de Grande-Bretagne  
31026 Toulouse cedex

## **P. Unal**

Centre de pédiatrie G de Clocheville - CHRU Tours

## **S. Vandergugten**

Orthopédie Pédiatrique - Cliniques Universitaires  
UCL Saint-Luc - 10 avenue Hippocrate, 1200 Bruxelles  
Grand Hôpital de Charleroi  
3 Grand'Rue - 6000 Charleroi - Belgique

## **P. Violas**

Service de chirurgie pédiatrique,  
Centre Hospitalier Universitaire De Rennes  
Hôpital Sud  
16 Boulevard de Bulgarie - 35203 Rennes Cedex

## **P. Wicart**

Hôpital Necker - Enfants Malades  
149 rue de Sèvres - 75015 Paris  
Université Paris Descartes

## Table of contents

<b>Conservative treatment of juvenile osteochondritis dissecans of the knee</b> V. RAMPAL	9
<b>Conservative management of ligamentous injuries of the knee</b> B. FRAISSE, G. LUCAS, S. MARLEIX, A. JOSSE, P. VIOLAS, P. GUILLEMOT	16
<b>Pathogenesis, diagnosis and management of spondylolysis and mild spondylolisthesis in athletic children and adolescents</b> P. VIOLAS, G. LUCAS, A. JOSSE, S. MARLEIX, B. FRAISSE, P. GUILLEMOT, C. LE GALL, R.E. JEANTET	22
<b>Conservative management of spondylolysis and spondylolisthesis in children and adolescents</b> E. EBERMEYER	30
<b>Physical conditioning for the prevention of overuse injuries in children</b> A.HAYS	44
<b>Surgical treatment of ligamentous injuries of the knee in children</b> E. CHOUFANI, S. PESENTI, F. LAUNAY, J-L. JOUVE	47
<b>Osteochondritis dissecans of the knee : pathophysiology and contributing factors</b> B. DE COURTIVRON, P. UNAL, S. KRISIAN, Th. ODENT	57
<b>Meniscal injuries in athletic children</b> S. VANDERGUGTEN, S. RAUX, F. CHOTEL	65
<b>Radiographic exploration in athletic children</b> H. DUCOU LE POINTE	81
<b>Radiographic diagnosis of osteochondritis of the knee</b> K. LAMBOT	91
<b>Is there a place for surgical management in adolescents with Scheuermann's disease?</b> F.X. LAMBERT, C. DECANTE, E. MAYRARGUE, S. GUILLARD, A. CHALOPIN, A. HAMEL	99
<b>Treatment of Scheuermann's disease</b> M. DELPONT, P. JOLY MONRIGAL, P. NEAGOE, D. LOUAHEM, C. JEANDEL, F. ALKAR, K. PATTE, J. COTTALORDA	110
<b>Spondylolysis and spondylolisthesis: operative treatment</b> R. COMPAGNON, M. BOLZINGER, J. SALES DE GAUZY	122
<b>Scheuermann's kyphosis : etiology and diagnosis</b> P. WICART	132
<b>Imaging of meniscal and ligamentous injuries of the knee in children</b> M. PANUEL, C. DESVIGNES, K. CHAUMOITRE, P. PETIT	139
<b>Overuse injuries in children</b> A.COURVOISIER	151
<b>Treatment of overuse injuries in children</b> R. GOURON	157
<b>Medical eligibility to participate in high-performance sports in children and adolescents</b> E. ALLADO, B. CHENUUEL	167
<b>Disability and sports</b> J. PORTERIE, A. SAFI, C. DURAN JOYA, J. SALES DE GAUZY	179
<b>Surgical management of osteochondritis dissecans of the knee</b> Y. LEFEVRE, A. LALIOUI, F. ACCADBLEL	184

# Conservative treatment of juvenile osteochondritis dissecans of the knee

Dr Virginie Rampal MD, PhD

Department of Pediatric Orthopaedic Surgery  
L'Enval University Children's Hospital  
06000 Nice, France

## 1. Definition

Osteochondritis dissecans (OCD) of the knee is defined as necrosis of the subchondral bone leading to lesions at the level of the articular cartilage. The juvenile form of osteochondritis dissecans (JOCD) appears in children [1,2].

The etiology of this pathology is thought to be multifactorial and multiple potential causes have been evoked (vascular, mechanical, or developmental mechanisms). It is important to note that the most frequently affected location (posterolateral aspect of the medial femoral condyle) is due to the load-bearing nature of the area, where there is maximal load. Osteochondral lesions at the level of the tibial spine may be due to significant traction [3].

Good outcomes of conservative treatment and cases with complete spontaneous resolution have been reported dating back 50 years ago [4-6]. Some cases of rapid spontaneous healing have also been reported: Only patients suffering from lingering and refractory pain for many months tend to seek medical care [7].

## 2. Type of treatment [8,9]

A panoply of conservative treatment modalities is available in the literature.

### 2.1. Medical treatment

Medical treatment has not shown to be effective in the management of JOCD [10].

### 2.2. Conservative treatment

#### 2.2.1 Reduced physical activity

Patients diagnosed with JOCD are primarily managed by a global reduction in physical activity, especially with a cessation of all contact sports, running, jumping, squatting, and prolonged standing positions [3,9,12].

#### 2.2.2 Physical therapy

Physical therapy may be useful as a complementary treatment modality and should be considered mainly for muscular strengthening. Iontophoresis (the use of continuous galvanic

currents which may cause burns or injury to the physes in children) or shock-wave therapy have not shown to be effective in the treatment of JOCD and are not recommended in children.

### 2.2.3 Limb unloading [9]

Unloading of the limb is generally indicated with either partial weight-bearing with the use of crutches, or non-weight-bearing with the use of a wheelchair.

Some authors recommend immobilization by casting or the use of an unloader brace with either a varus or a valgus deviation depending on the location of the osteochondral lesion [1,10]. The duration of the immobilization is usually 6 to 12 weeks and depends on the radiographic progression of the lesion. The use of unloading orthoses remains controversial as significant improvements in outcomes have yet to be shown [13].

## 3. Duration of treatment

The literature shows favorable results following conservative treatment for a duration ranging between 3 months to 2 years. A reduction in physical activity for a minimum of 6 to 12 months should be attempted before conclusions are made on the effectiveness of conservative treatment [10,12]. Frequent radiographic and clinical follow-up (every 6 to 8 weeks) are generally the norm and allow for a surveillance of the progression of the lesion, thereby guiding treatment. A follow-up MRI 4 to 6 months after initiation of treatment may be indicated. A reduction of at least 15% of the size of the lesion and a decrease in signal intensity in the bone surrounding the lesion indicate progression toward healing [10]. A gradual return to normal activity may thus be allowed, starting with certain activities with low impact on the knees (such as biking, swimming or walking).

Kocher et al. described a 3-phase protocol for the conservative treatment of JOCD [14]:

- Phase 1 consists of knee immobilization for a period of 4 to 6 weeks, partial weight-bearing and crutches. At the end of this stage, the knee should be pain-free.
- During phase 2 (weeks 6 to 12), brace-free weight bearing may be allowed. Physical therapy is usually initiated at this stage in order to regain range of motion and muscle conditioning.
- Phase 3 begins after 12 weeks when signs of clinical and radiographic healing begin to appear. This consists of a progressive return to sports after a follow-up MRI has been obtained.

## 4. Outcome assessment

A study conducted in 1999 by the French Society of Pediatric Orthopedics (SOFOP) defined the different outcomes of treatment of JOCD [8].

- The knee is said to be normal when it is free of pain, tenderness, and swelling. The lesion, as visualized on radiographs, must have either decreased in size or have

disappeared, without any evidence of intraarticular loose bodies or secondary osteoarthritis.

- The knee is said to be nearly normal if there is minimal pain or tenderness, with a persistent lesion on follow-up radiographs, without any cartilaginous damage.
- The knee is said to be abnormal if there is marked pain, gross joint effusion, a lesion >20mm in diameter, marked sclerosis, intraarticular loose body, or osteoarthritis.

Although radiographs are the primary diagnostic tool [11], an MRI of the knee is considered the mainstay in diagnosis as it allows a thorough assessment of the interface between the osteochondral fragment and the underlying bone [15,16].

Kramer's MRI-based grading system [17] (Table 1) has shown a high correlation to Guhl's arthroscopic grading system [18] (Table 2).

## 5. Indications

Even though many treatment modalities have been described for the management of JOCD, conservative treatment remains the preferred method.

### 5.1. Patient-dependent factors:

- Age, especially skeletal age: In patients with an open distal femoral physis, conservative treatment should be the preferred treatment modality [1,8,15,19,20,21]. Imminent closure of the physes (within 6 months of the initiation of management) is thought to be a factor of poor prognosis [21].
- Compliance of the patient, parent and surgeon to the treatment plan is indispensable [1,9,21], especially since cessation of physical activity for a minimum of 1 year may be extremely difficult for child athletes, especially elite athletes, to abide by. However, family members should be properly educated on the fact that conservative treatment often confers better outcomes than surgery, and that the duration of cessation of sports is not reduced by surgical intervention [21].
- The presence of functional signs (swelling, blocking) [9], which represent independent risk factors for the failure of functional treatment [10].
- Concomitant ipsilateral discoid meniscus [9,20].
- Delay between the start of symptoms and diagnosis (less or more than 6 months) [9,20].
- Body mass index [9].

### 5.2. Lesion-specific factors at the time of initiation of treatment:

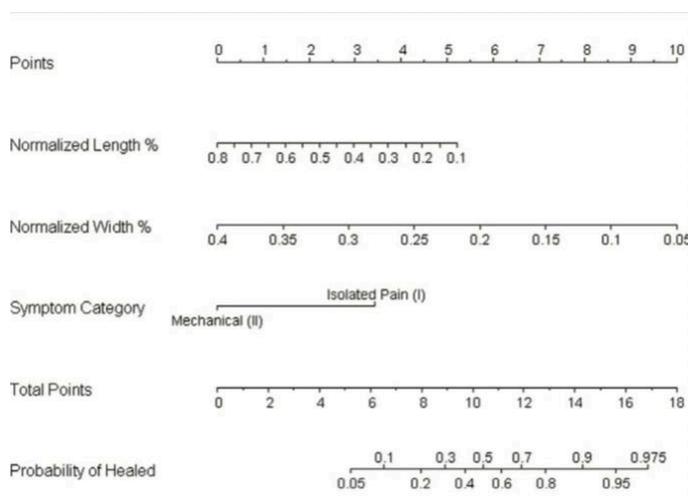
- Lesion stability, or absence of tears of the articular cartilage (as evaluated on T1-weighted images in all 3 planes) [1,8,15, 20, 21]: Stability of the lesion is one of the primary factors dictating the expected outcomes after conservative treatment. In one multicentric study, poor outcomes of conservative treatment were found in 25% of all patients. However, at the moment of initiation of treatment, only 10% of the patients who had favorable expected outcomes and 45% of those who had unfavorable expected outcomes, ended up with unfavorable outcomes [8].

The same study also showed that, when the expected outcome was favorable, conservative treatment showed better results compared than those treated surgically (10% vs 25% of abnormal knees, respectively). Inversely, when the expected outcome was unfavorable, surgical treatment yielded superior results (33% vs 44% of abnormal knees, respectively) [8].

The International Cartilage Repair Society (ICRS) classification [22] categorizes 4 different groups with decreasing lesion stability (I to IV). From this classification, groups I and II have shown up to 78% of favorable outcomes when treated conservatively [23].

- Location of the lesion: The classic location (Intercondylar notch of the medial condyle) is a factor of good prognosis compared to other locations [7-9]. Patellar lesions generally carry the worst prognosis [9].
- Size of the lesion: although no clear consensus exists between authors on threshold values, the values cited by most studies would be around 12mm [20,21].

Nevertheless, MRI data should not be considered in isolation as an indication for surgical treatment [24-28] due to the high sensitivity but low specificity of this imaging modality [10,28]. By combining both clinical and imaging data, in 2018, Wall et al. [1] proposed an algorithm predicting the probability of healing of a given lesion, with excellent intra- and inter-rater reliability [29].



The size of the lesion is therefore a primary prognostic factor predicting the progression of the lesion. The normalized size of the lesion along with the importance of clinical symptoms may predict the probability of healing. The equation relies on the size of the lesion on T1-weighted images in both the coronal and sagittal planes, which is then normalized according to both the maximal width and length of the femoral condyles. Cut-off values were reported in this article as being between 200 and 290 mm<sup>2</sup>.

## 6. Results

No significant differences have been reported in terms of final outcome between the different methods of conservative treatment [8]. Over 50% of lesions treated conservatively seem to heal within the first year [30], with some authors reporting healing rates of over 90% [31].

The primary goal of treatment is the relief of symptoms [7] with the use of crutches if necessary, and by respecting the 1-year period of sports cessation [32].

## 7. Conclusion

Conservative treatment in patients with JOCD is indicated in patients with:

- Open physes
- Classic locations (medial femoral condyle)
- Limited size of the lesion
- Fragment still attached to underlying bone (ICRS I or II)

Sports cessation, eventually accompanied by unloading of the limb (crutches) thereby leading to a pain-free life generally leads, within 1 year, to complete healing of the lesion [2,9,18].

Prematurely resorting to surgical fixation of the osteochondral fragment, especially in the absence of functional signs, must be avoided, even in cases where there is sequestration of the osteochondral lesion. In fact, the sequestered fragment may eventually reincorporate underlying bone [3], thereby transforming from an isolated lacunar image to a sequestrum that ends up progressively incorporating.

The primary issue remains the determination of the stability of the lesion. Actual classifications based on MRI do not allow for the accurate determination of lesion stability.

## References

1. Wall EJ, Vourazeris J, Myer GD, Emery KH, Divine JG, Nick TG, Hewett TE. The healing potential of stable juvenile osteochondritis dissecans knee lesions. *J Bone Joint Surg Am.* 2008 Dec;90(12):2655-64. doi: 10.2106/JBJS.G.01103.
2. Bruns J, Werner M, Habermann C. Osteochondritis Dissecans: Etiology, Pathology, and Imaging with a Special Focus on the Knee Joint. *Cartilage.* 2018 Oct;9(4):346-362. doi: 10.1177/1947603517715736. Epub 2017 Jun 22. Review.
3. Cahuzac JP, Mansat C, Clément JL, Pasquie M, Gaubert J. [The natural history of osteochondritis dissecans of the knee in children]. [Article in French] *Rev Chir Orthop Reparatrice Appar Mot.* 1988;74 Suppl 2:121-4.
4. Green WT, Banks HH. Osteochondritis dissecans in children *J Bone Joint Surg.* 1953 ; 35-A :26-47
5. Van Demark RE. Osteochondritis dissecans with spontaneous healing. *J Bone Joint Surg.* 1952 ; 34-A : 143-48
6. Clanton TO, DeLee JC. Osteochondritis dissecans. History, pathophysiology and current treatment concepts. *Clin Orthop Relat Res.* 1982 Jul;(167):50-64.
7. Cahill BR, Phillips MR, Navarro R. The results of conservative management of juvenile osteochondritis dissecans using joint scintigraphy. A prospective study. *Am J Sports Med.* 1989 Sep-Oct;17(5):601-5; discussion 605-6.
8. Hefti F, Beguiristain J, Krauspe R, Möller- Madsen B, Riccio V, Tschauner C, Wetzel R, Zeller R. Osteochondritis dissecans: a multicenter study of the European Pediatric Orthopedic Society. *J Pediatr Orthop B.* 1999 Oct;8(4):231-45

9. Andriolo L, Candrian C, Papio T, Cavicchioli A, Perdisa F, Filardo G Osteochondritis Dissecans of the Knee - Conservative Treatment Strategies: A Systematic Review..Cartilage. 2019 Jul;10(3):267-277. doi: 10.1177/1947603518758435. Epub 2018 Feb 22.
10. Yang JS, Bogunovic L, Wright RW. Nonoperative treatment of osteochondritis dissecans of the knee. Clin Sports Med. 2014; 33: 295-304
11. Nepple JJ, Milewski MD, Shea KG. Research in osteochondritis dissecans of the knee : 2016 update. J Knee Surg. 2016 ; 29 : 533-38
12. Jones MH, Williams AM. Osteochondritis dissecans of the knee: a practical guide for surgeons. J Bone Joint Surg. 2016: 98-B: 723-9
13. Tepolt FA, Kalish LA, Heyworth BE, Kocher MS. Non operative treatment of stable juvenile osteochondritis dissecans of the knee: effectiveness of unloader bracing. J Pediatr Orthop (B) 2019: 00-00
14. Kocher MS, Tucker R, Ganley TJ, Flynn JM. Management of osteochondritis dissecans of the knee: current concepts review. Am J Sports Med. 2006; 34: 1181-91
15. Jürgensen I, Bachmann G, Schleicher I, Haas H Arthroscopic versus conservative treatment of osteochondritis dissecans of the knee: value of magnetic resonance imaging in therapy planning and follow-up..Arthroscopy. 2002 Apr;18(4):378-86.
16. De Smet AA, Ilahi OA, Graf BK. Untreated osteochondritis of the femoral condyles: prediction of patient outcome using radiographic and PR findings. Skeletal radiol 1997; 26:463-7
17. Kramer J, Stiglbauer R, Engel A et al. MR contrast arthrography in osteochondrosis dissecans. J Comput Assist Tomogr. 1992; 16: 254-260
18. Guhl JF. Arthroscopic treatment of osteochondritis dissecans. Clin Orthop Relat Res. 1982; 167: 65-74
19. Green JP. Osteochondritis dissecans of the knee. J Bone Joint Surg. 48(B): 1966:82-91
20. Nakayama H, Iseki T, Kambara S, Yoshiya S. Analysis of risk factors for poor prognosis in conservatively managed juvenile osteochondritis dissecans of the lateral femoral condyle. Knee. 2016; 23: 950-54
21. Cahill BR, Ahten SM. The three critical components in the conservative treatment of juvenile osteochondritis dissecans (JOCD). Physician, parent, and child.Clin Sports Med. 2001 Apr;20(2):287-98, vi.
22. Brittberg M, Winalski CS. Evaluation of cartilage injuries and repair. J Bone Joint Surg. 2003 ; 85-A : 58-69
23. Ananthaharan A, Randsborg PH. Epidemiology and patient-reported outcome after juvenile osteochondritis dissecans in the knee. Knee. 2018 Aug;25(4):595-601.
24. Haeri Hendy S, de Sa D, Ainsworth K, Ayeni OR, Simunovic N, Peterson D. Juvenile Osteochondritis Dissecans of the Knee: Does Magnetic Resonance Imaging Instability Correlate With the Need for Surgical Intervention?.Orthop J Sports Med. 2017 Nov 14;5(11):2325967117738516.
25. Kijowski R, Blankenbaker D, Shinki K, et al. Juvenile versus adult osteochondritis dissecans of the knee: appropriate MR imaging criteria for instability. Radiology. 2008; 248: 571-78
26. O'Connor M, Palaniappan M, Khan N, et AL; Osteochondritis dissecans of the knee in children: a comparison of MRI and arthroscopic findings. J Bone Joint Surg. 2002; 84: 258-62
27. Samora WP, Chevillet J, Adler B, et al. Juvenile osteochondritis dissecans of the knee: predictors of lesion stability. J Pediatr Orthop. 2012; 32:1-4
28. Chen C, Liu Y, Chou P, et al. MR grading system of osteochondritis dissecans lesions: comparison with arthroscopy. Eur J Radiol. 2013; 82: 518-25

29. Uppstrom TJ, Haskel JD, Gausden EB, Meyer R, Shin YW, Nguyen JT, Green DW. Reliability of predictive models for non-operative healing potential of stable juvenile osteochondritis dissecans knee lesions. *Knee*. 2016 Aug;23(4):698-701.
30. Masquijo J, Kothari A. Juvenile osteochondritis dissecans (JOCD) of the knee: current concepts review. *EFORT Open Rev*. 2019 May 17;4(5):201-212. doi: 10.1302/2058-5241.4.180079. eCollection 2019 May
31. Sales de Gauzy J, Mansat C, Darodes P, et al. Natural course of osteochondritis dissecans in children. *J Pediatr Orthop (B)*. 1999 ; 8 : 26-8
32. Cepero S, Ullot R, Sastre S. Osteochondritis of the femoral condyles in children and adolescents: our experience over the last 28 years. *J Pediatr Orthop B*. 2005 Jan;14(1):24-9.

Table 1: Kramer's MRI stages [17]

MRI Stages	Definition
I	Hypointense T1-weighted signals, irregularly outlined subchondral bone
II	Hypointense T1- and T2-weighted signals, clear delimitation of the lesion
III	Hypointense T1-, moderate hyperintense T2-weighted signals
IV	Hypointense T1-, evident hyperintense T2-weighted signals
V	Loose body within joint

Table 2: Guhl's arthroscopic stages [18]

Arthroscopic stages	Definition
I	Irregular and softened cartilage , no visible fragment
II	Breached articular cartilage, non-displaceable fragment
III	Breached articular cartilage, displaceable but still partially attached fragment
IV	Loose body within joint

# Conservative management of ligamentous injuries of the knee

Dr Bernard Fraisse\*, Dr Grégory Lucas\*, Dr Sylvette Marleix\*,  
Dr Antoine Josse\*, Pr Philippe Violas\*, Dr Pierrick Guillemot\*\*

\* Department of Pediatric Orthopaedic Surgery,  
Rennes University Hospital: South hospital – 16 Boulevard de Bulgarie – 35203 Rennes CEDEX

\*\* Department of Sports Medecine,  
Rennes University Hospital – 2 Rue Henri Le Guilloux – 35033 Rennex CEDEX

## Introduction

The incidence of anterior cruciate ligament (ACL) injury in children older than 10 years of age is constantly increasing [1].

The management of an isolated ACL rupture in children must necessarily meet 3 objectives:

- The first is to restore the stability and proper function of the knee in order to lead a satisfactory life [2,3].
- The second is to reduce the risk of secondary meniscal and cartilaginous lesions in order to limit the risk of osteoarthritis [2].
- Finally, the risk of growth disorders must imperatively be limited [4,5,6].

Conservative treatment partially meets these objectives and is therefore an integral part of the available therapeutic arsenal.

Conservative treatment is actually an entirely separate entity that contains its own strict rules. Unfortunately, conservative management is frequently mistaken for lack of treatment.

Literature on this subject is relative sparse. In 2006, a literature review by Mahtadi et al. [4] found only seven studies on this subject, none of which were conducted on skeletally immature patients. As a result, the treatment plan suggested in this literature review remained vague and unclear. It was not before 2012 that Moksnes et al. [7] published their work on the principles of conservative treatment of ACL injuries in children.

## 1- Prerequisites: Recapitulation and definitions regarding rehabilitation

Conservative treatment requires strict cooperation between the patient, the physiotherapist, and the surgeon. It is therefore imperative that orthopedic surgeons be well versed in the concepts of rehabilitation medicine.

Muscle rehabilitation can be undergone in either an open or closed kinetic chain (OKC and CKC, respectively), and in either a dynamic (concentric or eccentric) or static (isometric) mode.

Dynamic exercises entail either a shortening or lengthening of the muscle fibers thereby provoking segmental movements of the limb: during concentric contraction, the muscle fibers shorten; during eccentric contraction, muscle fibers lengthen as the muscle is contracted thereby opposing the movement.

Static or isometric exercises entail muscle contractions against resistance without any effective muscular movement.

During OKC exercises of the lower limb, the foot is generally free and mobile compared to the knee. During CKC, the foot pushes against a support (e.g. the floor) and the knee is mobile compared to the foot.

Plyometrics are a group of bodybuilding exercises that focalize on striated skeletal muscle tonicity. They typically include certain activities such as sprinting, sudden changes in direction, and jumping as high as possible without momentum.

Isokinetic exercises, from the Greek iso- (ἴσος), signifying equal, and kinetic which relates to motion, are movements which may or may not be assisted by a machine that produce a constant speed.

## **2- Description of conservative treatment according to Moksnes et al. [7]**

Following an ACL rupture in children, the state of the meniscus will guide treatment.

When the ruptured ACL is associated with a meniscal tear that requires surgical management, or if the patient complains of significant instability of the knee, then operative treatment should be considered.

When there is no concomitant meniscal injury, then conservative treatment could be considered. In such patients, treatment algorithms generally recommend attempting conservative until maturity of the growth plate is achieved.

However, conservative treatment entails a certain number of conditions and requires strict cooperation between the patient, the physiotherapist, and the surgeon:

- Modification or restriction of certain physical activities and sports.
- A specific rehabilitation program.
- Custom-made articulated knee brace wear during physical exercise.
- Routine clinical follow-ups and MRIs. In our practice, an MRI is performed every six months along with a multidisciplinary follow-up with a sports physician and a pediatric orthopedic surgeon.

In patients who fail conservative treatment, surgical management should be considered [Fig.1]. Failed functional treatment is defined by the occurrence of a secondary meniscal lesion or the persistence of knee instability. In children, post-operative rehabilitation programs are generally less invasive than in adults and return to sports should be delayed, with recent trends moving towards even more prolonged restrictions of activity [8].

After initial urgent management with arthrocentesis, icing, and immobilization (in our experience with an adjustable articulated knee brace), the rehabilitation program takes place in 4 different phases and must be instated promptly. Each step of the program must be validated before proceeding to the next phase.

Consistent wear of the articulated knee brace is imperative during the entire duration of the rehabilitation program. There are no ideal knee braces and we recommend the use of knee braces with which the department is most accustomed.

### **Phase 1: Preparation and arthrocentesis (approximately six weeks)**

During the acute phase, the primary objectives are:

- Restoring active and passive knee extension (recurvatum and flexion > 120°) and resolving the intraarticular swelling.
- Reactivating the quadriceps and allowing for active locking of the knee
- Allowing for controlled double support with partial weight bearing.
- Obtaining symmetrical gait and sit-to-stand movement patterns.
- Painless participation in activities of daily living (especially going up and down the stairs).
- Initiating cardiovascular training.

*Means: Pool, Treadmill, BOSU Ball/Balance board, and Stairs.*

### **Phase 2: Muscle reinforcement and strengthening (6 to 12 weeks)**

During the 2<sup>nd</sup> phase, the primary objective is normalization of activities of daily living:

- Reinforcing the muscles of the lower limb surrounding the ruptured ACL (Triceps surae; Hamstrings; Quadriceps; Gluteus Medius and Maximus)
- Initiating open kinetic chain exercises from 90 to 45° only.
- Initiating single support proprioception training on stable surfaces.
- Highlighting the ligamentous and trunk compensation strategies during single support (unipedal squat under the supervision of the therapist) or double-leg jumps.
- Maintaining cardiovascular conditioning and optimizing core stability.

*Means: Cycling; Visual feedback; Lunges; Bipedal squats; Core strengthening; Targeted reinforcement*

After the completion of these two phases, objective isokinetic and laxity testing may be undertaken in order to assess muscle recuperation and knee stability. Nevertheless, these tests may be delayed until completion of phase three depending on the practices of the department.

### **Phase 3: preparation for running (12 weeks to 4 months)**

The primary goals during this phase include running without feeling of instability or intra-articular swelling, developing single leg hops with proper shock absorption, and avoidance of dynamic knee valgus.

This consists of:

- Controlled open kinetic chain exercises with full range of motion.
- Isolated eccentric exercises.
- Acquisition and optimization of unipedal squatting without utilizing ligamentous or trunk compensatory mechanisms.
- introduction to controlled plyometric exercises, starting with bipedal then unipedal stances.
- Optimization of unipedal proprioception on both stable and unstable surfaces.

*Means: Visual aids; Jumping; Unstable surfaces*

### **Phase 4: Isokinetic testing and return to running (4 to 5 months)**

- Optimization of muscle weakness through isokinetic testing.
- Optimization of proprioception.
- Introduction to controlled lateral hops
- Return to symmetric and economic running (joint stress)
- Quantification of applied forces for a gentle and progressive return to running

*Means: Functional exercises; Workout monitoring; Lateral hops*

### **Preparation for return to sports (5 to 6 months)**

- Multi-directional running with surprise changes in direction
- Functional exercises closely resembling (or simulating) the act of pivoting

*Means : Specific workouts closely resembling (or simulating) the patient's practiced sport*

### **Functional testing and return to workouts specific to patient's practiced sport (6 to 9 months)**

- Optimization of any lingering deficits as seen on functional tests
- Introduction to self-training at 6 months
- Increased workout load
- Introduction to group workouts at 7½ months

Regular MRI follow-ups should be ordered in order to identify any developing meniscal or osteochondral lesions. In fact, meniscal lesions on unstable knees are rarely symptomatic [9].

There is no consensus on the frequency of MRI follow-ups. We recommend the acquisition of an MRI every 6 months.

Failure of functional treatment is defined as the appearance of meniscal lesions or the persistence of knee instability. In case of failure, surgical treatment must be considered.

### **3- What can be expected from conservative treatment?**

The literature on this subject is relatively scarce. The first article published dates back to 1995 by Mizuta et al. [10] in which they argued against conservative treatment.

It was not before 2013 that Moksnes et al. [11] published their results of a series of isolated ACL ruptures treated by conservative treatment. This included a sample of 46 skeletally immature children aged 12 years and younger with isolated ACL ruptures diagnosed by MRI. These patients were followed-up for a minimum of 2 years after the initial incident. 78% of patients did not require surgical treatment; follow-up was carried out at regular intervals with clinical testing. However, 20% of these patients developed secondary meniscal lesions.

In 2018, after a symposium was held by the French Arthroscopy Society (SFA) in Marseille on the topic of ACL ruptures in children, Madelaine et al. [12] published the results of a multicentric study on the conservative treatment of ACL injuries in children. 53 patients were included with a median follow-up of 6.6 years. 21 patients were later operated, resulting in 37% therapeutic failure. Conservative treatment may also have consequences on the menisci, as 15% of patients presented a secondary meniscal lesion, with 1 patient requiring meniscectomy.

Risk factors for the failure of conservative treatment have been investigated with none being definitively identified. It would seem that instability in the first few months following the incident might be a predictor of therapeutic failure. Contrarily, patients who have yet to begin puberty (Tanner 1) seem to be protected. As a result, the onset of puberty seems to be a poor prognostic factor.

Conservative treatment is therefore a separate entity, with an increased risk of secondary cartilaginous and meniscal injury. It requires regular follow-ups in order to discontinue conservative treatment when the knee is considered to be in danger.

However, the risk of re-rupture after ACL reconstruction seems to be higher in children than in adults, and, according to the SFA symposium study in Marseille [13], ranges between 9% at two years and 22% at five years post-operatively. It should be noted that re-ruptures were more frequent with shorter tendon grafts.

## Conclusion

Conservative treatment and surgery are complementary modalities in the management of ACL ruptures in children.

Conservative treatment should not be considered for all patients and is rather reserved for isolated ACL lesions.

This type of management requires the patient's compliance and comprehension.

It would appear that younger patients have better expected outcomes. Management of a ruptured ACL in children is therefore neither "entirely surgical" nor is it "entirely conservative".

For the most part, conservative treatment meets the requirements for the treatment of ACL ruptures in children. If properly undertaken, it can restore the stability and function of the knee, with the running risk of secondary damage to the menisci and the articular cartilage. Nevertheless, one of the advantages of conservative treatment includes the lack of growth disturbances.

The ideal patient would be Tanner 1, without any meniscal tears, with no instability of the knee, and who is capable of adhering to the treatment protocol.

# Pathogenesis, diagnosis and management of spondylolysis and mild spondylolisthesis in athletic children and adolescents

Philippe Violas<sup>1</sup>, Gregory Lucas<sup>1</sup>, Antoine Josse<sup>1</sup>, Sylvette Marleix<sup>1</sup>, Bernard Fraisse<sup>1</sup>, Pierrick Guillemot<sup>2</sup>, Caroline Le Gall<sup>1</sup>, Rose Elisabeth Jeantet<sup>1</sup>

RENNES UNIVERSITY HOSPITAL, DEPARTMENT OF PEDIATRIC SURGERY, F-35033 RENNES, FRANCE

<sup>1</sup> Department of Pediatric Surgery, Rennes University Hospital, South Hospital, Boulevard de Bulgarie, 35203 Rennes

<sup>2</sup> Department of Sports Medicine, Rennes University Hospital, Pontchaillou Hospital, Rue Henri Le Guilloux, 35000 Rennes

## Introduction

During his talk at the 2001 French Society of Orthopedic Surgery & Traumatology (SOFOT) conference on lumbosacral spondylolysis (SL) and spondylolisthesis (SPL) in children and adolescents, Jouve [1] reviewed the historic, anatomic and pathogenic bases of this complex pathology. In 2015, during another conference on the same topic, a literature review of recent developments on the link between this pathology and global spinal sagittal alignment were presented, and the significant and still-present controversies relating to the treatment of SPL, especially in its severe forms, were highlighted [2]. However, the topic of SL was only briefly touched upon. As such, in this chapter, the controversial topic of the hypothetical post-traumatic type of SPL will be discussed, a type that differs from that of severe progressive SPL which is often dysplastic, even though other forms of SPL may be secondary to an initial SL and which are deemed “spondylolytic spondylolisthesis”.

## Definition

Spondylolysis is a defect of the pars interarticularis of the vertebral arch. This can present as either a uni- or bilateral defect, either simultaneously or developing over time. This may also be associated with an SPL. Spondylolisthesis signifies translation (olisthesis) of one vertebral segment (spondylo) over the one directly beneath it. This translation may be either anterior (anterolisthesis) or posterior (retrolisthesis). In children and adolescents, translation is mostly anterior.

The pars lesion is most frequently found at the level of L5 (71-95%), less frequently at the level of L4 (5-23%), and exceptionally at other levels [3-5].

## **Incidence and Pathogenesis**

Similar to SPL, SL is an acquired condition. The most frequently used classification for the categorization of SPL based on the severity of anterior translation of the vertebra is that of Meyerding.

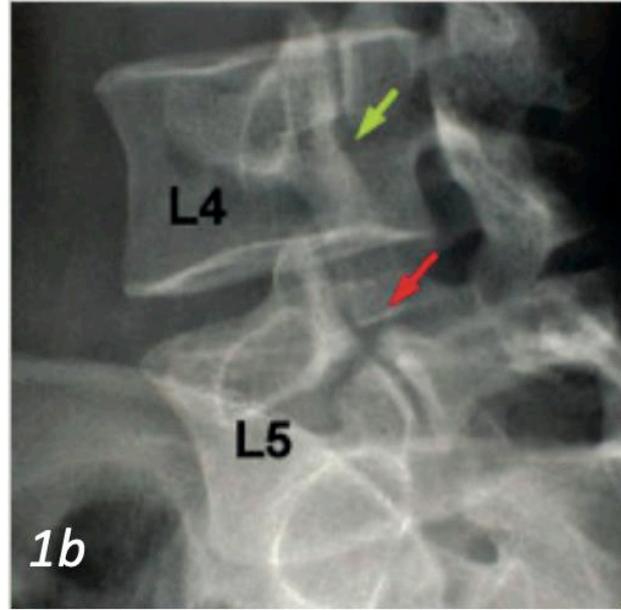
During childhood and adolescence, vertebral bone mass is generally low [6] and the posterior arches continue maturing until 20-25 years of age [7,8]. The increased elasticity of the intervertebral discs during adolescence compounds the stresses placed upon the neural arch, and specifically the pars interarticularis. Consequently, the principal etiologies of this pathology are often believed to be the traumatic and micro-traumatic events that arise secondary to repetitive compressive, torsional, and rotational constraints during certain types of sports in which these violent and cyclical movements are required [9,10]. As a result, even though the global prevalence of SL in athletes seems similar to that of the general population, certain types of sports, such as wrestling (30-35%) and Olympic-style weightlifting (23-30%), may lead to a significantly higher prevalence of SL. In fact, SL is fourfold more frequent in gymnasts compared to the rest of the female population [11].

## **Clinical presentation**

Athletic children with SL are usually asymptomatic, with incidental discoveries on radiographic images being undoubtedly the most frequent presentation. When SL is symptomatic, the patient usually presents with band-like or unilateral low-back-pain, which may or may not be associated with unilateral, bilateral, or alternating radicular pain. This pain is particularly reproduced by hyperextending the lumbar spine. Resumption of sports in spite of the pain might lead to a clinical scenario associating paravertebral muscle spasms, a flattening of lumbar lordosis, functional scoliosis, hamstring tightness, and rarely L5 radiculopathy. In such cases, imaging studies must be obtained in order to confirm the diagnosis and eliminate differential diagnoses.

## **Medical imaging**

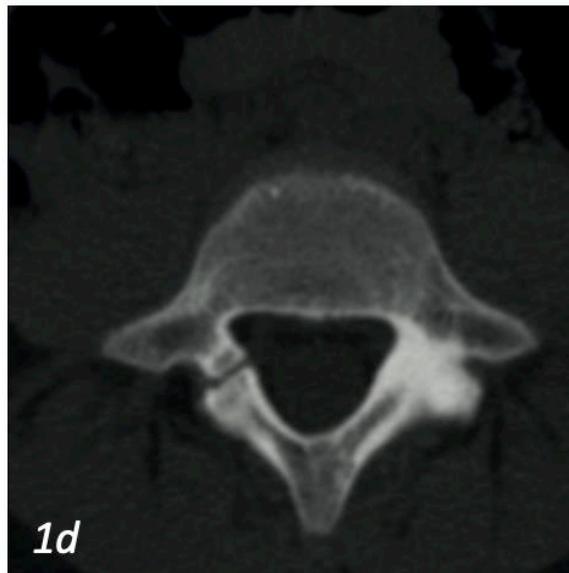
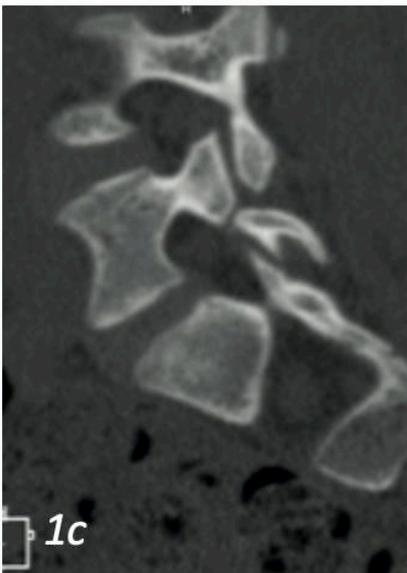
The diagnosis of SL can be made on conventional coronal and lateral radiographs of the lumbar spine, which may be completed with oblique views, ideal for the visualization of pars interarticularis defects.



*Unilateral L5S1 spondylolysis barely visible on the lateral view (1a), more easily recognizable on the oblique view (1b).*

Teleradiographic images of the spine on coronal and sagittal views, realized preferentially with the EOS® system in order to decrease radiation exposure, would complete the global morphological analysis of the spine, especially in the sagittal plane.

Conventional radiographs, and more commonly CT-scans can reveal the unilateral nature of SL, or contralateral sclerosis may sometimes also be identified, which is thought to be secondary to excessive mechanical load on the contralateral isthmus.



*CT-scans confirming the unilateral character of the isthmus lysis (1c) with contralateral sclerosis (1d)*

Conventional radiographs may appear normal initially. However, the best algorithmic approach for diagnostic imaging has yet to be established. Single-photon emission computed tomography (SPECT) is an imaging modality with high sensitivity but low specificity for the diagnosis of SL. CT-scans might serve useful as a complementary imaging modality for the visualization of bony anatomy, sclerosis, and bony healing, but with the inconvenience of high radiation exposure. MRI can be a useful diagnostic and follow-up tool with many advantages compared to conventional imaging modalities, but more data is required in order to assess its potential advantages compared to other techniques.

Therefore, there is actually no consensus regarding the choice of complementary imaging, especially between MRI and CT-scans [12]. West et al. [13] conducted a study with the aim of determining the precision of MRI and CT-scans in young athletes who had previously been diagnosed with SL through the use of SPECT. This cross-sectional study on 22 young athletes ( $14.7 \pm 1.5$  years old) where both an MRI and a CT-scan were obtained on the same day, showed the superiority of CT-scans for the diagnosis of SL. Their results showed 13 true positive (TP) and 9 false negative (FN) results for the MRI, compared to 17 TP and 5 FN results for the CT-scan. The sensitivity and FN rates of the MRI were 59.1% (95% Confidence Interval [95% CI] = 36.7% - 78.5%) and 40.9% (95% CI = 21.5% - 63.3%), respectively. The sensitivity and FN rates of the CT-scans were 77.3% (95% CI = 53.2% - 91.3%) and 22.7% (95% IC = 0.09% - 45.8%), respectively.

## **Treatment**

Initial management usually consists of conservative treatment for a period of several months and is generally effective by itself. Surgical management is usually only indicated after failure of conservative treatment.

### **1. Conservative treatment and the possibility of isthmic consolidation**

Conservative management is the initial treatment modality to be attempted and is generally sufficient. However, debate still exists within the framework of conservative treatment on whether a simple cessation of physical activity would suffice, or the use of a brace would be indicated, with even the choice of type of brace being controversial. Nevertheless, the effectiveness of conservative treatment and the non-requirement of immobilization was highlighted by a meta-analysis conducted by Klein et al. [14]. The primary goal of their study was to identify and summarize the evidence found in the literature on the effectiveness of conservative treatment in SL, including subjects with grade 1 SPL. The minimum follow-up period was that of one year. The results of the included studies were presented following two criteria: clinical outcome or radiographic evidence of consolidation of SL. Fifteen observational studies measuring the clinical outcomes were included, which showed weighted and pooled success rates of 83.9% in 665 patients. Subgroup analysis comparing the clinical outcomes of patients treated with or without immobilization did not show significant differences. Ten studies assessing radiographic healing of SL showed a pooled success rate of 28% (n=847). Subgroup analysis showed that unilateral defects healed with a pooled and weighted rate of 71% (n=92), which was significantly higher than bilateral defects with healing rates of 18.1% (n=446,  $p < 0.001$ ). A supplementary subgroup analysis showed that acute

defects healed at a rate of 68.1% (n=236), which was a significantly higher rate than chronic defects with healing rates of 28.3% (n=224, p<0.001). This meta-analysis of observational studies suggested that clinical results may not correlate well with radiographic evidence of pars defect consolidation. Acute lesions seemed to be more amenable to healing after conservative treatment, as were unilateral defects when compared to bilateral SL.

These conclusions should still be contemplated. It should also be noted that symptomatic patients may be treated more effectively, and sometimes even more quickly, if cessation of physical activity is associated with immobilization with a lumbar support or a short lumbar brace, such as the delordosing spondylogenic Boston brace [15].

The idea of a persistent pars defect, even after clinical improvement, remains widespread. Nonetheless, bony healing and consolidation of the isthmic lysis are possible even without surgery. As such, Sakai et al. [16] recently showed that, depending on the acute or chronic nature of the lesion (analysis by coupling CT-scans and MRI), as well as its uni- or bilateral character, significant and rapid healing may be achieved. For most patients, with the exception of those presenting with chronic isthmic lysis, conservative treatment comprised of rest and the wear of a thoraco-lumbo-sacral orthosis (TLSO).

## **2. Failure of conservative treatment: choosing from the types of surgery**

Isthmic reconstruction may be indicated in patients with SL without concomitant SPL or with only mild translation (grade 1, rarely grade 2). These generally include patients without predictors of severe disease, such as lumbosacral kyphosis. No neurological deficits are usually found, and surgery may be justified after conservative treatment has been attempted for a period of at least twelve months (cessation of physical activity, physiotherapy, lumbar support) without evidence of complete or even partial symptomatic improvement. The absence of anomalies at the level of the intervertebral discs confirmed by MRI is required during pre-operative planning. Otherwise, arthrodesis may be indicated. Isthmic reconstruction is more frequently attempted in patients who are at the end of their growth. This surgical approach preserves the mobility of the spine. The most frequent complication is non-union. An adapted construct is required in order to achieve isthmic healing in compression without bulky osteosynthesis, especially at the level of the subjacent articular mass.

Numerous isthmic reconstruction techniques have been described. Of note, the cerclage wire around the transverse and spinous processes technique of Nicol, temporary butterfly plate of Louis, and Buck's technique of direct fixation of the isthmic lysis. To this end, Bodman et al. [17] analyzed the clinical and radiographic results of Buck's modified fixation technique, in patients with symptomatic SL, with grade 1 SPL and normal L4-L5 and L5-S1 intervertebral discs, after failure of conservative treatment. Functional outcomes were quantified through the use of the Oswestry disability index (ODI). Healing of the pars defect was evaluated by conventional radiographs and CT-scanning. The motion of the L4-L5 and L5-S1 segments was measured on dynamic flexion/extension radiographs. In thirty-five patients with a mean follow-up of ten years, the authors reported excellent functional results in 22 patients and good results in 8 patients, with 5 patients in whom treatment had failed. Consolidation of the defect was found in 91.4% of patients. Other techniques may also be considered and may

entail fixation of the pedicles and laminae, such as Morscher's approach. Some authors have observed correction of theolisthesis in patients in whom isthmic reconstruction was undertaken during the early stages of the disease. Hefti et al. [18] and Preysson [19] noted that, in two and six cases respectively, complete healing of a grade 2 SPL was achieved by the end of growth after isthmic reconstruction was undertaken. Nevertheless, this endpoint does not represent the purpose behind isthmic reconstruction.

## **Are there any recommendations from sports federations?**

No specific recommendations have been put forth on this subject. After interviewing different national sports federations along with their medical staff, certain responses were obtained, such as: In the case of rugby, no specific indications exist for SL or SPL. Temporary contra-indications in relevance to injuries of the spine are transient neurological deficits of 1 to 4 limbs in the absence of exploration (MRI) and specialized opinion, non-operated herniated discs, and lumbar spinal stenosis. Definitive contra-indications are motor deficits due to injury to the spinal cord, confirmed tetra-pyramidal syndrome, three or more episodes of transitory tetra-paresis, severe cervical ligamentous sprains, spinal stenosis without safety margins on MRI, odontoid agenesis or hypoplasia, congenital or surgical fusion of 3 or more levels, intramedullary edema, a true syrinx, and Arnold-Chiari-type malformations of the cervico-occipital junction with occupation of the cisterna magna.

For the French swimming federation (FFN; swimming, diving, water-polo), the French federation for the education of underwater sports (FFESSM; Scuba diving), the French football (soccer) federation (FFF), and the French judo and jiu-jitsu federations, there are no specific indications. Physicians are thereby required to formulate temporary and absolute contra-indications depending on the patient.

In the case of boxing, the only absolute contra-indications found for spinal pathologies were reserved for herniated discs, without more detail being provided.

In the case of motocross, absolute contra-indications included non-consolidated affections that may jeopardize the stability of the spine. This definition remains ambiguous.

For the remainder of sporting activities, especially volleyball, handball, gymnastics, Olympic-style weightlifting, and athletics, absolute contra-indications are noted as being any severe static and/or dynamic morphological affections, particularly at the level of the thoracolumbar spine, running the risk of acute injury or accelerated degeneration. The indications here are also ambiguous.

In summary, there appear to be no clear recommendations by the different sports federations, who would rather leave the decision on the temporary or absolute contra-indications to the physicians. Since sporting federations have not provided recommendations, referring to different scientific societies may be a viable option in the future.

## Conclusion

The initial step in athletic children and adolescents presenting with low-back-pain should be to assess the presence or absence of SL. This approach may prove difficult, as pars interarticularis defects are frequently encountered in the general population, including in children. Imaging should therefore be utilized, with no evidence as to the modality of choice. MRI could be prescribed during the diagnostic workup of acute SL, and CT-scans may rather be useful in evaluating healing of the pars defect after conservative treatment. It would appear that bracing may not be necessary but may sometimes be a complementary means of limiting movement in order to further restrict activity in impatient children and parents, but also their coaches. In fact, the treatment of current or future athletes with SL and/or SPL may be difficult. Before deciding on surgery, one must be wary of any pressure by the patient's family or environment toward the rapid return to competition. Such pressures may lead to harm if a decision to operate has been made, and these motivations must necessarily be taken into account. After the failure of a well-conducted conservative treatment for an arbitrarily set period of one year, surgery may be indicated, although this may be only rarely necessary.

## References

1. Jouve JL. Spondylolyse et spondylolisthésis lombosacré de l'enfant et de l'adolescent. In : Cahiers d'enseignement de la SOFCOT. Paris : Expansion Scientifique Publications ; 2001. p. 171-92.
2. Violas P, Lucas G. L5S1 spondylolisthesis in children and adolescents. *Orthop Traumatol Surg Res.* 2016;102(1 Suppl):S141-7.
3. McCleary MD, Congeni JA. Current concepts in the diagnosis and treatment of spondylolysis in young athletes. *Curr Sports Med Rep;* 2007 6:62–66.
4. Kim HJ, Green DW. Spondylolysis in the adolescent athlete. *Curr Opin Pediatr;* 2011 23:68–72.
5. Tallarico RA, Madom IA, Palumbo MA. Spondylolysis and spondylolisthesis in the athlete. *Sports Med Arthrosc Rev.* 2008;16:32–38.
6. Fournier PE, Rizzoli R, Slosman DO. Asynchrony between the rates of standing height gain and bone mass accumulation during puberty. *Osteoporos Int* 1997;7:525–32
7. Cyron BM, Hutton WC. The fatigue strength of the lumbar neural arch in spondylolysis. *J Bone Joint Surg Br* 1978; 60:234–8.
8. Kim HJ, Green DW. Spondylolysis in the adolescent athlete. *Curr Opin Pediatr* 2011; 23:68–72.
9. Eddy D, Congeni J, Loud K. A review of spine injuries and return to play. *Clin J Sport Med* 2005; 15:453– 458.
10. Letts M, Smallman T, Afanasiev R, Gouw G. Fracture of the pars interarticularis in adolescent athletes: a clinical-biomechanical analysis. *J Pediatr Orthop* 1986; 6:40–46.
11. Stanitski CL. Spondylolysis and Spondylolisthesis in Athletes. *Oper Tech Sports Med* 2006; 14:141–146.
12. Cheung KK, Dhawan RT, Wilson LF, Peirce NS, Rajeswaran G. Pars interarticularis injury in elite athletes - The role of imaging in diagnosis and management Author links open overlay panel *European Journal of Radiology.* 2018; 108:28-42

13. West AM, d'Hemecourt PA, Bono OJ, Micheli LJ, Sugimoto D. Diagnostic Accuracy of Magnetic Resonance Imaging and Computed Tomography Scan in Young Athletes With Spondylolysis. *Clin Pediatr*. 2019 ;58:671-6.
14. Klein G, Mehlman CT, McCarty M. Nonoperative treatment of spondylolysis and grade I spondylolisthesis in children and young adults: a meta-analysis of observational studies. *J Pediatr Orthop*. 2009 ;29:146-56.
15. d'Hemecourt PA1, Zurakowski D, Kriemler S, Micheli LJ. Spondylolysis: returning the athlete to sports participation with brace treatment. *Orthopedics*. 2002 ;25: 653-7.
16. Sakai T, Tezuka F, Yamashita K, Takata Y, Higashino K, Nagamachi A, Sairyo K. Conservative Treatment for Bony Healing in Pediatric Lumbar Spondylolysis. *Spine* 2017 ; 42:E716-E720.
17. de Bodman C, Bergerault F, de Courtivron B, Bonnard C. Lumbo-sacral motion conserved after isthmic reconstruction: long-term results. *J Child Orthop*. 2014; 8: 97-103.
18. Hefti F, Seelig W, Morsher E. Repair of lumbar spondylolysis with a hook-screw. *Int Orthop*, 1992, 16 : 81-5.
19. Preyssas P. Consolidation isthmique selon la technique de Buck modifiée dans les spondylolyses et spondylolisthésis de grade I chez l'enfant et l'adolescent. A propos de 26 cas. thèse médecine, faculté de médecine de Tours, 2000.

# Conservative management of spondylolysis and spondylolisthesis in children and adolescents

Dr Eric Ebermeyer

Spine Department, Bellevue University Hospital, Boulevard Pasteur 42030 St Etienne CEDEX2

## Introduction

The estimated prevalence of spondylolysis in the general population, which may or may not be associated with spondylolisthesis, is approximately 5%. It is more frequently found in young athletes and up to 20 to 30% in certain high-intensity sports.

Patients are infrequently symptomatic in these patients and the incidental diagnosis on conventional radiographs is rather the norm. It is therefore considered as a compensatory mechanism allowing for the maintenance of a satisfactory sagittal alignment. Nevertheless, the diagnosis of spondylolysis or spondylolisthesis requires frequent follow-ups and certain preventive measures must be adopted in order to avoid deterioration.

In general, 80% of children presenting with isthmic lyses are symptomatic, and over 80% of these children are relieved by medical management alone [1,4,5]. **Conservative management and physiotherapy may ensure the stabilization of the lesion and pain relief.**

The clinical semiology of isthmic lysis is typical and reproduceable and allows to make the diagnosis: It is characterized by lumbosacral pain, on either the right or left side, rarely bilateral initially, which is aggravated on exertion, improved with rest, and which may appear either acutely or progressively over weeks. Upon resumption of physical activity, there is typically recurrence of the pain at increasingly lower levels of exertion.

Two factors are generally present: one predisposing factor (such as a sagittal alignment favoring lumbosacral hyperextension), and repetitive spinal microtrauma during growth.



**Figure 1:** *isthmic lysis*

## 1-Definitions

**Spondylolysis:** A defect at the level of the interarticular isthmus (pars interarticularis) without displacement of the vertebra. The isthmus may be drawn out, thinned or condensed, brittle, or broken off, but may also consist of fibrous tissue or progress into pseudarthrosis.



**Figure 2:** non-union

This bony defect is most frequently found at the level of L5. As a result, the sole elements supporting L5 over the sacrum and preventing its translation are the lumbo-sacral intervertebral disk (L5-S1), the paraspinal muscles that are found in a permanently contracted state and are at the origin of pain, the joint capsules, and the ligaments. Spondylolysis may be rarely encountered at L4 or L3.

**Spondylolisthesis:** A bilateral isthmic defect may be accompanied by slippage of L5 over S1. This translation may be measured in percentage or a grade may be attributed depending on the degree of translation relative to the width of the sacral plate, or according to the Meyerding classification by dividing the sacral base into 4 parts: Grade 1 is slippage within the 1<sup>st</sup> quartile, grade 2 within the 2<sup>nd</sup> quartile, until stage 4. When the vertebra is no longer in contact with the sacrum, it is known as spondyloptosis [2].



**Figure 3:** Stage 1 spondylolisthesis over bilateral isthmus lysis

## 2- Clinical diagnosis in the young athlete

- Medical history: The diagnosis may be incidental. In fact, isthmic lysis, which may be bilateral and associated with spondylolisthesis, may take place during growth and non-athletic children may even be pain-free. It is thought to be a compensatory mechanism secondary to fragility of the isthmic region and to the vertical posture which places mechanical strain on this region. As a result, not all patients with spondylolisthesis or spondylolysis are symptomatic. No relationships have been found between severity of the slippage (as visible on radiographs) and functional impact: anatomical and clinical dissociation is one of the characteristics of spondylolisthesis. The diagnosis may also be evoked during painful episodes. Acute low-back-pain is usually secondary to a uni- or bilateral isthmic fracture. Consequently, this fracture is believed to occur as a result of indirect trauma, movements of hyperextension (e.g. gymnastics), or sudden load (e.g. rugby, basketball).

Pain secondary to isthmic lysis or spondylolisthesis has certain characteristics:

- Localized at the lumbo-sacral region and lateralized to one side
- Activated by extension of the spine
- Relieved by rest, especially in the fetal position
- May sometimes irradiate to the gluteal region
- Awakened by physical activity at increasingly earlier phases of a workout usually requiring the child to stop the activity
- After a prolonged period of cessation (at least 15 days), pain reappears after resumption of physical exercise

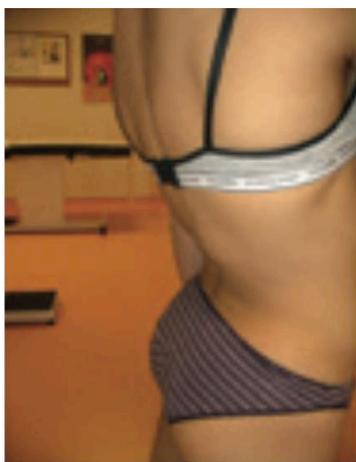
The assessment of sagittal alignment is primordial and may be done with a plumb line allowing for the assessment of the sagittal curves.

Upon palpation of the L5 spinous process, pain may be elicited. Pelvic retroversion can often be found with reactionary distal hyperlordosis and lumbosacral kyphosis. Plumb line assessment may highlight any anterior sagittal imbalance.

Dynamic examination is also important, where the patient is asked to perform flexion and extension of the spine: back pain and stiffness are usually evident especially during painful episodes. Harmony of the curves must be noted: in flexion, the disappearance of the lordosis or its maintenance, in extension the capacity to realize extension of the thoraco-lumbar region or the existence of a break in the lumbo-sacral region (figure 5). Extension of the hip and spine are evaluated in the prone position by passively raising the straightened lower limbs: the presence of pain and the flexibility extension must be noted.



**Figure 4:** *appropriate thoraco-lumbar extension*



**Figure 5:** *distal thoracic hyperkyphosis with disruption of the harmonious lumbo-sacral alignment*

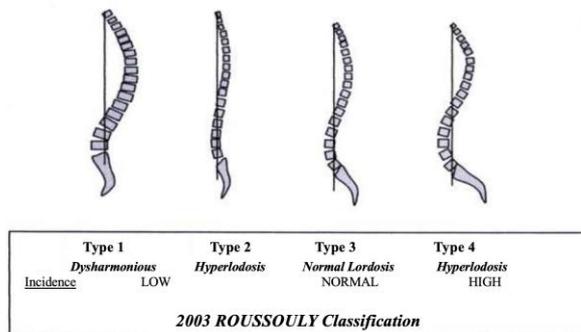
Hamstrings tightness may be identified on physical exam and, in case of advanced spondylolisthesis, pelvic retroversion, lumbar hyperlordosis, lumbosacral kyphosis, hip and knee flexion contracture, and tightness of the triceps surae may also be found.



**Figure 6:** *extension test in prone position*

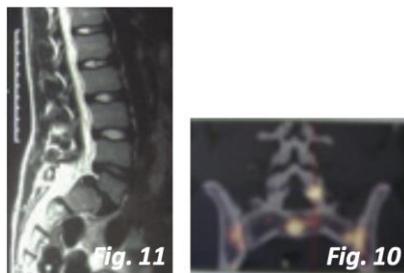
### 3- Radiographic diagnosis

Spondylolisthesis may be diagnosed on conventional radiographs. With the use of the EOS® system, radiation doses may be decreased while maintaining excellent image quality. Based on this imaging modality, the sagittal profile may be classified into one of 4 different types of backs, as per the Roussouly classification [9].



**Figure 7:** Roussouly classification

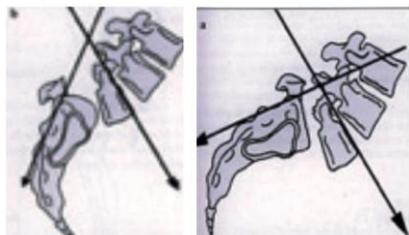
However, isthmic lysis without spondylolisthesis may not always be diagnosed on conventional radiographs. In young athletes, early diagnosis is imperative, and MRI or scintigraphy are more frequently resorted to. CT-scans are generally avoided due to the high doses of radiation but may lead to the diagnosis of isthmic lysis in certain patients in whom the diagnosis is uncertain.



**Figure 11:** intervertebral disc disease above and below the level of spondylolisthesis

**Figure 10:** isthmic lysis as seen on scintigraphy

The angle of lumbosacral kyphosis or slip angle is most frequently measured using the Louis method and is defined as the angle between the tangent to the posterior edge of S1 and the superior plateau of L5. If this angle falls below 90°, the sacrum is considered vertical, and if the lumbosacral kyphosis is superior to 100°, the sacrum is considered horizontal [3].



**Figure 8:** vertical sacrum

**Figure 9:** horizontal sacrum

While assessing the sagittal alignment, pelvic incidence (as described by Duval-Beaupère) and the position of the sacrum are essential for the interpretation of lumbo-sacral sagittal imbalance [8,10].

#### **4-Risk factors**

The configuration of the sagittal curves of the spine plays an important role in spinal pathology. In fact, both high and low pelvic incidence may increase strain on the isthmus by increasing traction or slippage forces anteriorly, or by increasing the shear forces exerted by the L4 articular processes over the L5 isthmus, respectively. Similarly, the abnormal or repetitive shear or compressive forces which are imposed on the spine could have similar consequences: the incidence of spondylolysis increases notably in practitioners of certain types of sports (diving, Olympic-style weightlifting, wrestling, gymnastics, etc.).

Most frequently, the pars defect does not consolidate. This may be simply be an adaptation of the spine to situations where, among others, spinal balance is dictated by the pelvic incidence. During growth, progressive anterior displacement of the vertebra may take place. After skeletal maturity, progressive slippage is rare and may be due to secondary degeneration of the intervertebral disc.

When spondylolysis occurs in the context of low pelvic incidence, secondary dysplasia takes place: the L5 vertebra takes a cuneiform shape and dysplasia of the dome of S1 is observed. When pelvic incidence is higher, dysplasia and displacement are often less significant. Thereby, 3 primary risk factors may be determined:

- Familial: probably genetic, linked to isthmic fragility
- Postural: Global hyperlordosis (Roussouly type 4) or short lumbosacral hyperlordosis (Roussouly type 1)
- Repetitive microtrauma

Two ulterior factors may also be found: On the one hand, anomalies of the lumbosacral junction with partial lumbarization of S1 which could lead to instability of L5 over S1. On the other hand, sacralization of L5 would increase strain at the level of L4, especially if there is highly localized extension at the same level.

A final intervening factor: the growth element. Isthmic lysis with a fragilized vertebral growth plate arising around the time of puberty may rapidly progress if the physéal lesion reaches

the superior endplate of S1. The Example of a high-level gymnast who presented with isthmic lysis in 2007 with progressive erosions of the anterior edge of the S1 endplate is presented in figures 12 to 15.



*Figure 12 to 15: progression of the isthmic lysis between 2004 and 2011*

## **5- Conservative treatment**

It is crucial to underline the fact that the majority of patients respond well to functional or orthopedic treatment and do not require surgical management [1,4,5].

### **5.1 Rehabilitation**

Our experience with gymnasts of the French Pole of St Etienne, a population in whom isthmic lysis and spondylolisthesis are frequently found, allows us to better define the objectives of rehabilitation in these patients.

Said objective is generally to maintain an adapted level of physical activity with little strain on the lumbosacral junction while avoiding hyperextension of the spine.

The basis of rehabilitation relies on the core conditioning of the lumbar region and stretching the hip and thigh muscles. Core conditioning consists of exercises that maintain isometric contraction (i.e. without modifying the length of the muscle fibers) of multiple agonistic and antagonistic muscle groups simultaneously thereby locking many different joints. The patient must remain immobile in a fixed position for a certain amount of time. The muscles reinforced during these exercises are specifically the deep and postural muscles (vertebral and para-vertebral, abdominal, gluteal, hamstrings, etc...).



**Figure 16:** *abdominal muscle conditioning*

**Figure 17:** *Paravertebral muscle reinforcement*

Stretching exercises should concentrate on hip and thigh muscles, particularly the hamstrings, as well as the anterior chain, psoas, and quadriceps. These exercises can be undertaken with the help of a physiotherapist as muscle energy techniques, or by maintaining certain postures for a certain amount of time, such as the Mézière method for global stretching of the posterior chain.

**Obtaining a harmonious relationship** between the sagittal curves is imperative. As a result, rehabilitation must take into account the child's type of back: In patients with a global hyperlordosis with a type 4 back (Roussouly's classification), the objective is to reduce both lordosis and kyphosis through core strengthening, pelvic retroversion, active spinal elongation, and global postural rehabilitation. The Mézière techniques involving certain postures which stretch the posterior chain are particularly useful.



**Figure 18:** *stretching of the posterior chain and reinforcement of paravertebral muscles*

In situations where the lumbar spine is hyperlordotic over a short segment with underlying thoracolumbar kyphosis and a flat back (Roussouly type 1), the objective is to harmonize the lordosis in order to further extend it proximally into the thoracolumbar region and to lock the lumbosacral junction. Reinforcement of the paravertebral muscles in the thoracolumbar region is thus indicated. In high-level young athletes, rehabilitation must take place in collaboration with the trainer. **Modification of body movement during physical activity is indispensable** along with the active locking the lumbosacral region. Eccentric reinforcement of the abdominals in order to better control lumbar extension has been developed specifically for gymnasts along with an increase in flexibility of the shoulders in order to decrease strain on the lumbosacral region during the bridge maneuver.



*Figure 19: Stretching of the thoracolumbar region*



*Figure 20: Gain in extension of the thoracolumbar spine while protecting the lumbosacral region*



*Figure 21: Gain in shoulder extension*



**Figure 22:** *isokinetic exercises*

Isokinetic exercises may be justified for high-level athletes while privileging eccentric reinforcement (especially abdominals for their role of limiting lumbar hyperextension).

## **5-2 Orthopedic management**

Patients who have recently been diagnosed with symptomatic spondylolysis, with or without associated spondylolisthesis, benefit from orthopedic treatment.

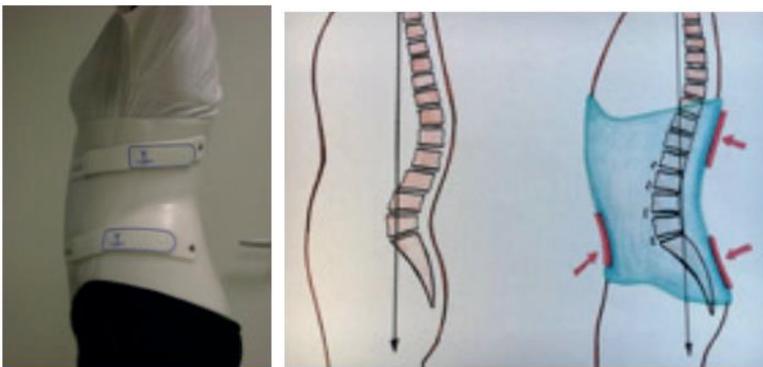
The objectives are to quickly relieve the patient's pain, modify maladapted postures, and finally to ensure continued rehabilitation, which should initially be undergone while wearing a brace.

Three possible orthopedic treatment modalities have been described:

- Initial cast immobilization in order to achieve consolidation of the isthmic lysis. This is generally undertaken for a period of 3 months.
- A brace with unilateral femoral support for the limitation of motion at the lumbosacral region. This brace should be worn for 23 hours a day for a period of 3 months with the objective of obtaining consolidation.
- Generally, consolidation is not achievable and relative immobilization with a bivalve polyethylene brace may suffice. Time of brace wear is reduced to approximately 8 hours a day for a period of 3 months. Weaning must be done progressively, and the brace must be worn for a period of 1 to 2 hours after physical exercise.



**Figure 23:** *Brace with unilateral femoral support.*



**Figure 24 and 25:** *bivalve brace with maintenance of a certain degree of lordosis*

**No matter the type of brace chosen, the following principles must be observed (figure 25):**

- No excessive decrease in lumbar lordosis: The lumbosacral junction must be immobilized in a position that best reproduces the parallel positions of the surfaces of the superior plateau of S1 and inferior endplate of L5. The pelvis should not be retroverted during the application and molding of the cast.
- Placement of pressure points at the sub-umbilical and pelvic levels are indispensable for the repositioning of L5 over the sacrum.
- If the patient presents with a high pelvic incidence (Rousouly type 4), lumbar lordosis must be respected but decreased with abdominal and sacral pressure points. The

latter must not be situated too distally over the sacrum in order to avoid overly retroverting the pelvis.

- If the patient presents with low pelvic incidence (Roussouly type 1), lordosis at the level of the thoracolumbar junction must be extended and slightly reduced at the lumbosacral region with a more proximal placed pressure point with the sacrum. Another pressure point must be placed between the breasts in order to allow for proper thoracolumbar extension.
- Anterior imbalance, which favors pelvic retroversion and lumbosacral kyphosis, must be avoided.
- During molding of the cast, the pressure points must to place the patient in an antalgic position. As a result, it is preferable to mold the cast using either plaster of Paris or fiberglass and in the standing position rather than utilizing 3D computer-aided design and manufacturing (CAD/CAM), which does not allow for the proper placement of pressure points in order to obtain an antalgic position.
- Rehabilitation while wearing a brace is indispensable. During the casting phase or while wearing the femoral brace, muscle mass must be maintenance with stretching exercises and static contractions of the paravertebral and abdominal muscles. While wearing the bivalve polyethylene brace, core conditioning is intensified during brace wear with progressive reconditioning to physical activity.
- Carbon braces with anterior windows should be avoided since they do not allow for sub-umbilical support.

#### **Indications:**

- In symptomatic patients in whom the acute nature of the lesion is confirmed via scintigraphy or MRI, consolidation of the pars defect may be attempted as long as pelvic biomechanical characteristics remain favorable; a very high pelvic incidence (>70°) would lead to extremely high shearing forces and may lead to non-union.
- If the patient presents with an unstable spine along with daily pain with minimal changes in position, femoral support may be indicated for a period of 1 to 3 months. Relay with a bivalve brace may then be authorized.
- In all other cases, a bivalve brace according to the previously cited principles is indicated.

Clinical and radiographic follow-up at 6-month intervals is indicated. In patients with a recurrence of symptoms, the bracing period is prolonged until complete relief of pain is achieved. The brace may sometimes even be required for a period of 6 months to 2 years.

## **CONCLUSION**

Conservative management of isthmic lysis and spondylolisthesis in children and adolescents is the standard treatment modality. It comprises of rehabilitative and orthopedic management and is indicated in either symptomatic patients or those presenting with progressive exacerbation of spondylolisthesis during growth.

The objectives of management are to achieve quick pain relief along with adaptation of inappropriate postures which could have been, in part, the source of the isthmic lysis.

Rehabilitation is initiated while wearing a brace. Generally, physical activity is contraindicated for a period of 3 months, which may be shortened if the patient is managed at a high-level rehabilitation center with daily therapy sessions. Physical activity is then reinstated while maintaining brace-wear after physical activity. Surveillance during the entire period of growth is essential. Reestablishing an appropriate sagittal alignment and developing lumbosacral locking reflexes during physical exercise maximize the chances of a successful outcome after conservative management.

## References

1. Klein G, Mehlman CT, McCarty M. Nonoperative treatment of spondylolysis and grade I spondylolisthesis in children and young adults: a meta-analysis of observational studies. *J Pediatr Orthop* 2009;29(2):146-56.
2. Meyerding H. Spondylolisthesis. *Surg Gynecol Obstet* 1932;54:371-7.
3. Jouve J. Spondylolyse et spondylolisthésis lombosacrée de l'enfant et de l'adolescent. In: *Conférences d'enseignement de la SOFCOT*; 2001; Paris: Cahiers d'enseignement de la SOFCOT; 2001. p. 171-192.
4. Hu SS, Tribus CB, Diab M, Ghanayem AJ. Spondylolisthesis and spondylolysis. *J Bone Joint Surg Am* 2008;90(3):656-71.
5. Logroscino G, Mazza O, Aulisa G, Pitta L, Pola E, Aulisa L. Spondylolysis and spondylolisthesis in the pediatric and adolescent population. *Childs Nerv Syst* 2001;17(11):644-55.
6. Hammerberg KW. New concepts on the pathogenesis and classification of spondylolisthesis. *Spine (Phila Pa 1976)* 2005;30(6 Suppl):S4-11.
7. Labelle H, Roussouly P, Berthonnaud E, Dimnet J, O'Brien M. The importance of spino-pelvic balance in L5-s1 developmental spondylolisthesis: a review of pertinent radiologic measurements. *Spine (Phila Pa 1976)* 2005;30(6 Suppl):S27-34.
8. Labelle H, Roussouly P, Berthonnaud E, Transfeldt E, O'Brien M, Chopin D, et al. Spondylolisthesis, pelvic incidence, and spinopelvic balance: a correlation study. *Spine (Phila Pa 1976)* 2004;29(18):2049-54.
9. Roussouly P, Gollogly S, Berthonnaud E, Labelle H, Weidenbaum M. Sagittal alignment of the spine and pelvis in the presence of L5-s1 isthmic lysis and low-grade spondylolisthesis. *Spine (Phila Pa 1976)* 2006;31(21):2484-90.
10. Legaye J, Duval-Beaupere G, Hecquet J, Marty C. Pelvic incidence: a fundamental pelvic parameter for three-dimensional regulation of spinal sagittal curves. *Eur Spine J* 1998;7(2):99-103.
11. Fu KM, Smith JS, Polly DW, Jr., Perra JH, Sansur CA, Berven SH, et al. Morbidity and mortality in the surgical treatment of six hundred five pediatric patients with isthmic or dysplastic spondylolisthesis. *Spine (Phila Pa 1976)*;36(4):308-12.
12. Cheung EV, Herman MJ, Cavalier R, Pizzutillo PD. Spondylolysis and spondylolisthesis in children and adolescents: II. Surgical management. *J Am Acad Orthop Surg* 2006;14(8):488-98.
13. Gill GG, Manning JG, White HL. Surgical treatment of spondylolisthesis without spine fusion; excision of the loose lamina with decompression of the nerve roots. *J Bone Joint Surg Am* 1955;37-A(3):493-520.
14. Agabegi SS, Fischgrund JS. Contemporary management of isthmic spondylolisthesis: pediatric and adult. *Spine J*;10(6):530-43.
15. Lenke LG, Bridwell KH. Evaluation and surgical treatment of high-grade isthmic dysplastic spondylolisthesis. *Instr Course Lect* 2003;52:525-32.
16. Transfeldt EE, Mehbood AA. Evidence-

based medicine analysis of isthmic spondylolisthesis treatment including reduction versus fusion in situ for high-grade slips. *Spine (Phila Pa 1976)* 2007;32(19 Suppl):S126-9.

17. Hresko MT, Hirschfeld R, Buerk AA, Zurakowski D. The effect of reduction and instrumentation of spondylolisthesis on spinopelvic sagittal alignment. *J Pediatr Orthop* 2009;29(2):157-62.

# Physical conditioning for the prevention of overuse injuries in children.

Hays Arnaud

Institute of Movement Sciences,  
Aix Marseille University

Tim Grover, the legendary physical trainer (Michael Jordan, Scottie Pippen, Charles Barkley, and Shaun Livingston, among others) has declared:

“Hundreds of games, thousands of hours, since they were old enough to pick up a ball. Peewee. Youth leagues. Summer camps. Travel teams. AAU. High school. College. A relentless schedule of games, practice, travel, and training, sometimes for multiple teams and leagues, with multiple trainers and programs. No time for rest or recovery. No time to play or train for other sports. End result: The same muscles, ligaments, tendons, and joints are used over and over again, in the same direction, the same angles, the same motions. What piece of machinery doesn’t eventually give out from repeated use over many years?”

This requires deep reflection in an age when, on a daily basis, dozens of new workout programs are conceived for increasingly younger populations. However, children are meant to be active, play, use their entire bodies, and not continuously work on the same aspects time and time again.

“Everyone wants to go fast and hard, but without the ability to decelerate, what happens? Eventually you crash. Any racecar driver can go at top speed, but elite drivers know when to speed up, when to slow down, when to stop. Explosiveness without the ability to decelerate will almost always result in injury. To me, it’s one of the most critical elements of effective training. But how many athletes want to learn to slow down and stop? It’s not sexy. But it’s essential.”

In an interview in 2013, Tim Grover summarized what researchers were able to later conclude between 2015 and 2019. As Tim Gabbet noted [1]: “the problem is not with training per se, but more likely the inappropriate training that is being prescribed.”

As noted by Duncan in 2019 [2]: “With the emergence of technology and dwindling physical education in schools, there is a severe lack of fundamental movement skills (FMS) among today’s youth. Meanwhile, participation in sports is rising and many young people are not prepared to handle the sport-specific workload they experience; leading to suboptimal performance and increased risk of injury.”

“When youth and adolescent athletes do engage in training, it is imperative that they are not treated as mini-adults and given overly advanced programs. Rather than specializing at a

young age, there should be a system in place to establish a well-rounded base that serves as the framework for the development of new and future motor skills. As the athlete matures, their adaptive capabilities and tolerance to training loads will be dependent on this. Thus, early sampling or diversification is recommended over early specialization and has been shown to correlate with less psychological “burn-out”, fewer injuries, and greater future performance [3].”

In fact, physical development precedes cognitive development in the adolescent population. Physical development is most prominent from early to mid-adolescence, whereas cognitive development, which includes structural changes at the level of the brain and cognition, continues maturing until the end of the third decade. Physical and cognitive changes of puberty and the rate with which these changes occur might ultimately lead to a temporary increase in the risk of injury and may even lead to overtraining syndrome in adolescents [4].

The risks of injury and overuse increase proportionately with the degree of the child’s sport specialization and not with the training load [5,6]. In fact, there is a higher risk of serious injury due to overuse in athletes who spend twice as much time participating in organized sports compared to those partaking in recreational play, even though both exert the same overall load (7).

In order to conclude the concept of training load, the primary factor in overuse injuries, it was previously shown that an optimal load exists that allows a decrease in the risk of injury through a progressive phase of adaptation taking into account the acute and global loads in a systematic fashion [1,8]. This is achieved with a predominance of circuit exercises, thereby avoiding monotony, coupled with regular unwinding periods [9]. The risk factor could be modeled with the following formula :

*Risk factor =*

$$\frac{V_{\text{volume}} \times I_{\text{intensity}}}{PR_{\text{Physical Recuperation}}} \times \frac{MF_{\text{Monotony factor}} \times SF_{\text{Specialization factor}} \times Y_{\text{Years of specialization}} \times E_{\text{Demand for result}}}{CR_{\text{Cognitive recuperation}} \times PR_{\text{Psychological recuperation}} \times PF_{\text{play factor}} \times TF_{\text{Time factor}} \times CF_{\text{Capacity factor}}}$$

Moreover, aside from training load, the contents of training and particularly of physical conditioning play a predominant role in avoiding injuries [10,11]. Thus, physical preparation in children, as was underlined by Tim Grover, must not be tied solely on improving performance. In fact, power and speed are especially linked to the physiological age of the young athlete [12]. Therefore, it is fundamental to develop neuromuscular prophylactic measures combining both global postural and intermuscular coordination exercises [14] with an adapted load, all the while teaching the importance of rest [13]. Complex and varied motricity programs must be integrated in order to enrich neuromotor programs and neuromuscular qualities in a well-proportioned manner [2]. Fine and adaptable motor coordination, which indicate adaptable movements, allow the prevention of injuries and optimal performance in athletic children [15].

## References :

1. Gabbett TJ. The training—injury prevention paradox: should athletes be training smarter and harder? *Br J Sports Med.* 2016 Mar;50(5):273–80.
2. Duncan F, Rodriguez N, Liebenson C. Self care Part 1. Athletic Development: Youth Considerations. *J Bodyw Mov Ther.* 2019 Jul;23(3):619–21.
3. Côté J, Lidor R, Hackfort D. ISSP position stand: To sample or to specialize? Seven postulates about youth sport activities that lead to continued participation and elite performance. *Int J Sport Exerc Psychol.* 2009 Jan;7(1):7–17.
4. McKay D, Broderick C, Steinbeck K. The Adolescent Athlete: A Developmental Approach to Injury Risk. *Pediatr Exerc Sci.* 2016 Nov;28(4):488–500.
5. Post EG, Trigsted SM, Riekena JW, Hetzel S, McGuine TA, Brooks MA, et al. The Association of Sport Specialization and Training Volume With Injury History in Youth Athletes. *Am J Sports Med.* 2017 May;45(6):1405–12.
6. Myer GD, Jayanthi N, Difiori JP, Faigenbaum AD, Kiefer AW, Logerstedt D, et al. Sport Specialization, Part I: Does Early Sports Specialization Increase Negative Outcomes and Reduce the Opportunity for Success in Young Athletes? *Sports Health Multidiscip Approach.* 2015 Sep;7(5):437–42.
7. Jayanthi NA, LaBella CR, Fischer D, Pasulka J, Dugas LR. Sports-Specialized Intensive Training and the Risk of Injury in Young Athletes: A Clinical Case-Control Study. *Am J Sports Med.* 2015 Apr;43(4):794–801.
8. Murray A. Managing the Training Load in Adolescent Athletes. *Int J Sports Physiol Perform.* 2017 Apr;12(s2):S2-42-S2-49.
9. Myer GD, Jayanthi N, DiFiori JP, Faigenbaum AD, Kiefer AW, Logerstedt D, et al. Sports Specialization, Part II: Alternative Solutions to Early Sport Specialization in Youth Athletes. *Sports Health Multidiscip Approach.* 2016 Jan;8(1):65–73.
10. Zouita S, Zouita ABM, Keksi W, Dupont G, Ben Abderrahman A, Ben Salah FZ, et al. Strength Training Reduces Injury Rate in Elite Young Soccer Players During One Season: *J Strength Cond Res.* 2016 May;30(5):1295–307.
11. Bounemri SZ, Mimouni S, Mimouni N, Massarelli R. Effet d'un programme de développement des qualités physiques sur l'organisme. :8.
12. Malina RM, Eisenmann JC, Cumming SP, Ribeiro B, Aroso J. Maturity-associated variation in the growth and functional capacities of youth football (soccer) players. *Eur J Appl Physiol.* 2004 May 1;91(5–6):555–62.
13. Müller L, Hildebrandt C, Müller E, Fink C, Raschner C. Long-Term Athletic Development in Youth Alpine Ski Racing: The Effect of Physical Fitness, Ski Racing Technique, Anthropometrics and Biological Maturity Status on Injuries. *Front Physiol.* 2017 Aug 31;8:656.
14. Zwolski C, Quatman-Yates C, Paterno MV. Resistance Training in Youth: Laying the Foundation for Injury Prevention and Physical Literacy. *Sports Health Multidiscip Approach.* 2017 Sep;9(5):436–43.
15. Hamill J, Palmer C, Van Emmerik RE. Coordinative variability and overuse injury. *BMC Sports Sci Med Rehabil.* 2012;4(1):45.

# Surgical treatment of ligamentous injuries of the knee in children

Elie Choufani<sup>1,2</sup>, Sébastien Pesenti<sup>1,2</sup>, Franck Launay<sup>1,2</sup>, Jean-Luc Jouve<sup>1,2</sup>

<sup>1</sup> AP-HM Department of Pediatric Orthopaedic Surgery, Timone-Enfants Hospital, 264 Rue St Pierre, 13005 Marseille, France

<sup>2</sup> Aix-Marseille University, Faculty of Medicine, 27 Boulevard Jean Moulin, 13005 Marseille, France

Corresponding author:

Dr Elie Choufani MD

AP-HM Department of Pediatric Orthopaedic Surgery, Timone-Enfants Hospital, 264 Rue St Pierre, 13005 Marseille, France

Knee trauma is very frequent in children. Hemarthrosis is a sign of severity and is found in up to 18% of patients presenting with knee trauma [1]. When hemarthrosis is encountered, three main types of lesions should be suspected:

- 1- Ligamentous injuries (1/3 of cases)
- 2- Meniscal injuries (1/3 of cases)
- 3- Femoropatellar lesions (1/3 of cases)

This chapter will focus on ligamentous injuries of the knee and their surgical treatment.

## **Particularities of the ligaments of the knee in children:**

The ligaments in children are more elastic in their younger years. Since the growth plate presents a lower resistance than the ligaments in young children, epiphyseal separations and/or apophyseal avulsions are more frequently encountered. In preadolescents and adolescents, ligaments are less elastic, and the resistance of the cartilage becomes superior to that of the ligament. According to Young's principle of elasticity, the higher the stretching forces, the greater the elastic deformity of the ligament, until reaching plastic deformity (or even rupture), a threshold after which ligaments could never return to their original state.

These ligamentous injuries are increasingly frequent, and for a multitude of reasons:

- Modifications of the sporting activities of children and adolescents

- Increasing pressure by trainers and parents
- Easier access to MRIs
- Development of arthroscopic techniques

According to the 2006 French Society of Orthopedic Surgery & Traumatology (SOFOT) symposium [2], the different ligaments of the knee are injured in the following proportions: 70% medial collateral ligament (MCL), 20% lateral collateral ligament (LCL), 10% cruciate ligaments.

## Peripheral sprains (MCL/LCL)

Injuries to the collateral ligaments of the knee are more frequently the causes of sprains. Hemarthrosis is not usually found in these types of injuries, except if they are associated with injuries to the cruciate ligaments. The classification into 3 grades is of both therapeutic and prognostic importance:

- Grade 1: stretching (no laxity at 20° of knee flexion)
- Grade 2: partial tear (joint laxity but with a firm end point)
- Grade 3: complete tear (joint laxity without an end point)

Treatment depends on severity of the injury :

- Grades 1 and 2: immobilization for 1 to 3 weeks in order to relieve pain (plaster knee brace or removable knee brace depending on the age) with full weight-bearing being authorized.
- Grade 3: cast immobilization or hinged knee brace for 4 to 6 weeks. If there is associated cruciate ligament injury, collateral ligament management would be surgical with suture ± ligamentous reinforcement.



*MCL: Jacob et al. Insights imaging 2013 [3]*

*a/ Normal aspect of the MCL, appearing thicker in its proximal compared to its distal end.*



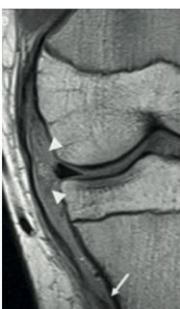
*b/ Grade 1 sprain: low-grade partial tear of the deep fibers with tear of the medial meniscomfemoral ligament with intact superficial fibers.*



*c/ Grade 2 sprain: high-grade tear of both the deep and superficial fibers which remain attached at their proximal and distal ends*



*d/ Grade 3: complete MCL tear at the level of the joint space*



*e/ Grade 3: complete MCL tear at its tibial insertion*

## Central sprains:

Age plays an important role in treatment decision-making:

In children younger than 12 years of age, cruciate ligament injuries are in 80% of cases associated to bony avulsions [2] or a tibial eminence fracture (TEF). In children younger than 8 years of age presenting with hemarthrosis and a normal radiograph, a purely cartilaginous avulsion must be considered [4]. In children older than 12 years of age, ligamentous tears are more frequent and represent approximately 90% of cruciate ligament injuries. Lesions of the PCL and multi-ligamentous injuries are far less frequently found.

## Tibial eminence fracture

The global incidence of TEF is around 1 per 300,000 inhabitants [5] and is more frequently found in children aged between 8 and 13 years old.

The mechanism of injury is a direct blow to the knee in flexion. The ligament has higher resistance than its bony insertion, thereby leading to avulsion fractures. However, this does not prevent distension of the ligaments before the avulsion has occurred, a notion described by Noyer in 1974 [6]. Association with meniscal injury (6 to 8%) is far less frequent than in ligamentous injuries (50%). The diagnosis is generally made on conventional radiographs (lateral views). The most frequently utilized classification is that of Meyers and McKeever (figure 1) and is of therapeutic importance, including 4 different types:

- Type 1: Non-displaced (20%)
- Type 2: Anterior displacement but with posterior continuity (50%)
- Type 3: Complete displacement
- Type 4: Complete displacement with rotation and comminution

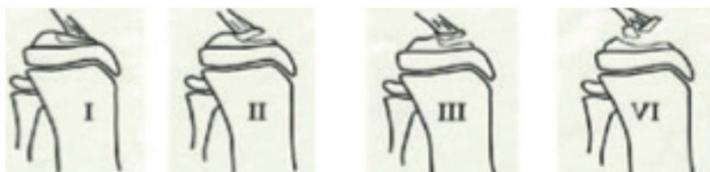


Figure 1: Meyers and McKeever classification of tibial eminence fractures

### Treatment:

- Type 1: treatment is conservative and involves immobilization in a long leg cast or a leg cylinder cast for a period of 4 to 6 weeks with slight (10° to 20°) flexion on the knee.
- Type 2: even though treatment leans toward surgical repair, conservative treatment may be attempted if anatomic reduction of the avulsed fragment is ensured without secondary displacement with minimal flexion. In this case, treatment is similar to that of type 1 fractures. Surgical treatment is indicated if an anatomic and stable reduction is impossible to achieve or if the anterior horn of the medial or lateral menisci or the inter-meniscal ligament is entrapped within the fracture site, an often-encountered

entity (54% of cases according to Kocher) [7]. The treatment of entrapment is either by retraction or resection of the incarcerated fragment [8].

- Types 3 and 4: treatment is surgical and involves arthroscopic or open repair with the aim of stabilizing the avulsed fragment and reestablishing articular congruency.

Means of fixation: The aim of treatment is to reestablish the articular congruency and to secure a stable fixation. This is achieved either by sutures, anchors, screws, or endobuttons. Hunter showed comparative results between arthroscopic suture and screw fixation techniques [8].

## Results

An anatomic reduction does not eliminate the risk of residual laxity. This may be due to the plastic distension due to stretching of the ligament. Nevertheless, multiple studies have shown excellent functionality (absence of instability) regardless of residual laxity [9-11].

## Complications

1. Joint stiffness is the most frequent complication and is essentially due to prolonged immobilization. Rigid fixation systems are recommended for early mobilization (figure 2).



Figure 2: Stage 3 tibial eminence fracture with screw fixation [12].

2. Residual laxity is a frequent complication: Smith et al. showed that 100% of cases were found to have residual laxity at 7 years follow-up and 50% of patients were still symptomatic [13]; Willis et al. reported 74% laxity at 4 years follow-up and 10% were symptomatic [14]. More recent publications have shown lower numbers, with Janarv et al. reporting 38% of cases with residual laxity [15] and Iborra et al. reporting 33% at 7 years follow-up [3]. Moreover, this laxity is rarely associated with clinical instability or secondary meniscal injury. Countersinking, which involves fixing the fracture fragment below its original position, allows to counteract the potential stretching and distension of the ACL.

## Ligamentous tears of the ACL

### Acute phase:

The acute primary repair of an ACL tear has been regaining traction lately, and the two following conditions must be met:

- Proximal tears, as classified on an MRI according to the remaining percentage of ligament distal to the site of ACL rupture: Type 1 (distal remainder > 90%), type II (75%-90%), and type III (25%-75%)
- Good quality of the tendon based on an evaluation by an MRI, classified into good, fair, and poor.

This repair is undertaken by arthroscopy with anchors  $\pm$  internal brace. Recent studies in skeletally immature children have reported good results as long as the prerequisites have been strictly respected (type I proximal rupture and good ligamentous quality). Nonetheless, these studies included only small sample sizes and sometimes utilized synthetic braces:

- Smith et al. (KSSTA 2016): 3 patients with internal brace (removed 3 months post-operatively), 2-year follow-up [17].
- Bigoni et al. (Knee 2017): 5 patients without internal brace with 4-year follow-up (figure 3) [18].

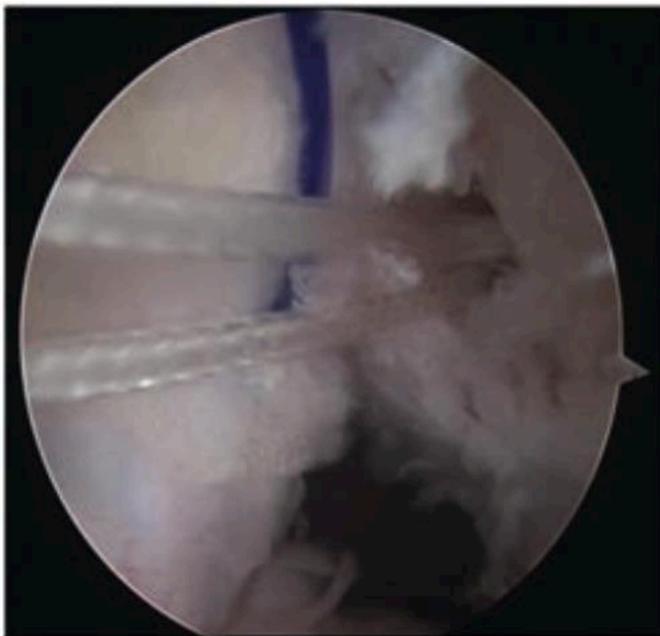


Figure 3 : *Anchor-lacing without internal brace* [18].

### Chronic phase:

When an ACL tear has been confirmed in skeletally immature children, two therapeutic options exist: conservative or operative treatment.

Conservative treatment does not signify abstention of therapy. A rigorous rehabilitation program is necessary along with frequent medical follow-ups allowing for the child to return to pivoting. Adepts of conservative treatment base their arguments on the potential growth of the knee in children which is at increased risk with adult ACL reconstruction techniques. Surgical treatment is indicated when there is knee instability after conservative treatment has been attempted (failure of conservative treatment with episodes of clinical instability and meniscal lesions evidencing instability). Adepts of surgical treatment base their arguments on pediatric-specific surgical techniques (extraphyseal, transphyseal, epiphyseal or mixed).

Multiple studies during the 1990s [19-21] argued that conservative treatment of ACL ruptures in children is no different than its natural history with the following consequences (figure 4):

- 1- Instability in over 90% of patients at 2 years follow-up.
- 2- Secondary medial meniscal lesions in 50% of cases at one-year follow-up.
- 3- Cartilaginous injuries and early osteoarthritis with 50% of patients having abnormal radiographs at 5 years follow-up.

In a study by the Francophone Arthroscopy Society (SFA) in 2017 [22], conservative treatment was assessed in a cohort of 53 patients with a mean follow-up of 31.5 months with a rigorous rehabilitation program (OSLO protocol). Specific inclusion criteria were adopted: children complaining of major clinical instability (>2 episodes of instability); children with an initial meniscal tear were excluded. The rate of success of conservative management was 81%, with 17% developing secondary meniscal tears, 36% complaining of instability, and 40% undergoing ACL reconstruction. Factors that predicted the eventual need for surgery included clinical instability and meniscal injuries.

Review of conservative management of ACL rupture in literature.

Study	Year of publication	Number of patients	Mean age at inclusion (years)	Mean follow-up (years)	Meniscal tears at last follow-up	ACL reconstruction
Graf et al. [10]	1992	12	14.5 (11.7-16.3)	Min 2.0	7 (58%)	0
Mizuta et al. [4]	1995	18	12.8 (10-15)	4.3	6 (33%)	6 (33%)
Woods et al. [11]	2004	13	13.8 (11.0-16.0)	5.8	6 (46%)	13 (100%)
Moksnes et al. [12]	2013	46	11.8 (9.0-14.5)	3.2 (± 1.1)	4 (9%)	10 (22%)
Madeleine et al.	2017	53	11.7 (9.2-14.2)	3.2 (± 2.0)	9 (15%)	21 (40%)

Figure 4: Results of conservative treatment found in the literature [22].

### Anterior cruciate ligament reconstruction techniques:

Multiple techniques have been described for the reconstruction of the ACL in children (figure 5). These techniques differ depending on the type of graft that is used (hamstrings [quadrupled hamstring autograft], bone-patellar tendon-bone, tensor fascia latae, soft baguette technique) and the location of the tunnels (epiphyseal, extraphysal, transphysal, or mixed).

The key in these techniques is avoiding any iatrogenic injury to the physes may hamper the growth of the knee while keeping in mind that femoral and tibial tunnels used in adult reconstructions transverse the growth plate. In fact, these growth plates are responsible for 65% of the growth of the knee in children (1.2cm per year at the level of the femur and 0.8cm per year at the level of the tibia). These risks require surgeons to respect certain security measures:

- Bone should not be harvested from the tibial tuberosity (Kenneth-Jones technique) thus avoiding any damage to tibial tuberosity growth plate which may lead to recurvatum.
- The transphyseal tunnel must not surpass 9mm in diameter.
- Reaming must be slow when approaching the physis.
- Respect the perichondrial ring.
- The tibial tunnel must be more vertical than in adult techniques.
- The bone tunnels must be filled with fibrous tissue.
- Avoid placing interference screws across the physis.

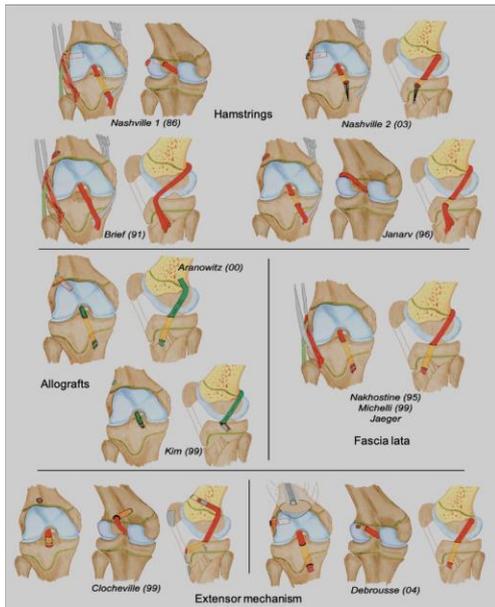


Figure 5: Anterior cruciate ligament reconstruction techniques in children (Chotel collection)

A study by the SFA published in 2017 [23] on 71 skeletally immature patients having undergone ACL reconstruction did not find clinical or radiographic evidence of growth disturbances (limb length discrepancy >10mm or axis deviations >5°). In contrast, MRI analysis found that 20% of cases developed physeal bone bridges. Risk factors for the development of physeal bone bridges include (figure 6):

- Femoral: type of graft (BPTB) as well as diameter ( $\geq 9\text{mm}$ ) and location of the tunnel (epiphyseal).
- Tibial: Type of graft (patellar tendon) and type of fixation (interference screws)

Relative risk of the occurrence of physeal bone bridges at the femur.

Parameter	RR and 95% CI	p
STG	2.1 [0.6–7.6]	NS
SG	0.5 [0.1–2.1]	< 0.05
QT	0.8 [0.1–5.7]	NS
FL	1.3 [0.2–9.1]	NS
Tunnel $\geq 9$ mm	1.7 [0.3–9.2]	NS
Epiphyseal tunnel	1.6 [0.4–5.9]	NS
Transphyseal tunnel	1.2 [0.2–6]	NS
Over-the-top	0	< 0.05

Relative risk of the occurrence of physeal bone bridges at the tibia.

Parameter	RR and 95% CI	p
STG	1.4 [0.4-4.5]	NS
SG	0.3 [0.1-1.5]	NS
QT	3.6 [1.2-10.8]	< 0.05
FL	0	< 0.05
Epiphyseal tunnel	0	< 0.05
Screw fixation	3.7 [0.8-16.1]	NS
Hyperintensity/screw	2.4 [-0.3-7.7]	NS
Non-absorbable screw	1.2 [0.3-5.3]	NS
Transphyseal screw	1 [0.3-3.6]	NS

Figure 6: Relative risk of the occurrence of physeal bone bridges [23]

Return to sports and risk of graft failure:

In a French multicentric study conducted by the SFA in 2017 [24], return to sports and the risk of graft failure was evaluated in patients who had undergone ACL reconstruction. In the “open physis” group, 20% of patients had unsatisfactory results (graft failure or IKDC scores of C or D), with a risk of graft failure of 9% compared to 2.8% in the “closed physis” group.

Tibial and femoral transphyseal tunnels were created in 95% and 60% of cases, respectively (clear tendency for transphyseal techniques). These findings were independent of the technique utilized (epiphyseal, transphyseal, extraphyseal, or mixed techniques) and the choice of graft (BPTB, quadrupled hamstring autograft, or fascia latae). Return to sports was longer compared to adults (13 months for return to training and 14 months for return to competition), with a rate of return to competition of 63.5% in the “open physis” group and 55% in the “closed physis” group (table 1).

Physis	Open	Closed
Sample size	100	178
Running (months)	10.5	9
Training (months)	13	12
Competition (months)	14	12
Return to previous level	80%	77%
Return to competition	63.5%	55%
Graft failure	9%	2.8%
Contralateral rupture	6%	5%
Unsatisfactory result	20%	14.7%

Table 1: Summary of return to sports and graft failure risk in both the “open physis” and “closed physis” groups [24].

In conclusion, actual tendencies for the treatment of ligamentous ruptures of the ACL in children can be summarized as such:

- Increasing interest in arthroscopic repair of ACL tears in the acute setting, a technique that was long abandoned, if the prerequisites of proximal rupture and good ligamentous quality are met. Outcomes in the adult population are

encouraging. A small number of pediatric series have begun to emerge, and longer follow-up and larger sample sizes are required in order to validate this therapeutic option in the pediatric population.

- Well conducted conservative treatment is indicated when no clinical instability or initial or secondary meniscal tears have occurred, thus the necessity of regular follow-ups with annual MRIs allowing for the detection of secondary meniscal tears which may sometimes be asymptomatic.
- The different techniques for ACL reconstruction in the pediatric population have shown comparable results to adult techniques and confirm the definitive nature of reconstructions in the pediatric population, rather than merely spanning while the patient awaits definitive surgery after skeletal maturity has been reached. Recent French multicentric studies have shown a tendency toward transphyseal techniques (95% at the level of the tibia and 60% at the level of the femur) with an absence of postoperative complications in terms of limb length or lower limb axis. Nevertheless, MRI studies have shown the formation of physeal bone bridges at the level of the femur and the tibia, but which remain asymptomatic. This confirms the need for experienced pediatric surgeons when treating ACL injuries in the pediatric population, allowing to monitor of potential growth disturbances.
- The rate of graft failure is significantly higher in pediatric patients compared to adults, thereby inciting a longer return to sports than adults (13-14 vs. 6 months, respectively). This is due to a slower ligamentization of the graft in children [25]. Studies attempting to increase rotational stability, which is difficult to control, are actually underway and are evaluating the effect of combined ACL and anterolateral ligament (ALL) reconstruction techniques. This is an old concept that is once again gaining traction in an attempt to increase the stability of the construct. Graft failure and return to previous level of competition [24] are important notions to discuss with the patients and their parents during preoperative consultations. This would allow for the postoperative rehabilitation to integrate the required time for proper preparation and readaptation, and would allow ample time to reassess the high-level student-athlete future of the child.

# Osteochondritis dissecans of the knee: Pathophysiology and contributing factors

De Courtivron B, Unal P, Krissian S, Odent Th

Clocheville Pediatric Hospital – Tours University Hospital

The term osteochondritis dissecans of the knee was first coined by König in 1887 who suggested an inflammatory or even infectious cause to this disease. However, subsequent studies could not confirm the presence of local or systemic inflammatory processes.

The lesion was described by Hughston as being a fragment of avascular bone covered by articular cartilage that is separated from the normal underlying bone by a layer of fibrous tissue forming a barrier preventing revascularization. The fragment may either detach completely and lead to a loose body, allow for revascularization and spontaneously heal, or lead to non-union and remain in its crater within the femoral condyle [1,2].

The primary cause of this localized devascularization remains unknown and is thought to be multifactorial. A meta-analysis conducted by Andriolo et al. [3] provides an insight into the diversity of recent studies in this domain (table 1).

An analysis of the hypotheses presented today in relation to the etiology of osteochondritis provides a framework for the assessment of possible risk factors.

## **I - Epidemiology**

The incidence of osteochondritis of the knee is estimated to range between 9 and 12 per 100,000 in those aged younger than 19 years. It is generally not encountered before 6 years of age, with 25% appearing between the ages of 6 and 11 years old and 75% between 11 and 19 years old. Even though it is 2 to 4 times more prevalent in boys compared to girls, the incidence of osteochondral lesions in girls appears to be on the rise [4]. It appears equally as prevalent on either knee and 15% of cases are bilateral.

The medial condyle is most commonly affected with up to 80% of cases appearing in this region, especially on the lateral border, with the remaining 20% of cases involving the lateral condyle.

Some cases have been described with lesions at the level of the trochlea, patella, and even the tibial plateau [5]. The natural history after conservative treatment of benign forms is generally favorable. In more severe forms with osteochondral defects, secondary osteoarthritis may appear in up to 50% of patients at 20 years follow-up in the absence of surgical repair [6].

## **II- Pathophysiology**

### A – Mechanical origin

#### **1 - Traumatic**

In all published studies, it has been suggested that micro-traumatic events secondary to physical exercise may be the primary cause of osteochondritis dissecans. This is further confirmed by an increase in the incidence of lesions found in girls who are increasingly partaking in sports requiring large amounts of pivoting or high impact on the knees. However, in multicentric studies conducted by the European Pediatric Orthopedic Society (EPOS), only 55% of subjects affected with osteochondritis dissecans engaged regularly in sports [7].

Even though some cases have been reported in the literature and documented by MRI, an acute traumatic cause is generally not found [8].

Moreover, lesions that resemble osteochondritis have been reproduced experimentally through repeated contusions of the knees, with the development of subchondral fracture that later progressed toward non-union [6].

#### **2 - Biomechanical**

Certain types of sports induce high pressures on the knees due to certain positions, such as kneeling in baseball players. These young athletes present more frequently with osteochondritis of the posterior aspects of the condyles [9].

Furthermore, a higher prevalence of bilateral posterior and bicondylar osteochondritis was reported in patients with cerebral palsy ambulating in a crouched gait pattern [10]. However, in a series of 34 subjects presenting with trochlear osteochondritis, Price et al. found that 90% participated in sports that placed extreme pressure on the femoro-patellar joint, such as Basketball and football (soccer) [11].

Jacobi noted that most subjects presenting with osteochondritis of the lateral condyle were in slight genu valgum, with genu varum leading to medial osteochondritis [5].

#### **3 – Anatomical**

Many Japanese publications have studied the incidence of lateral osteochondritis in subjects with a discoid meniscus, a variant that is especially prevalent in Japan. Osteochondritis was found in 5% of patients who had not been previously operated on the meniscus, whereas 20% of patients who had been previously operated by meniscoplasty later developed osteochondritis, with larger meniscal resections and younger age being the primary risk factors [12].

Finally, following Fairbanks in 1933 and Smilie in 1957, a number of studies have been conducted in search of impingement between the medial intercondylar eminence (tibial spine) and the lateral aspect of the medial condyle. Moreover, this mechanism is reproduced

during the diagnostic maneuvers of Wilson: pain is elicited when the leg is placed in internal rotation with the knee in flexion. A team of researchers from Toulouse, France, showed a significantly raised tibial spine [13], and Chow et al. found a narrow intercondylar notch in patients with signs of impingement [14]. Other biomechanical factors, such as tibial slope, lateral tibial torsion, and hyperlaxity may also be associated leading to repeated impingement between the tibial spine and the most frequent location of the osteochondral lesion. Furthermore, the proximal attachment of the posterior cruciate ligament could also cause increased traction on this area [15] (figure 1).



Obesity also increases the risk of osteochondritis of all locations. The elbows and ankles are especially affected in heavier patients, with the knees being the more frequent location in children who are moderately overweight. Incidentally, it is difficult to determine whether the risk factors are mainly mechanical or biological [16]. As a result, certain activities and anatomical characteristics either increase the frequency or affect the location of osteochondral lesions due to repetitive localized trauma. Nevertheless, these factors may not fully explain this pathology, which is often found in the absence of these factors.

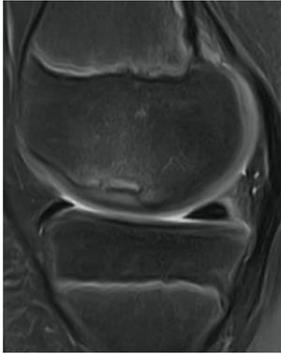
### B – Genetic origin

A genetic predisposition is suspected but difficult to demonstrate. Gornitzky et al. [17] showed that 14% of subjects diagnosed with osteochondritis had a relative with the same pathology, and Yellin et al. attempted to identify genetic loci associated with the pathogenesis of osteochondritis [18].

### C- Biological origin

#### **1- Osteonecrosis**

Intraoperative histologic studies (thereby denoting more severe forms) in search of local and general markers of regeneration confirmed that the deterioration of bony and cartilaginous matrices was situated at the level of the subchondral bone and was associated with the potential for regeneration. Osteonecrosis was a frequent but inconstant finding. A more consistent element was the separation between the undersurface of the osteochondral fragment and the subchondral bone, regardless of its macroscopic stability [20]. Cellular and serologic markers of bony and cartilaginous regeneration can be found in the area of separation. In some cases, the lesions do not involve the subchondral bone and the separation takes place directly beneath the articular cartilage. The cartilage itself is rarely the site of necrosis and can sometimes even be hypertrophied, a finding which may explain the asymptomatic nature of certain lesions [20] (figure 2).



These findings are not compatible with Enneking's hypothesis, which was based on the fact that condylar subchondral bone is vascularized by terminal arterioles. According to this hypothesis, osteochondritis dissecans could be secondary to intra-osseous vascular pathology.

## **2 – Animal studies: Epiphyseal growth plate**

Animal studies may provide further insight into the pathophysiology of this disease. In fact, horses are known to frequently suffer from osteochondritis of the knees, such that racehorses may even benefit from systematic screening. Veterinarians have thus searched for animal models that are easy to study, an endeavor that led them to the miniature pig. These pigs are known to present preclinical forms of osteochondritis dissecans, lesions that were first described in 1978 and termed osteochondrosis [21].

Studies based on histologic and MRI data have revealed frequent ossification irregularities at the level of the epiphyseal growth plate. This is usually found between the secondary ossification center of the condyle and the articular cartilage. Contrary to hyaline cartilage, these sites of irregularity contain a temporarily fragilized terminal vascular bed which flows back and forth within the cartilaginous canals. During growth, as the canal is progressively pervaded through the process of chondrogenesis or ossification, this vascularization is interrupted.

Animal models have also shown that osteochondrosis is due to the early obstruction of these canals leading to ischemia and necrosis of the chondrocytes that are situated at the ends of these canals. During the progression of the ossification site, this area of chondral necrosis resists to ossification and thus leads to late focal enchondral ossification. This may evolve toward:

- 1- Healing, which is the most frequent case
- 2- Formation of cysts within the subchondral bone
- 3- osteochondritis dissecans [22]

Since animal models allow easy reproduction of osteochondral lesions by surgically interrupting the vascular supply to the growth plate without affecting that of subchondral bone, histological studies in these models have been thorough [23]. This has led to a better understanding of these injuries, especially why a fragment of osteochondritis dissecans

contains rather necrotic subchondral bone or is completely formed by rather necrotic hyaline cartilage. Necrotic subchondral bone and cartilage therefore merely represent the natural history of this disease process.

Increased signal intensity, which may be of liquid or cystic origin, may be found even in the absence of articular cartilage fissures and signify instability. These are generally secondary to the presence of chondroblast-rich granulation tissue.

### 3 – In children

The femoral epiphyseal cartilage of the miniature pig appears to be very similar to that of children. Lesions that are equivalent to chondrosis in animals have been sought on human femur samples aged 1 month to 11 years. Significant lesions have been found on over 50% of these samples, which are sometimes multiple and bilateral, and are located at the usual sites of osteochondritis dissecans in children.

As these lesions, specifically those affection the posterior condyles, leave no sequelae and are frequently found in children, they are generally considered as normal variants (figure 3).



Comparing these lesions to osteochondrosis in animals suggests that ischemic chondral necrosis in children is a reality and may even progress toward osteochondritis dissecans [22]. Therefore, this entity is probably due to pathological processes that start early on in life, well before the age of 6 years, and well before any clinical or even radiographic manifestations. Progression toward osteochondritis dissecans seems to be influenced by certain factors that promote the persistent separation between the epiphysial ossification center and the subchondral bone, and thus its instability (a primordial sign of poor prognosis) and secondary necrosis.



Figure 4 illustrates partial spontaneous healing of an extensive lesion.

### **III- Risk factors**

Osteochondritis dissecans is the result of a frequent, old lesion in which the process of spontaneous healing is impeded by multiple factors.

Genetic factors probably caused the initial osteochondrosis, whereas anatomical and biomechanical factors seem to influence progression toward actual osteochondritis.

Overweight also seems to be an authentic risk factor.

In addition, certain types of sports may play a role in the emergence of symptomatic osteochondritis. Nevertheless, there is no single type of sport or activity level that would be particularly responsible for the occurrence of osteochondritis of the knee, contrarily to, for example, the relationship that is frequently found between high-level gymnastics and osteochondritis of the elbow. The only exception would be baseball players, especially receivers, who may develop osteochondritis of the knee due to prolonged periods in the kneeling position.

It would appear that athletes are more frequently affected by this pathology. However, these patients are also usually more concerned by any limitation in their physical ability. In fact, the same lesions may be asymptomatic in some subjects, such as musicians, but invalidating to a football player.

Mechanical axis deviations of the lower limbs cannot be held responsible either, even though they may influence the location of said lesion.

The roles of certain anatomical factors must also be mentioned:

- A discoid meniscus seems to lead to lesions of the lateral condyle, a finding that has been essentially reported in the Japanese population.
- The prominence of the medial tibial spine probably explains the frequency of osteochondritis dissecans of the lateral border of the medial condyle, which is undoubtedly associated with other dynamic elements.

### **Conclusion**

This seemingly well-known lesion appears to originate from a histologic structure that is still unknown: The epiphyseal growth plate of the chondroepiphysis with its vascular networks and its multidirectional canals. A discrete vascular event that is considered to be normal, is frequently found and often resolves spontaneously. However, in certain difficult to control circumstances, this vascular event may later progress into a necrotic osteochondral fragment that would place the child's activity and future of the knee in peril.

Further studies in animal models and humans may give insight into this vascular event, one of the objectives of the ROCK (Research on OsteoChondritis of the Knee) group that was founded in 2010 [24].

### **Images**

Table 1: Recent studies on the etiology of osteochondritis dissecans

Type of study	Number of studies
Genetic	27
Ossification of chondro-epiphysis	12
Endocrinology	9
Overuse	18
Tibial spine	5
Discoid meniscus	16
Biomechanical	20

Figure 1: Association of two anatomical characteristics: both a hypertrophic medial intercondylar eminence (tibial spine) and a narrow, asymmetric intercondylar notch are risk factors for impingement with the lateral border of the medial femoral condyle.

Figure 2: High signal intensity within the bone; the subchondral bone is thin, and the articular cartilage is hypertrophic.

Figure 3: Irregular ossification of the femoral condyle.

Figure 4: Natural history at 3 years follow-up: The posterior aspect of the osteochondral lesion has healed without the need for specific treatment while the anterior aspect, constrained by higher mechanical loads, has persisted.

## References

1. Accadbled F, Vial J, Sales de Gauzy J. Osteochondritis dissecans of the knee. *Orthop Traumatol Surg Res* 2018;104:S97-S105.
2. Masquijo R, Kothari A. Juvenile osteochondritis dissecans (JOCD) of the knee : current concepts review. *EFORT Open Rev* 2019;4:201-212.
3. Andriolo L, Crawford DC, Reale D, Zaffagnini S, Candrian C, Cavicchioli A, Filardo G. Osteochondritis Dissecans of the Knee : Etiology and pathogenetic mechanisms. A systematic review. *Cartilage*.2018 Jul 1. 4. Ananthaharan A, Randsborg PH. Epidemiology and patient-reported outcome after juvenile osteochondritis dissecans of the knee. *The Knee* 2018;25:595-601.
5. Jacobi M, Wahl P, Bouaicha S, Jakob RP, Gautier E. Association between mechanical axis of the leg and osteochondritis dissecans of the knee. *Am J Sports Med*.2010;38(7):1425-1428.
6. Bruns J, Werner M, Habermann C. Osteochondritis dissecans : etiology, pathology, and imaging with a special focus on the knee joint. *Cartilage* 2018;9(4):346- 362.
7. Hefti F, Beguiristain J, Krauspe R, Moller-Madsen B, Riccio V, Tschauer C, Wetzel R, Zeller R. Osteochondritis dissecans: a multicenter study of the european pediatric orthopedic society. *J Pediatr Orthop B* 1999;8(4):231-245. 8. Shea KG, Jacobs JC, Grimm NL, Pfeiffer R. Osteochondritis dissecans development after bone contusion in the skeletally immature : a case series. *Knee Surg Sports Traumatol Arthrosc* 2013;21:403-407.

9. McElroy MJ, Riley PM, Tepolt FA, Nasreddine AY, Kocher MS, MD. Catcher's knee: posterior femoral condyle juvenile osteochondritis dissecans in children and adolescents. *J Pediatr Orthop* 2018;38:410–417.
10. Nhan DT, Robles Garcia M, Lee RJ. Bilateral bicondylar osteochondritis dissecans in a child with spastic diplegia and crouch gait. *J Bone Joint Surg Case Connector*.2018;8(2).
11. Price M, Tuca M, Nguyen J, Silberman J, Luderowski E, Uppstrom T, Green D. Juvenile osteochondritis dissecans of the trochlea : a cohort study of 34 trochlear lesions associated with sporting activities that load the patellofemoral joint. *J Pediatr orthop* 2018;0:00.
12. Mochizuki T, Tanifuji O, Sato T, Watanabe S, Endo N. Predictive factors for developing osteochondritis dissecans after surgery for discoid lateral meniscus are younger age and shorter meniscal width. *Knee Surg Sports Traumatol Arthrosc* 2019(Oct).
13. Cavaignac E, Perroncel G, Thepaut M, Vial J, Accadbled F, Sales de Gauzy J. Relationship between tibial spine size and the occurrence of osteochondritis dissecans : an argument in favour of the impingement theory. *Knee Surg Sports Traumatol Arthrosc* 2017;25:2442- 2446.
14. Chow RM, Guzman MS. Intercondylar notch width as a risk factor for medial femoral condyle osteochondritis dissecans in skeletally immature patients. *J Pediatr Orthop* 2016;36(6):640-644.
15. Markolf KL, Du PZ, McAllister DR. Contact force between the tibial spine and medial femoral condyle : a biomechanical study. *Clin Biomech* 2018;60:9-12.
16. Kessler JJ, Jacobs JC, Cannamela PC, Shea KG, Weiss JM. Childhood obesity is associated with osteochondritis dissecans of the knee, ankle and elbow in children and adolescent. *J Pediatr orthop* 2018;38(5):e296-e299.
17. Gornitzky AL, Mistovich RJ, Atuahene B, Storey EP, Ganley TJ. Osteochondritis dissecans lesions in family members : does a positive family history impact phenotypic potency ? *Clin Orthop Relat Res* 2017;475(6):1573-1580.
18. Yellin JL, Trocle A, Grant S, Hakonarson H, Shea K, Ganley T. Candidate loci are revealed by an initial genome- wide association study of juvenile osteochondritis dissecans. *J Pediatr Orthop* 2017;37(1):e32-e36.
19. Shea KG, Jacobs JC, Carey JL, Anderson AF, Oxford J. Osteochondritis dissecans knee histology studies have variable findings and theories of etiology. *Clin Orthop Relat Res* 2013;471:1127-1136.
20. Gabusi E, Manferdini C, Paoletta F, Gambari L, Kon E, Filardo G, Mariani E, Lisignoli G. Clinical and Biological Signature of Osteochondritis Dissecans in a Cross- Sectional Study. *BioMed Research International*. 2018:1- 9. 545870
21. Toth F, Johnson CP, Mills B, Nissi M, Nykänen O, Ellermann J, Ludwig K, Tompkins M, Carlson C. Evaluation of the suitability of miniature pigs as an animal model of juvenile osteochondritis dissecans. *J Orthop Res* 2019;37:2130-2137.
22. Olstad K, Shea KG, Cannamela PC, Polousky JD, Ekman S, Ytrehus B, Carlson CS. Juvenile osteochondritis dissecans of the knee is a result of failure of the blood supply to growth cartilage and osteochondrosis. *Osteoarthritis Cartilage* 2018;26:1691-1698.
23. Olstad K, Hendrickson EHS, Carlson CS, Ekman S, Dolvik NI. Transection of vessels in epiphyseal cartilage canals leads to osteochondrosis and osteochondritis dissecans in the femoro-patellar joint of foals : a potential model of juvenile osteochondritis dissecans. *Osteoarthritis Cartilage* 2013;21:730-738.
24. Nepple JJ, Milewski MD, Shea KG. Research in Osteo Chondritis of the Knee : 2016 update. *J Knee Surg*.2016;29(7):533-538.

# Meniscal injuries in athletic children

Simon Vandergugten\*, Sébastien Raux\*\*, Franck Chotel\*\*

\* Pediatric Orthopedics, Saint-Luc UCL University Clinics, 10 Avenue Hippocrate, 1200 Bruxelles, Charleroi Grand Hospital, 3 Grand'Rue, 6000 Charleroi, Belgium

\*\* Department of Pediatric Orthopaedic Surgery, Hospital Woman Mother Child, 59 Boulevard Pinel, 69699 Bron

## Introduction

Meniscal injuries in athletic children have become a recurrent issue for pediatric orthopedic surgeons. They appear especially frequently in children partaking in pivoting sports, of which football (soccer) is a prime example. There is a male predominance, especially in children aged 8 years and older. In fact, isolated meniscal injuries are more frequent in the pediatric population than in adults.

Meniscal injuries have become more frequent in pediatric orthopedics: On the one hand, children are increasingly being introduced to pivoting sports at earlier ages. On the other hand, MRI, a medical imaging modality not based on ionizing radiation, has become more accessible and easily makes the diagnosis of meniscal injury. The pediatric orthopedic surgeon is thus frequently interrogated on the management of such injuries, interrogations that may intensify by the patients' clubs and/or trainers who are eager to see young athletes return to their previous level of activity.

In this chapter, isolated meniscal tears on a stable knee will be distinguished from those associated with a ruptured anterior cruciate ligament (ACL), and thus on an unstable knee. These two injury patterns are different on the pathophysiological level, and in each of these two entities, the clinical specificities, the different management strategies (primarily surgical) and their results, all of which are to be considered in relation to certain elements (type of lesion, age of patient, etc...), will be detailed. Furthermore, meniscal injuries on congenital meniscal pathologies, such as a discoid meniscus, will not be discussed, even though these abnormalities are often detected as a result of a sports-related accident.

### 1. Isolated meniscal injuries on a stable knee

The difficulty in the management of isolated meniscal tears is their diagnosis. Treatment is primarily surgical, even though it is sometimes possible to attempt conservative treatment for small, acute lesions .

#### a. Epidemiology

Similar to ACL ruptures, meniscal tears occur as a result of a sudden twisting movement on a hyperflexed knee. 80 to 90% of these injuries occur as a result of a sports injury: football (soccer), basketball, rugby, or skiing [1-6]. In fact, 50% of patients aged between 7 and 18 years presenting with a posttraumatic hemarthrosis of the knee have a meniscal injury [7].

The exact prevalence of these injuries remains to be elucidated, although isolated meniscal lesions are known to be more frequent in skeletally immature children than adults, even though the meniscal tears in general are less frequently found on stable knees compared to unstable knees [5,6,8-11]. Meniscal injuries are encountered most frequently in adolescents [6,11,12] and are rare in children without congenital abnormalities. Meniscal tears may rarely appear in children younger than 8 years [13,14]. Multiple types of meniscal lesions may be encountered, with simple longitudinal and bucket handle tears being the most frequent, followed by horizontal and complex tears. Radial tears and meniscal root avulsions are less common [5,6,11,12].

## b. Diagnosis

### - Clinical

A meniscal tear should be suspected in children presenting with pain at the femorotibial joint line that increases in the kneeling position with difficulty passing to the standing position. Children may feel a blocking sensation that may be permanent or intermittent, with a sensation of popping (sign of meniscal instability) or a limitation in knee range of motion [1,3,5]. Mild hemarthrosis is usually present initially.

Physical examination should assess for joint effusion and compare the range of motion to the contralateral knee in search of a deficit in flexion, flexion contracture or more frequently a deficit in hyperextension.



**Figure 1:** Ruler sign indicating a deficit in hyperextension of the right knee.

Injuries to the medial meniscus elicit pain on forced varus of the knee or the figure-of-four maneuver, and are relieved with forced valgus, and vice versa for the lateral meniscus. The Beighton score determines the laxity of the knee, which constitute a risk factor for meniscal injury [2]. Specific meniscal tests are then realized:

1. Palpation of the anterior, middle, and posterior horns of the medial and lateral menisci.
2. McMurray test: repeatedly passing from flexion to extension with external rotation and valgus for medial meniscal tears, and internal rotation and varus for lateral meniscal tears.



**Figure 2:** McMurray test: repeatedly passing from flexion to extension with external rotation and valgus for medial meniscal tears, and internal rotation and varus for lateral meniscal tears.

3. Apley Grind test (GT): The child is placed prone, the knee is flexed to 90° and is then pressed against the examination table with external rotation for medial meniscal tears and internal rotation for lateral meniscal tears.



**Figure 3:** Apley Grind test (GT): The child is placed prone, the knee is flexed to 90° and is then pressed against the examination table with external rotation for medial meniscal tears and internal rotation for lateral meniscal tears.

4. Thessaly test (TT): difficult to realize in the pediatric population, the child is asked to stand on a single leg with the knee flexed to 20°. The patient is then asked to turn to the left and then to the right thereby causing external rotation of the knee to test for medial meniscal tears and internal rotation to test for lateral meniscal tears.



**Figure 4:** *Thessaly test (TT): difficult to realize in the pediatric population, the child is asked to stand on single support with the knee flexed to 20°. The patient is then asked to turn to the left and then to the right thereby causing external rotation of the knee to test for medial meniscal tears and internal rotation to test for lateral meniscal tears.*

According to the literature [3,15], these specific tests have a very low sensitivity (average 40%) but high specificity (>80%) in children. As a result, they should be taken into account only if they are positive. In children with lateral meniscal injuries, a discoid meniscus must always be ruled out [16].

#### - Medical imaging

Conventional radiographs have little value in the diagnosis of meniscal injuries, although they are systematically ordered in patients presenting with pivoting injuries of the knee. Their utility is in eliminating other lesions, such as osteochondral injuries, Segond fractures, and tibial eminence fractures [3]. The diagnosis of a meniscal injury is generally made on MRI, as was described in the previous chapter. Care should be taken not to misdiagnose a normal variant as a pathological finding. In fact, increased signal intensity of the posterior horn of the medial meniscus or of the popliteal hiatus of the lateral meniscus is often of vascular nature and may be mistaken for a horizontal tear of the medial meniscus or a vertical tear of the posterior horn of the lateral meniscus, respectively [17].

The diagnosis of a meniscal injury is thus made by associating the above described symptoms and physical exam in conjunction with imaging findings as to avoid misinterpreting a physiologic vascular increased signal of the posterior horn of the medial meniscus as a meniscal injury in a patient presenting with femoral patellar pain.

#### b. Treatment

- Conservative treatment

Acute, short meniscal tears may benefit from conservative treatment with limb unloading and abstention from pivoting sports, especially in tears of the peripherally vascularized “red/red” zone [3]. These patients require close follow-up with both physical and MRI exams. Follow-ups should be arranged at a minimum of 3-month intervals before allowing progressive return to high-risk sports. Anatomically, longitudinal tears measuring less than 10mm may be managed conservatively [3].

- Operative treatment

The majority of meniscal tears must be sutured, either because the diagnosis has been delayed (thus proving that the tear did not heal), or due to a complex meniscal injury or an extended bucket handle tear leading to an increased risk of joint instability [4,5,8,9,11,12].

> Principles of treatment:

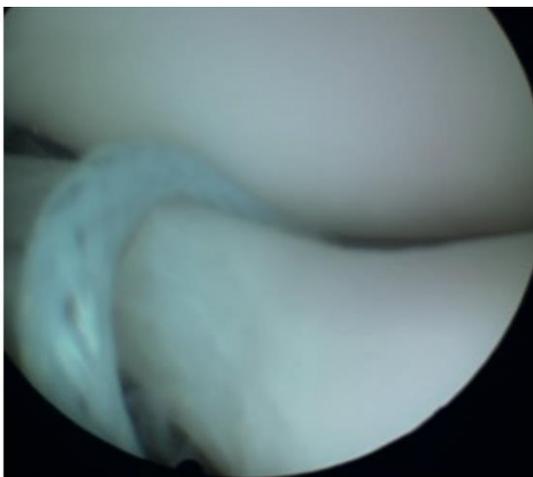
- Unstable lesions must be repaired. Instability may be diagnosed clinically by a sensation of clicking, blocking or limited range of motion, or on arthroscopy by testing the meniscal tear with a probe.

- Regardless of the location of the injury, conserving as much of the meniscal tissue as possible is primordial, and no tear should be considered unreparable, even those in the avascular “white/white” zones [6,10-12,18]. The only exceptions to this rule are radial tears, which are exceptional in children.

> Techniques:

Certain rules must be followed:

- Begin by preparing and debriding the edges of the tear with a rasp. In meniscocapsular separations, the meniscal edges as well as the capsule should be debrided.
- Heavy non-absorbable braided suture (e.g. Mersuture 1) should be used, since the healing time of a meniscal tear is generally longer (4-6 months) than the absorption-time of absorbable sutures.
- Privilege vertical or oblique mattress sutures over horizontal mattress sutures due to their higher biomechanical resistance [19,20].
- Do not place sutures too close to the lesion in order to include a maximum of meniscal tissue within the suture [20].
- In extended bucket handle tears, “total knots” can be used: the knots are passed over the superior edge of the meniscus towards the inferior edge thus grabbing the free edges (figure 5) [18,20].



**Figure 5:** “Total knot” grabbing the free edges of the meniscus.

- Multiply the sutures (separated by 5mm intervals) for better tension distribution between the different sutures while tightening them.

Dedicated devices for meniscal repair can be found, and each medical device company advances its own “unique and revolutionary” product. Regardless, 3 types of suture techniques should be differentiated based on the type of knot: from the outside to the inside

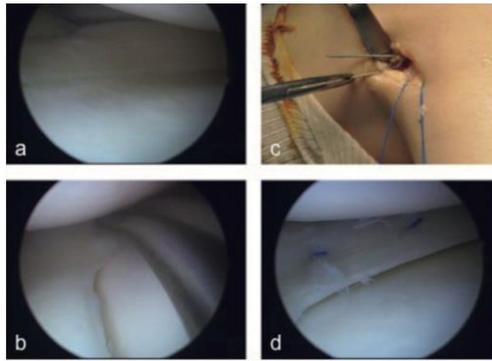
of the knee (outside-to-inside), from the inside to the outside of the knee (inside-to-outside), or completely intraarticular (all-inside).

Our preferred techniques will be detailed depending on the location of the meniscal tear (figure 6):



**Figure 6:** three different techniques of meniscal suture: from the outside to the inside of the knee (outside-to-inside) for the anterior horn, from the inside to the outside of the knee (inside-to-outside) for the body, or completely intraarticular (all-inside) for the posterior horn.

- Tears of the anterior horn of the meniscus are often repaired by an outside-to-inside approach. Using arthroscopy, a hollow needle is first introduced in an ascending fashion through the tear. A heavy non-absorbable braided suture is then passed through the lumen of the needle. A second needle is passed in the same direction, placed either 5mm more lateral to the previous or, if a “total knot” is desired, passing through the other side of the meniscus. The suture is then passed through the lumen of the second needle using either a prefabricated lasso system or a handmade lasso fashioned out of non-braided suture loop (e.g. size 0 PDS suture). The suture bridges the tear within the joint with both ends exiting the skin. A small vertical cutaneous incision is made between the exit points of the suture ends. The sutures are then drawn subcutaneously using an arthroscopic hook and passed through the newly created skin incision. Under arthroscopic guidance, the knots are made against the joint capsule using a slip knot. Slip knots allow better tension distribution between the different knots. For tears with a very anterior extension where laxity of the joint capsule in this area may be problematic, it is preferable to secure the knot on an anchor placed over the anterior tibial epiphysis (meniscopexy) in order to avoid loosening.



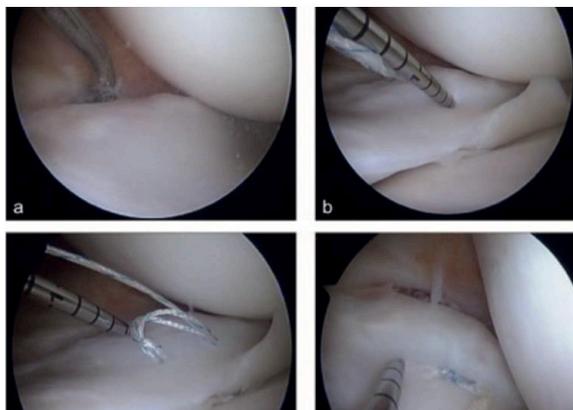
**Figure 7:** Inside-to-outside technique. *a: debridement of the lesion using a rasp. b: Insertion of the cannula and then the needle through the tear. c: extraction of the needles and sutures through a skin incision at the level of the lateral meniscus in order to avoid the common fibular nerve. Note that the free ends of the first suture are extracted anteriorly, and the needles of the 2<sup>nd</sup> suture are extracted posteriorly. d: the two knots after tightening against the capsule.*

- Tears of the body of the meniscus are repaired by an inside-to-outside approach (figure 7). A curved suture cannula is passed through the opposite portal (anteromedial portal for the lateral meniscus and anterolateral portal for the medial meniscus), with the convexity of the cannula facing the cruciate ligaments. One hand is used to apply the distal end of the cannula against the meniscus in a slightly descending fashion (for a vertical mattress suture) at its anterior-most edge. A heavy non-absorbable braided suture is passed through the eye of a long needle that is adapted to the cannula, and the needle is passed through the cannula with a needle driver, passing through the meniscal tears and exiting the skin. The cannula must always face anteriorly in order to avoid the needle passing too posteriorly. In lateral meniscal tears, the needle must exit anterior to the fibular head as to avoid damaging the common fibular nerve. The needle and one end of the suture are passed through the skin, and the other end (still inside the cannula) is once again passed through the eye of the needle. The cannula containing the suture is then moved under arthroscopic guidance at least 5mm away from the first meniscal entry point either laterally or to the other side of the meniscus (if a “total knot” is desired) all the while maintaining the same direction. The needle and the second end of the suture are finally passed once again through the cannula and through the meniscal tear and the skin in a manner similar to the previously described. Similar to the outside-to-inside technique, the suture bridges the tear within the joint and its ends lie outside the skin. As previously described, the suture is passed through a small vertical skin incision with the help of an arthroscopic hook. The sutures are tied under the skin incision via arthroscopic guidance with a slip knot, and the knots are applied against the joint capsule. For tears of the lateral meniscus where the sutures are made relatively close to the fibular head, the cutaneous incision is extended in order to ensure the passage of the sutures in front of the biceps femoris tendon, thus avoiding the common fibular nerve. This suturing technique is preferred since it allows a solid fastening of the knot on the joint capsule [4,18]. The use of a cannula also aids in the reduction and stabilization of bucket handle tears. Finally, in the particular case of horizontal tears

of the lateral meniscus with an associated meniscal cyst, a direct lateral approach at the level of the cyst may be beneficial in order to excise the cyst during meniscal repair.

- Tears of the posterior horn of the meniscus are usually inaccessible by the inside-to-outside approach, since the exit points of the needles are too posterior and run the risk of neurovascular damage. The all inside technique (figure 8) is preferred in such cases. Its advantages are ease of use, lower neurovascular risk, and limited number of cutaneous incisions [21]. Nevertheless, this suturing technique is considered less robust than the previous two, since it is supported by a non-absorbable anchor that is theoretically applied against the meniscal wall of the posterior capsule. In reality, the exact position of the anchor in the posterior soft tissues and its mechanical value are unknown. As a result, the anchors risk loosening and floating within the knee joint if the sutures fail.

In the all-inside technique, disposable industrial devices are generally used: The needle is passed vertically through the meniscal tear at a mean depth of 18mm (20mm in older adolescents and 16mm in younger children), the first anchor is deployed, the needle is removed from the meniscus and moved 5mm from the first meniscal entry point either laterally or to the other side of the meniscus (if a “total knot” is desired) and then passed through the meniscal tear, the second anchor is deployed, and the needle is removed from the knee. In order to bring the two anchors closer together and lock the knots, continued traction is applied on the ends exiting the skin with a counterpressure being applied on the meniscus with a hook, and eventually liberating one of the two sutures linking the two anchors. The suture is then cut flush with the meniscus.

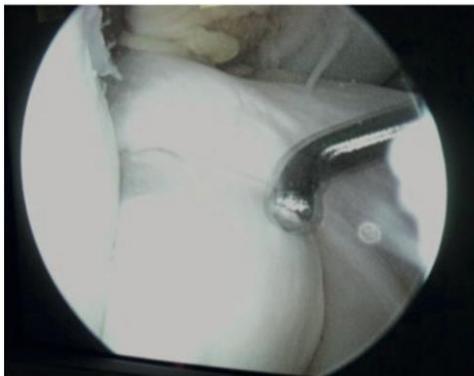


**Figure 8:** All-inside technique. *a: preparation and debridement of the tear using a rasp. b: the first suture is passed through the meniscal tear; the first anchor is deployed behind the meniscus. c: two anchors are placed; the two sutures are spaced 5mm apart. d: appearance of the first knot under tension and preparation of the second knot.*

> postoperative management: immobilization by with a knee immobilizer with the knee in extension for a period of 6 weeks. The knee may be mobilized from 0 to 60° by a physiotherapist 3 times per week in the immediate postoperative period. If the lesion was considered as unstable during arthroscopic hook examination, the patient must be placed non-weight-bearing with crutches for 6 weeks. Physical exercise is prohibited for a period of 4 to 6 months depending on the expected stability of the meniscal repair.

#### d. Results

Healing rates in patients operated for meniscal repair are still debated and depend extensively from the complexity of the lesion and the quality of the repair. Although postoperative follow-up MRI is rarely obtained in patients with a doubtful clinical follow-up, it is actually estimated that healing rates are around 80% in children, compared to 30% in adults [2,4,6,8,10-12,18,22]. A team at Mayo Clinic recently published their results of isolated meniscal repairs (on stable knees) in 32 children and adolescents with a mean follow-up of 17.6 years: The functional International Knee Documentation Committee (IKDC) score significantly increased from 65.3 preoperatively, to 92.3 at final follow-up, independently from the complexity of the lesion, although there was a significantly higher percentage of early failure requiring revision surgery in complex lesions (80% failure rate) compared to simple lesions (18.2% failure rate) and bucket handle tears (47% failure rate) [11]. A recently-published systematic review including 8 studies and 287 patients on the outcomes of meniscal repair (of which 60% were on unstable knees with an associated ACL tear) in children aged younger than 18 years confirms the previously-reported good functional results, with most patients being asymptomatic at final follow-up regardless of the type and location of the lesion or type of suture used [6]. Finally, a systematic review of the same 8 studies (287 patients and 301 meniscal lesions) also reported a good mean postoperative Lysholm functional score ranging from 85 to 96, a mean failure rate of 17.3%, and a low complication rate (1 incidence of temporary paralysis of the common fibular nerve and 1 septic arthritis) [12]. Children generally have high meniscal plasticity: as a result, when treating a chronic dislocated bucket handle tear, the free edge of the torn meniscus can be easily reduced since the dislocated portion will remodel and thin out over time (figure 9).



**Figure 9:** reduction of a chronic bucket handle tear, the thick free edge showing remodeling and progressive thinning.

As a result, all meniscal tears in children and adolescents should be sutured. Meniscectomy is an extremely detrimental procedure that must be avoided. For pediatric orthopedic surgeons, meniscal tears that are believed to be beyond the surgeon's expertise must be referred to a colleague (even an adult orthopedic surgeon) rather than be removed.

## 2. Meniscal tears on an unstable knee

### a. Epidemiology

A study published in 2011 reported a 10-year increase in the prevalence of ACL injuries in children and adolescents and, with it, an unavoidable increase in the prevalence of meniscal tears [23]. In a study by Stracciolini, ACL tears represented up to 10% of sports injuries in 13 to 17-year-olds [24]. In fact, ACL injuries increase the risk of synchronous meniscal lesions, but also metachronous tears due to chronic instability of the knee [25].

In a study on secondary meniscal injuries in patients with a ruptured ACL, Raad et al. found a high prevalence of rugby and football (soccer) players and a relationship between meniscal tears and high BMI, attempted functional treatment, and delay until surgery [26].

Meniscal tears with concomitant ACL injuries were discussed during the 2017 symposium of the Francophone Arthroscopy Society which was centered around ACL injuries in children and adolescents. In a prospective study titled "ACL injuries – open physes", the authors found that 48% had associated meniscal tears. The lateral meniscus was injured in 49% of cases (posterior horn: 83%; anterior horn: 0%, body of meniscus: 10%, body and posterior horn: 7%). The medial meniscus was injured in 51% of cases (posterior horn: 86%, posterior horn and body: 7%, body: 7%, anterior horn: 0%). Tears were vertical in 60% of cases, bucket handle tears in 12%, horizontal tears in 10%, radial tears in 5%, and complex tears in 13%.

In a retrospective study "ACL injuries – open physes", the authors found meniscal tears in 24% of cases (medial meniscus 62%, lateral meniscus 19%, both menisci 19%).

The percentage of meniscal tears in patients with a defective ACL thus varies depending on the study and dictates the indications for surgical intervention, with some studies reporting meniscal tears in up to 85% of ACL deficient knees [27]. These differences may be explained by the delays until surgical management in some studies (1 year for Ramski et al. [28]), which increases the incidence of meniscal injury, especially the medial meniscus [27,29]. Contrarily, in patients operated in the acute setting, a lower incidence of secondary meniscal injuries was found.

In a prospective study "ACL injury – closed physes", the authors report meniscal tears in 49% of patients with ACL ruptures who were operated, with once again a predominance of injury to the medial meniscus.

In fact, ruptures of the ACL lead to a specific pattern of meniscal injury: injuries of the medial meniscal ramp, initially described as a posterior meniscocapsular injury of the medial meniscus [30]. Some studies have recently suggested that these injuries may be associated with lesions of the attachment of the meniscotibial ligament on the posterior meniscal horn. These injuries are often difficult to identify on MRI due to patient positioning with the knee in extension. The prevalence of these injuries depends on the authors and ranges from 24 to 28% in children and adolescents with a ruptured ACL [30,31].

Peltier et al. found that injury to the meniscal ramp increases anterior tibial translation, rotation and pivot-shift [32], and increases instability [33]. As a result, Stephen et al.

recommend repairing the meniscal ramp simultaneously with the ACL, due to its potential implication in persistent instability of the knee [34]. If left untreated, increased instability of the knee would lead to higher strain on the newly repaired ACL.

#### b. Diagnosis

The most frequently reported symptoms in the context of an acutely unstable knee after an exaggerated valgus moment and external rotation include pain and episodes of locking or pseudo-locking. The physical exam would also search for:

- Classic signs of instability: anterior/posterior drawer test, Lachman test, positive pivot-shift test.
- Signs of meniscal pathology: joint line tenderness on the medial or lateral side, positive McMurray test, pain on the Apley and Thessaly tests.

The physical exam may be particularly difficult to perform in the acute phase, especially in bucket handle tears. In this case, the patient must be reassessed in 2 to 3 weeks.

Conventional radiographs of the knee are not particularly useful, although findings of a Segond fracture (avulsion of the anterolateral ligament) are pathognomonic of an ACL tear.

MRI is the modality of choice and would allow a complete assessment of the entire knee:

- ACL rupture: complete rupture of the ACL, signal anomalies, Blumensaat angle superior to  $10^\circ$ .
- Injuries secondary to ACL rupture: bone contusion, anterior drawer sign from anterior tibial translation
- Meniscal injury especially of the posterior horn, dislocations of bucket handle tears within the intercondylar notch with a classic double PCL sign.

#### c. Treatment

Patients are most often seen at a distance from the acute incident. In fact, indications for the emergent treatment of ACL are limited.

As such, meniscal tears associated with an ACL injury discovered on MRI must prompt the immediate repair of the meniscus. In fact, the finding of a meniscal lesion is the main indication for concomitant ACL reconstruction. Moreover, ACL tears are not known to spontaneously heal, and the secondary knee instability that is caused by such an injury would prevent any meniscal tear from healing, even when repaired.

Nevertheless, not all meniscal tears should be systematically repaired: partial-thickness tears of the posterior horn of the lateral meniscus are generally concomitant with the ACL injury. These tears are frequently encountered and have a high potential for healing in the absence of renewed episodes of knee instability. If these tears are found to be stable during hook testing or solely partial thickness, repair is generally not necessary.

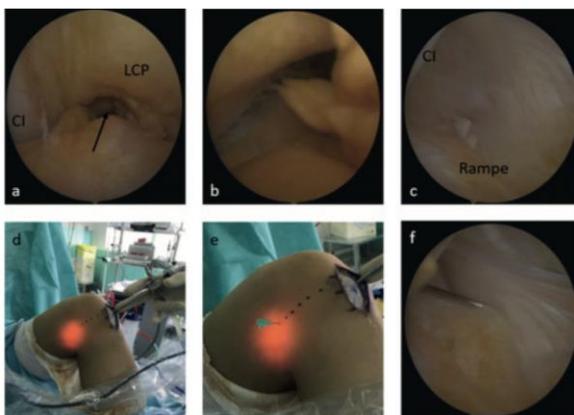
Surgical management is undergone via arthroscopy, under a tourniquet, and under general anesthesia. In children, the technique used for ACL reconstruction generally depends on the surgeon's preferences and skeletal maturity.

Meniscal repair in unstable knees generally follows the same principles as in isolated meniscal tears: tears measuring 10mm or more must be repaired, while those measuring less may be simply debrided. Different suturing techniques are summarized depending on the location of the tear:

- All-inside technique (previously fast-fix) for injuries to the posterior horn.
- Inside-to-outside technique (previously Acufex) for injuries to the body of the meniscus.
- Outside-to-inside technique (previously Meniscus Mender) for injuries to the anterior horn.

Although the inside-to-outside technique by non-absorbable sutures requires a lateral counter incision on the knee, it remains the gold-standard treatment in terms of suture stability.

Injury to the meniscal ramp also requires arthroscopic repair. The diagnosis is usually made on MRI and the tear is classified according to Thaunat's system, although the type of injury may be accurately established only after arthroscopic examination [35]. Injuries of the meniscal ramp are generally identified by a sequential arthroscopic approach, including a transcondylar (passing between the PCL and the medial femoral condyle) and posteromedial approach. Visualization of meniscal ramp injuries may be simplified by using a 70° arthroscope. A needle, placed through a posteromedial approach, may be used to elevate the meniscus from the distal end of the ramp and assess its integrity (figure 10).



**Figure 10:** Exploration of the medial condylar ramp. a: the transcondylar approach is made between the medial femoral condyle (MC) and the posterior cruciate ligament (PCL, arrowhead). b: Arthroscope advancement (arrow). c: exposition of the posteromedial aspect of the knee with the ramp underneath. d: introduction of the needle by a posteromedial approach over transilluminated area. e: penetration of the needle at the level of the joint line (dotted) at its posterior aspect. f: once the posteromedial space has been breached, the needle may elevate the soft tissues from the extremity of the ramp thus evaluating the tears.

The tear is prepared and debrided using a shaver or rasp through a posteromedial approach. Through the same approach, a 45° dedicated hook is introduced facing to the right for the left knee (and vice versa for the right knee). This dedicated hook, also known as a Kuntscher awl, is used to pierce the meniscus from the bottom-up, passing completely through the tear. The lasso system is then released, and the suture is passed through the awl and the lasso. The suture is fastened onto the lasso and the awl is removed leaving both ends of the suture protruding from the skin. A slip knot is then made (figure 11) while pulling on one strand and lowering the knot onto the meniscal tear. Once the knot is in place, it is locked with a couple of throws and finally a surgeon's knot. The free ends of the suture are then severed under arthroscopic guidance, and its stability is tested using a hook.

In extensive meniscal tears that have been treated with sutures, a strict postoperative regimen must be followed: No weight bearing for 6 weeks while wearing a knee brace and mobilizing with crutches or a wheelchair. Anticoagulants are prescribed based on the patient's pubertal status (menarche in girls, and appearance of pubic hair in boys). Following immobilization, physiotherapy may prove useful as an aid for ambulation, reinforcement of the quadriceps and hamstrings, and recuperation of knee range of motion. Physical training and knee hyperflexion are contraindicated for 4 months postoperatively.

As for return to all types of sports (including pivoting), most authors recommend particular care be taken postoperatively in children, with return to sports note being authorized before 12 months postoperatively, sometimes even 14 months in younger patients. In fact, this delayed return to sports stems primarily from the slower ligamentization of the graft seen on MRI in children compared to adults, but also from an increased risk of ACL failure from early return to sports [36,37].



**Figure 11:** Slip knot tightened deep into the operative field. a: the left hand holds both strands as the suture is passed in the lesion, the right strand being the longer one. b: the right strand is passed three times around these two. c: the right strand is then passed from the top to the bottom through the preformed loop and held between the thumb and index of the left hand. d: while the assistant stabilizes the knee, the left hand pulls on its strand and the knot slips and is lowered until reaching the lesion area. e: The knot is fastened by a throw and a two surgeon's knots (bulky knots must be avoided, especially in a smaller knee, in order to reduce subcutaneous irritation).

#### d. Results

Outcomes of meniscal repair in children have been inconsistent in the literature: In a multivariate analysis, Ferrari et al. reported healing rates of 33 to 100% in children treated with meniscal repair [6].

Adults treated with both meniscal repair and ACL reconstruction simultaneously have shown superior results when compared to patients with isolated meniscal injury. In children, these differences are less evident, as was shown in a study by Yang et al. [38]. Moreover, meniscal healing is more difficult to assess in children, and some patients may be completely asymptomatic and show no signs of meniscal healing on imaging [22].

Lucas et al. found that up to 68% of patients treated with isolated meniscal tears and who were treated by sutures showed signs of healing, whereas Krych et al. reported that up to 74% of patients with both a meniscal tear and an ACL injury showed signs of healing [10,39]. Thus, outcomes between children and adults seem comparable, although studies directly comparing meniscal healing rates between isolated meniscal tears and meniscal tears associated with ACL injuries in children are still lacking.

Concerning the repair of the meniscal ramp, data on the outcomes remain scarce. In one systematic review, Alessio-Mazolla et al. found a failure rate of 8.3% in these cases [40].

## Conclusion

The dogma of meniscal preservation is more appropriate than ever in children. Meniscal tears must be identified, and MRI must be abundantly prescribed. The majority of these injuries must be repaired by sutures via arthroscopy, an intervention requiring a high level of expertise. Meniscectomy is contraindicated in children or must be limited to the debridement of small unrepairable tears. Meniscal repair is justified by its good outcomes in children, and by the absolute necessity of preventing the development of early osteoarthritis as early as young adulthood.

## References

1. Andrish JT. Meniscal Injuries in Children and Adolescents: Diagnosis and Management. *J Am Acad Orthop Surg* 1996;4(5):231-7.
2. Bonnard C, Chotel F. [Knee ligament and meniscal injury in children and adolescents]. *Rev Chir Orthop Reparatrice Appar Mot* 2007;93(6 Suppl):95-139.
3. Bellisari G, Samora W, Klingele K. Meniscus tears in children. *Sports Med Arthrosc Rev* 2011;19(1):50-5.
4. Vanderhave KL, Moravek JE, Sekiya JK, Wojtys EM. Meniscus tears in the young athlete: results of arthroscopic repair. *J Pediatr Orthop* 2011;31(5):496-500.
5. Wilson PL, Wyatt CW, Romero J, Sabatino MJ, Ellis HB. Incidence, Presentation, and Treatment of Pediatric and Adolescent Meniscal Root Injuries. *Orthop J Sports Med* 2018;6(11):2325967118803888.
6. Ferrari MB, Murphy CP, Gomes JLE. Meniscus Repair in Children and Adolescents: A Systematic Review of Treatment Approaches, Meniscal Healing, and Outcomes. *J Knee Surg* 2019;32(6):490-8.
7. Stanitski CL, Harvell JC, Fu F. Observations on acute knee hemarthrosis in children and adolescents. *J Pediatr Orthop* 1993;13(4):506-10.
8. Krych AJ, McIntosh AL, Voll AE, Stuart MJ, Dahm DL. Arthroscopic repair of isolated meniscal tears in patients 18 years and younger. *Am J Sports Med* 2008;36(7):1283-9.

9. Kraus T, Heidari N, Svehlik M, Schneider F, Sperl M, Linhart W. Outcome of repaired unstable meniscal tears in children and adolescents. *Acta Orthop* 2012;83(3):261-6.
10. Lucas G, Accadbled F, Violas P, Sales de Gauzy J, Knorr J. Isolated meniscal injuries in paediatric patients: outcomes after arthroscopic repair. *Orthop Traumatol Surg Res* 2015;101(2):173-7.
11. Hagmeijer MH, Kennedy NI, Tagliero AJ, Levy BA, Stuart MJ, Saris DBF, et al. Long-term Results After Repair of Isolated Meniscal Tears Among Patients Aged 18 Years and Younger: An 18-Year Follow-up Study. *Am J Sports Med* 2019;47(4):799-806.
12. Liechti DJ, Constantinescu DS, Ridley TJ, Chahla J, Mitchell JJ, Vap AR. Meniscal Repair in Pediatric Populations: A Systematic Review of Outcomes. *Orthop J Sports Med* 2019;7(5):2325967119843355.
13. Bloome DM, Blevins FT, Paletta GA, Jr., Newcomer JK, Cashmore B, Turker R. Meniscal repair in very young children. *Arthroscopy* 2000;16(5):545-9.
14. Shea KG, Archibald-Seiffer N, Kim KM, Grimm NL. Bucket-handle meniscal tear in a 5-year-old child. *Knee Surg Sports Traumatol Arthrosc* 2012;20(11):2291-3.
15. Kocher MS, DiCanzio J, Zurakowski D, Micheli LJ. Diagnostic performance of clinical examination and selective magnetic resonance imaging in the evaluation of intraarticular knee disorders in children and adolescents. *Am J Sports Med* 2001;29(3):292-6.
16. Ellis HB, Jr., Wise K, LaMont L, Copley L, Wilson P. Prevalence of Discoid Meniscus During Arthroscopy for Isolated Lateral Meniscal Pathology in the Pediatric Population. *J Pediatr Orthop* 2017;37(4):285-92.
17. Bouju Y, Carpentier E, Bergerault F, De Courtivron B, Bonnard C, Garaud P. The concordance of MRI and arthroscopy in traumatic meniscal lesions in children. *Orthop Traumatol Surg Res* 2011;97(7):712-8.
18. Noyes FR, Barber-Westin SD. Arthroscopic repair of meniscal tears extending into the avascular zone in patients younger than twenty years of age. *Am J Sports Med* 2002;30(4):589-600.
19. Kocabey Y, Taser O, Nyland J, Doral MN, Demirhan M, Caborn DN, et al. Pullout strength of meniscal repair after cyclic loading: comparison of vertical, horizontal, and oblique suture techniques. *Knee Surg Sports Traumatol Arthrosc* 2006;14(10):998-1003.
20. Kocabey Y, Taser O, Nyland J, Ince H, Sahin F, Sunbuloglu E, et al. Horizontal suture placement influences meniscal repair fixation strength. *Knee Surg Sports Traumatol Arthrosc* 2013;21(3):615-9.
21. Beck JJ, Shifflett K, Greig D, Ebramzadeh E, Bowen RE. Defining a Safe Zone for All-Inside Lateral Meniscal Repairs in Pediatric Patients: A Magnetic Resonance Imaging Study. *Arthroscopy* 2019;35(1):166-70.
22. Accadbled F, Cassard X, Sales de Gauzy J, Cahuzac JP. Meniscal tears in children and adolescents: results of operative treatment. *J Pediatr Orthop B* 2007;16(1):56-60.
23. Shea KG, Grimm NL, Ewing CK, Aoki SK. Youth sports anterior cruciate ligament and knee injury epidemiology: who is getting injured? In what sports? When? *Clin Sports Med* 2011;30(4):691-706.
24. Stracciolini A, Casciano R, Levey Friedman H, Meehan WP, 3rd, Micheli LJ. Pediatric sports injuries: an age comparison of children versus adolescents. *Am J Sports Med* 2013;41(8):1922-9.
25. Samora WP, 3rd, Palmer R, Klingele KE. Meniscal pathology associated with acute anterior cruciate ligament tears in patients with open physes. *J Pediatr Orthop* 2011;31(3):272-6.
26. Raad M, Thevenin Lemoine C, Berard E, Laumonerie P, Sales de Gauzy J, Accadbled F. Delayed reconstruction and high BMI z score increase the risk of meniscal tear in paediatric and adolescent anterior cruciate ligament injury. *Knee Surg Sports Traumatol Arthrosc* 2019;27(3):905-11.

27. Guenther ZD, Swami V, Dhillon SS, Jaremko JL. Meniscal injury after adolescent anterior cruciate ligament injury: how long are patients at risk? *Clin Orthop Relat Res* 2014;472(3):990-7.
28. Ramski DE, Kanj WW, Franklin CC, Baldwin KD, Ganley TJ. Anterior cruciate ligament tears in children and adolescents: a meta-analysis of nonoperative versus operative treatment. *Am J Sports Med* 2014;42(11):2769-76.
29. Henry J, Chotel F, Chouteau J, Fessy MH, Berard J, Moyon B. Rupture of the anterior cruciate ligament in children: early reconstruction with open physes or delayed reconstruction to skeletal maturity? *Knee Surg Sports Traumatol Arthrosc* 2009;17(7):748-55.
30. Malatray M, Raux S, Peltier A, Pfirrmann C, Seil R, Chotel F. Ramp lesions in ACL deficient knees in children and adolescent population: a high prevalence confirmed in intercondylar and posteromedial exploration. *Knee Surg Sports Traumatol Arthrosc* 2018;26(4):1074-9.
31. Seil R, Mouton C, Coquay J, Hoffmann A, Nuhrenborger C, Pape D, et al. Ramp lesions associated with ACL injuries are more likely to be present in contact injuries and complete ACL tears. *Knee Surg Sports Traumatol Arthrosc* 2018;26(4):1080-5.
32. DePhillipo NN, Moatshe G, Brady A, Chahla J, Aman ZS, Dornan GJ, et al. Effect of Meniscocapsular and Meniscotibial Lesions in ACL-Deficient and ACL- Reconstructed Knees: A Biomechanical Study. *Am J Sports Med* 2018;46(10):2422-31.
33. Peltier A, Lording TD, Lustig S, Servien E, Maubisson L, Neyret P. Posteromedial meniscal tears may be missed during anterior cruciate ligament reconstruction. *Arthroscopy* 2015;31(4):691-8.
34. Stephen JM, Halewood C, Kittl C, Bollen SR, Williams A, Amis AA. Posteromedial Meniscocapsular Lesions Increase Tibiofemoral Joint Laxity With Anterior Cruciate Ligament Deficiency, and Their Repair Reduces Laxity. *Am J Sports Med* 2016;44(2):400-8.
35. Thaunat M, Jan N, Fayard JM, Kajetanek C, Murphy CG, Pupim B, et al. Repair of Meniscal Ramp Lesions Through a Posteromedial Portal During Anterior Cruciate Ligament Reconstruction: Outcome Study With a Minimum 2-Year Follow-up. *Arthroscopy* 2016;32(11):2269-77.
36. Pauvert A, Robert H, Gicquel P, Graveleau N, Pujol N, Chotel F, et al. MRI study of the ligamentization of ACL grafts in children with open growth plates. *Orthop Traumatol Surg Res* 2018;104(8S):S161-S7.
37. Geffroy L, Lefevre N, Thevenin-Lemoine C, Peyronnet A, Lakhil W, Fayard JM, et al. Return to sport and re-tears after anterior cruciate ligament reconstruction in children and adolescents. *Orthop Traumatol Surg Res* 2018;104(8S):S183-S8.
38. Yang BW, Liotta ES, Paschos N. Outcomes of Meniscus Repair in Children and Adolescents. *Curr Rev Musculoskelet Med* 2019;12(2):233-8.
39. Krych AJ, Pitts RT, Dajani KA, Stuart MJ, Levy BA, Dahm DL. Surgical repair of meniscal tears with concomitant anterior cruciate ligament reconstruction in patients 18 years and younger. *Am J Sports Med* 2010;38(5):976-82.
40. Alessio-Mazzola M, Lovisolo S, Capello AG, Zanirato A, Chiarlone F, Formica M, et al. Management of ramp lesions of the knee: a systematic review of the literature. *Musculoskelet Surg* 2019.

# Radiographic exploration in athletic children

Hubert Ducou Le Pointe

Department of Pediatric Radiology – Armand-Trousseau Hospital  
26 Avenir du Dr A Netter – 75012 Paris

There has been an increase in sports-related trauma in children during the past decade. Musculoskeletal injuries in child and adolescent athletes may be related to acute trauma or excessive repetitive load.

Similar to the adult population, acute trauma may lead to fractures or dislocations. However, dislocations are quite rare in younger children. Fractures from a direct impact are quite particular since they risk damaging the growth plate and occur most often in skeletons with a thick periosteum composed of a strong cartilaginous component.

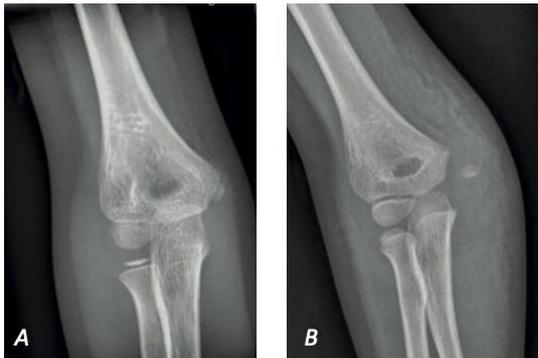
Acute lesions of the soft tissues, ligaments, and tendons are less frequently encountered in children and require particular attention during diagnostic evaluation [1]. Bony structures and physes are weaker than the ligaments and tendons, thus accounting for the differences in the proportions of these injuries in the pediatric population. Furthermore, the growth plate is especially fragile during growth spurts and around the time of fusion.

Injuries secondary to excessive and repetitive load on an immature skeleton are often due to inappropriate training regimens in the context of high-level competition sports. There is often associated overinvestment from the family in the child's sporting career [1].

Imaging modalities must be chosen depending on the suspected diagnosis and the anatomic location of interest. Different imaging techniques assess different anatomical structures, possess specific limitations, and are not equally accessible, all of which must be taken into consideration when choosing an imaging modality.

**Conventional radiographs** are generally the primary modality of choice and are the standard for the evaluation of bony structures. These are obtained after a thorough physical exam, thus justifying the need for complementary investigations. Requests for imaging must be made in a written format and must abide by public health regulations, thereby favoring dialogue between the requesting physician and the radiologist. Conventional radiographs may be used for the diagnosis of acute fractures, lesions secondary to excessive load, or guide the diagnosis toward an infectious or tumoral cause. Radiographs must be of good quality and undertaken only after proper pain management has been prescribed. Two orthogonal views are required and are eventually completed with complementary views depending on the physical assessment. Comparative radiographs are not recommended. In case of doubt on a pathological finding, references containing the different anatomical variants must be

consulted instead. If there is a doubt depending on the child's skeletal age (appearance of the epiphysis and ossification centers), the findings should instead be compared to other radiographs found in the picture archiving and communications systems (PACS) of the imaging department. The skeletons of children differ from those of adults due to the secondary ossification centers at the epiphyses and the apophyses. The growth plate separating the epiphysis from the metaphysis and the apophysis must not be misdiagnosed as fractures. Their aspect may lead to confusion especially on oblique views. Similarly, certain synchondroses, secondary ossification centers (figure 1), or accessory bones such as sesamoids (figure 2) may further lead confusion [2,3].



**Figure 1:** Anteroposterior radiographs of the right elbow of a 6-year-old girl; A) Normal aspect of the medial epicondyle not to be interpreted as a fracture; B) The medial epicondyle is displaced from the epiphysis with surrounding soft tissue edema leading to the diagnosis of a fracture.



**Figure 2:** Right ankle radiograph of a 17-year-old boy. Presence behind the talus of an os trigonum.

Of all the synchondroses, the ischiopubic synchondrosis, a simple anatomical variant, is most often the source of confusion in symptomatic patients. In fact, its radiographic appearance may sometimes be confused with a stress injury.

Secondary ossification centers, especially at the level of the elbow, may lead to misdiagnosis. Knowledge of the age of appearance of these ossification centers is of great importance (table 1).

**Table 1:** Age of appearance of ossification centers

Ossification center	Age of appearance (years)
Capitellum	1-3
Radial head	5-6
Internal condyle	5-8
Trochlea	11
Olecranon	10-13
External condyle	10-12

Concerning the secondary ossification centers, the pelvis is an anatomical region containing a large number of apophyses which may be the source of acute or chronic avulsions (figure 3)



**Figure 3:** Anteroposterior radiograph centered over the right hip in a 14-year-old boy. Presence of an acute avulsion fracture of the secondary ossification center of the ischium.

Apophyses generally appear on radiographs between 13 and 15 years of age [4]. Acute avulsions are easily diagnosed, but when the diagnosis is missed, healing takes place with an extensive callus which may be mistaken for a tumor. As a result, knowledge of the age of appearance and fusion of the apophyses is of great clinical value. Every apophysis is the site of muscular insertions that are stressed by specific movements during sports (table 2).

**Table 2:** Pelvic apophyses: Muscular insertions and specific activities leading to avulsion fractures.

Apophysis	Muscles	Sport
Anterior superior iliac spine	Sartorius and tensor fasciae latae	Sprinting
Anterior inferior iliac spine	Rectus femoris	Football (Soccer)
Ischial tuberosity	Semimembranosus, semitendinosus, biceps femoris	Jumping, hurdling, gymnastics
Iliac crest	Internal and external oblique, transverse abdominis, Gluteus medius, tensor fasciae latae	Jumping, hurdling, gymnastics
Lesser trochanter	Iliopsoas	Jumping, dancing, gymnastics

Accessory ossicles of the ankle and foot are frequent findings and must not be misdiagnosed as avulsion fractures. The absence of edema and a periosteal reaction on follow-up radiographs can make the distinction.

Stress fractures must be recognized on conventional radiographs. The proximal tibia is the most frequent site of stress fractures in children and adolescents, whereas the 2<sup>nd</sup> metatarsal is more frequently encountered in adults. The fracture line is generally visible as a dense metaphyseal line that lies orthogonal to the axis of the tibia (figure 4).



**Figure 4:** Anteroposterior radiograph of the left tibia in a 6-year-old boy. The dense metaphyseal line orthogonal to the axis of the tibia makes the diagnosis of a stress fracture.

These fractures may be accompanied by a periosteal reaction that may be misdiagnosed as an Ewing's sarcoma. The diagnosis may be more difficult if the fracture line is not visualized initially. In such cases, an MRI may be of use.

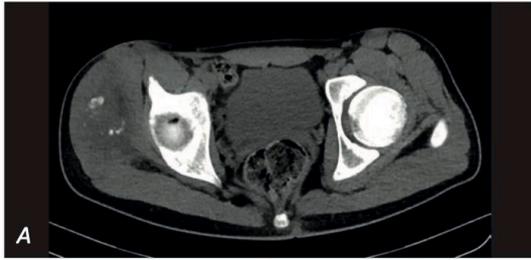
Not all lesions require extensive explorations in order to make their diagnosis. Such lesions include periosteal desmoids (microtraumatic lesions at the distal insertion of the 3<sup>rd</sup> adductor tendon situated at the posteromedial aspect of the distal femoral metaphysis) and osteochondroses. The essential osteochondroses include those located at the tip of the patella (Sinding-Larsen-Johansson syndrome), tibial tuberosity (Osgood-Schlatter disease) (figure 5), insertion of the Achilles tendon on the calcaneus (Sever's disease), metatarsal heads (Frieberg disease), and navicular bone (Köhler disease) [5].



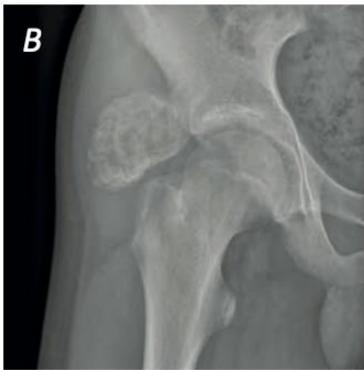
**Figure 5:** Lateral Radiograph of the left knee in a 13-year-old football (soccer) player presenting with chronic mechanical pain at the tibial tuberosity. The patellar tendon is thickened at its insertion on the tibial tuberosity. This appearance confirms the clinical diagnosis of Osgood-Schlatter disease.

Certain repetitive microtraumatic lesions may be responsible for osteochondritis in athletic children, most notably at the level of the elbow in pitchers, as well as the knee [5,6]. The diagnosis is made on conventional radiographs. Complementary imaging using MRI or an arthro-scanner may be required if radiographs are non-diagnostic and symptoms suggest an injury to the articular cartilage. Similarly, repetitive stress of the growth plate may lead to irregular thickening, as is frequently found at the distal ends of both bones of the forearm in gymnasts [7].

The diagnosis of myositis ossificans circumscripta (heterotopic proliferation of bone and cartilage within muscular structures) should be made on conventional radiographs. A previous traumatic injury is found in half of patients and is accompanied by pain and edema. Conventional radiographs may initially be normal, but rapid progression toward calcifications with a zonal phenomenon (mineralization at the periphery with progression toward the center of the lesion) is observed. These two characteristics make the diagnosis of myositis ossificans circumscripta. Initially, CT-scans may be used to better display the zonal phenomenon (figure 6).



**Figure 6:** A) CT-scan with slices passing through the roof of the right acetabulum in a 12-year-old boy presenting with supra-trochanteric inflammatory swelling. The diagnosis of myositis ossificans circumscripta may be made based on peripheral calcifications of an ill-defined mass of the gluteus medius effacing the fascia.



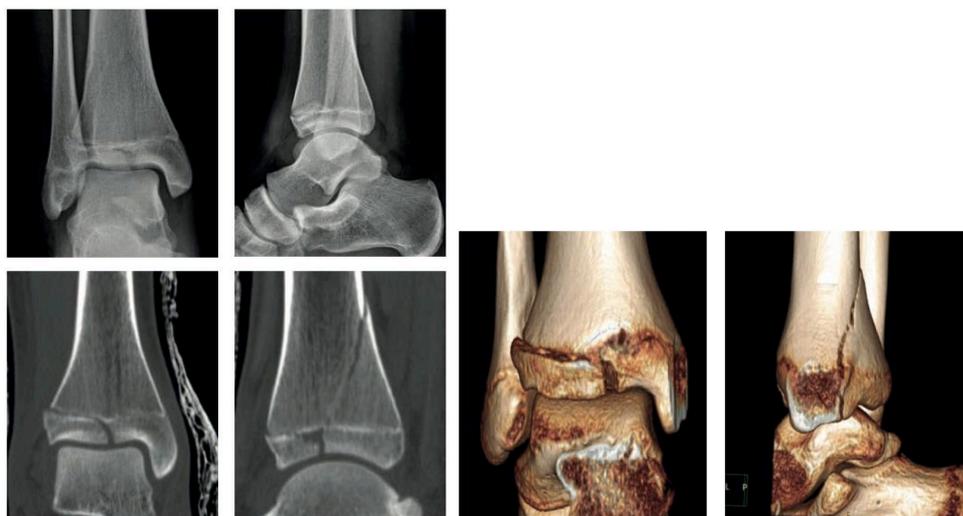
B) Anterolateral radiograph of the right hip one month after the CT-scan confirming the diagnosis due to its zonal effect and the rapid ossification of the lesion.

The diagnosis on MRI is made using the same criteria as previously. Gradient echo sequences are useful for the visualization of peripheral ossification. Furthermore, a severe inflammatory reaction may be found and should not rule out the diagnosis of myositis ossificans circumscripta.

Knowledge of the characteristics of the disease on conventional radiography avoids misdiagnosing normal variants as pathological or the false diagnosis a tumor leading to unnecessary complementary examinations, or even biopsies, which may be troublesome for pathologists with limited experience in musculoskeletal pathologies.

Follow-up radiographs should be ordered after a period of physical inactivity and immobilization with clinical follow-ups. Nevertheless, cartilaginous structures and soft tissue injuries (tendons, ligaments, and muscles) are not directly visualized on conventional radiographs.

**CT-scans** are also widely available, and most patients do not require sedation. Certain anatomical locations may be difficult to assess on conventional radiographs and require secondary evaluation by CT-scans, including the skull, spine, pelvis, hindfoot, shoulder, and wrist. Certain comminuted, complex fractures are also better analyzed using CT-scans [8]. One example is a triplane fracture of the ankle (figure 7).



**Figure 7:** 15-year-old boy presenting with a triplane fracture of the right ankle. A) Anteroposterior and B) lateral radiographs; C) CT-scan with coronal and D) lateral reconstructions; E) Anterior and F) lateral views of a 3D reconstruction using CT-scanning. Multiplane and 3D reconstructions allow for a more detailed evaluation of the relationships between the different fragments and that of the joint line.

CT-scans allow for a better pre-operative surgical evaluation. It also visualizes any intra-articular loose bodies and may also confirm the diagnosis of a stress fracture. CT-scans may be useful in certain avulsion fractures of the posterior apophyseal ring of the spine (limbus), thus differentiating it from a herniated disc, a rare occurrence in children and young adolescents. CT-scans may be ordered in order to evaluate spinal deformities and spondylolysis. Even though it is a powerful tool for the study of cortical bone, it exposes to extremely high doses of ionizing radiation. Moreover, it is limited in the study of muscles, tendons, ligaments, and both hyaline and fibrous cartilage. When MRI is contra-indicated, the evaluation of cartilage requires an arthro-scan, a powerful diagnostic tool.

**Ultrasonography** has multiple advantages: widely available, inexpensive, and based on ultrasounds rather than ionizing radiation. It is often considered as complementary to the physical exam allowing the evaluation in real-time of the area of interest and allows both dynamic maneuvers and comparative views. It is very useful in the assessment of joint effusion, hematomas, and soft tissue injuries (figure 8).



**Figure 8:** 16-year-old boy. Axial image using ultrasonography of the tibia showing a hematoma of the antero-medial aspect of the left tibia after a football (soccer) game.

Ultrasonography is under-employed since it requires the presence of a trained radiologist during image capture. Soft tissue lesions are generally rare in pediatrics since bones in children are weaker than the surrounding muscles and tendons, and the tendons of a large number of muscles do not insert directly on the bone at this age, but via an apophysis. Even though they are rare, muscle injuries can be identified by ultrasound. These lesions are more easily identified after a delay of 24 to 48 hours to the traumatic incident. During the first few hours follow the traumatic incident, soft tissue lesions may be isoechoic, which could be falsely reassuring [9]. Muscular lesions in older adolescents may be analyzed as in adults. Depending on the mechanism of injury, two types of lesions must be differentiated: extrinsic lesions responsible mostly for contusions, hematomas and dilacerations; intrinsic lesions secondary to muscular stretching. These injuries may be responsible for injuries either at the level of the connective tissue, ranging from thickening of the muscular septum to ruptures of said connective tissue, or of the tendon-bone junction, ranging from simple modifications of muscle structure without disruption, to stripping or even avulsion and hematoma formation [9]. Ultrasound may also allow the grading of lesions [10]. Findings on sonography are generally conveyed by specialized sonographers.

Sonography is sometimes requested by specialized teams in order to assess the severity of a ligamentous injury and to guide management. This modality may sometimes diagnose certain fractures but may also misdiagnose certain bony lesions. As a result, in the assessment of bony injury, ultrasonography should be considered as a complementary technique to radiography.

MRI is a technique that does not rely on ionizing radiation and, due to its high spatial resolution and excellent contrast resolution, is the best available all-in-one method for the exploration of anatomical structures in their entirety, especially when radiographs are non-diagnostic. Due to its excellent contrast resolution, it may differentiate cartilaginous structures (joint cartilage, epiphysis, fibrocartilage, and growth plate). It is routinely utilized in the evaluation of the knee and, more particularly, in the assessment of meniscal (figure 9) and ligamentous injuries. These injuries are similar to those found in adults, and their frequency may vary according to sex; injury to the anterior cruciate ligament is less frequently encountered in girls compared to boys [11].



**Figure 9:** MRI of the knee in a 15-year-old boy. Sagittal view using proton density and fat saturation images passing with slices passing through the medial meniscus. A linear meniscal tear is seen extending toward the free edge of the meniscus.

MRI also allows better visualization of traumatic osseocartilaginous lesions that are frequently encountered at the level of the knee. At the level of the hip, osseous lesions (osteochondritis, slipped capital femoral epiphysis) have been thoroughly studied, but no

relations to sports have as of yet been elucidated. However, labral tears have shown a clear association to sports, and may be evaluated using MRI [12]. MRI is an effective technique for the exploration of the spine, even though CT-scans may be used but limited in their utility. It is the modality of choice in the exploration of spinal cord and nerve root injuries, especially when neurological signs are encountered on physical exam. MRI is particularly useful for the assessment of pathologies of the intervertebral discs and the limbus. As a result, MRI may differentiate between a herniated disc and an avulsion fracture of the posterior apophyseal ring. MRI is generally not the imaging modality of choice for the evaluation of muscles in general except for the muscles of the shoulder and pelvic girdles, especially in patients complaining of pain in the inguinal area [13]. It is also indicated in patients presenting with pain refractory to conservative treatment. Moreover, MRI is useful in the diagnosis of bony contusions and may provide insight in patients presenting with chronic pains without identifiable lesions (figure 10).



**Figure 10:** 16-year-old girl presenting with right knee pain 3 months after initial trauma without identifiable bony lesions. Coronal view using T2-weighted sequences with fat saturation, more specifically Short-Tau Inversion Recovery (STIR). An isolated bony contusion is identified explaining the pain experienced by this patient.

MRI is an essential technique when an incidental finding of a potentially aggressive lesion is made on conventional radiographs in the setting of sports-related trauma and must also precede any biopsies. Contrarily, immediate use of an MRI instead of conventional radiographs may lead to misdiagnosis and unnecessary complementary examinations, including biopsy. These may prove difficult to analyze by pathologists and may lead to further errors in diagnosis (e.g. stress fractures and myositis ossificans).

Even though the demand for MRI is increasing, urgent accessibility remains limited. It also requires a cooperative child since certain positions during acquisition may be uncomfortable. This technique is very sensitive, and the quality of the image may be altered by numerous artefacts, two of the most frequent being movement artefacts and artefacts due to metallic foreign bodies. Absolute contra-indications to MRI (electronic materials, metallic bodies susceptible of being dislodged or causing burns) must be eliminated before the exam is undertaken. T1-weighted images are obtained in order to verify bone marrow signal, a signal that normally varies with age due to fatty infiltration of the bone marrow. The most sensitive

sequence for the detection of bony and soft tissue lesions is Short Tau Inversion Recovery (STIR): T2-weighted sequence associated with fat saturation. The majority of these lesions have a decreased signal intensity on T1 and an increased signal intensity on T2-weighted images. T2\* allows for a better visualization of the cartilages and, more specifically, the growth plate. Proton density with fat saturation are optimal in the assessment of the different joints.

Injection of contrast material has not been approved in France for use in children. As a result, pediatric orthopedic surgeons working in France avoid ordering arthro-MRIs. Intravenous injections of gadolinium are generally not necessary but may be useful in the analysis of tissue perfusion, especially when the diagnosis of osteonecrosis is considered, or the vascular supply of the growth plates or related pathologies must be evaluated. When infection or malignancy is shown, IV injection of contrast is recommended.

In conclusion, imaging modalities that provide digital slices of the body have become common practice in the exploration of a large number of clinical scenarios related to sports in pediatric patients. However, as a rule, radiography remains the initial imaging modality. Ultrasonography is utilized for the exploration of muscular and tendinous lesions, rare findings in the pediatric population, CT-scanning is limited in its indications, and MRI has excellent contrast resolution and its radiation-free nature has many uses.

# Radiographic diagnosis of osteochondritis of the knee

Dr Karen Lambot

Cabinet de Radiologie  
17 Cours Joseph Thierry 13001 Marseille  
17 Avenue William Booth, 13012 Marseille

Radiology Department, North Hospital, Chemin de Bourrely, 13015 Marseille

## Introduction

Osteochondritis dissecans of the knee is a localized alteration of subchondral bone that affects the bordering articular cartilage that may progress to instability of the fragment. The natural history of the disease may lead, in its most severe form, to loosening of the subchondral osseous fragment and liberation within the joint [1].

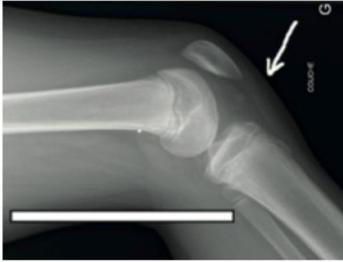
The juvenile form must be differentiated from the adult form. It manifests during infancy; most frequently starting at preadolescence and affects children with open physes [2]. The pathophysiology is not well elucidated, although repetitive microtrauma is known to play a major role in physically active children, especially in high-level athletes.

## Radiographic diagnosis

Conventional radiographs

### *Technique*

Most cases of osteochondritis dissecans can be diagnosed on conventional radiographs, the primary imaging modality of choice in patients complaining of knee pain [3]. Along with the classic anteroposterior and lateral views, complementary views may be useful, such as a tunnel view, where the beam is directed with a certain inclination relative to the film thereby optimally visualizing the posterior aspect of the femoral condyles, a frequent location of these lesions (figure 1) [2]. A tunnel view is more easily realized than the Schuss view, in which the patient must be placed in a standing position, sometimes difficult to perform in children.



**Figure 1:** Tunnel view

*Location and diagnosis*

On lateral views of the knee, the majority of these lesions are located within the subchondral bone at the level of the medial femoral condyle and situated between two lines: The line first is extended anteriorly relative to the intercondylar roof (Blumenstaat line); The second is extended distally from the posterior cortex of the distal femoral diaphysis (figure 2) [4].



**Figure 2:** Locations of osteochondral lesions on a lateral radiograph of the knee.

On anteroposterior radiographs of the knee, osteochondral lesions are most often located on the medial femoral condyle (80% of cases). The most frequent and classic location is the lateral (intercondylar) aspect of the medial femoral condyle, followed by the extended classic and infero-central locations (figures 3 and 4), and finally the lateral condyle (20% of cases) [2].



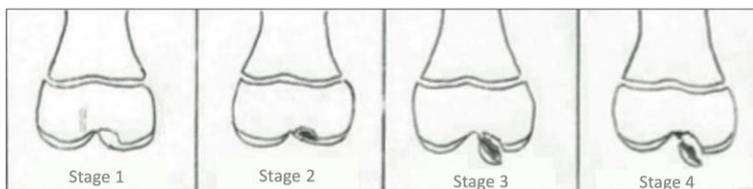
**Figure 3:** Usual locations of osteochondral lesions on anteroposterior radiographic views of the knee.



**Figure 4:** Anteroposterior and lateral radiographs of the knee. Stage 3 extended intercondylar osteochondritis dissecans at the level of the middle third of the medial femoral condyle (arrow).

The lesions may be divided into 4 stages based on a simplified radiographic classification (figures 4 and 5) [5]:

- Stage 1: lacunar lesions with radiolucent recess.
- Stage 2: nodular structure with condensed osseous sequestrum that remains at the same level as the condyle.
- Stage 3: Sleigh-bell aspect with condensed sequestra that are often dislodged from the bony surface of the condyle.
- Stage 4: Intra-articular loose body.



**Figure 5:** Radiographic classification of osteochondritis dissecans into 4 stages.

Conventional radiographs are a reliable method for situating the lesion (differentiating medial and lateral condylar lesions) and measuring its size. When the fragment is ossified, the reliability of conventional radiographs for the evaluation of fragmentation, displacement, central radiographic density, borders, and contours is moderate to good. However, reliability decreases when it comes to estimating the radiographic density of the surrounding borders and epiphyseal bone [6].

There is no consensus on the radiographic characteristics used to monitor the healing process of osteochondral lesions. In fact, even a general definition of radiographic healing is lacking [3]. Despite these limitations, conventional radiographs have excellent reliability for the global evaluation of these lesions [7].

Finally, conventional radiographs may also be used to assess skeletal maturity and rule out other bone lesions [2,6].

#### Arthro-scan

An arthro-scan is an imaging technique consisting in a fluoroscopy-guided injection of contrast material within the joint. A CT-scan is then undergone which visualizes the surface of the articular cartilage. In the context of osteochondritis, an arthro-scan may demonstrate the partially detached character of an osteochondral fragment. It is more frequently utilized in adults than children owing to its high radiation exposure. It is often substituted with an MRI: a powerful, non-invasive and radiation-free imaging modality.

## MRI

### *Technique*

The evaluation of child with a painful knee using MRI generally requires a T1-weighted sequence – most often sagittal views – and sequences with high sensibility to bony anomalies, especially bone edema, such as fat saturation proton density (or T2-weighted images) images with sagittal, coronal, and axial views.

### *Location and diagnosis*

On sagittal views, if the femoral condyle is divided into 3 parts, 1/3 of osteochondral lesions are located at the middle third, 1/3 at the posterior third, and 1/3 between the middle and posterior thirds [2].

On coronal views, utilizing a similar division system, the majority of lesions are located at the intercondylar notch and the middle third of the femoral condyle (73%) [2].

In patients with early disease and a stable lesion, there is visible hypertrophy of the epiphyseal cartilage at the level of the osteochondral fragment. These alterations may be visualized on both MRI and histology [2,8,9].

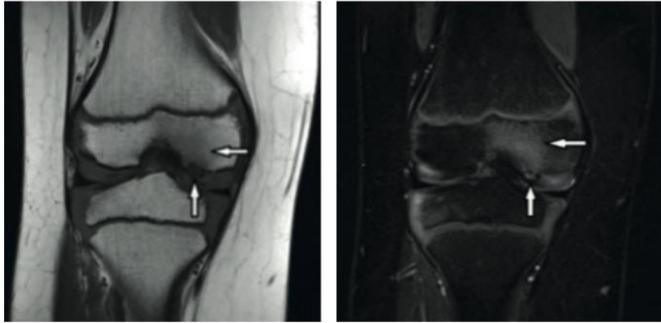
The secondary physis, which is spherical in nature and responsible for the growth of the epiphysis, undergoes a process of circumferential enchondral ossification, similar to that of the primary physis which is responsible for the longitudinal growth of long bones. This secondary physis is clearly identified on MRI of skeletally immature children as a thin line with increased signal intensity, an aspect similar to that of the primary physis. Some authors describe an interruption of the secondary physis at the level of the osteochondral lesion, whereas it remains continuous at the sane portion of the femoral condyle or the contralateral condyle (figure 6) [2].



**Figure 6:** Coronal fat saturation proton density images. Primary and secondary physes (black arrows); osteochondritis dissecans with interruption of the secondary physis (white arrow).

Bone edema of the epiphysis is frequently found in children with osteochondritis dissecans and is generally isolated to the subchondral bone or deeper (figure 7) [2,8,10].

Upon histological examination, no necrosis or inflammation is found [8]. This could be the reflection of repetitive micro-trauma or a direct impact during physical activity and may be in relation with the pain described by the patient [2].

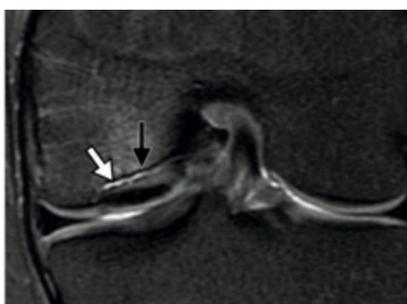


**Figure 7:** T1-weighted image (left) and fat saturation T2-weighted image (right); osteochondritis dissecans (vertical arrow) and deep edema of epiphyseal bone (horizontal arrow).

#### *Instability of the lesion*

The classic signs of instability of the osteochondral fragment described in adults are sensitive (100%) but non-specific (11%) in children [11]. As a result, revised criteria were described for the juvenile form with the addition of 3 secondary signs that, when combined, increase specificity to 100% [12]:

- High T2 signal intensity rim: indicating instability only if it has the same signal intensity as adjacent joint fluid, most often appearing as a fluid-filled cleft between the osteochondral fragment and the underlying bone (figure 8) [8], not to be confused with certain clefts with high T2 signal intensity that correspond to fibro-vascular tissue [13];
- Outer rim of low T2 signal intensity: may represent organized fibrous tissue or sclerotic bone at the bone-fragment interface (figure 8) [8];



**Figure 8:** Coronal fat saturation T2 image. Signs of instability: High T2 signal intensity rim surrounding the osteochondral lesion with similar signal intensity to adjacent joint fluid (white arrow) and outer rim of low T2 signal intensity (black arrow).

- Multiple breaks in the subchondral bone plate on T2 images: may indicate a larger degree of disorganization at the bone-cartilage interface with extensive replacement with fibro-vascular tissue (figure 9) [8].
- Cysts surrounding the lesion: indicating instability only if they are multiple or single with a diameter >5mm (figure 9) [3,4,12].



**Figure 9:** Sagittal fat saturation T2 image. Signs of instability: Multiple breaks in the subchondral bone plate (arrowhead) and single cyst measuring >5mm in diameter (curved arrow).

However, MRI criteria of instability are absent in most lesions [12].

In symptomatic unstable lesions, fragment characteristics on MRI will guide surgical management depending on the estimated viability of the fragment. An unstable fragment is deemed non-viable or irreversible when it is purely cartilaginous (lack of subchondral bone), is composed of multiple pieces, or presents a loss or damage of articular cartilage [3].

**Differential diagnosis: normal variants of femoral condyle ossification.**

Differentiating between typical development and early osteochondritis dissecans is not always a simple task, especially if the child complains of non-specific knee pain [2,14]. Demographically, normal ossification precedes the age of appearance of osteochondral lesions, which are more frequently encountered in adolescence [14]. Jan et al. reported that age is an important factor when differentiating between these two entities, since variations in ossification do not appear in girls older than 10 years of age and in boys older than 13. Furthermore, osteochondritis dissecans is generally not seen in children younger than 8 years of age. Moreover, if the growth plate is almost fused and the lesion extends toward the intercondylar region, then an ossification variant is less probable [10]. Distinguishing between typical development and osteochondral lesions is often possible on MRI [14], with signs of normal variants of ossification including signal intensity anomalies located exclusively at the posterior inferocentral condyle, an intact articular cartilage, accessory ossification centers (sometimes visible as spiculations), absence of bone edema, and no intercondylar extension in the coronal plane (figure 10) [2,3,14].



**Figure 10:** Sagittal T1 image. Variant of normal ossification (arrow).

Finally, the continuity of the secondary physis may also differentiate between normal ossification variants and osteochondral lesions [2].

If an osseous defect of the femoral condyle is considered as a normal ossification variant, surveillance with follow-up imaging is generally not indicated, except if the pain persists [14].

## Conclusion

The radiographic diagnosis of osteochondritis of the knee is made most often on anteroposterior and lateral radiographs. These radiographs are indicated when a patient presents with knee pain, which is sometimes chronic and ill-localized. Radiographs are used to determine the location of the lesion, its extension, and the radiographic stage. MRI further confirms the diagnosis, allows the analysis of the articular cartilage, and displays signs of instability. The main differential diagnosis of osteochondritis dissecans of the knee is a normal ossification variant of the femoral condyle and depends on the clinical scenario, notably the age of the child, the location of the lesion, and its characteristics on medical imaging.

## References

1. Edmonds EW, Shea KG. Osteochondritis dissecans: editorial comment. *Clin Orthop Relat Res.* 2013 Apr;471(4):1105-6.
2. Laor T, Zbojnicz AM, Eismann EA, Wall EJ. Juvenile osteochondritis dissecans: is it a growth disturbance of the secondary physis of the epiphysis? *AJR* 2012 Nov;199(5):1121-8.
3. Gorbachova T, Melenevsky Y, Cohen M, Cerniglia BW. Osteochondral Lesions of the Knee: Differentiating the Most Common Entities at MRI. *Radiographics* 2018 Sep-Oct;38(5):1478-1495.
4. Harding WG 3rd. Diagnosis of osteochondritis dissecans of the femoral condyles: the value of the lateral x-ray view. *Clin Orthop Relat Res.* 1977 Mar- Apr;(123):25-6.
5. Rey JC, Bedouelle J, Cirotteau Y coll E. Symposium sur les nécroses des condyles fémoraux chez l'enfant et l'adulte. *Ann Orthop Ouest* 1975;7:119-75.
6. Wall EJ, Polousky JD, Shea KG, Carey JL, Ganley TJ, Grimm NL, Jacobs JC Jr, Edmonds EW, Eismann EA, Anderson AF, Heyworth BE, Lyon R; Research on Osteochondritis Dissecans of the Knee (ROCK) Study Group. Novel radiographic feature classification of knee osteochondritis dissecans: a multicenter reliability study. *Am J Sports Med.* 2015 Feb;43(2):303-9.

7. Wall EJ, Milewski MD, Carey JL, Shea KG, Ganley TJ, Polousky JD, Grimm NL, Eismann EA, Jacobs JC Jr, Murnaghan L, Nissen CW, Myer GD; Research in Osteochondritis of the Knee (ROCK) Group, Weiss J, Edmonds EW, Anderson AF, Lyon RM, Heyworth BE, Fabricant PD, Zbojniewicz A. The Reliability of Assessing Radiographic Healing of Osteochondritis Dissecans of the Knee. *Am J Sports Med.* 2017 May;45(6):1370-1375.
8. Zbojniewicz AM, Stringer KF, Laor T, Wall EJ. Juvenile Osteochondritis Dissecans: Correlation Between Histopathology and MRI. *AJR* 2015 Jul;205(1):W114-23.
9. Yonetani Y, Nakamura N, Natsuume T, Shiozaki Y, Tanaka Y, Horibe S. Histological evaluation of juvenile osteochondritis dissecans of the knee: a case series. *Knee Surg Sports Traumatol Arthrosc.* 2010 Jun;18(6):723-30.
10. Jans LB, Jaremko JL, Ditchfield M, Huysse WC, Verstraete KL. MRI differentiates femoral condylar ossification evolution from osteochondritis dissecans. A new sign. *Eur Radiol.* 2011 Jun;21(6):1170-9.
11. De Smet AA, Ilahi OA, Graf BK. Reassessment of the MR criteria for stability of osteochondritis dissecans in the knee and ankle. *Skeletal Radiol.* 1996 Feb;25(2):159-63.
12. Kijowski R, Blankenbaker DG, Shinki K, Fine JP, Graf BK, De Smet AA. Juvenile versus adult osteochondritis dissecans of the knee: appropriate MR imaging criteria for instability. *Radiology.* 2008 Aug;248(2):571-8.
13. O'Connor MA, Palaniappan M, Khan N, Bruce CE. Osteochondritis dissecans of the knee in children. A comparison of MRI and arthroscopic findings. *J Bone Joint Surg Br.* 2002 Mar;84(2):258-62.
14. O'Dell MC, Jaramillo D, Bancroft L, Varich L, Logsdon G, Servaes S. Imaging of Sports-related Injuries of the Lower Extremity in Pediatric Patients. *Radiographics.* 2016 Oct;36(6):1807-1827.

# Is there a place for surgical management in adolescents with Scheuermann's disease?

F.X. Lambert, C. Decante, E. Mayrargue, S. Guillard, A. Chalopin, A. Hamel

## Introduction

Scheuermann's disease, or osteochondritis deformans juvenilis dorsi, was first described in 1920 by Danish orthopedic surgeon and radiologist Dr Holger Wefel Scheuermann who reported a stiff thoracic hyperkyphosis in young apprentice watchmakers [1]. Scheuermann's disease is one of the most frequent causes of back pain in adolescents [2-6], with an incidence varying between 0.04 and 10% [2,7-10]. Nevertheless, an increase in prevalence was observed between 2003 and 2012, increasing from 3.6 to 7.5 per 100,000 [11]. Scheuermann's disease occurs most frequently in children aged 8 to 12 years old, with stiffer forms concerning primarily the ages 12-16 years [9]. There is a clear male predominance with a male to female ratio of 2:1 [11].

The etiology of Scheuermann's disease remains unknown. Mechanical factors have been considered with associations to a bad posture or obesity, but no evidence has been found. According to a Danish cohort study on 35,000 twins, 74% of cases were hereditary [12] and 1.8% were due to a syndrome, mostly Prader-Willi and Marfan's syndrome [11].

The treatment of Scheuermann's disease includes physiotherapy, orthopedic treatment with bracing, and occasionally surgery. Scheuermann's disease is considered a developmental disorder as it progresses during growth. Nevertheless, the natural history of this disease during adulthood remains unknown, thus making surgical intervention controversial.

## Clinical features

Scheuermann's disease is characterized by thoracic or thoracolumbar hyperkyphosis associated with lumbar and cervical compensatory hyperlordosis in order to maintain proper sagittal balance. Progression toward stiffness may cause pain and important aesthetic complaints in adult patients. Hamstring and iliopsoas tightness, and stiffness of the shoulder girdle may also be associated. Furthermore, one third of patients present with non-structural scoliosis and/or L5-S1 spondylolisthesis [2].

There is often confusion between Scheuermann's disease and postural kyphosis. Clinically, patients with postural kyphosis have a flexible kyphotic deformity and do not present with hamstring tightness [13].

Psychological distress may also be associated with Scheuermann's disease and is often underestimated. In a series of 1,070 pediatric subjects, Hom et al. found an associated 5.5%

rate of depression and 4.5% rate of anxiety in patients with Scheuermann's disease, compared to only 1.7% and 0.8% in the general population, respectively [11].

## **Radiographic features**

The radiographic diagnosis of Scheuermann's disease relies on certain criteria established by Sorensen in 1964 [14]. Anterior wedging across 3 consecutive vertebrae superior to 5° confirms the diagnosis [14]. Vertebral endplate irregularities with intervertebral disc herniations through the vertebral endplate (Schmorl nodes) may also be found [2,15].

The stiffness of the kyphotic deformity is best evaluated on hyperextension views.

Alterations in spinal sagittal balance on whole spine sagittal radiographs must be evaluated by drawing a vertical line passing through the center of the auditory meati. This line usually passes just posterior to the femoral heads. Global sagittal alignment is also assessed by measuring the spino-sacral angle, spinal tilt, and spino-pelvic tilt [16]. Alterations in spinal sagittal balance have a much more significant impact on the quality of life of these patients than on aesthetics and determine the progression of the disease [17-20].

MRI may be useful as a complement to conventional radiographs in order to assess the severity of inter-vertebral disc disease and to evaluate the entirety of the Schmorl nodes and endplate irregularities. Finally, spinal cord anomalies that may or may not be secondary to Scheuermann's disease can also be screened [5,21,22].

## **Natural history of Scheuermann's disease**

Scheuermann's disease is a spinal pathology occurring primarily during the later stages of growth, with a natural history that remains difficult to elucidate. Nonetheless, symptoms tend to decrease during adulthood, whereas a slight increase in the kyphotic deformity may be observed during aging. However, in some patients, the kyphotic deformity progresses during adulthood and causes, besides important aesthetic consequences, mechanical pain and neurological complications [23].

Bartynski et al. studied the mean thoracic kyphosis and lumbar lordosis in patients without spinal disease [24]. Their results showed that, in young adults (18-35 years old), thoracic kyphosis was in average 27°, whereas in patients aged 65 years or older, it was 42°. Other authors have suggested that a "normal" kyphosis varies between 27 and 44° in adolescents [15,25], and between 20 and 50° in adults [26-28]. Stagnara et al. suggested that a "normal" kyphosis or lordosis does not exist, and that these values are only indicative and not normative [28].

In a study published in 2017, Ristolainen et al. followed 19 patients with Scheuermann's disease who were treated non-operatively with an average follow-up of 46 years and concluded that the deformity progressed slowly [29]. This progression, however, did not predict the appearance of symptoms. Mean age at final follow-up was 64.7 years, and

patients had a mean kyphosis of 60°. In their series, kyphosis progressed by 14° but non-uniformly. Progression of the kyphotic deformity <10° was found in 42% of patients, and >20° in 32% of patients. A progression of 32° was reported in 3 cases (one female aged 65 years old in whom kyphosis increased from 48° to 80° over 36 years; two males, one aged 73 years old in whom kyphosis increased from 28° to 60° and one aged 76 years old in whom kyphosis increased from 50° to 82° over 59 years). The severity of the kyphotic deformity at the time of diagnosis did not predict progression of the deformity. No significant differences in terms of quality of life were found in patients with more progressive curves. Moreover, a significant increase in vertebral body wedging and lumbar lordosis was observed.

In a study conducted on 67 subjects with Scheuermann's disease and a control group, Murray et al. found that, after a mean follow-up of 32 years, patients who had not been operated presented certain functional restrictions but without major limitations to activities of daily living [30]. Patients with Scheuermann's disease also reported more back pain compared to controls, with no limitations in activities of daily living or during work. In fact, no differences were found based on the type of work, the number of days absent from work due to low-back pain, aesthetic complaints, painkiller use, participation in hobbies, and the presence of numbness in the lower extremities. Furthermore, patients with thoraco-lumbar deformities presented with more functional limitations than purely thoracic deformities. No differences were found between subjects with Scheuermann's disease and controls in terms of marital status, but patients with a kyphosis >85° were more frequently single and had a lower pulmonary capacity.

In a study conducted on 49 subjects with Scheuermann's disease who were treated non-operatively with a mean follow-up of 37 years, Ristolainene et al. found a higher prevalence of back pain and limitations in activities of daily living compared to the general population [31]. However, this increased prevalence was not correlated to the severity of the kyphosis. In fact, no differences in the intensity of the back pain or functional limitations were found between subjects with a kyphosis <40° and >60°.

## **Treatment**

### **Conservative treatment**

The treatment of Scheuermann's disease depends primarily on the intensity of the pain, the development of neurological or cardiopulmonary complications, aesthetic considerations, and the degree and progression of the deformity, all the while taking into account the residual growth of the spine.

Physiotherapy includes softening of the hips and stretching of the hamstrings as well as the spinal erectors and stabilizers. Although physiotherapy does not slow the progression of the disease, it is still recommended for symptomatic patients presenting with a stiff curve and may even be complementary to bracing in order to counteract the stiffness of the deformity. In a study conducted on 351 subjects with Scheuermann's disease aged 17 to 21 years old, Weiss et al. found a significant decrease of pain (16-32%) with physiotherapy, thereby suggesting the positive effects of this type of management on the primary complaint of this disease in young adults.

Treatment by bracing in patients during growth may improve the kyphotic deformity and even lead to vertebral remodeling. Nonetheless, once the brace has been weaned, a loss of correction is often observed and may even reach 30% [30-34]. Flexible kyphosis, early management with a deformity  $<65^\circ$ , an initial correction  $>15^\circ$  with a brace, and a residual growth of the spine of at least 1 year are factors of good prognosis when treatment with a brace is considered [32,33]. A stiff kyphosis with a deformity  $>65^\circ$ , vertebral wedging  $>10^\circ$ , and a finished spinal growth are considered as risk factors for failure of treatment by bracing.

## **Surgical treatment**

### Indications

Indications for surgical management are controversial, and objective assessments are scarce in the literature. The large variability of the natural history of Scheuermann's disease renders an estimation of the risk-benefit ratio of surgical management difficult to establish. Additionally, surgery in Scheuermann's disease is complex and may lead to potentially serious complications [34]. According to the Scoliosis Research Society, less than 1% of spine surgeries are conducted on patients with Scheuermann's kyphosis [35]. The frequency of patients with Scheuermann's disease who are managed operatively is actually stable [11], with recently published series indicating an apparent increase [36].

Although the severity of the deformity in the sagittal plane is a major criterion leading to surgical management in patients with Scheuermann's disease, the angular kyphotic value does not seem to be the primary factor in surgical decision-making. The threshold of the sagittal Cobb angle indicating the need for surgery varies greatly between publications [34,37-44]. Furthermore, Polly et al. found no differences in maximal Cobb angle between operated ( $70^\circ$ ) and non-operated ( $73^\circ$ ) subjects [45]. The majority of authors recommend surgical management in progressive deformities that are superior to  $60-75^\circ$  and not controlled by bracing, in patients with back pain resistant to lifestyle modifications (physical activity, NSAIDs  $>6$  months), appearance of neurological or cardiopulmonary complications, or in case of significant aesthetic complaints [23,34,38-40,46-55]. Patients and their families must be made aware of the expected benefits and the risks of surgery. The evaluation of global sagittal alignment is paramount, especially in thoraco-lumbar deformities, since these locations disrupt the harmony of the different curves in the sagittal plane and alter the sagittal alignment of the spine.

Care must be taken as parents of children with Scheuermann's disease suffering from back pain and aesthetic complications may pressure surgeons into adopting certain treatment modalities [45].

### Surgical technique: Anterior, posterior or combined approach?

In 1975, Bradford et al. were the first to report a series of 22 patients who were treated surgically by posterior fusion using only the Harrington technique [56]. They reported a mean correction of  $25^\circ$  (reduction from  $72$  to  $47^\circ$ ) and a mean loss of correction of  $21^\circ$  in 16 patients (72% of cases) [23].

In order to avoid this loss of correction, a combined approach with anterior release and posterior fusion was long considered the gold standard in treatment and was particularly recommended for the treatment of severe and stiff deformities [37,43,46,55,57-61]. As other surgical options were developed, specifically pedicle screws, very satisfying results by posterior approach only have been reported. In fact, multiple studies have concluded that the quality of the correction is comparable between posterior-only and combined approaches.

In fact, combined approaches are associated with higher complication rates than a posterior-only approach [7,11,41,62]. The posterior-only approach leads to less blood loss, decreased operative times, and seems to decrease the risk of adjacent segment disease [42,62,63]. In their case series, Riouallon et al. found no significant differences in complication rates between the two methods and reported 3 types of complications that were specific to the anterior approach [61]. Mizashahi et al. did not show any complications in patients treated with a posterior-only approach [64]. Furthermore, the length of hospital stay was longer for patients operated by a combined approach [11,36].

The majority of authors report no significant loss of correction in the posterior-only approach compared to the combined approach [58,61,62,65,66], except for Temponi et al. who found that the combined approach showed less loss of correction [7]. As a result, the number of patients with Scheuermann's kyphosis treated by posterior fusion without anterior release has significantly increased from 34 to 78% between the years 2000 and 2008 [11,36]. Furthermore, complication rates have decreased from 22.6% to 15.5% during the same time frame (a rate that still remains especially high) [11,36].

Ponte or Smith-Peterson osteotomies during a posterior-only correction allows for better correction [57,58,64,65,67]. Correction of the deformity by posterior compression without osteotomy lengthens the anterior column of the spine and increases the risk of spinal cord elongation and of anterior spinal artery spasms. One or more osteotomies around the apex of the deformity would shorten the posterior column without lengthening the spine anteriorly, and thereby decrease the risk of neurological complications.

Implant density has also been evaluated. It is commonly believed that an increase in the density of the implants favors the correction of kyphosis. However, Behrbalk et al. showed a similar correction between an implant density of 100% and 50% around the apex of the deformity [38]. The decrease in implant density is associated with a decrease of complications by 50% and surgical cost by 32% [38]. Finally, no significant differences were found between a posterior-only approach and a combined approach in terms of pain, functional outcome, and aesthetic satisfaction [61,62].

### Level of instrumentation

Surgery for Scheuermann's disease must include the entire kyphotic deformity. The choice of proximal and distal instrumentation levels and the degree of correction is not clearly elucidated in the literature. For some authors, correction of the kyphotic deformity must not surpass 50% [34], with a post-operative thoracic kyphosis ranging between 40 and 50° [16,43,53,68-70]. In fact, over-correction increases the risk of proximal junctional kyphosis (PJK), whereas under-correction would maintain the compensatory hyperlordosis at the

lumbar level which would accelerate spinal degeneration in the long term [71,72]. In order to prevent the occurrence of PJK, the upper instrumented vertebra should be chosen as the most proximal vertebra that is included within the thoracic kyphotic deformity [55,60].

Recently, some authors have studied the relationship between pelvic and sagittal parameters. An analysis of these parameters allows to predict the estimated post-operative lumbar lordosis, and by conjunction, the thoracic kyphosis [16,19,69,70,73]. It has been shown that patients with Scheuermann's disease developing PJK after correction of their hyperkyphosis were those with a high pelvic incidence and a significant post-operative deficit in lumbar lordosis [41,74]. As a result, the correction of thoracic kyphosis must take into account the pelvic incidence. Nasto et al. have established an equation for the prevention of such a phenomenon [74]: %LL correction = 0.66 x (%TK correction) – 2.

The choice of lower instrumented vertebra remains controversial in the literature. This choice must preserve the maximum of lumbar mobility, all the while preventing distal adjacent segment disease, especially distal junctional kyphosis (DJK). DJK has not been well-defined in the literature. Zhu et al. defined DJK as a sagittal Cobb angle >10° between the superior endplate of the lower instrumented vertebra and the inferior endplate of the immediately adjacent distal vertebra [75]. Another risk factor for the development of DJK is a significant shift from lordosis to neutral or kyphosis of the intervertebral disc immediately distal to the lower instrumented vertebra. In a meta-analysis by Gong Y et al. evaluating four studies and 173 patients, 20.8% of patients who were operated for Scheuermann's disease developed DJK post-operatively, and 27.8% of those with DJK required revision surgery [76].

Cho et al. developed the concept of the stable sagittal vertebra (SSV) [77]. This is defined as the most proximal lumbar vertebral body bisected by the vertical line from the posterior-superior corner of the sacrum. The first lordotic vertebra (FLV) is defined as the vertebra lying immediately distal to the most proximal lordotic disc. SSV and FLV can sometimes be superimposed. On the one hand, the choice of the FLV as the lower instrumented vertebra does not lead to higher incidences of DJK and allows for the preservation of distal motion segments [15,53,78]. On the other hand, the choice of the SSV as lower instrumented vertebra decreases the risk of adjacent segment disease [76,77,79-82]. In fact, in a series including 20 subjects with Scheuermann's disease operated by spinal fusion ending on the FLV, Cobden et al. found that 15% of patients developed DJK postoperatively [65].

In subjects with Scheuermann's disease with a thoraco-lumbar kyphotic curve, Zhu et al. found that ending the construct on the FLV was sufficient, but that in thoracic curves, it was necessary to include the SSV in order to decrease the risk of DJK [75].

### Complications

Complication rates after surgery for Scheuermann's disease are especially high. Around 10% of subjects treated by posterior fusion report at least one complication, and 20% of those treated by a combined approach [11,36]. Huq et al. published a meta-analysis including 1,829 subjects with Scheuermann's disease treated by surgery between the years 1950 and 2017 [66]. Correction of the kyphotic deformity was associated with significant neurological complications up to 8% in this meta-analysis. The authors reported 25% instrument failure, 14% PJK, and 14% DJK in posterior-only approaches, with 10% requiring revision surgery, as

well as 26% PJK, 20% DJK, 21% respiratory complications, and 6% cardiovascular complications in combined approaches, with 11% requiring revision surgery.

## Conclusion

Scheuermann's disease is a deformity of the growing spine with a relatively benign long-term natural history. Nevertheless, the deformity may progress in adulthood and lead to mechanical pain and neurological complications such as adult spinal deformity.

It is difficult to predict which patients will be symptomatic during adulthood, and which patients could benefit from surgical treatment. A comprehensive analysis of the sagittal alignment must be undertaken before proposing operative treatment, which presents with a high rate of severe complications. If surgery is considered, recent data suggest the need for vertebral osteotomies and posterior spinal fusion ending on the stable sagittal vertebra.

## Bibliographie

1. Scheuermann HW. Kyphosis dorsalis juvenalis. *Ugeskr Laeger.* 1920;(82):38593.
2. Ali RM, Green DW, Patel TC. Scheuermann's kyphosis. *Curr Opin Pediatr.* févr 1999;11(1):705.
3. Greene TL, Hensinger RN, Hunter LY. Back pain and vertebral changes simulating Scheuermann's disease. *J Pediatr Orthop.* févr 1985;5(1):17.
4. Cleveland RH, DeLong GR. The relationship of juvenile lumbar disc disease and Scheuermann's disease. *Pediatr Radiol.* févr 1981;10(3):1614.
5. Paajanen H, Alanen A, Erkintalo M, Salminen JJ, Katevuo K. Disc degeneration in Scheuermann disease. *Skeletal Radiol.* 1989;18(7):5236.
6. Lowe TG. Scheuermann's kyphosis. *Neurosurg Clin N Am.* avr 2007;18(2):30515.
7. Temponi EF, de Macedo RD, Pedrosa LOG, Fontes BPC. Scheuermann's kyphosis: comparison between the posterior approach associated with smith-petersen osteotomy and combined anterior-posterior fusion. *Rev Bras Ortop.* déc 2011;46(6):70917.
8. Scoles PV, Latimer BM, Digiovanni BF, Vargo E, Bauza S, Jellema LM. Vertebral alterations in Scheuermann's kyphosis. *Spine.* mai 1991;16(5):50915.
9. Papagelopoulos PJ, Mavrogenis AF, Savvidou OD, Mitsiokapa EA, Themistocleous GS, Soucacos PN. Current concepts in Scheuermann's kyphosis. *Orthopedics.* 2008;31(1):528; quiz 5960.
10. Nissinen M. Spinal posture during pubertal growth. *Acta Paediatr Oslo Nor* 1992. mars 1995;84(3):30812.
11. Horn SR, Poorman GW, Tishelman JC, Bortz CA, Segreto FA, Moon JY, et al. Trends in Treatment of Scheuermann Kyphosis: A Study of 1,070 Cases From 2003 to 2012. *Spine Deform.* 2019;7(1):1006.
12. Damborg F, Engell V, Nielsen J, Kyvik KO, Andersen MØ, Thomsen K. Genetic epidemiology of Scheuermann's disease. *Acta Orthop.* oct 2011;82(5):6025.
13. Hart ES, Merlin G, Harisiades J, Grottkau BE. Scheuermann's thoracic kyphosis in the adolescent patient. *Orthop Nurs.* déc 2010;29(6):36571; quiz 3723.
14. Sorensen KH. Scheuermann's juvenile kyphosis : clinical apperances, radiography, etiology and prognosis. *Munksgaard. Ann Arbor, MI;* 1964.
15. Wenger DR, Frick SL. Scheuermann kyphosis. *Spine.* 15 déc 1999;24(24):26309.

16. Mac-Thiong J-M, Labelle H, Berthonnaud E, Betz RR, Roussouly P. Sagittal spinopelvic balance in normal children and adolescents. *Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc.* févr 2007;16(2):22734.
17. Blondel B, Schwab F, Ungar B, Smith J, Bridwell K, Glassman S, et al. Impact of magnitude and percentage of global sagittal plane correction on health-related quality of life at 2-years follow-up. *Neurosurgery.* août 2012;71(2):3418; discussion 348.
18. Glassman SD, Bridwell K, Dimar JR, Horton W, Berven S, Schwab F. The impact of positive sagittal balance in adult spinal deformity. *Spine.* 15 sept 2005;30(18):20249.
19. Lafage V, Schwab F, Vira S, Patel A, Ungar B, Farcy J-P. Spino-pelvic parameters after surgery can be predicted: a preliminary formula and validation of standing alignment. *Spine.* juin 2011;36(13):103745.
20. Mac-Thiong J-M, Transfeldt EE, Mehbod AA, Perra JH, Denis F, Garvey TA, et al. Can c7 plumbline and gravity line predict health related quality of life in adult scoliosis? *Spine.* 1 juill 2009;34(15):E519-527.
21. Gokce E, Beyhan M. Radiological imaging findings of scheuermann disease. *World J Radiol.* 28 nov 2016;8(11):895901.
22. Solomou A, Kraniotis P, Rigopoulou A, Petsas T. Frequent Benign, Nontraumatic, Noninflammatory Causes of Low Back Pain in Adolescents: MRI Findings. *Radiol Res Pract.* 2018;2018:7638505.
23. Bradford DS, Moe JH, Montalvo FJ, Winter RB. Scheuermann's kyphosis. Results of surgical treatment by posterior spine arthrodesis in twenty-two patients. *J Bone Joint Surg Am.* juin 1975;57(4):43948.
24. Bartynski WS, Heller MT, Grahovac SZ, Rothfus WE, Kurs-Lasky M. Severe thoracic kyphosis in the older patient in the absence of vertebral fracture: association of extreme curve with age. *AJNR Am J Neuroradiol.* sept 2005;26(8):207785.
25. Propst-Proctor SL, Bleck EE. Radiographic determination of lordosis and kyphosis in normal and scoliotic children. *J Pediatr Orthop.* juill 1983;3(3):3446.
26. Bernhardt M, Bridwell KH. Segmental analysis of the sagittal plane alignment of the normal thoracic and lumbar spines and thoracolumbar junction. *Spine.* juill 1989;14(7):71721.
27. Boseker EH, Moe JH, Winter RB, Koop SE. Determination of « normal » thoracic kyphosis: a roentgenographic study of 121 « normal » children. *J Pediatr Orthop.* déc 2000;20(6):7968.
28. Stagnara P, De Mauroy JC, Dran G, Gonon GP, Costanzo G, Dimnet J, et al. Reciprocal angulation of vertebral bodies in a sagittal plane: approach to references for the evaluation of kyphosis and lordosis. *Spine.* août 1982;7(4):33542.
29. Ristolainen L, Kettunen JA, Kujala UM, Heinonen A, Schlenzka D. Progression of untreated mild thoracic Scheuermann's kyphosis - Radiographic and functional assessment after mean follow-up of 46 years. *J Orthop Sci Off J Jpn Orthop Assoc.* juill 2017;22(4):6527.
30. Murray PM, Weinstein SL, Spratt KF. The natural history and long-term follow-up of Scheuermann kyphosis. *J Bone Joint Surg Am.* févr 1993;75(2):23648.
31. Ristolainen L, Kettunen JA, Heliövaara M, Kujala UM, Heinonen A, Schlenzka D. Untreated Scheuermann's disease: a 37-year follow-up study. *Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc.* mai 2012;21(5):81924.
32. Weiss H-R, Dieckmann J, Gerner H-J. Effect of intensive rehabilitation on pain in patients with Scheuermann's disease. *Stud Health Technol Inform.* 2002;88:2547.
33. Riddle EC, Bowen JR, Shah SA, Moran EF, Lawall H. The duPont kyphosis brace for the treatment of adolescent Scheuermann kyphosis. *J South Orthop Assoc.* 2003;12(3):13540.

34. Arlet V, Schlenzka D. Scheuermann's kyphosis: surgical management. *Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc.* nov 2005;14(9):81727.
35. Coe JD, Smith JS, Berven S, Arlet V, Donaldson W, Hanson D, et al. Complications of spinal fusion for scheuermann kyphosis: a report of the scoliosis research society morbidity and mortality committee. *Spine.* 1 janv 2010;35(1):99103.
36. Jain A, Sponseller PD, Kebaish KM, Mesfin A. National Trends in Spinal Fusion Surgery For Scheuermann Kyphosis. *Spine Deform.* janv 2015;3(1):526.
37. Bradford DS, Ahmed KB, Moe JH, Winter RB, Lonstein JE. The surgical management of patients with Scheuermann's disease: a review of twenty-four cases managed by combined anterior and posterior spine fusion. *J Bone Joint Surg Am.* juill 1980;62(5):70512.
38. Behrbalk E, Uri O, Parks RM, Grevitt MP, Rickert M, Boszczyk BM. Posterior-only correction of Scheuermann kyphosis using pedicle screws: economical optimization through screw density reduction. *Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc.* oct 2014;23(10):220310.
39. Geck MJ, Macagno A, Ponte A, Shufflebarger HL. The Ponte procedure: posterior only treatment of Scheuermann's kyphosis using segmental posterior shortening and pedicle screw instrumentation. *J Spinal Disord Tech.* déc 2007;20(8):58693.
40. Koptan WMT, Elmiligi YH, Elsebaie HB. All pedicle screw instrumentation for Scheuermann's kyphosis correction: is it worth it? *Spine J Off J North Am Spine Soc.* avr 2009;9(4):296302.
41. Lonner BS, Newton P, Betz R, Scharf C, Michael O'Brien, Sponseller P, et al. Operative management of Scheuermann's kyphosis in 78 patients: radiographic outcomes, complications, and technique. *Spine.* 15 nov 2007;32(24):264452.
42. Lee SS, Lenke LG, Kuklo TR, Valenté L, Bridwell KH, Sides B, et al. Comparison of Scheuermann kyphosis correction by posterior-only thoracic pedicle screw fixation versus combined anterior/posterior fusion. *Spine.* 15 sept 2006;31(20):231621.
43. Hosman AJ, Langeloo DD, de Kleuver M, Anderson PG, Veth RP, Slot GH. Analysis of the sagittal plane after surgical management for Scheuermann's disease: a view on overcorrection and the use of an anterior release. *Spine.* 15 janv 2002;27(2):16775.
44. Poolman RW, Been HD, Ubags LH. Clinical outcome and radiographic results after operative treatment of Scheuermann's disease. *Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc.* déc 2002;11(6):5619.
45. Polly DW, Ledonio CGT, Diamond B, Labelle H, Sucato DJ, Hresko MT, et al. What Are the Indications for Spinal Fusion Surgery in Scheuermann Kyphosis? *J Pediatr Orthop.* juin 2019;39(5):21721.
46. Herndon WA, Emans JB, Micheli LJ, Hall JE. Combined anterior and posterior fusion for Scheuermann's kyphosis. *Spine.* avr 1981;6(2):12530.
47. Taylor TC, Wenger DR, Stephen J, Gillespie R, Bobeck WP. Surgical management of thoracic kyphosis in adolescents. *J Bone Joint Surg Am.* juin 1979;61(4):496503.
48. Lowe TG. Double L-rod instrumentation in the treatment of severe kyphosis secondary to Scheuermann's disease. *Spine.* mai 1987;12(4):33641.
49. Otsuka NY, Hall JE, Mah JY. Posterior fusion for Scheuermann's kyphosis. *Clin Orthop.* févr 1990;(251):1349.
50. Tribus CB. Scheuermann's kyphosis in adolescents and adults: diagnosis and management. *J Am Acad Orthop Surg.* févr 1998;6(1):3643.
51. Tsirikos AI. Scheuermann's Kyphosis: an update. *J Surg Orthop Adv.* 2009;18(3):1228.

52. Speck GR, Chopin DC. The surgical treatment of Scheuermann's kyphosis. *J Bone Joint Surg Br.* mars 1986;68(2):18993.
53. Lowe TG, Kasten MD. An analysis of sagittal curves and balance after Cotrel-Dubouset instrumentation for kyphosis secondary to Scheuermann's disease. A review of 32 patients. *Spine.* 1 août 1994;19(15):16805.
54. Lenke LG. Kyphosis of the thoracic and thoracolumbar spine in the pediatric patient: prevention and treatment of surgical complications. *Instr Course Lect.* 2004;53:50110.
55. Papagelopoulos PJ, Klassen RA, Peterson HA, Dekutoski MB. Surgical treatment of Scheuermann's disease with segmental compression instrumentation. *Clin Orthop.* mai 2001;(386):13949.
56. Palazzo C, Sailhan F, Revel M. Scheuermann's disease: an update. *Jt Bone Spine Rev Rhum.* mai 2014;81(3):20914.
57. Johnston CE, Elerson E, Dagher G. Correction of adolescent hyperkyphosis with posterior-only threaded rod compression instrumentation: is anterior spinal fusion still necessary? *Spine.* 1 juill 2005;30(13):152834.
58. Koller H, Lenke LG, Meier O, Zenner J, Umschlaeger M, Hempfing A, et al. Comparison of Anteroposterior to Posterior-Only Correction of Scheuermann's Kyphosis: A Matched-Pair Radiographic Analysis of 92 Patients. *Spine Deform.* mars 2015;3(2):1928.
59. Lim M, Green DW, Billingham JE, Huang RC, Rawlins BA, Widmann RF, et al. Scheuermann kyphosis: safe and effective surgical treatment using multisegmental instrumentation. *Spine.* 15 août 2004;29(16):178994.
60. Denis F, Sun EC, Winter RB. Incidence and risk factors for proximal and distal junctional kyphosis following surgical treatment for Scheuermann kyphosis: minimum five-year follow-up. *Spine.* 15 sept 2009;34(20):E729-734.
61. Riouallon G, Morin C, Charles Y-P, Roussouly P, Kreichati G, Obeid I, et al. Posterior-only versus combined anterior/posterior fusion in Scheuermann disease: a large retrospective study. *Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc.* 2018;27(9):232230.
62. Yun C, Shen CL. Anterior release for Scheuermann's disease: a systematic literature review and meta-analysis. *Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc.* 2017;26(3):9217.
63. Etemadifar M, Ebrahimzadeh A, Hadi A, Feizi M. Comparison of Scheuermann's kyphosis correction by combined anterior-posterior fusion versus posterior-only procedure. *Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc.* 2016;25(8):25806.
64. Mirzashahi B, Chehrassan M, Arfa A, Farzan M. Severe rigid Scheuermann kyphosis in adult patients; correction with posterior-only approach. *Musculoskelet Surg.* déc 2018;102(3):25760.
65. Cobden A, Albayrak A, Camurcu Y, Sofu H, Tacal T, Kaygusuz MA. Posterior-Only Approach with Pedicle Screws for the Correction of Scheuermann's Kyphosis. *Asian Spine J.* août 2017;11(4):5139.
66. Huq S, Ehresman J, Cottrill E, Ahmed AK, Pennington Z, Westbroek EM, et al. Treatment approaches for Scheuermann kyphosis: a systematic review of historic and current management. *J Neurosurg Spine.* 1 nov 2019;113.
67. Ponte A, Vero B, Siccardi G. Surgical treatment of Scheuermann's hyperkyphosis. In: *Progress in spinal fusion : kyphosis.* Winter RB. Bologna: Aulo Gaggi; 1984. p. 7581.
68. Winter RB, Hall JE. Kyphosis in childhood and adolescence. *Spine.* déc 1978;3(4):285308.

69. Kim YB, Lenke LG, Kim YJ, Kim Y-W, Blanke K, Stobbs G, et al. The morbidity of an anterior thoracolumbar approach: adult spinal deformity patients with greater than five-year follow-up. *Spine*. 15 avr 2009;34(8):8226.
70. Boulay C, Tardieu C, Hecquet J, Benaim C, Mouilleseaux B, Marty C, et al. Sagittal alignment of spine and pelvis regulated by pelvic incidence: standard values and prediction of lordosis. *Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc*. avr 2006;15(4):41522.
71. Koller H, Juliane Z, Umstaetter M, Meier O, Schmidt R, Hitzl W. Surgical treatment of Scheuermann's kyphosis using a combined antero-posterior strategy and pedicle screw constructs: efficacy, radiographic and clinical outcomes in 111 cases. *Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc*. janv 2014;23(1):18091.
72. Lafage V, Bharucha NJ, Schwab F, Hart RA, Burton D, Boachie-Adjei O, et al. Multicenter validation of a formula predicting postoperative spinopelvic alignment. *J Neurosurg Spine*. janv 2012;16(1):1521.
73. Mac-Thiong J-M, Labelle H, Roussouly P. Pediatric sagittal alignment. *Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc*. sept 2011;20 Suppl 5:58690.
74. Nasto LA, Perez-Romera AB, Shalabi ST, Quraishi NA, Mehdian H. Correlation between preoperative spinopelvic alignment and risk of proximal junctional kyphosis after posterior-only surgical correction of Scheuermann kyphosis. *Spine J Off J North Am Spine Soc*. avr 2016;16(4 Suppl):S26-33.
75. ZhuW, SunX, PanW, YanH, LiuZ, QiuY, et al. Curve patterns deserve attention when determining the optimal distal fusion level in correction surgery for Scheuermann kyphosis. *Spine J Off J North Am Spine Soc*. sept 2019;19(9):152939.
76. GongY, YuanL, HeM, YuM, ZengY, LiuX, et al. Comparison Between Stable Sagittal Vertebra and First Lordotic Vertebra Instrumentation for Prevention of Distal Junctional Kyphosis in Scheuermann Disease: Systematic Review and Meta-analysis. *Clin Spine Surg*. oct 2019;32(8):3306.
77. Cho K-J, Lenke LG, Bridwell KH, Kamiya M, Sides B. Selection of the optimal distal fusion level in posterior instrumentation and fusion for thoracic hyperkyphosis: the sagittal stable vertebra concept. *Spine*. 15 avr 2009;34(8):76570.
78. Yanik HS, Ketenci IE, Coskun T, Ulusoy A, Erdem S. Selection of distal fusion level in posterior instrumentation and fusion of Scheuermann kyphosis: is fusion to sagittal stable vertebra necessary? *Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc*. févr 2016;25(2):5839.
79. Lundine K, Turner P, Johnson M. Thoracic hyperkyphosis: assessment of the distal fusion level. *Glob Spine J*. juin 2012;2(2):6570.
80. Kim HJ, Nemani V, Boachie-Adjei O, Cunningham ME, Iorio JA, O'Neill K, et al. Distal Fusion Level Selection in Scheuermann's Kyphosis: A Comparison of Lordotic Disc Segment Versus the Sagittal Stable Vertebrae. *Glob Spine J*. mai 2017;7(3):2549.
81. Mikhaylovskiy MV, Sorokin AN, Novikov VV, Vasyura AS. Selection Of The Optimal Level Of Distal Fixation For Correction Of Scheuermann's Hyperkyphosis. *Folia Med (Plovdiv)*. mars 2015;57(1):2936.
82. Dikici F, Akgul T, Sariilmaz K, Korkmaz M, Ozkunt O, Sar C, et al. Selection of distal fusion level in terms of distal junctional kyphosis in Scheuermann kyphosis. A comparison of 3 methods. *Acta Orthop Traumatol Turc*. janv 2018;52(1):711.

# Treatment of Scheuermann's disease

Delpont M, Joly Monrignal P, Neagoe P, Louahem D, Jeandel C, Alkar F, Patte K, Cattalorda J

Department of Pediatric Surgery – Montpellier University Hospital

Department of Physical and Rehabilitation Medicine – Institute Saint-Pierre – Palavas les flots

## Introduction

Holger Werfel Scheuermann, a Swedish physician, described in 1920 a painful thoracic hyperkyphosis which he linked to the “apprentice kyphosis”, a deformity previously described by Schanz in 1891 [1]. Scheuermann's disease is a disorder affecting the growth of the vertebral bodies. It corresponds to a spinal osteochondritis in children, appropriately named osteochondritis deformans juvenilis dorsi. The prevalence of this disease in the general population varies from 2 to 10% [2]. However, these estimates are generally for the typical form of Scheuermann's kyphosis, thereby underestimating its prevalence. Furthermore, some studies show a higher prevalence in males compared to females [2].

Scheuermann's kyphosis is a disco-vertebral pathology due to the bipedal nature and erect spine of human beings. It occurs at the onset of puberty, at around 11 years of age in girls and 13 in boys. The vertebral bodies at this age are more fragile and vulnerable, and mechanical load leads to irregularities at the bony contours and to vertebral deformities [2]. The etiology of Scheuermann's disease is not well understood and is believed to be multifactorial. A multitude of factors have been suggested: Genetic predisposition; repetitive microtrauma (for example intensive practice in certain types of sports), especially between 8 and 12 years old (i.e. at the midpoint through the growth spurt); sagittal balance alterations leading to microtraumatic events with repetitive load on the anterior part of the vertebral bodies [2-4]. Growth is thus delayed at the anterior portion of the vertebral bodies and remains normal posteriorly, thus causing an increasingly hyperkyphotic spine during growth. The resulting cartilaginous and vertebral lesions are irreversible, even though the progression of the disease is halted at the end of growth. Patients may experience pain during adulthood secondary either to the vertebral deformities or to the appearance of intervertebral disc herniations that are probably due to the disease process itself.

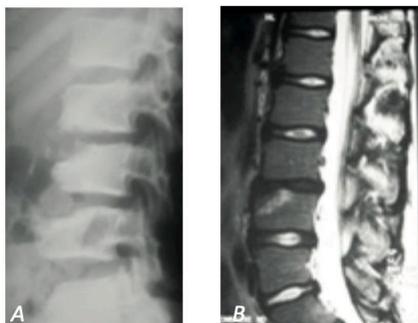
Scheuermann's kyphosis is characterized by a thoracic kyphosis superior to 45°. Sorensen [5] described, in 1964, the radiographic characteristics of the disease. Of note, Anterior wedging across 3 consecutive vertebrae superior to 5° associated with a T5-T12 kyphosis superior to 45°. The concept of Scheuermann's disease, which was initially strictly thoracic in its topography, was later extended to other spinal segments, which are deemed atypical. The latter are distinguished from the typical form either by a different location of the deformity (i.e. thoraco-lumbar or lumbar), or by the presence of distinct radiographic features. As a result, new radiographic criteria were described by Cleveland in 1981 [6]. In these atypical

forms, the presence of kyphosis is no longer a requirement. Actually, Scheuermann's disease includes the aforementioned typical and atypical forms, and is characterized by the presence of at least two of the following radiographic criteria: Anterior wedging across 3 consecutive vertebrae superior to 5° , T5-T12 thoracic kyphosis superior to 45° , or endplate irregularities.

Scheuermann's disease should ideally be treated in its early forms in order to benefit from the potential residual growth of the spine and limit the development of hyperkyphosis. Unfortunately, patients tend to present for consultation with a spine that is already stiff and painful. Physiotherapy may be beneficial, especially in the preservation of mobility, and surgery is generally not indicated except in severe forms.

### 1. What is the natural history of Scheuermann's disease?

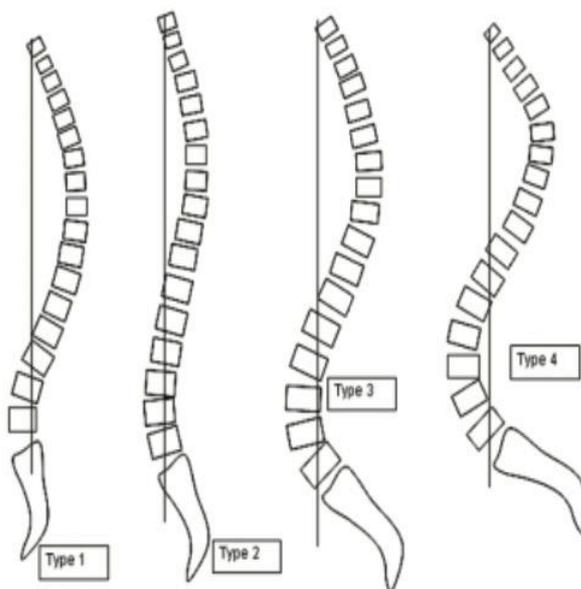
It is estimated that 26% of adults have sequelae of Scheuermann's disease [7] and 76% of the general population have Schmorl nodes [8]. The earlier the onset of Scheuermann's disease, the higher the risk of disease progression [4]. Less progressive forms are known to be benign. In the absence of marked degradation in sagittal alignment and severe inter-vertebral disc disease, the pain encountered during adolescence decreases with skeletal maturity and is rarely carried over into adulthood [9-10]. Nevertheless, it is difficult to imagine that a painful, deforming, and stiffening disease of adolescence could be asymptomatic during adulthood. Scheuermann's disease may be associated with persistent back pain [11-12] and spondylolysis [13]. Intervertebral disc disease is irreversible and may be exacerbated at the onset of adolescence [14-16] (figures 1A and A). Thoracic kyphosis superior to 70° generally leads to poorer functional results [17]. Thoracolumbar and lumbar kyphosis (low pelvic incidence and lumbar lordosis) are not well tolerated and deteriorate faster than kyphosis purely of the thoracic spine. During adulthood, patients rarely complain of the aesthetic appearance of their backs but have a tendency to work less physically demanding jobs than their counterparts. Restrictive lung disease may also be present, but only in severe kyphosis superior to 85°. Some neurological complications secondary to kyphosis are possible but exceptional (sensory deficits at the level of the trunk, nerve root or spinal cord compression by herniated discs, and myelopathy at the apex of the kyphosis) [18].



**Figures 1A and B:** *Scheuermann's disease of the lumbar spine with degenerations of the intervertebral discs and the anterior margins of the vertebrae, confirmed by MRI.*

Recent studies classify the different types of backs according to the magnitude of the sagittal curves and spinopelvic parameters. The primary classification differentiating between varying types of backs is that of Roussouly [19] (figure 2). Roussouly's classification is based on the

sacral slope (SS) and pelvic incidence (PI) and classifies backs into 4 different morphotypes. Sacral slope is the angle formed between the horizontal and the sacral plate. Pelvic incidence is the angle formed between a line perpendicular to the sacral plate at its midpoint and a line connecting this point to the femoral head axis. The advantage of pelvic incidence is that it is relatively stable in a given individual and varies only slightly with age. A type 1 back is defined as a SS <35°, with a low PI and a short and disharmonious lumbar lordosis (called a junctional kyphosis). Type 2 backs also have a SS <35° and a low PI and a more prolonged and harmonious lumbar lordosis. Type 3 backs have a SS between 35 and 45° with a good sagittal alignment. Type 4 backs have a SS >45° and a high PI, which characterize a spine with marked thoracic and lumbar curves. The lower the SS or the PI, the stiffer and flatter the spine, due to a lower lumbar lordosis. Contrarily, the higher these parameters, the higher the lumbar lordosis. Backs with a Roussouly type 1 and 2 are considered as flat backs, and types 3 and 4 as having harmonious curves. From this classification, certain at-risk situations may be identified. In fact, “flat” backs do not allow appropriate adaptation to situations that are highly stressful on the spine. This is especially true when excessive axial load is placed on the spine, as well as during hyperextension of the spine. In the general population, Roussouly types 1 and 2 are at increased risk of developing Scheuermann’s disease, independently from their participation in physical exercise [20-21]. This was shown by Jiang who compared 55 adolescents with Scheuermann’s disease to 60 control subjects [22]. Adolescents with Scheuermann’s disease had a tendency to present a significantly lower pelvic incidence compared to control subjects (32 vs. 45°, respectively).



**Figure 2:** Roussouly’s classification.



**Figure 3:** Junctional kyphosis (Roussouly type 1).

## 2. Can the natural history of Scheuermann's disease be prevented?

The natural history of Scheuermann's disease may be only partially prevented. By focusing on aspects that are secondary to repetitive microtrauma, it may be possible to slow the progression of the deformity. In fact, the only strategies having shown favorable outcomes in this regard are corrective orthopedic and surgical treatments.

### 2.1. Prevention

Screening of at-risk individuals is paramount, including subjects with family history of Scheuermann's disease, and especially patients with low pelvic incidence (predisposing lumbar and thoracolumbar osteochondritis, which are less well tolerated than the thoracic forms). Preventive strategies in children with kyphosis must be undertaken at the youngest possible age. This includes postural exercises with shoulder retropulsion.

The following general preventive measures must be adhered to:

- Adaptation of the profession: Prolonged sitting increases constraints on the anterior portion of the spine. As a result, seated positions with a curved back must be avoided as much as possible, and patients with Scheuermann's disease must favor non-seated professions.
- Adaptation of the work environment (chairs, tables, workstations): Ergonomic kneeling chairs (figure 4) may be used during prolonged sitting (homework, video games, etc...). This would force the child into a straighter posture. Although no studies have shown its effectiveness, ergotherapy may allow proper adaptation to the professional environment.
- Adaptation of practiced sports: Allowed types of sports include those with limited jumps, shocks, blows or falls.
- Avoiding overweight with the help of nutritionists if necessary.
- Avoiding wearing heavy items on back (backpacks).



**Figure 4:** Ergonomic kneeling chair. The patient's weight is supported by the knees, the chair is lightly inclined. As a result, the patient is forced to maintain an upright posture.

## 2.2. What types of sports may be safely practiced?

Physical education at school is rarely contra-indicated. If required, the physician may provide a written note partially excusing the patient from sports placing high stressors on the spine according to the official bulletin of the ministry of education. Physical exercise is authorized in a recreational rather than performance capacity in order to reduce microtrauma. Activity levels must be monitored and limited, sports unloading the spine preferred, sufficient recovery time provided, and a healthy lifestyle promoted [23]. Scheuermann's disease and high-performance sports are not always compatible and the link between the two have been well established [23]. As a rule, thoracic kyphosis and lumbar lordosis have a tendency to increase with the number of hours per week of physical exercise [24,27]. The prevalence of Scheuermann's disease of the lumbar spine increases with the intensity of training, especially in boys carrying heavy weights [25].

In his thesis, Cubillé discussed Scheuermann's disease and its associated factors in 97 young high-performance skiers [26]. His study showed an increased prevalence of back pain and Scheuermann's disease in this population. Partaking in intense, competition-level alpine ski seems to be the principal driving force behind the appearance of this pathology. The mechanical loads placed on the spine during these activities are extremely high, thus placing all professional skiers at risk [26].

Furthermore, intensive gymnastics increases thoracic kyphosis, especially in male athletes [27]. A specialist should be consulted in order to ascertain each patient's actual risk factors. Certain sports to avoid in an intensive manner are those which place high strains on the spine, such as activities requiring flexion of the spine thereby increasing the thoracic kyphosis. This includes horseback riding [28], wrestling, rugby, football (soccer), judo, field hockey, diving, parachuting, skiing [26,29], tennis, table tennis, and hockey [30] among others. Sports that theoretically should be recommended instead include those that extend spine, including dancing [31], rhythmic gymnastics, fitness, volleyball, handball, and backstroke swimming among others. Nevertheless, there is no consensus between the different studies as to the effects that football (soccer) [32], volleyball [33], handball [34], and tennis [35] have on Scheuermann's disease. Swimming increases muscle strength while reducing the load on the spine, but intensive swimming, especially butterfly strokes, may increase Scheuermann's disease of the thoracic spine [30].

During the painful stages of the disease, sports is contra-indicated for an extended period of time. If the pain has completely disappeared and radiographic signs have stabilized for at least 3 months, progressive return to sports may be allowed.

When no contra-indications to physical exercise have been found during a consultation, preventive strategies must be highlighted. Care must be taken when physical activity surpasses 8 to 10 hours per week, or when joining a school sports team, sports training facilities or a national team. Adjustments to the volume or intensity of training may be suggested if they are judged to be unsuitable or poorly experienced. Children requesting permission for “playing-up” require a specific medical exam.

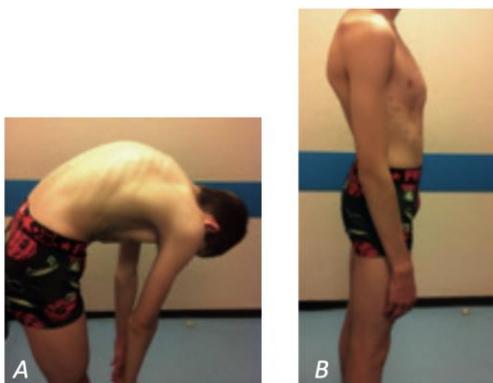
### 3. Functional treatment

#### 3.1. Symptomatic treatment

Even though hyperkyphosis is localized to the thoracic spine, pain is generally located outside of the stiff area, especially at the thoracolumbar junction and at the lumbar segment. The pain is persistent, mechanical in nature with occasional inflammatory episodes, and may progress in flares that may be disabling. Ergotherapy, physiotherapy, shockwave therapy, infrared therapy, and electrotherapy may provide pain relief. Consultation with a pediatric pain specialist may be particularly helpful.

#### 3.2. Rehabilitation

According to a recent meta-analysis [36], rehabilitation has a positive effect on thoracic kyphosis. strength training in particular should be preferred: abdominal, lumbar and erector spinae muscles, and postural correction [29]. Effects are much less tangible in terms of lumbar lordosis, for which strength training and stretching must be simultaneously undertaken (posterior chain and iliopsoas). Hamstring tightness are a known risk factor for sagittal decompensation [37,38] (figures 5A and B). Postural exercises counteract posterior shift of the spine, pelvic retroversion, shoulder antepulsion, and anterior shift of the head [4]. Unfortunately, these exercises do not prevent the progression of the deformity, but may decrease the stiffness of the spine, especially when associated with bracing.



**Figures 5A and B:** Adolescent with thoracic kyphosis and hamstring tightness.

## 4. Bracing

Nonoperative treatment has two goals:

- Limit progression of the disease and preserve the intervertebral discs by reducing the load on the anterior portion of the spine
- Control the structural deformity of the thoracic kyphosis and prevent the appearance of compensatory curves at the lumbar and cervical segments [36].

In patients with higher remaining potential for spinal growth (Risser <2), correction of the deformity is generally more durable if spinal hyperextension during treatment allowed for compensatory growth of the anterior portion of the spine [39].

There is actually no consensus on the optimal type of brace and length of treatment. The effectiveness of treatment is assessed based on the restoration of acceptable sagittal alignment, correction of the anterior wedging of the vertebral bodies, and the appearance of signs of healing in case of a fracture of the marginal borders of the vertebra. Complete correction or ad integrum restitution of the kyphotic deformity is generally not possible, except for some very early cases. Intervertebral disks generally remain deformed and only moderate correction of the vertebral wedging is possible [40]. Bracing is generally necessary for a long period of time and maintained until skeletal maturity, which tends to discourage adolescents who are often unable to tolerate it. Even though the effectiveness of bracing has been established by some authors, the overall outcomes are deceiving. After weaning of the brace, loss of correction is expected (between 5 and 20°), suggesting that, even though bracing expands the anterior intervertebral space, its effects are temporary [14]. Effects are even less significant in the treatment of lumbar disease. Treatment must therefore be undertaken in the early stages of the disease, before wedging of the vertebral bodies has taken place.

### 4.1. Immobilization

Temporary immobilization is indicated only in acute, painful episodes with the aim of relieving pain, and is not recommended for prolonged use, as it may lead to atrophy of the paravertebral muscles.

### 4.2. Corrective treatment

When bracing is considered, a multitude of options exist, all of which must include a posterior support at the apex of the deformity, and 2 anterior supports parasternally in order to retropulse the shoulders.

#### 4.2.1. Corrective cast brace

The use of a cast brace has the ultimate goal of reducing thoracic hyperkyphosis, thereby decreasing the mechanical load on the anterior portion of the vertebral bodies. This is meant to restore the normal growth of the anterior part of the vertebrae and reduce wedging. The cast brace is inspired by the elongation-derotation-flexion (EDF) brace used in scoliotic patients. The indication for orthopedic treatment is generally progressive kyphosis >60° with residual

potential growth (and thereby the potential exacerbation in the absence of treatment) [9]. The spine must be relatively flexible in order to undertake this treatment (supine fulcrum extension test). This treatment modality is undertaken in association with physiotherapy and postural exercises.

The cast is molded on a Cotrel traction table [41]. The primary posterior support must be situated beneath the apex of the kyphosis, on both sides of the spinous processes. Two anterior supports opposing the previous one are placed at the level of the pelvis and the manubrium sterni. Flexion of the hips during cast molding reduces lumbar lordosis by retroverting the pelvis. Windows allowing for the expansions of the rib cage and the abdominal cavity must be conceived. The anterior thoracic window must be relatively large allowing for the expansion of the rib cage during breathing. Depending on brace tolerance, sequential casting is undertaken at 2- to 3-week intervals until correction is obtained. Occipital-mandibular support is not recommended in this case and should be reserved for patients with a deformed hyperkyphosis.

Cutaneous complications may develop at the level of the cast-skin points of contact. At the level of the pelvis, the window must also be large enough to allow flexion of the hips and avoid entrapment of the lateral femoral cutaneous nerve in the seated position.

#### 4.2.2. Progressive correction by the addition of felt

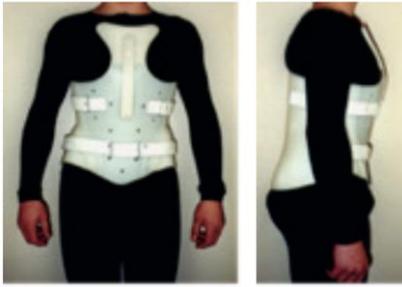
Correction is obtained by progressively increasing pressure at the zones of contact with the addition of layers of felt to the initial cast, 2 to 3 weeks after initiation of treatment. An extra layer of felt is added on a weekly basis with surveillance of the skin at the zones of contact. Radiographic follow-up is necessary in order to assess the effectiveness of the correction.

#### 4.2.3. Milwaukee brace

The principal of the Milwaukee brace may be tempting by its active auto-elongation effect on the trunk which tends to decrease the spinal curves in the sagittal plane. It is more frequently reserved for young children, although some authors have utilized it in adolescents. It may be undertaken as a standalone treatment or as a continuity of correction by casting [41]. Unfortunately, it is often badly tolerated by the patient and discontinued by almost 50% patients, especially those who are a little older [41]. Some authors have recommended the use of a thoraco-lumbo-sacral orthosis (TLSO) instead of the Milwaukee brace [42].

#### 4.2.4. Rigid brace (figure 6)

Since the Milwaukee brace is often badly tolerated, it may be replaced by a bivalve brace [13]. This type of brace is molded on the trunk. During the molding process, the patient must be in a standing position with the hips partially flexed in order to decrease lumbar lordosis. Once more, cutaneous complications at the contact points must be monitored. Brace-wear must be continued during the entire period of residual growth of the trunk. A diurnal-only use of the brace may be allowed. A hypercorrective night-time brace may also be prescribed, although it places excessive stress on the spine and may not be well tolerated.



**Figure 6:** *Bivalve rigid brace*

## 5. Operative treatment

The need for operative treatment in Scheuermann's disease remains exceptional and controversial. A detailed chapter has been consecrated to this end (see "Is there a place for surgical management in adolescents with Scheuermann's disease?" by Antoine Hamel). Surgery is considered as the final recourse for pediatric patients who are at the end of their growth or in adults. It may be indicated in patients with severe kyphosis ( $>70^\circ$ ), back pain, neurological symptoms, and resistance to all other forms of treatment [37,38,41]. The surgical approach may include posterior or combined spinal fusion, with or without osteotomies of the apex of the deformity. Instrumentation may be hybrid or an all-screw construct. Fusion must be extensive, include 10-12 vertebrae, and must include the first lordotic intervertebral segment (often T2/T3 to L2/L3). Shorter constructs lead to post-operative loss of correction with the appearance of badly tolerated junctional kyphosis. In the setting of preoperative assessment, an MRI is ordered in search of spinal cord anomalies, herniated discs, or spinal cord compression at the apex of the deformity since the major risk of this surgery is the development of paraplegia. Nevertheless, in order to minimize neurological complications, it is recommended not to overcorrect the kyphosis.

## 6. Indications

Indications for the choice of treatment depend primarily on the age of the child, stiffness of the deformity, location and number of vertebrae implicated, and refractory pain [42]. Thoracolumbar and lumbar disease often lead to moderate angular deformities which are more often painful due to the presence of Schmorl nodes; These are generally treated by conservative management, except in rare cases where neurological signs may be present, in which case surgical spinal cord decompression along with posterior spinal fusion would be necessary. In younger patients with thoracic disease, the deformity is rarely severe, is always flexible, and is manageable by conservative treatment. Rehabilitation is extremely effective in improving the posture, especially when the thoracic spine is flexible, and the sagittal curve is not extreme ( $45-55^\circ$ ). The rehabilitation strategy should concentrate on hamstring and pectoral stretching, as well as strengthening the extensors of the spine. When the curve magnitude is superior to  $50-55^\circ$ , a full-time brace may be indicated (TLSO or Milwaukee). Rehabilitation may be associated with brace-wear but is in no way sufficient by itself as a means of correcting an already-structuralized kyphotic deformity. Conservative treatment must be pursued until the end of growth in order to prevent any loss of correction [12-13]. In adolescents, the majority of authors agree on the effectiveness of conservative treatment in flexible forms or in patients with a Risser  $<3$ . For patients with a stiff deformity, serial

corrective casting may be attempted as an initial treatment modality. A loss of correction of 10-20° after discontinuation of conservative treatment has been reported in at least 30% of patients [11]. For patients with a kyphosis >70°, functional results are less satisfactory [17]. Severe postural alterations that accompany more advanced forms of Scheuermann's disease are sources of discogenic pain at the junction between the structuralized, stiff deformity and the mobile segments [14]. Moreover, compensatory curves, such as lumbar and cervical hyperlordosis, are also sources of painful mechanical spinous process impingement. Scheuermann's disease tends to resolve in males between 16 and 18 years of age, with possible radiographic sequellae that may not be painful.

## Conclusion

The optimal treatment modality in Scheuermann's disease is prevention. However, the majority of patients present at advanced stages where bony deformity has already been established and is almost impossible to reverse. The primary goal of treatment is to attempt to delay progression of the deformity. Conservative treatment by bracing is protracted and often badly tolerated by adolescents. Surgical management is exceptional and may be indicated at the end of growth and only as a last resort.

## References

1. Scheuermann H. Kyphosis dorsalis juvenilis. *Ugeskr Laeger* 1920;82:385-93.
2. Nectoux E. Maladie de Scheuermann. [https://oer.clouvain.be/jspui/bitstream/123456789/274/1/Eric\\_Nectoux\\_Cyphoses.pdf](https://oer.clouvain.be/jspui/bitstream/123456789/274/1/Eric_Nectoux_Cyphoses.pdf)
3. Blumenthal SL, Roach J, Herring JA. Lumbar Scheuermann's. A clinical series and classification. *Spine* 1987;12:929-32.
4. Laumonier F, Lechevallier J. Maladie de Scheuermann et dystrophie rachidienne de croissance. *Appareil Locomoteur*, Paris: Elsevier Masson; 2008, p. 15-865-A-10.
5. Sorensen KH. Scheuermann's juvenile kyphosis, clinical appearances, radiography, actiology and prognosis. Munksgaard, Copenhagen; 1964.
6. Cleveland RH, Delong GR. The relationship of juvenile lumbar disc disease and Scheuermann's disease. *Pediatr Radiol* 1981;10:161-4.
7. Serre H, Barjon M, Simon L. Les séquelles des dystrophies rachidiennes de croissance chez l'adulte. *Rev Rhum Mal Osteoartic* 1964;31:392-412.
8. Greene TL, Hensinger RN, Hunter LY. Back pain and vertebral changes simulating Scheuermann's disease. *J Pediatr Orthop* 1985;5:1-7.
9. Murray PM, Weinstein SL, Spratt KF. The natural history and long-term follow-up of Scheuermann kyphosis. *J Bone Joint Surg Am* 1993;75:236-48.
10. Montgomery SP, Erwin WE. Scheuermann's kyphosis : long-term results of Milwaukee braces treatment. *Spine* 1981;6:5-8.
11. Harreby M, Neergaard K, Hesselsøe G, Kjer J. Are radiologic changes in the thoracic and lumbar spine of adolescents risk factors for low back pain in adults? A 25-year prospective cohort study of 640 school children. *Spine* 1995;20:2298-302.
12. Ristolainen L, Kettunen JA, Heliövaara M, Kujala UM, Heinonen A, Schlenzka D. Untreated Scheuermann's disease: a 37-year follow-up study. *Eur Spine J* 2012;21:819-24.
13. Stoddard A, Osborn JF. Scheuermann's disease or spinal osteochondrosis: its frequency and relationship with spondylosis. *J Bone Joint Surg Br* 1979;61:56-8.
14. Paajanen H, Alanen

- A, Erkintalo M, Salminen JJ, Katevuo K. Disc degeneration in Scheuermann disease. *Skeletal Radiol* 1989;18:523-6.
15. Osti OL, Fraser RD. MRI and discography of annular tears and intervertebral disc degeneration. A prospective clinical comparison. *J Bone Joint Surg Br* 1992;74:431-5.
  16. Salminen JJ, Erkintalo MO, Pentti J, Oksanen A, Kormanen MJ. Recurrent low back pain and early disc degeneration in the young. *Spine* 1999;24:1316-21.
  17. Soo CL, Noble PC, Esses SI. Scheuermann kyphosis: long-term follow-up. *Spine J* 2002;2:49-56.
  18. Cho W, Lenke LG, Bridwell KH, Hu G, Buchowski JM, Dorward IG, et al. The prevalence of abnormal preoperative neurological examination in Scheuermann kyphosis: correlation with X-ray, magnetic resonance imaging, and surgical outcome. *Spine* 2014;39:1771-6.
  19. Roussouly P, Gollogly S, Berthonnaud E, Dimnet J. Classification of the normal variation in the sagittal alignment of the human lumbar spine and pelvis in the standing position. *Spine* 2005;30:346-53.
  20. Tyrakowski M, Janusz P, Mardjetko S, Kotwicki T, Siemionow K. Comparison of radiographic sagittal spinopelvic alignment between skeletally immature and skeletally mature individuals with Scheuermann's disease. *Eur Spine J* 2015;24:1237-43.
  21. Tyrakowski M, Mardjetko S, Siemionow K. Radiographic spinopelvic parameters in skeletally mature patients with Scheuermann disease. *Spine*. 2014;39:E1080-5.
  22. Jiang L, Qiu Y, Xu L, Liu Z, Wang Z, Sha S, et al. Sagittal spinopelvic alignment in adolescents associated with Scheuermann's kyphosis: a comparison with normal population. *Eur Spine J* 2014;23:1420-6.
  23. Hellström M, Jacobsson B, Swärd L, Peterson L. Radiologic abnormalities of the thoracolumbar spine in athletes. *Acta Radiol* 1990;31:127-32.
  24. Wojtys EM, Ashton-Miller JA, Huston LJ, Moga PJ. The association between athletic training time and the sagittal curvature of the immature spine. *Am J Sports Med* 2000;28:490-498.
  25. Lowe TG. Scheuermann disease. *J Bone Joint Surg Am* 1990;72:940-5.
  26. Cubillé V. Etude de la dystrophie rachidienne de croissance en imagerie EOS et de ses facteurs associés chez 97 jeunes skieurs de haut niveau. Thèse de Médecine, 2018, Université de Grenoble Alpes.
  27. Sanz-Mengibar JM, Sainz-de-Baranda P, Santonja-Medina F. Training intensity and sagittal curvature of the spine in male and female artistic gymnasts. *J Sports Med Phys Fitness* 2018;58:465-71.
  28. Förster R, Penka G, Bösl T, Schöffl VR. Climber's back-form and mobility of the thoracolumbar spine leading to postural adaptations in male high ability rock climbers. *Int J Sports Med* 2009;30:53-9.
  29. Alricsson M, Werner S. Young elite cross-country skiers and low back pain-A 5-year study. *Phys Ther Sport* 2006;7:181-4.
  30. Keene JS, Drummond DS. Mechanical back pain in the athlete. *Compr Ther* 1985;11:7-14.
  31. Nilsson C, Wykman A, Leanderson J. Spinal sagittal mobility and joint laxity in young ballet dancers. A comparative study between first-year students at the Swedish Ballet School and a control group. *Knee Surg Sports Traumatol Arthrosc* 1993;1:206-8.
  32. Wodecki P, Guigui P, Hanotel MC, Cardinne L, Deburge A. Sagittal alignment of the spine: comparison between soccer players and subjects without sports activities. *Rev Chir Orthop Reparatrice Appar Mot* 2002;88:328-36.
  33. Grabara M. Comparison of posture among adolescent male volleyball players and non-athletes. *Biol Sport* 2015;32:79-85.

34. Grabara M. A comparison of the posture between young female handball players and non-training peers. *J Back Musculoskelet Rehabil* 2014;27:85-92.
35. Muyor JM, Sánchez-Sánchez E, Sanz-Rivas D, López-Miñarro PA. Sagittal spinal morphology in highly trained adolescent tennis players. *J Sports Sci Med* 2013;12:588-93.
36. González-Gálvez N, Gea-García GM, Marcos- Pardo PJ. Effects of exercise programs on kyphosis and lordosis angle: A systematic review and meta-analysis. *PLoS ONE* 2019;14:e0216180.
37. Hosman AJ, Langeloo DD, de Kleuver M, Anderson PG, Veth RP, Slot GH. Analysis of the sagittal plane after surgical management for Scheuermann's disease: a view on overcorrection and the use of an anterior release. *Spine* 2002;27:167-75.
38. Tsirikos AI, Jain AK. Scheuermann's kyphosis; current controversies. *J Bone Joint Surg Br* 2011;93:857-64.
39. Riddle EC, Bowen JR, Shah SA, Moran EF, Lawall H. The duPont kyphosis brace for the treatment of adolescent Scheuermann kyphosis. *J South Orthop Assoc* 2003;12:135-40.
40. Sachs B, Bradford D, Winter R, Lonstein J, Moe J, Willson S. Scheuermann kyphosis. Follow-up of Milwaukee-brace treatment. *J Bone Joint Surg Am* 1987;69:50-7.
41. Abelin K, Vialle R, Morin C, Leclair-Richard D. Traitement orthopédique des hypercyphoses en période de croissance. *Techniques chirurgicales- Orthopédie-Traumatologie*, Elsevier Masson SAS; 2009, p. 44192.
42. Miladi L. Cyphoses régulières et angulaires chez l'enfant. *Conférences d'enseignement de la SOFCOT*, 2012. Elsevier Masson éditeurs.

# Spondylolysis and spondylolisthesis: operative treatment

Roxane Compagnon, Manon Bolzinger, Jérôme Sales De Gauzy

Department of Pediatric Orthopaedic Surgery, Children's Hospital, Toulouse University Hospital  
330 Avenue de Grande-Bretagne 31026 Toulouse CEDEX

## Introduction

Surgical management is rarely indicated in children and adolescents with spondylolysis and spondylolisthesis when considering the high prevalence of these two entities in the general population. Abstention of treatment and conservative management are much more frequently employed management strategies. The focus of this chapter will be on the surgical strategies of L5-S1, the most frequent location of spondylolysis and spondylolisthesis.

Numerous surgical techniques have been described and the decision on which to employ remains controversial for both low-grade and high-grade spondylolisthesis. Different techniques, their results and their complications will be discussed, along with a management strategy based on the degree of vertebral translation and stability.

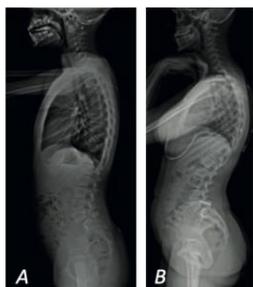
## Surgical techniques

### 1. Pars repair [15]

First described by Kimura in 1968, the goal of pars repair is to restore the continuity of the isthmus with a bone graft. This intervention has the theoretical advantage of conserving the mobility of the L5-S1 segment and requires a healthy L5-S1 intervertebral disc pre-operatively verified on MRI. The patient is placed prone, and a posterior midline approach is made. After the posterior arch is cleared, fibrous tissue at the level of the pars interarticularis is excised. The autologous bone graft, harvested from the iliac crest using the same incision, is placed.

In order to ensure the graft is held firm in place, multiple fixation techniques have been described [5]:

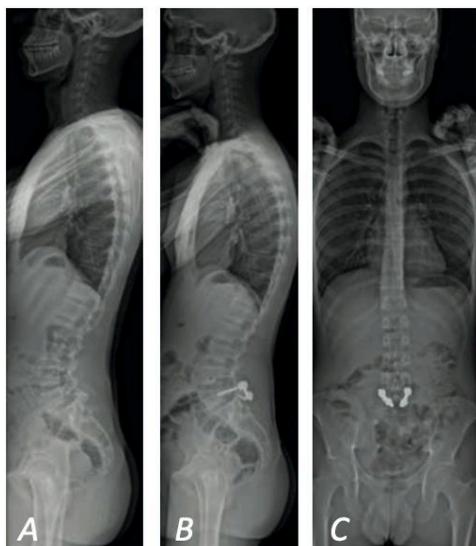
- Screw fixation through the superior articular process with an associated laminar hook (Morscher technique).
- Direct fixation of the isthmus (Buck repair) (figure 1): lag screw placement through the lamina is often difficult due to the laminar dysplasia.



**Figure 1:** A) 10-year-old girl with symptomatic grade I spondylolisthesis resistant to conservative treatment. B) isthmic repair by Buck's technique. Results at 6-year follow-up.

- Cerclage wire around the transverse and spinous processes (Scott repair).
- perforated screw with passage of a polyester ligament with the strands placed around the L5 lamina (Bonnard's technique). The construct is combined with a Kirschner wire passing through the lamina and the pedicle in order to neutralize shearing forces.

Our preference is the combination of a pedicle screw and a sublaminar hook along with a short rod in between (figure 2). This construct may be undertaken with most commonly used spinal instrumentations and allows adequate compression of the grafted area. The increased stability renders post-operative immobilization unnecessary [5].

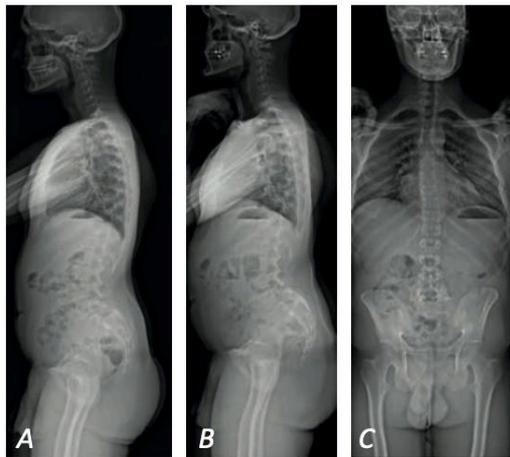


**Figure 2:** A) 18-year-old boy with symptomatic grade I spondylolisthesis resistant to conservative treatment. B,C) pars repair with pedicle screws and sublaminar hooks. Results at 1-year follow-up.

These different instrumentation techniques were tested experimentally by Fan et al. [3], who confirmed increased stability with the screw-hook construct.

Return to full sporting activities is usually authorized 6 months postoperatively.

## 2. Posterolateral fusion (PLF) [15] (figure 3)



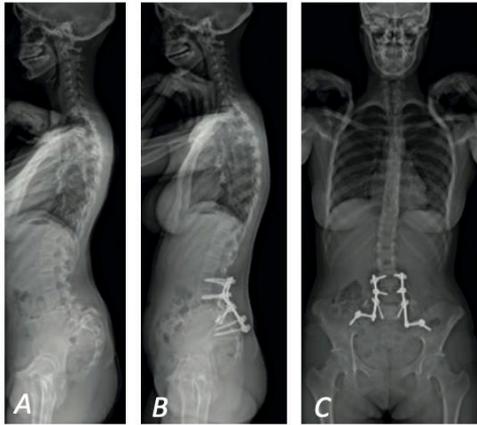
**Figure 3:** A) 15-year-old boy with symptomatic, unstable, grade III spondylolisthesis. B,C) L4-S1 posterolateral fusion. Results at 6-year follow-up. No further translation of L5 over S1 was observed.

Posterolateral fusion is undergone according the Wiltse's technique with the aim of in-situ L5-S1 and sometimes L4-S1 fusion without reduction or instrumentation. Laminectomy or exploration of the spinal canal are not necessary. A posterior midline incision is made centered on L5-S1. Subcutaneous dissection is done approximately 5cm lateral to the midline. After incision of the aponeurosis and passage between the multifidus and longissimus muscles, the L5 transverse process, the lateral portions of the L5-S1 facet joints, and the sacral ala are exposed. A rongeur is used to excise the fibrous tissue at the level of the pars interarticularis (isthmic hook) since it can be a source of localized and radiating radicular pain. Decortication of the L5 transverse processes, lateral portion of the L5-S1 facet joint, and sacral ala is carried out. Finally, cortical and cancellous bone grafts harvested from the iliac crest through the same incision are placed. Immobilization with a thermoformed back brace, crafted prior to the intervention, is prescribed for a period of 3 months.

## 3. Closed reduction and fusion [15].

This technique is used in high-grade spondylolisthesis. The goal is to achieve progressive reduction of the lumbosacral kyphosis followed by fusion in the reduced position. This technique is highly constringent and requires a cooperative child and family. Reduction is achieved through bipolar traction and support with a hammock. The hammock is fashioned out of a 20cm-wide piece of cloth with the superior ends passing directly over the anterosuperior iliac spines. The hammock is then progressively tightened by adding a weight on both sides. The weight is then progressively increased until half of the child's weight is reached and until the buttocks are suspended in the air. Reduction may be obtained within the first 24 to 48h. Radiographs are obtained to confirm the reduction of the lumbosacral kyphosis. A cast is then fashioned in the reduced position and includes both thighs. Circumferential (anterior and posterior) fusion is then performed while the patient is still in the cast, through a window that is created beforehand. The cast is left in place until consolidation is achieved. This technique is no longer commonly used due to its constringent nature.

#### 4. Surgical reduction and fusion [15] (figure 4).



**Figure 4:** A) grade IV spondylolisthesis. B,C) surgical correction with reduction with L5-S1 anterior and L4-S1 posterior fusion. Postoperative results at 1-year follow-up.

The goal of reduction and fusion in spondylolisthesis is the restoration of sagittal balance with correction of the lumbosacral kyphosis rather than reduction of the translated vertebrae. An L5 laminectomy is realized through a posterior approach, and the L5-S1 roots are located. The L5 roots must be protected throughout the procedure due to the risk of compression within the foramen or stretching after reduction is achieved, with the risk of a permanent postoperative deficit. L4-L5 pedicular screws are then placed. At the level of the pelvis, fixation may be achieved with S1-S2 screws, iliac screws, or by trans-sacral rods as per the Jackson technique. Under fluoroscopy, the sacral dome is resected passing by both sides of the dural sac using an osteotome. The rods are bent beforehand and positioned allowing for a progressive reduction of the lumbosacral kyphosis. At the end of correction, the L5-S1 roots must be checked for entrapment. The iliac bone grafts are finally placed in front of the L5-S1 space, and posterolaterally between L4, L5 and S1. In order to increase stability, an intersomatic cage may be placed at the level of L5-S1, which also aids in restoring the L5-S1 intervertebral disc height. Postoperatively, in order to release the L5 root, the patient's hips and knees must be kept in flexion with the support of a pillow. Extension is allowed progressively over the following days, and then a cast brace or rigid lumbar brace with inclusion of the thighs is prescribed for at least 3 months.

## 5. Trans-sacral screw fixation [15] (figure 5).



**Figure 5:** A) 18-year-old girl with Spondylolisthesis. B,C) circumferential fusion through a posterior approach with placement of a trans-sacral screw. Postoperative radiographs at 3-month follow-up.

Trans-sacral screw fixation is indicated in high-grade spondylolisthesis. The patient is placed on cushions in the prone position, allowing partial reduction of the intervertebral translation and lumbosacral kyphosis. An L5-S1 laminectomy is carried out. The nerve roots and the dural cul de sac are retracted in order to expose the posterior aspect of the S2 vertebra. Under fluoroscopic control, a Kirschner wire is placed through S2 at its midline in the direction of the posterosuperior corner of L5. A perforated drill bit is used over the guidewire and the dedicated screws are placed (Medicalex®); The proximal end of the screw is curtailed allowing for the attachment of the posterior aspect of the S2 vertebra after adequate compression has been achieved. The protruding dome of S1 is resected in order to liberate the S1 vertebra and the L5 nerve root is checked for entrapment. L5-S1 discectomy is then carried out, and debridement of the L5 and S1 endplates is achieved using an osteotome and a curette. Finally, the iliac bone grafts are placed anteriorly in the L5-S1 intervertebral space and posteriorly around the posterolateral area.

## 6. Lumbosacral fusion without instrumentation by anterior approach [15].

Lumbosacral fusion without instrumentation using an anterior approach is indicated in high-grade spondylolisthesis as a complement to L5-S1 posterolateral fusion. The patient is placed supine in a hyperlordotic position. A Pfannenstiel incision is made, and the anterior aspect of L5 is reached by subperiosteal dissection. Using an osteotome, the middle third of L5 is resected. The L5-S1 intervertebral disc and the sacral dome are accessed and the L5-S1 disc is excised. An autologous graft harvested from either the tibia or the iliac crest is then placed. Postoperative immobilization is prescribed for 4 months.

## Results

### 1. Low-grade spondylolisthesis.

The most frequently utilized techniques are PLF and pars repair. Clinically, pars repair has satisfactory results in the majority of the reported series with 80 to 90% of the studies

reporting good results, ranging between 56 and 100% [5]. Return to physical exercise at the previous level is observed in the majority of patients. However, the isthmus may not consolidate in all patients. Furthermore, uncertainty persists on the long-term effects of these interventions on the L5-S1 disc. After a 15-year follow-up, Schlenzka et al. found identical results between PLF and pars repair for both symptoms and lumbar mobility [19].

## **2. High-grade spondylolisthesis (superior to 50%).**

Multiple series have shown good results with in-situ fusion, all of which were carried out with long-term follow-up; 10 years for Seitsalo et al. [18] and 17 years for Lamberg et al. [11]. Circumferential fusion has been shown to provide better results than isolated posterior or anterior fusion [2,11]. No significant advantages were found between fusion with or without reduction. Alzakri et al. [1] and Mac-Thiong et al. [14] reported better results after restoration of the pelvic sagittal balance. Contrarily, Poussa et al. found improved results after in-situ fusion compared to reduction and fusion [16]. Longo et al. found identical clinical outcomes and no significant differences in terms of neurological complications in a systematic review comparing in-situ fusion (101 patients) to reduction and fusion (165 patients) [12], and the only difference found was the risk of non-union, with higher rates non-union in non-instrumented cases. No advantages were found for fusion with reduction. In a recent study, Joelson et al. confirmed the good clinical and functional outcomes after a 30-year follow-up in patients treated with circumferential fusion without reduction [8]. Nevertheless, these patients reported a deteriorated self-image compared to the general population and related it to their lumbosacral deformity [9].

## **Complications**

In a series with over 600 patients, Fu et al. reported complication rates exceeding 10% [4]. In 50% of these patients, neurological deficits were noted mostly at the level of L5 root, with cauda equina syndrome also being reported. Damage to the L5 nerve root is estimated at around 10 to 50%. The deficit is most often temporary, although it can rarely be permanent. The risk of neurological damage is increased in patients in who had undergone reduction [12]. Nonetheless, this complication may also arise in patients treated by posterolateral fusion without reduction, and especially in high-grade spondylolisthesis [12,17].

Perioperatively, the L5-S1 roots must be protected. Perioperative monitoring of the L5-S1 roots has been proposed, although no objective results have been reported in the literature [12]. As was previously discussed, patients must be placed with hips and knees in flexion over a cushion in the postoperative period. Progressive extension is allowed in the days following surgery.

Other complications include a 3% risk of infection, with no significant differences between reduction and in-situ fusion [4].

The risk of non-union is higher in patients treated by in-situ fusion (18%) compared to surgical reduction (5.5%) [12].

Complications specific to the anterior approach include vascular complications secondary to the proximity of the aorto-bi-iliac bifurcation and the ilio-cava venous confluence, and neurovegetative complications (reduced fertility) due to the proximity of the hypogastric plexus.

## Indications

Operative treatment remains controversial. This is true for both the indications for surgical management and the surgical technique to use. The most essential element is the analysis of sagittal balance in order to assess the stability of the deformity [7,10,14,20].

Two factors are essential: sacral slope and pelvic incidence.

- In patients with a horizontal sacrum, the deformity is generally stable due to the vertical orientation of the sacral plate, thereby conferring sufficient lumbar lordosis that is harmonious with the pelvic incidence. This is true even in patients with high pelvic incidence.
- In patients with a vertical sacrum, the deformity is generally unstable due to the horizontal orientation of the sacral plate, thereby limiting the lumbar spine's ability to increase lordosis and to adapt to the pelvic incidence.

As a result, if pelvic incidence is high, the slippage of L5 over S1 will usually progressively increase and may even rotate around the anterosuperior corner of S1 in an attempt to increase lordosis. This is characterized by lumbosacral kyphosis with an L5-S1 angle  $<90^\circ$  (as described by Dubousset). In the majority of cases, this leads to a decompensated sagittal alignment and a progressive translation of L5 over S1 leading to spondyloptosis.

### 1. Spondylolysis and low-grade spondylolisthesis (translation $<50\%$ )

#### Stable forms

Operative treatment in stable forms is rarely indicated and is recommended only in patients who remain symptomatic after conservative treatment has been attempted. As previously mentioned, pars repair and PLF have similar outcomes. Our preference is operative treatment of these patients with a direct pars repair by pedicular screws and sublaminar hooks.

#### Unstable forms

Unstable forms are rarely encountered in patients with low-grade spondylolisthesis. Nevertheless, knowledge of these forms is still important. In our institution, surgery is absolutely indicated in order to avoid further slippage of L5 over S1. L5-S1 PLF is sufficient to ensure stability. In fact, fusion between L5 and S1 will modify the lumbosacral alignment, with the superior endplate of L5 becoming the new base upon which the lumbar spine will adapt its lordosis.

## **2. High-grade spondylolisthesis (translation >50%)**

### Stable forms

Various studies have shown that conservative management with close follow-up is possible in stable forms [2,6,13]. However, after an 18-year follow-up, Harris and Weinstein [6] showed that patients who had been previously operated were more active. As such, in both symptomatic and asymptomatic patients, surgical intervention is preferred [2]. Our preference in this case is to operate with in-situ fusion without reduction.

### Unstable forms

Surgery is absolutely indicated in these unstable forms with large displacement in order to avoid progressive translation of L5 over S1 and to relieve pain, a very complaint.

Circumferential fusion is necessary in these patients. However, the question of reduction is controversial. As was previously discussed, the goal is reduction of the lumbosacral kyphosis rather than the translation of L5 over S1.

Our attitude actually depends on the amount of translation. It must be taken into consideration that the per-operative prone position often allows partial reduction of the translation and, more significantly, the L5-S1 kyphosis. In grade III spondylolisthesis, reduction with posterior fusion and intersomatic L5-S1 cage placement may be indicated. In spondyloptosis, in-situ fusion with trans-sacral screws is preferred. In grade IV, choosing between the two techniques may be difficult.

## **3. Spondylolisthesis by isthmic elongation**

This particular type of spondylolisthesis is rare but must be mentioned. The isthmus is elongated but not fractured and translation is generally moderate (grades I or II). Progression of slippage of L5 over S1 may be responsible for poorly tolerated lumbar spinal stenosis requiring laminectomy along with L5-S1 fusion and instrumentation.

## **Conclusion**

Surgical management of spondylolisthesis must be suggested with care. It is rarely indicated in forms with low translation, with PLF or pars repair both providing good results in symptomatic patients not responding to conservative treatment. Surgery is more formally indicated in forms with a large displacement where circumferential fusion is indicated. Reduction of the deformity remains controversial.

## **References**

1. Alzakri A, Labelle H, Hresko MT, Parent S, Sucato DJ, Lenke LG, Marks MC, Mac-Thiong JM. Restoration of normal pelvic balance from surgical reduction in high- grade spondylolisthesis. Eur Spine J. 2019;28(9):2087- 2094.

2. Crawford CH 3rd, Larson AN, Gates M, Bess RS, Guillaume TJ, Kim HJ, Oetgen ME, Ledonio CG, Sanders J, Burton DC. Current Evidence Regarding the Treatment of Pediatric Lumbar Spondylolisthesis: A Report From the Scoliosis Research Society Evidence Based Medicine Committee. *Spine Deform.* 2017 Sep;5(5):284-302.
3. Fan J, Yu GR, Liu F, Zhao J, Zhao WD. A biomechanical study on the direct repair of spondylolysis by different techniques of fixation. *Orthop Surg.* 2010 ;2(1):46-51.
4. Fu KM, Smith JS, Polly DW Jr, Perra JH, Sansur CA, Berven SH, Broadstone PA, Choma TJ, Goytan MJ, Noordeen HH, Knapp DR Jr, Hart RA, Donaldson WF 3rd, Boachie-Adjei O, Shaffrey CI. Morbidity and mortality in the surgical treatment of six hundred five pediatric patients with isthmic or dysplastic spondylolisthesis. *Spine.* 2011, 15;36(4):308-12.
5. Gagnet P, Kern K, Andrews K, Elgafy H, Ebraheim N. Spondylolysis and spondylolisthesis: A review of the literature. *J Orthop.* 2018,17;15(2):404-407.
6. Harris IE, Weinstein SL. Long-term follow-up of patients with grade-III and IV spondylolisthesis: treatment with and without posterior fusion. *J Bone Joint Surg Am.* 1987;69:960–969.
7. Hresko MT, Labelle H, Roussouly P, Berthonnaud E. Classification of high-grade spondylolisthesis based on pelvic version and spine balance: possible rationale for reduction. *Spine.* 2007,15;32(20):2208-13.
8. Joelson A, Danielson BI, Hedlund R, Wretenberg P, Frennered K. Sagittal Balance and Health-Related Quality of Life Three Decades After in Situ Arthrodesis for High-Grade Isthmic Spondylolisthesis. *J Bone Joint Surg Am.* 2018 Aug 15;100(16):1357-1365.
9. Joelson A, Diarbakerli E, Gerdhem P, Hedlund R, Wretenberg P, Frennered K. Self-Image and Health-Related Quality of Life Three Decades After Fusion In Situ for High-Grade Isthmic Spondylolisthesis. *Spine Deform.* 2019 Mar;7(2):293- 297.
10. Jouve JL. Spondylolyse et spondylolisthesis lombosacr  de l'enfant et de l'adolescent. In : Cahiers d'enseignement de la SOFCOT. Paris : Expansion Scientifique Publications ; 2001. P.171-92
11. Lamberg T, Remes V, Helenius I, Schlenzka D, Seitsalo S, Poussa M. Uninstrumented in situ fusion for high-grade childhood and adolescent isthmic spondylolisthesis: long-term outcome. *J Bone Joint Surg Am.* 2007 Mar;89(3):512-8.
12. Longo UG, Loppini M, Romeo G, Maffulli N, Denaro V Evidence-based surgical management of spondylolisthesis: reduction or arthrodesis in situ. *J Bone Joint Surg Am.* 2014 Jan 1;96(1):53-8.
13. Lundine KM, Lewis SJ, Al-Aubaidi Z, Alman B, Howard AW. Patient outcomes in the operative and nonoperative management of high-grade spondylolisthesis in children. *J Pediatr Orthop.* 2014 Jul-Aug;34(5):483-9.
14. Mac-Thiong JM, Hresko MT, Alzakri A, Parent S, Sucato DJ, Lenke LG, Marks M, Labelle H. *Eur Spine J.* 2019 Sep;28(9):2060-2069.
15. Morin C, Sales de Gauzy J, Jouve JL. *Orthop die P diatrique Rachis et Thorax.* Editions Elsevier Masson. 2016.
16. Poussa M, Remes V, Lamberg T, Tervahartiala P, Schlenzka D, Yrj nen T, Osterman K, Seitsalo S, Helenius I. Treatment of severe spondylolisthesis in adolescence with reduction or fusion in situ: long- term clinical, radiologic, and functional outcome. *Spine (Phila Pa 1976).* 2006 Mar 1;31(5):583-90; discussion 591-2.
17. Schoenecker PL, Cole HO, Herring JA, Capelli AM, Bradford DS. Cauda equina syndrome after in situ arthrodesis for severe spondylolisthesis at the lumbosacral junction. *J Bone Joint Surg Am.* 1990 ;72 : 369-77

18. Seitsalo S, Osterman K, Hyvärinen H, Schlenzka D, Poussa M. Severe spondylolisthesis in children and adolescents. A long-term review of fusion in situ. *J Bone Joint Surg Br.* 1990 Mar;72(2):259-65.
19. Schlenzka D, Remes V, Helenius I, Lamberg T, Tervahartiala P, Yrjönen T, Tallroth K, Osterman K, Seitsalo S, Poussa M. Direct repair for treatment of symptomatic spondylolysis and low-grade isthmic spondylolisthesis in young patients: no benefit in comparison to segmental fusion after a mean follow-up of 14.8 years. *Eur Spine J.* 2006 Oct;15(10):1437-47.
20. Violas P, Lucas G. L5S1 spondylolisthesis in children and adolescents. *Orthop Traumatol Surg Res.* 2016 Feb;102(1 Suppl):S141-7.

# Scheuermann's kyphosis: Etiology and diagnosis

Philippe Wicart

Necker-Enfants Malades Hospital, 149, Rue de Sèvres – 75015 Paris  
Paris Descartes University

## Introduction

Over a century has passed since Holder Scheuermann described a kyphotic deformity with anterior wedging of the vertebral bodies and characteristic irregularities of the vertebral endplates [1].

The first signs usually appear during the growth spurt at the start of puberty and no radiographic signs have been reported before the age of 10 years.

Scheuermann's kyphosis affects around 0.4 to 10% of adolescents aged between 10 and 14 years old. When even minor irregularities of the ossification of endplates are considered, the prevalence increases to 40%.

## Pathophysiology

### 1. Histology

Scheuermann suggested a form of aseptic necrosis of the growth plate at the level of the lateral vertebral margins. Schmorl and Junghans considered endplate herniations as typical lesions found in Scheuermann's disease and hypothesized that alterations in the consistency of the cartilaginous endplates may be the cause of Scheuermann's disease [2]. However, the occurrence of such irregularities may be seen outside of the kyphotic area and even in patients without Scheuermann's disease.

Histologic studies have revealed anomalies of the vertebral endplates and growth plates suggesting that the etiology may be an alteration of endochondral ossification. Aufdermaur and Spycher [3], and Ippolito and Ponseti [4] identified similar anomalies with a lack of osteoporosis or avascular necrosis of the lateral margins. In fact, Aufdermaur and Spycher [3] suggest that the primary histologic lesion may be an alteration of the connective tissue fibrils of the vertebral endplates that are disrupted, irregular, and may be fragmented, which lead to weakening of the connective sheath of the vertebral endplates. Ascani and Montanaro suggested different elements [5], with the primary anomaly being mosaic ≈ alterations of the cells and the extracellular matrix of the vertebral growth plates and endplates. Rarefaction and thinning of the collagenous fibers and excessive proteoglycans characterize this pathological extracellular matrix. The cellular phenomenon of endochondral ossification is altered, slowed, or absent with bone formation taking place directly from cartilage without

the process of physiological differentiation. These phenomena alter the longitudinal growth of the vertebra.

Scheuermann's disease is, in fact, secondary to a "slowing in growth" rather than a process of destruction. This finding contrasts with the fact that the vertebral endplates lying outside of the deformed zone are not affected by this pathological process. In fact, these zones are characterized by accelerated growth and hyperplasia of the lateral margins, thus leading to a progressive increase in vertebral wedging. According to Pierre Stagnara, this could be due to defective adaptation of the spine to the standing or sitting position [6]. Studies showing similar anomalies in large primates who are quadrupedal but only occasionally bipedal, conform to this hypothesis [7]. Other studies measuring the bone mineral density in patients with Scheuermann's disease have shown that a kyphosis superior to 45° is accompanied by notable osteoporosis [8], leading to possible therapeutic options.

## **2. Genetics**

A high incidence of Scheuermann's disease has been noted within families. Halal et al. studied the genetic transmission of Scheuermann's disease in 5 families with high incidences of this pathology and suggest an autosomal mode of transmission with a high degree of penetrance and variable expressivity [9]. The existence of this anomaly in the homozygous twin brother of a patient with Scheuermann's kyphosis is observed in almost 75% of cases. Within a 36-member family in whom signs of Scheuermann's kyphosis have been identified, the previously described observations have been found in 30% of the members, while only 4% are found in control subjects [10]. This anomaly may be seen in both males and females without an established sex ratio, although some authors have shown that boys are twice as likely to be affected than girls [10].

## **3. Mechanical**

Patients with Scheuermann's disease are larger and heavier with a higher body mass index than control subjects [11]. Nevertheless, this finding is not correlated to the severity of the deformity and may correspond to a hormonal profile that is associated rather than causal. In fact, bone age is higher for chronological age in patients with Scheuermann's disease and the role of transitory growth hormone hypersecretion has been hypothesized but not proven [12].

Moreover, vertebral overloading with repetitive microtrauma has been incriminated in the development of the lumbar type of Scheuermann's disease, especially in male subjects.

## **Conclusions on the pathophysiology**

The actual etiology of Scheuermann's disease remains unknown with variable and sometimes contradictory data resulting from histological studies of resected specimens. Typical disruption of the vertebral endochondral ossification may be the result rather than the cause of this pathology. Nonetheless, an evident genetic context and possible mechanical factors exist that could predispose to or aggravate the disease.

## Functional signs

Primary reasons for consultation are twofold, with the first being obviously kyphotic deformity. This kyphosis is generally uniform in the upright position and appears angular when the patient bends forward (Adams forward bending test) (figure 1).



**Figure 1:** uniform thoracic kyphosis in the upright position that becomes angular when the patient bends forward (Adams forward bending test) – The fingertip-to-floor distance is reduced and is probably due to hamstring tightness.

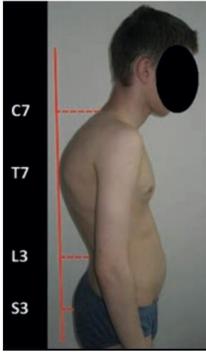
The kyphosis in Scheuermann's disease is associated with a significant limitation in sagittal spinal mobility both in flexion and in extension [13].

Pain is also a frequent reason for consultation. Pain at the apex of the deformity may be secondary to degenerative phenomena of the vertebra endplates and the intervertebral discs, as may be seen on MRI [14]. The rigidity of the thoracic kyphosis may explain the excess mobility of the adjacent cervicothoracic and thoracolumbar junctions [11]. The increased cervical and lumbar lordosis restoring the sagittal balance on either end of the thoracic kyphosis (figure 2) may in and of itself be the cause of the pain due to posterior interarticular impingement. The pain often radiates to the paraspinal area and distal to the level of the deformity [15]. Patients with Scheuermann's disease of the thoracolumbar spine, more specifically the lumbar spine, are especially prone to present with pain from a disrupted sagittal alignment.

The disordered global sagittal alignment of the spine with anterior translation of the head leads to muscle contractions that may damage certain muscles, such as the pectorals or the hamstrings [16] (figure 1).

## Physical exam

Similar to all spinal deformities, the physical exam starts by measuring the patient's length in the standing position, then only the head and trunk segments in the seated position. Clinical measurement of the sagittal curves may be done using a plumb line: distance between the plumb line running tangent to the apex of the kyphosis and the protrusion of C7, the apex of the lumbar lordosis, and the convexity of the sacrum (figure 2).



**Figure 2:** Exaggeration of cervical and lumbar lordosis on either end of the thoracic kyphosis restoring the sagittal balance – the plumb line allows for the measurement of the sagittal curves (C7: 7<sup>th</sup> cervical vertebra, T7: 7<sup>th</sup> thoracic vertebra, L3: 3<sup>rd</sup> lumbar vertebra, S3: 3<sup>rd</sup> sacral vertebra)

These measurements quantify the deformity and assess the results of the suggested treatment. In severe forms, the plumb line that runs tangent to the apex of the deformity lies greater than 2cm than the clinical contour of the sacrum (figure 2). A minor gibbosity (rib hump) may be found indicating a small scoliotic curve, often of the lumbar spine, that is often a non-structural deformity. Moderate pectus excavatum with a caved-in appearance of the thorax beneath the mammary glands, induced at least partially by the patient collapsing into kyphosis, accompanies quite often such kyphotic deformities (figure 3).



**Figure 3:** Moderate pectus excavatum with caved-in appearance beneath the mammary glands induced by the collapse into kyphosis.

Pectus carinatum is also possible but rare. Tightening of the hamstrings (figure 1) may be noted and evaluated with the popliteal angle. In some cases, posterior horizontal stretch marks may be seen at the apex of the kyphosis (figure 4), thought to be due to the increased straining on the cutaneous and subcutaneous tissues.



**Figure 4:** posterior horizontal stretch marks at the apex of the kyphosis.

Examining the limbs, the overlying skin, and a complete neuro-orthopedic exam complete the physical assessment.

Finally, the psychological burden of the disease felt by the child must be assessed, who is often introverted. This global attitude of collapsing in oneself may be the consequence of the deformity but may also be an element exacerbating the kyphosis.

### **Radiographic signs**

Radiographic diagnosis is based on whole-spine radiographs in the standing position using the EOS® system with measurement of the sagittal Cobb angle (between the tangent to the superior vertebral endplate of the upper end vertebra and the inferior vertebral endplate of the lower end vertebra) (figure 5). A sagittal Cobb angle superior to 40° at the level of the thoracic spine is deemed as pathological [17]. Pierre Stagnara considered that this cutoff value may be too stringent, suggesting instead that every patient has their own, personal sagittal alignment [6]. The sagittal Cobb angle is an indicator of the functional prognosis and is considered of poor prognosis if it is superior to 75°. Sagittal angular alterations are generally less important in patients with thoracolumbar or lumbar deformities with a limited wedging of the vertebra, giving way instead to Schmorl nodes. The Cobb angles of adjacent lordotic curves as well as the variable differences in sagittal balance allow a complete analysis of the deformity (figure 5).



**Figure 5:** Sagittal spinal radiograph of a subject with Scheuermann's disease of the thoracic spine: Wedging of the vertebral bodies around the apex of the kyphosis with vertebral endplate irregularities.

The radiographic diagnosis of Scheuermann's disease requires four criteria established by Sørensen (18):

Anterior wedging across 3 consecutive vertebrae around the apex superior to 5°, vertebral endplate irregularities, intervertebral disc space narrowing, and intravertebral disc herniations. All of the above criteria must not necessarily be met for the diagnosis to be made. Schmorl nodes correspond to a depression of the vertebral endplate and result from the penetration of the nucleus pulposus into adjacent cancellous bone. This finding is not specific to Scheuermann's disease and may be found even in subjects without vertebral pathology. Separation of the anterior vertebral margins (limbus vertebrae) at the level of the lumbar spine is secondary to the same pathophysiologic mechanism [19] (figure 6).



**Figure 6:** Schmorl node and separation of the anterior vertebral margins (limbus vertebrae) at the level of the lumbar spine.

## Differential diagnosis

Postural kyphosis differs from Scheuermann's disease by a reducible deformity both passively and actively (figure 7), and by the absence of vertebral wedging and other radiographic typical signs of Scheuermann's kyphosis. Kharrat and Dubousset described a type of kyphosis, probably congenital in nature, with progressive anterior fusion of the intervertebral discs which, prior to the appearance of these anomalies, could be falsely labeled as Scheuermann's disease [21]. Other types of kyphosis (spondyloepiphyseal dysplasia, type I neurofibromatosis, dysraphism) are characterized by distinct radiographic signs, relevant history and clinical elements allowing to rapidly establish the correct diagnosis.



**Figure 7:** Actively reducible [ostural thoracic kyphosis.

In sum, Scheuermann's kyphosis is a frequently encountered deformity that may lead to functional decline. The consideration of certain elements as well as knowledge of the natural history of the disease will guide management.

## References

1. Scheuermann HW (1921) : Kyphosis dorsalis juvenilis. *Orthop Chir* 41 : 305.
2. Schmorl G, Junghans H (1932) : Die gesunde und kranke wirbelsacule in roent. Liezig : Thieme Verlag.
3. Aufdermaur M, Spycher M (1986). Pathogenesis of osteochondrosis Juvenilis Scheuermann. *J Orthop Res.* 1986;4(4):452-7.
4. Ippolito E, Ponseti IV (1981) Juvenile kyphosis, histological and histochemical studies ? *J Bone Joint Surg Am.* 63:175.
5. Ascani E, Montanaro A (1985) : Scheuermann disease. In : Bradford, Hensinger RM, eds *The pediatric spine.* Berlin ; Thieme Verlag ; p 97.
6. Stagnara P (1982). Cyphoses thoraciques régulières pathologiques. In *Modern Trends in Orthopaedics.* Bologna : Gaggi de.p. 268.
7. Farrell BM, Kuo CC, Tang JA, Phan S, Buckley JM, Kondrashov DG. Scheuermann kyphosis in nonhuman primates. *Spine (Phila Pa 1976).* 2012 Nov 1;37(23):E1432-7.
8. Lopez RA, Burke SW, Levine DB, Schneider R. Osteoporosis in Scheuermann's disease. *Spine (Phila Pa 1976).* 1988 Oct;13(10):1099-103.
9. Halal F, Gledhill R, Fraser C (1978) : Dominant inheritance of Scheuermann's juvenile kyphosis. *Am J Dis Child* 132 ; 1105.
10. Damborg F, Engell V, Nielsen J, Kyvik KO, Andersen MØ, Thomsen K. Genetic epidemiology of Scheuermann's disease. *Acta Orthop.* 2011 Oct;82(5):602-5.
11. Fotiadis E, Kenanidis E, Samoladas E, Christodoulou A, Akritopoulos P, Akritopoulou K. Scheuermann's disease: focus on weight and height role. *Eur Spine J.* 2008 May;17(5):673-8.
12. Ascani E, Montanaro A (1985) : Scheuermann disease. In : Bradford, Hensinger RM, eds *The pediatric spine.* Berlin ; Thieme Verlag ; p. 307.
13. Stagnara P(1985). In : *Les déformations du rachis : scolioses, cyphoses, lordoses.* Masson ed, Paris New- York Barcelone Milan Mexico Sao Paulo, p 177.
14. Kalifa G, Cohen PA, Hamidou A. The intervertebral disk: a landmark for spinal diseases in children. *Eur Radiol.* 2002 Mar;12(3):660-5.
15. Lehmann TR, Brand RA, Gorman TWO 1983 : A low-back pain rating pain. *Spine* 8 : 308.
- 16 . Murray PM, Weinstein SL, Spratt KF. () The natural history and long-term follow-up of Scheuermann's kyphosis. *J Bone Joint Surg.* 199375A :236-248.
17. Lowe TG (1987) Mortality – morbidity committee report. Presented at Annual Meeting of the Scoliosis Research Society, Vancouver, British Columbia, Canada 1987.
18. Sørensen KH (1964) Scheuermann's juvenile kyphosis. Copenhagen :Mundsgaard.
19. Blumental S, Roach J, Harring J (1987). Lumbar Scheuermann's. *Spine*, 12 : 929.
20. Kharrat K, Dubousset J. Progressive anterior vertebral fusion in children. *Orthop Traumatol Surg Res* 1980, 66(8):485-92.

**Acknowledgements:** Georges Finidori and Lotfi Miladi for their friendly collaboration and abundant teachings.

# Imaging of meniscal and ligamentous injuries of the knee in children

Panuel Michel<sup>1,3</sup>, Desvignes Catherine<sup>1,2</sup>, Chaumoitre Kathia<sup>1,3</sup>, Petit Philippe<sup>1,4</sup>

<sup>1</sup> Department of Medical Imaging – North Hospital – Center for Medical Imaging AP-HM

<sup>2</sup> Department of Pediatric Radiology – Timone Children’s Hospital – Center for Medical Imaging AP-HM

<sup>3</sup> Aix-Marseille University, CNRS, EFS, ADES, Marseille, France

<sup>4</sup> Aix-Marseille University, EA 3279, Marseille France

## Introduction

Knee trauma is a common reason for presentation to the pediatric orthopedic clinic in children and adolescents. In fact, sports related accidents are more frequent than motor vehicle accidents [1-3]. The most frequently encountered injuries in this setting include fractures (contusions, growth plate injuries, osteochondral lesions), lesions of the extensor mechanism, and meniscal tears with or without ligamentous rupture. The imaging modalities for the diagnosis of meniscal and ligamentous injuries as well as their semiology will be discussed in this chapter.

## Imaging modalities

The recommendations of the American College of Radiology (ACR) on the radiographic evaluation of knee trauma based on the mechanism of injury and physical exam underline the necessity for conventional radiographs and MRI in most patients [4]. Other imaging modalities are less commonly indicated.

### 1. Conventional radiographs

Two orthogonal views are required and are generally sufficient in acute trauma or pain experienced during physical exercise: anteroposterior and true lateral views with the knee in extension. The following findings may be seen on conventional radiographs: 1. Knee joint effusion on lateral radiographs appearing as a thickening of the suprapatellar recess; 2. Traumatic bone or osteochondral lesions; 3. Dysplastic deformities (trochlear dysplasia, patellar dysplasia, etc.). Comparative radiographs are not indicated in this setting and should be avoided.

### 2. Magnetic resonance imaging (MRI)

Magnetic resonance is the preferred imaging modality for the assessment of intra-articular derangements of the knee in both adults and children. This allows a minute analysis of the meniscal and ligamentous structures, extensor mechanism, cartilage, bone marrow signal,

and presence or absence of edema of the adjacent soft tissues. The MRI exam must include views in the 3 planes and in various sequences. As a rule, fat saturation proton density, T1, and eventually echo gradient T2\* images must be obtained [5-10]. A standard MRI exam lasts for 20 minutes. With the development of new imaging techniques, three-dimensional reconstructions can be obtained in a shorter period of time with an excellent diagnostic value. However, this setting is not available on all MRI machines [11]. There is no consensus on the optimal moment to order an MRI; too early and post-traumatic edema may overestimate the lesion; Too late and important diagnostic information may be delayed. In addition, the use of an adapted antenna may be impossible in the presence of an immobilization apparatus such as a cast, thereby diminishing the quality of the acquired images. Injection of contrast media such as Gadolinium is usually unnecessary when the traumatic nature of the injury has been established.

Indications for MRI include the suspicion of internal derangements of the knee, the search for an occult fracture, persistent posttraumatic pain, and hemarthrosis.

### **3. Computed tomographic scan**

Computed tomography is usually not indicated except when a detailed analysis of an associated bony lesion is required [4].

### **4. Ultrasonography**

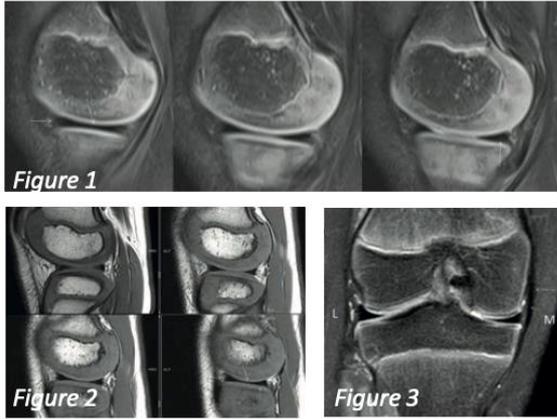
Ultrasonography is a powerful tool for the evaluation of joint effusion, the collateral ligaments, and the tendons of the extensor mechanism. It is also more sensitive than MRI in the evaluation of meniscal injuries [12]. However, ultrasonography is not recommended by the ACR in the diagnostic workup of meniscal and ligamentous injuries of the knee since it does not allow an evaluation of the entirety of neither the menisci nor the cruciate ligaments.

## **Normal findings**

Normal findings on an MRI exam of the knee will be developed in this section.

### **1. Menisci**

The medial meniscus is shaped like a “U” or “open C”, and the lateral meniscus, being more circular, is shaped like an interrupted “O”. Both menisci have a triangular appearance when sectioned with the base at the periphery and a thin central free edge. From front to rear, the meniscus is subdivided into an anterior horn, a body, and a posterior horn [13] (figures 1-3).



**Figure 1:** Contiguous sagittal views using fat saturation proton density MRI of the medial meniscus in a six-year-old child. Horizontal arrow: Anterior horn; oblique arrow: body; vertical arrow: posterior horn.

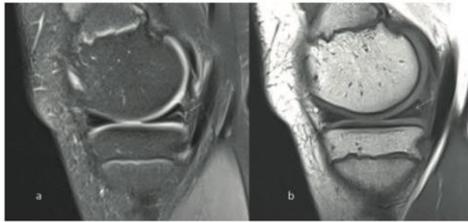
**Figure 2:** Contiguous sagittal views using T1 MRI of the lateral meniscus in the same patient as figure 1. Horizontal arrow: anterior horn; oblique arrow: body; vertical arrow: posterior horn.

**Figure 3:** Coronal view using fat saturation proton density MRI of the knee in a 12-year-old child showing the body of the meniscus and the medial collateral ligament (arrow). L: lateral, M: Medial.

The menisci grow in size with age. However, the growth of the lateral meniscus is slower than the corresponding surface of the tibial epiphysis [14]. The body is the narrowest part of the menisci. The structures that ensure the attachment of the menisci are easily recognizable, when they exist. These consist of the anterior intermeniscal and menisiofemoral ligaments. On the medial meniscus, the menisiofemoral and menisiotibial ligaments are prolongations of the deep fibers of the medial collateral ligament; the capsular attachment of the lateral meniscus is looser, but the fascicles of the menisioptiteal ligament facing the posterior horn are identifiable.

MRI of the menisci shows a low intensity signal on all sequences owing to their fibrocartilaginous nature. In practice, it is impossible to distinguish the two structural zones of the menisci: the richly vascularized peripheral “red zone” and the central avascular “white zone”. Meniscal signal on MRI, as initially described by Cruess et al. in 1987, is classified into four grades: Grade 0 corresponds to a globally homogeneous, low intensity signal of the meniscus; Grade 1 corresponds to a zone with a punctiform or ovoid higher intensity signal, without communication with an articular surface or the menisiocapsular junction; Grade 2 corresponds to linear or arcuate increased signal intensity without communication with the articular surfaces, although it may reach the menisiocapsular junction; Grade 3 is a linear or arcuate intrameniscal increased signal that is rather large and communicates with one or both articular surfaces. Grades 1 and 2 in both children and adults do not signify meniscal tear [5]. In children, increased signal intensity is due to a highly vascularized area and is found in 60% of children younger than 13 years of age; in adults, this is often due to mucoid degeneration

of the meniscus. The increased signal intensity is generally localized to the posterior horn of the medial meniscus [15] (figure 4).

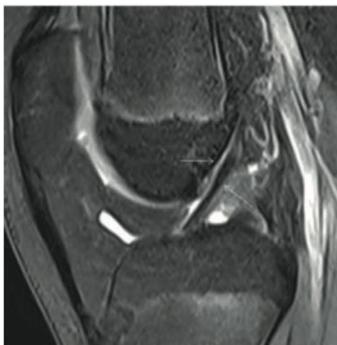


**Figure 4:** Sagittal view using fat saturation proton density (a) and T1 (b) MRI of the knee in an 11-year-old child. An area of relative increased signal intensity can be seen at the level of the posterior horn of the medial meniscus (oblique arrows). This aspect is not abnormal.

The aspects of grades 1 and 2 are classic pitfalls, often falsely interpreted as a meniscal tear [16]. A folded appearance of the free edge of the medial meniscus may be observed on the sagittal view without signifying pathology [16,17].

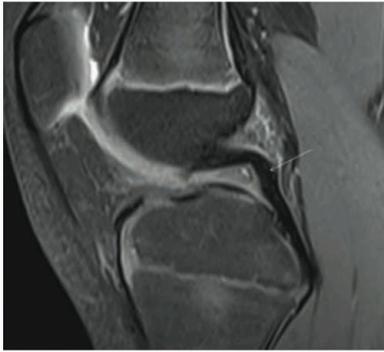
## 2. Cruciate ligaments

The anterior cruciate ligament (ACL) is an oblique fibrous band, easily recognizable on sagittal views of the knee, and stretched between the medial intercondylar eminence on the front and bottom, and the posterior part of the medial edge of the lateral femoral condyle on the rear and top (figure 5).



**Figure 5:** Sagittal view using fat saturation proton density MRI of the knee in an 11-year-old child illustrating the morphology of the ACL (oblique arrow): linear, and with a sharp anterior edge that is virtually parallel to the Blumensaat line (horizontal arrow).

When the knee is in extension, the ACL is linear and forms an angle of  $10^\circ$  or less with the Blumensaat line (roof of the intercondylar notch), with a low signal intensity on all sequences, and a relatively striated aspect. On coronal views, the constitutive bundles (anteromedial and posterolateral) are discernable. Recognizing the ACL in the coronal, sagittal and axial planes is essential in order to detect partial tears [18-20]. The posterior cruciate ligament (PCL) is longer but thicker than the ACL, extends from the posterior, median part of the tibial epiphysis on the bottom and rear, to the anterior, intercondylar edge of the medial condyle; In extension, the PCL forms a concave curve on the bottom and front (figure 6).



**Figure 6:** Sagittal view using fat saturation proton density MRI of the knee of the same patient as in figure 5. The morphology of the LCP (oblique arrow) can be seen with its anteroinferior concavity and its characteristic thickness.

### 3. Other ligaments

The collateral ligaments are easily identifiable on MRI and on ultrasound. The proximal, femoral insertions of the collateral ligaments are entirely epiphyseal [21]. A significantly lower intensity is seen on MRI with sharp and regular contours (figure 3). The anterolateral ligament is difficult to visualize in children younger than 15 years old [22].

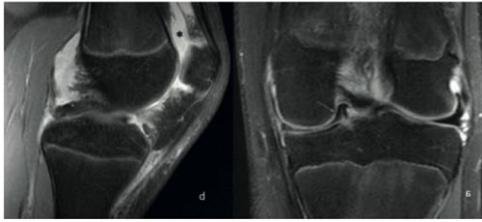
## Meniscal injury

The prevalence of meniscal injuries is lower in children compared to adults.

### 1. Meniscal tears

Meniscal tears may be isolated or associated with ligamentous and/or osteochondral lesions. In the context of trauma, the medial meniscus is more often damaged than the lateral meniscus, and its posterior horn more often injured than the anterior horn or body. The strong correlations between the aspect of the meniscus on MRI and surgery has long been established, with a sensibility of 85% and a specificity of 88 to 100% [7,8].

Different types of meniscal tears exist. However, only signal intensity alterations that communicate with the articular surface are considered pathological (grade 3). Meniscal tears may be horizontal, vertical, radial or oblique, peripheral or central, or of the bucket handle type. Bucket handle tears are distinct entities, and the diagnosis is easily made when a large fragment is projected within the intercondylar notch, giving rise to a “double PCL” sign on the sagittal views, and an absence or marked alteration of one of the meniscal horns or the body (figure 7). A meniscal flap may be projected anteriorly and give the aspect of a “double anterior horn”. A separation between the capsule and the meniscus or a loose meniscal fragment may be difficult to identify in children [5,23]. Finally, tears of the posterior horn of the lateral meniscus may be underestimated [24].



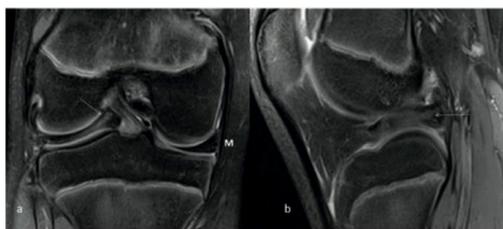
**Figure 7:** Coronal (a) and sagittal views (b) using fat saturation proton density MRI of the knee in a 13-year-old adolescent illustrating a bucket handle tear of the medial meniscus. The loose fragment (oblique arrows) is situated in the intercondylar notch on the coronal view (a) and gives the appearance of a “double PCL” on sagittal views (b). \*: hemarthrosis.

## 2. Discoid meniscus

A discoid deformity of the meniscus constitutes anomalies in both the shape and attachment of the meniscus. Discoid menisci are frequently encountered and affect the lateral meniscus (1.5% to 15% of the population depending on the series) more frequently than the medial meniscus (0.3% of the population) and are bilateral in almost 1/3 of cases. Multiple anatomical classifications have been developed [5,25-32]. The etiology remains unknown. A discoid meniscus may be asymptomatic but predisposes to complications which may be manifested clinically (pain, blocking). A clear history of trauma or intense physical exercise that may have started at an earlier age compared to other types of meniscal tears is often absent. On anteroposterior radiographs, lateral tibiofemoral joint widening, largening of the lateral femoral condyle with a squared shape, and cupping of the lateral tibial plateau may be seen. On MRI, the diagnosis is easily made in the absence of meniscal avulsion. The discoid meniscus is generally larger and thicker than regular menisci, and the anterior and posterior horns are seen on three contiguous sagittal slices [25]. A relatively thick horizontal fissure is consistently found (figure 8). If completely displaced, the discoid nature of the meniscus may be difficult to confirm [33] (figure 9).



**Figure 8:** Coronal view using fat saturation proton density (a) and sagittal view using T1 (b) MRI of the knee in a 7-year-old child. A discoid lateral meniscus is seen (horizontal arrow). M: medial.



**Figure 9:** Coronal (a) and sagittal (b) views using fat saturation proton density MRI of the knee in a 12-year-old adolescent illustrating a lateral meniscal tear over a discoid meniscus with a centrally and posteriorly displaced fragment (arrows). M: medial.

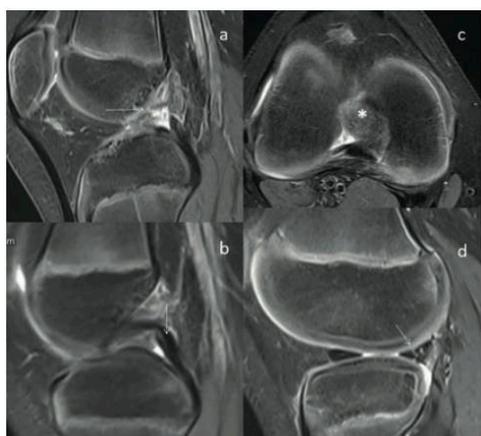
### 3. Meniscal cyst

Rarely found in children and adolescents, a meniscal or parameniscal cyst presents as painful and recurrent swelling over the femorotibial joint line. Ultrasonography is non-specific since the lesions may have a finely echoic, or even heterogenic content. On MRI, a meniscal tear, most often longitudinal, is consistently found; In the absence of such a tear, the differential diagnosis of a soft tissue mass should be considered [6,34].

## Ligamentous lesions

### 1. Anterior cruciate ligament lesions

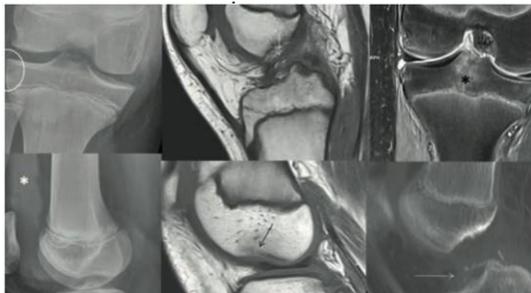
The preferred imaging modality for the assessment of the ACL is MRI. Both the direct and indirect signs that are described in adults may be applied to children as well [18,35]. Direct signs of an ACL tear include discontinuity of the fibers, modification of their orientation, and an abnormal signal (difficult to assess when there is significant joint effusion). Indirect signs include verticalization of the PCL, anterior shift of the tibia, posterior translation of the posterior horn of the lateral meniscus, joint effusion, and bone contusions (lateral femoral condyle and medial tibial plateau) (figure 10).



**Figure 10:** Sagittal (a,b,d) and transverse views (c) using fat saturation proton density MRI of the knee in an 11-year-old child illustrating a complete ACL tear (a, horizontal arrow; c, asterisk) with marked angulation of the PCL (b, vertical arrow), and vertical tear in the posterior horn of the lateral meniscus (d, oblique arrow).

These signs are rarely observed in their totality. One study found a sensitivity of 95% and a specificity of 88% in diagnosing ACL tears using MRI [35]. The diagnosis of a partial ACL tear is difficult to make and, based on the findings during the physical exam, the MRI may be repeated.

Tibial eminence fractures are more commonly encountered in children than purely ligamentous injuries and correspond to an osseocartilaginous avulsion of the distal insertion of the ACL. Multiple subtypes of tibial eminence fractures exist and are classified according to the Meyers and McKeever classification, which was later modified by Zaricznyj (36). The classification is based on a sagittal radiograph of the knee but may also be applied to MRI or CT-scan. Type I corresponds to no or minimal displacement, type II (the most frequent) corresponds to an anterior displacement of the fragment with an intact posterior hinge, type III is characterized by complete displacement of the osteochondral fragment, and type IV corresponds to complete displacement with rotation and comminution (figure 11). As a general rule, in avulsion fractures of the intercondylar eminence, the ACL is normal. Postoperative MRI evaluation after surgical repair of the ACL will not be discussed in this chapter.



**Figure 11:** 13-year-old adolescent presenting with trauma to the right knee. The initial radiographs (a,b) show a bony avulsion of the lateral border of the tibial epiphysis signaling a Segond fracture (a, circle), abundant hemarthrosis (b, asterisk), and a marked lateral condylar notch (b). MRI three weeks after the incident (c,d : sagittal views using T1 images; e: coronal view using fat saturation proton density images) show a continuous but serpentine aspect of the ACL (c, vertical arrow), impaction of the lateral condyle (d, arrow), and edema at the level of the tibial eminence (e, asterisk). CT-scan obtained at the same time as the MRI (f) shows a comminuted fracture of the intercondylar eminence (f, horizontal arrow).

## 2. Other ligamentous injuries

Injuries to the PCL are rare in children and adolescents and are generally secondary to hyperextension of the knee. A PCL rupture is diagnosed using MRI and shows an interruption of the fibers and/or a global or focal increased signal of the ligament. It is rarely an isolated injury. A mechanism of injury similar to that of ACL injuries may lead to an avulsion of the tibial insertion of the PCL (figure 12).



**Figure 12:** Sagittal views using T1 (a) and fat saturation proton density (b) MRI of the knee in a 13-year-old adolescent who had presented 3 months prior with trauma to the knee with persisting pain and instability. An osteochondral avulsion fracture of the posterior insertion of the PCL (oblique arrows) can be seen.

Injury to the collateral ligaments is rarely isolated, and medial compartment injuries are much more frequently observed than at the lateral compartment. These injuries may be assessed using ultrasound or MRI. On MRI, collateral ligament injuries are classified into three grades: grade 1, edema of the ligament and adjacent soft tissues with normal appearance; grade 2, partial disruption with thickening and edema of the ligament; grade 3, complete disruption of the ligament. In these lesions also, an osseocartilaginous avulsion fracture that is generally located at the proximal insertion may be secondary to the same mechanism of injury (figure 13).



**Figure 13:** 11-year-old child presenting with trauma to the right knee. The initial radiograph (a) shows an avulsion fracture of the lateral edge of the femoral epiphysis (a, oblique arrow). MRI obtained on the same day (b: sagittal view using T1 images; C: transverse view using fat saturation proton density images) confirms the avulsion fracture (oblique arrow) at the proximal insertion of the lateral collateral ligament (b, horizontal arrow) and the popliteus tendon.

## Associated lesions

Clinical and radiographic signs of hemarthrosis of the knee is an indication for an MRI. In fact, significant injuries (lesions of the extensor mechanism, osteochondral lesions, meniscoligamentous lesions, occult fractures) are observed in almost 50% of cases, with meniscoligamentous injuries found in almost 25% of cases [37-39]. Meniscal and ligamentous injuries may be isolated or associated with numerous other lesions, especially of the medial meniscus, ACL, and medial collateral ligament [20,40,41].

Findings of bone edema may provide some insight into the mechanism of injury and potential associated meniscoligamentous lesions must be ruled out [42]. Contrary to adults, traumatic

bone contusion in children may be isolated without associated meniscoligamentous lesions [41,43,44]. On conventional radiographs, the careful search for an osseous injury is imperative: e.g. in a Segond fracture, avulsion of the lateral edge of the lateral tibial plateau may be discrete and may even be absent on MRI; Its discovery must lead to an assessment of the ACL in order to rule out rupture, tibial eminence fracture and/or a lateral meniscal tear (figure 11).

## Messages

- MRI is the imaging modality of choice in meniscoligamentous injuries in children and adolescents as a complement to physical exam and conventional radiographs.
- In acute injuries, there is no consensus on the optimal moment for the realization of an MRI.
- Meniscal lesions may be isolated or, more frequently, associated with other injuries.
- An isolated hemarthrosis is an absolute indication for an MRI.

## References

1. Brown T, Moran M. Pediatric Sports-Related Injuries. *Clin Pediatr (Phila)*. févr 2019;58(2):199-212.
2. Kraus T, Švehlík M, Singer G, Schalamon J, Zwick E, Linhart W. The epidemiology of knee injuries in children and adolescents. *Arch Orthop Trauma Surg*. juin 2012;132(6):773-9.
3. Siow HM, Cameron DB, Ganley TJ. Acute Knee Injuries in Skeletally Immature Athletes. *Phys Med Rehabil Clin N Am*. 1 mai 2008;19(2):319-45.
4. Appropriateness Criteria [Internet]. [cité 29 déc 2019]. Disponible sur: <https://acsearch.acr.org/list/GetAppendix?TopicId=78&PanelName=Musculoskeletal>
5. Francavilla ML, Restrepo R, Zamora KW, Sarode V, Swirsky SM, Mintz D. Meniscal pathology in children: differences and similarities with the adult meniscus. *Pediatr Radiol*. 1 août 2014;44(8):910-25.
6. Gill KG, Nemeth BA, Davis KW. Magnetic Resonance Imaging of the Pediatric Knee. *Magn Reson Imaging Clin N Am*. nov 2014;22(4):743-63.
7. Pai DR, Strouse PJ. MRI of the Pediatric Knee. *Am J Roentgenol*. mai 2011;196(5):1019-27.
8. Strouse PJ. MRI of the knee: key points in the pediatric population. *Pediatr Radiol*. avr 2010;40(4):447-52.
9. Stein-Wexler R, Wootton-Gorges SL, Ozonoff MB, éditeurs. *Pediatric Orthopedic Imaging* [Internet]. Berlin Heidelberg: Springer-Verlag; 2015 [cité 30 déc 2019]. Disponible sur: <https://www.springer.com/gp/book/9783642453809>
10. Sanchez R, Strouse PJ. The knee: MR imaging of uniquely pediatric disorders. *Magn Reson Imaging Clin N Am*. août 2009;17(3):521-37, vii.
11. Fritz J, Ahlawat S, Fritz B, Thawait GK, Stern SE, Raithel E, et al. 10-Min 3D Turbo Spin Echo MRI of the Knee in Children: Arthroscopy-Validated Accuracy for the Diagnosis of Internal Derangement. *J Magn Reson Imaging JMRI*. juin 2019;49(7):e139-51.
12. Cook JL, Cook CR, Stannard JP, Vaughn G, Wilson N, Roller BL, et al. MRI versus ultrasonography to assess meniscal abnormalities in acute knees. *J Knee Surg*. août 2014;27(4):319-24.

13. Beaufils P, Verdonk R, éditeurs. The meniscus. Heidelberg: Springer Verlag; 2010. 407 p.
14. Bedoya MA, Barrera CA, Chauvin NA, Delgado J, Jaramillo D, Ho-Fung VM. Normal meniscal dimensions at different patient ages-MRI evaluation. *Skeletal Radiol.* avr 2019;48(4):595-603.
15. Takeda Y, Ikata T, Yoshida S, Takai H, Kashiwaguchi S. MRI high-signal intensity in the menisci of asymptomatic children. *J Bone Joint Surg Br.* mai 1998;80(3):463-7.
16. De Smet AA, Nathan DH, Graf BK, Haaland BA, Fine JP. Clinical and MRI Findings Associated with False-Positive Knee MR Diagnoses of Medial Meniscal Tears. *Am J Roentgenol.* juill 2008;191(1):93-9.
17. Tan K, Yoong P, Toms AP. Normal anatomical variants of the menisci and cruciate ligaments that may mimic disease. *Clin Radiol.* nov 2014;69(11):1178-85.
18. Lo IK, Bell DM, Fowler PJ. Anterior cruciate ligament injuries in the skeletally immature patient. *Instr Course Lect.* 1998;47:351-9.
19. Ho-Fung VM, Jaimes C, Jaramillo D. MR imaging of ACL injuries in pediatric and adolescent patients. *Clin Sports Med.* oct 2011;30(4):707-26.
20. Prince JS, Laor T, Bean JA. MRI of Anterior Cruciate Ligament Injuries and Associated Findings in the Pediatric Knee: Changes with Skeletal Maturation. *Am J Roentgenol.* 1 sept 2005;185(3):756-62.
21. Tschauner S, Sorantin E, Singer G, Eberl R, Weinberg A-M, Schmidt P, et al. The origin points of the knee collateral ligaments: an MRI study on paediatric patients during growth. *Knee Surg Sports Traumatol Arthrosc Off J ESSKA.* janv 2016;24(1):18-25.
22. Helito CP, Helito PVP, Leão RV, Louza ICF, Bordalo- Rodrigues M, Cerri GG. Magnetic resonance imaging assessment of the normal knee anterolateral ligament in children and adolescents. *Skeletal Radiol.* sept 2018;47(9):1263-8.
23. Dunoski B, Zbojniec AM, Laor T. MRI of displaced meniscal fragments. *Pediatr Radiol.* 1 janv 2012;42(1):104-12.
24. Gans I, Bedoya MA, Ho-Fung V, Ganley TJ. Diagnostic performance of magnetic resonance imaging and pre-surgical evaluation in the assessment of traumatic intra-articular knee disorders in children and adolescents: what conditions still pose diagnostic challenges? *Pediatr Radiol.* févr 2015;45(2):194-202.
25. Araki Y, Yamamoto H, Nakamura H, Tsukaguchi I. MR diagnosis of discoid lateral menisci of the knee. *Eur J Radiol.* mai 1994;18(2):92-5.
26. Bisicchia S, Botti F, Tudisco C. Discoid lateral meniscus in children and adolescents: a histological study. *J Exp Orthop.* 24 sept 2018;5(1):39.
27. Hagino T, Ochiai S, Senga S, Yamashita T, Wako M, Ando T, et al. Arthroscopic treatment of symptomatic discoid meniscus in children. *Arch Orthop Trauma Surg.* janv 2017;137(1):89-94.
28. Jung JY, Choi S-H, Ahn JH, Lee SA. MRI findings with arthroscopic correlation for tear of discoid lateral meniscus: comparison between children and adults. *Acta Radiol Stockh Swed* 1987. mai 2013;54(4):442-7.
29. Kocher MS, Logan CA, Kramer DE. Discoid Lateral Meniscus in Children: Diagnosis, Management, and Outcomes. *J Am Acad Orthop Surg.* nov 2017;25(11):736-43.
30. Kushare I, Klingele K, Samora W. Discoid Meniscus: Diagnosis and Management. *Orthop Clin North Am.* oct 2015;46(4):533-40.
31. Ahn JH, Lee YS, Ha HC, Shim JS, Lim KS. A Novel Magnetic Resonance Imaging Classification of Discoid Lateral Meniscus Based on Peripheral Attachment. *Am J Sports Med.* 1 août 2009;37(8):1564-9.

32. Rao SK, Sripathi Rao P. Clinical, radiologic and arthroscopic assessment and treatment of bilateral discoid lateral meniscus. *Knee Surg Sports Traumatol Arthrosc Off J ESSKA*. mai 2007;15(5):597-601.
33. Yaniv M, Blumberg N. The discoid meniscus. *J Child Orthop*. juill 2007;1(2):89-96.
34. Campbell SE, Sanders TG, Morrison WB. MR Imaging of Meniscal Cysts: Incidence, Location, and Clinical Significance. *Am J Roentgenol*. août 2001;177(2):409-13.
35. Lee K, Siegel MJ, Lau DM, Hildebolt CF, Matava MJ. Anterior cruciate ligament tears: MR imaging-based diagnosis in a pediatric population. *Radiology*. déc 1999;213(3):697-704.
36. Casalonga A, Bourelle S, Chalencon F, De Oliveira L, Gautheron V, Cottalorda J. Tibial intercondylar eminence fractures in children: The long-term perspective. *Orthop Traumatol Surg Res OTSR*. sept 2010;96(5):525-30.
37. Abbasi D, May MM, Wall EJ, Chan G, Parikh SN. MRI findings in adolescent patients with acute traumatic knee hemarthrosis. *J Pediatr Orthop*. déc 2012;32(8):760-4.
38. Askenberger M, Ekström W, Finnbogason T, Janarv P-M. Occult Intra-articular Knee Injuries in Children With Hemarthrosis. *Am J Sports Med*. juill 2014;42(7):1600-6.
39. Wessel LM, Scholz S, Rüsç M, Köpke J, Loff S, Duchêne W, et al. Hemarthrosis after trauma to the pediatric knee joint: what is the value of magnetic resonance imaging in the diagnostic algorithm? *J Pediatr Orthop*. juin 2001;21(3):338-42.
40. Samora WP, Palmer R, Klingele KE. Meniscal pathology associated with acute anterior cruciate ligament tears in patients with open physes. *J Pediatr Orthop*. mai 2011;31(3):272-6.
41. Shea KG, Grimm NL, Laor T, Wall E. Bone bruises and meniscal tears on MRI in skeletally immature children with tibial eminence fractures. *J Pediatr Orthop*. mars 2011;31(2):150-2.
42. Zhang L, Hacke JD, Garrett WE, Liu H, Yu B. Bone Bruises Associated with Anterior Cruciate Ligament Injury as Indicators of Injury Mechanism: A Systematic Review. *Sports Med Auckl NZ*. mars 2019;49(3):453-62.
43. Snearly WN, Kaplan PA, Dussault RG. Lateral- compartment bone contusions in adolescents with intact anterior cruciate ligaments. *Radiology*. janv 1996;198(1):205-8.
44. Gómez JE, Molina DD, Rettig SD, Kan JH. Bone Bruises in Children and Adolescents Not Associated With Ligament Ruptures [corrected]. *Orthop J Sports Med*. 2018;6(7):2325967118786960.

# Overuse injuries in children

Pr Aurélien Courvoisier

Department of Pediatric Orthopedics  
Couple/Child Hospital  
Grenoble Alpes University Hospital

## Introduction

In its most classic definition, an overuse injury is a chronic injury due to a high level of physiological load exerted on the musculoskeletal system without allowing ample amount of rest between training sessions [1,2]. As a result, a quantified amount of overuse cannot be specifically defined for the entire pediatric population.

There has been a recent increase in overuse injuries in children, injuries which are often ignored or trivialized and are categorized as “growing pains”. However, growth is not painful and musculoskeletal pathologies are responsible for the pain, an invalidating complaint for many children. These pains are especially aggravated during pubertal growth spurts when the lower limbs develop too quickly and the entire musculoskeletal system is overloaded [3].

This chapter will mostly focus on sports-related injuries, even though overuse injuries may occur in children who practice little to no sports. Even non-athletic children complaining of chronic pains that are typical of these types of injuries frequently seek medical attention.

Instead of simplifying the issue as one affecting hyperactive athletic children in general, it would be preferable to compare it to a mismatch between “supply”, i.e. the musculoskeletal system’s ability to cope with a given activity over a certain period of time during growth, and “demand”, i.e. the intensity, frequency, and duration of one or multiple activities; It should be noted that, in certain children, merely activities of daily living may be considered as physical activity.

The most common overuse injuries of one or multiple extremities will be discussed in this chapter while concentrating on the more typical sports-related injuries. In order to simplify the presentation, reasons for consultation will be discussed first, i.e. pain in either the lower or upper limbs.

## Lower limb pain

Pain located at the knee or tibia is a frequent complaint in preadolescent children seeking medical attention. The most common overuse injuries include:

- Osgood-Schlatter disease
- Sinding-Larsen-Johansson syndrome

These two entities represent 18% of all causes of overuse injuries in the pediatric population and form part of the grand family of osteochondroses [4].

Osgood-Schlatter disease is defined as chronic inflammation of the patellar tendon at its insertion on the tibial tuberosity. In its most frequent form, Osgood-Schlatter disease affects girls between 8 and 13 years old and boys between 10 and 15 years old and is the primary ailment suffered in France by boys engaging in professional football (soccer) [5]. While striking the ball, the quadriceps is locked, the knee is in full extension, and the entire extensor mechanism is stressed. Pain, in the absence of appropriate management, may become invalidating to the point of preventing the child from participating.

The typical clinical presentation includes pain during physical activity, tenderness, and, in its more severe forms, swelling and erythema at the level of the tibial tuberosity. Conventional radiographs are often normal; fragmentation of the secondary ossification center of the tibial tuberosity is a normal finding. Contrarily, in its chronic form, ossification of the patellar tendon may be rarely seen. Further complementary examination is unnecessary. Radiographs themselves are not always required, especially when the clinical findings are typical, and the pain is bilateral [6,7]. Care must be taken in case of clinical doubt and radiographs must be obtained in the search for a differential diagnosis, particularly bony tumors.

Sinding-Larsen-Johansson syndrome is the equivalent of Osgood-Schlatter disease of the proximal insertion of the patellar tendon [3]. Clinically, the child complains of pain on the anterior aspect of the knee; the patient cannot always precisely localize the pain. Palpation of the tip of the patella with the knee in extension and the quadriceps relaxed elicits intense pain. As in Osgood-Schlatter disease, radiographs are most commonly normal. In more chronic presentation, a spur may be seen on the inferior pole of the patella.

At the level of the lower limbs, the type of overuse injury also depends on the type of sporting activity. In runners, in addition to the two previously discussed injuries, one of the most frequent ailments is iliotibial band syndrome [8,9]. Findings are characteristic, since they occur mainly during the act of running. The pain is localized on the lateral side of the knee, precisely on the lateral aspect of the lateral femoral condyle. The pain is so intense that the child will inevitably discontinue the physical activity. The lateral condyle is tender, and rest provides relief. However, return to running unavoidably reawakens the pain. However, participating in other sporting activities that load the knee in a different manner than running may be possible without awakening the pain. Iliotibial band syndrome is generally due to excessive friction between the distal iliotibial band and the lateral femoral epicondyle.

In girls, pain during the act of running is most frequently localized over the anterior tibia [9]. Two entities are to be distinguished: Medial tibial stress syndrome (MTSS), and stress fractures. MTSS is more commonly encountered in girls, those with little experience in running, those with a previous MTSS, and those with a high body mass index (BMI). Contrarily, stress fractures are found in high-level adolescent runners [10-12]. It is important to note that stress fractures occur on a normal bone. Conventional radiographs are therefore essential in the workup of the pain, although they are often insufficient for the official diagnosis. A common finding is thickening of the anterior cortex of the tibia. However, the fracture line may be subtle and difficult to identify. MRI or CT-scans are sometimes necessary in order to

make an official diagnosis [4]. Physicians must be prudent when treating children with overuse injuries since they may be pressured by the child's parents, especially since diagnostic delay often leads to apprehension. In some cases, if a tumor is included within the differential diagnoses, complementary exams must be ordered in order to rule out the diagnosis. MRIs are not always beneficial in stress fractures since cortical bone density is high, and the low spatial resolution of the MRI does not allow identification of the fracture line.

Sever disease is an osteochondrosis similar to Osgood-Schlatter disease and is localized at the level of the insertion of the Achilles tendon on the calcaneus [1-4]. Sever disease is very frequently encountered in children during growth spurts. Patients typically complain of pain at the level of the heel that is exacerbated while walking and particularly during running. For some patients, mere contact between the heel and the floor may be impossible due to excruciating pain, leading these patients to walk on the tips of their toes. On physical exam, there is tenderness while pinching the heel or while palpating the insertion of the Achilles tendon. Radiographs are rarely required except when there is diagnostic doubt. Fragmentation of the secondary ossification center of the calcaneus is a normal finding and is not a sign of Sever disease. Stress fractures may also occur level of the foot. Nevertheless, their prevalence remains too low thereby precluding a discussion in this chapter. In patients presenting with foot pain in the setting of intense physical exercise, a stress fracture must be considered, and an MRI – the preferred imaging modality – must be ordered.

## **Upper limb pain**

Overuse injuries of the upper limbs are usually due to excessive traction or compression at the level of a joint. The 3 most frequently encountered injuries in the literature are:

- Gymnast's wrist
- Little leaguer's shoulder
- Little leaguer's elbow

The last two listed injuries are extremely rare in Europe where baseball is not a commonly practiced sport, although they are common entities in adolescent pitchers in the United States. Moreover, gymnastics is a pediatric sport that is almost exclusively high-performance, even though the Olympic games are reserved for those older than 16 years of age. Gymnasts reach their highest level of performance even before puberty, with 75% of gymnasts having already reported wrist pain during practice or competition [4].

Gymnast's wrist is an injury that arises due to repetitive compressions of the wrist in hyper-extension. As a result, there is premature closure of the distal physis of the radius. Initially, pain is felt at the level of the wrists, and radiographs are most commonly normal, and MRI may show edema of the distal metaphyses of the radius and ulna [13,14].

Little leaguer's elbow is a generic term to describe a group of injuries of the child's elbow that occur in baseball players [4,15]. However, Little leaguer's elbow specifically affects the medial epicondyle of the elbow and is caused by repetitive traction in children aged between 11 to 15 years old thus leading to chronic widening of the physis of the medial epicondyle. Nonetheless, its occurrence in Baseball players is exceptional in France, although it may be

seen in tennis players. The famous “tennis elbow” encountered in adults may be translated in children by a lesion of the ossification center of the medial epicondyle. Radiographs show widening of the physis and the diagnosis is confirmed by signs of inflammation on an MRI.

Little leaguer’s shoulder corresponds to epiphysiolysis of the proximal humerus due to the repetitive motion of pitching [15,16]. This injury is exceptional in France.

#### TREATEMENT AND PREVENTION [1,6]

The treatment of overuse injuries is discussed in another chapter. Nevertheless, the essential concepts of treatment and especially the prevention of overuse injuries will be touched upon.

Once the diagnosis is made, it is fundamental to approach these injuries in their entirety, and risk factors should be identified promptly. Intrinsic and extrinsic risk factors should be differentiated [4,17].

Intrinsic risk factors include the child’s internal personal factors. These may be further divided into modifiable and non-modifiable risk factors.

Modifiable intrinsic risk factors include BMI, strength, and flexibility [3,4,8,18]. Non-modifiable intrinsic risk factors include age, height, timing of the pubertal growth spurt, and previous overuse injuries. Moreover, having previously been diagnosed with an overuse injury is the primary risk factor for the occurrence of another.

Extrinsic risk factors are, by definition, modifiable and are related to the child’s exercise regimen: Volume, intensity, type of coaching, variability of the exercises, and the level of competition before the start of the present season [4,8,9,17].

There is no consensus on prevention strategies. However, a period of active rest after a competition cycle is indicated. Adapted training with more variability is also recommended [17].

There is no consensus of the optimal treatment approach either. Some studies suggest complete cessation of sporting activities, while others suggest adapting the physical activity based on the child’s symptoms in order to limit the psychological impact of cessation of sports in these young athletes. Some authors suggest programs with stretching and physical conditioning [19]. However, no studies have compared the different therapeutic approaches. It is always recommended, independently of the adopted therapeutic strategy, not to return to intensive competition or sports until after the pain has completely subsided [4].

The relationship between the sports medicine physician, surgeon, sports instructor, parents, and child must be fluid and transparent in order to, firstly reassure the patient and their family, and secondly to accompany the often-protracted healing process. This may sometimes even lead to a reconsideration of the practiced sports. However, numerous prejudices often persist, and it may sometimes be difficult to discuss these sensitive topics with the patients and their families. Nevertheless, it is paramount to remain centered on the initial objective and to remain focused on the child’s well-being rather than their performance.

## REFERENCES

1. Launay F. Sports-related overuse injuries in children. *Orthop Traumatol Surg Res.* 2015;101(1 Suppl):S139-47. Epub 2014/12/30. doi: 10.1016/j.otsr.2014.06.030. PubMed PMID: 25555804.
2. Stracciolini A, Casciano R, Friedman HL, Meehan WP, Micheli LJ. A closer look at overuse injuries in the pediatric athlete. *Clin J Sport Med.* 2015;25(1):30-5. doi: 10.1097/JSM.000000000000105. PubMed PMID: 24926911.
3. Wu M, Fallon R, Heyworth BE. Overuse Injuries in the Pediatric Population. *Sports Med Arthrosc Rev.* 2016;24(4):150-8. doi: 10.1097/JSA.000000000000129. PubMed PMID: 27811514.
4. Arnold A, Thigpen CA, Beattie PF, Kissenberth MJ, Shanley E. Overuse Physeal Injuries in Youth Athletes. *Sports Health.* 2017;9(2):139-47. Epub 2017/02/06. doi: 10.1177/1941738117690847. PubMed PMID: 28165873; PubMed Central PMCID: PMC5349397.
5. Leppänen M, Pasanen K, Clarsen B, Kannus P, Bahr R, Parkkari J, et al. Overuse injuries are prevalent in children's competitive football: a prospective study using the OSTRC Overuse Injury Questionnaire. *Br J Sports Med.* 2019;53(3):165-71. Epub 2018/08/14. doi: 10.1136/bjsports-2018-099218. PubMed PMID: 30108062.
6. Journeau P, Polirzstok E, Launay F, Barbier D. [OVERUSE INJURIES IN THE YOUNG ATHLETE]. *Rev Prat.* 2015;65(8):1084-90. PubMed PMID: 26749713.
7. Chang GH, Paz DA, Dwek JR, Chung CB. Lower extremity overuse injuries in pediatric athletes: clinical presentation, imaging findings, and treatment. *Clin Imaging.* 2013;37(5):836-46. Epub 2013/06/04. doi: 10.1016/j.clinimag.2013.04.002. PubMed PMID: 23759208.
8. Hogan KA, Gross RH. Overuse injuries in pediatric athletes. *Orthop Clin North Am.* 2003;34(3):405-15. doi: 10.1016/s0030-5898(03)00006-3. PubMed PMID: 12974490.
9. Seto CK, Statuta SM, Solari IL. Pediatric running injuries. *Clin Sports Med.* 2010;29(3):499-511. doi: 10.1016/j.csm.2010.03.005. PubMed PMID: 20610035.
10. Shelat NH, El-Khoury GY. Pediatric stress fractures: a pictorial essay. *Iowa Orthop J.* 2016;36:138-46. PubMed PMID: 27528851; PubMed Central PMCID: PMC5349397.
11. Changstrom BG, Brou L, Khodae M, Braund C, Comstock RD. Epidemiology of stress fracture injuries among US high school athletes, 2005-2006 through 2012-2013. *Am J Sports Med.* 2015;43(1):26-33. Epub 2014/12/05. doi: 10.1177/0363546514562739. PubMed PMID: 25480834.
12. Heyworth BE, Green DW. Lower extremity stress fractures in pediatric and adolescent athletes. *Curr Opin Pediatr.* 2008;20(1):58-61. doi: 10.1097/MOP.0b013e3282f370c0. PubMed PMID: 18197040.
13. Bell DR, Post EG, Biese K, Bay C, Valovich McLeod T. Sport Specialization and Risk of Overuse Injuries: A Systematic Review With Meta-analysis. *Pediatrics.* 2018;142(3). Epub 2018/08/22. doi: 10.1542/peds.2018-0657. PubMed PMID: 30135085.
14. Lomasney LM, Lim-Dunham JE, Cappello T, Annes J. Imaging of the pediatric athlete: use and overuse. *Radiol Clin North Am.* 2013;51(2):215-26. Epub 2012/12/25. doi: 10.1016/j.rcl.2012.09.014. PubMed PMID: 23472587.
15. Mautner BK, Blazuk J. Overuse throwing injuries in skeletally immature athletes--diagnosis, treatment, and prevention. *Curr Sports Med Rep.* 2015;14(3):209-14. doi: 10.1249/JSR.000000000000155. PubMed PMID: 25968854.

16. Heyworth BE, Kramer DE, Martin DJ, Micheli LJ, Kocher MS, Bae DS. Trends in the Presentation, Management, and Outcomes of Little League Shoulder. *Am J Sports Med.* 2016;44(6):1431-8. Epub 2016/03/16. doi: 10.1177/0363546516632744. PubMed PMID: 26983458.
17. Paterno MV, Taylor-Haas JA, Myer GD, Hewett TE. Prevention of overuse sports injuries in the young athlete. *Orthop Clin North Am.* 2013;44(4):553-64. Epub 2013/08/29. doi: 10.1016/j.ocl.2013.06.009. PubMed PMID: 24095071; PubMed Central PMCID: PMC3796354.
18. Brown T, Moran M. Pediatric Sports-Related Injuries. *Clin Pediatr (Phila).* 2019;58(2):199-212. Epub 2018/11/27. doi: 10.1177/0009922818810879. PubMed PMID: 30477308.
19. Pengel KB. Common overuse injuries in the young athlete. *Pediatr Ann.* 2014;43(12):e297-308. doi: 10.3928/00904481-20141124-09. PubMed PMID: 25486038.

# Treatment of overuse injuries in children

Richard Gouron

Chief of Department of Pediatric Surgery. Department of Pediatric Orthopaedic Surgery Amiens University Hospital. SSPC Laboratory (Simplification des Soins des Patients Chirurgicaux Complexes) Picarde Jules Verne University.  
Institut Fédératif GRECO (Groupe de Recherche En Chirurgie rObotique) Picarde Jules University.

## Introduction

The intensity of physical exercise in children is constantly increasing [1,2] and many of these children practice multiple types of sports with decreasing periods of rest at the end of the sporting season. This leads to permanent overuse of the musculoskeletal system [3]. Moreover, children have a tendency to specialize in one type of sport precociously, with a constant premature increase in intensity, thereby leading to an overt risk of overuse injuries [4].

Repeatedly stressing the bones, muscles and tendons without appropriate recuperation and healing times leads to microtrauma of these structures. The growth plate is physiologically vulnerable, especially at the level of the apophyses where the strained tendons attach, leading to specific injuries. The physis and the epiphysis might also be stressed which could lead to intraarticular lesions. Finally, repetitive compression and traction of a bone may lead to bone marrow edema and stress fractures of the diaphysis, metaphysis, or the spine (spondylolysis).

The treatment of these injuries must be aimed at the entire pathophysiological process of the overuse injury and, of course, at the specific lesions and local consequences cause by this increased load.

## General principles

Rest is the basis of the treatment of overuse injuries and should be absolute, unless severe intra-articular injuries have occurred. Depending on the severity of the lesion and pain, training may be simply adapted to the patient's injury or the specific movement that is overloading a certain joint modified [5].

Immobilization is sometimes useful in alleviating pain or in facilitating the healing of a bony fragment.

Conventional painkillers or NSAIDs are rarely prescribed since artificial pain relief might encourage the child to return to sports prematurely, thereby leading to further overuse and exacerbation of the injury. Furthermore, peritendinous or peri-apophyseal injections must be avoided [5].

Surgery may be indicated in some cases in order to facilitate healing, remove or repair an loose osteochondral fragment, or treat sequelae of a previous injury.

Patience is key in overuse injuries since a significant proportion of these injuries (of the physes or apophyses) will spontaneously heal during puberty as the growth plates begin to fuse and the pelvis reaches Risser grade 1. Overuse injuries are diverse, and every anatomical site and type of injury (apophysis, epiphysis, physis or stress fracture) has its own characteristics.

## **Treatment of apophyseal injuries**

### General principles [6]

The first step in the management of apophyseal injuries is relative rest. The presence of residual pain will guide management to either need for further rest or return to sports. If pain is experienced at the level of the apophysis during physical exercise, sports should be restricted for a period of days to weeks. Return to sports may be achieved progressively with, at first, restricted training times, until the patient's previous level of intensity is reached. If symptoms reoccur, activity is once again either reduced or withheld.

The family, school and trainer must be informed that adjusting the training volume depending on the child's pain may be considered. For high-performance athletes, a sporting activity that spares the injured joint may be authorized in order to maintain cardiovascular conditioning.

Extremely painful episodes may be managed by temporarily immobilizing the limb. This immobilization must not be too prolonged in order to avoid muscular atrophy.

Painkillers may be prescribed, although local treatments such as icing are preferable. Finally, physical therapy is sometimes useful in increasing the tightened muscle's flexibility and length – an integral part of the pathophysiology of apophysitis – and should be initiated as soon as the apophyseal pain has subsided. In fact, continued strain on the apophysis during rehabilitation may maintain the microlesion that is at the source of the symptoms that the physical therapist is attempting to treat. Daily stretching programs may therefore be simpler and more useful than formal physical therapy.

Return to normal sporting activity is allowed depending on the non-recurrence of the injury by maintaining adequate flexibility and proper muscular length.

### Elbow [3]

Medial epicondyle apophysitis, secondary to valgus loading in patients with open medial epicondylar growth plates (between 9 and 12 years), is frequently encountered in tennis and baseball players. The initial treatment is limiting the activity causing the increased load (e.g. pitching) along with local treatments such as icing. Adequate joint range of motion should be maintained as soon as symptoms decrease in order to avoid stiffness. Furthermore, young athletes should maintain some sort of physical activity in order to preserve their cardiovascular conditioning. Return to sports is achieved progressively while limiting the number of pitches or serves in tennis players. In addition, premature return to sports may

lead to recurrence of the injury, medial epicondylar hypertrophy, or an apophyseal avulsion fracture requiring surgical fixation.

### Pelvis and hip

Apophyseal injuries of the iliac spine, ischium, or lesser trochanter are essentially avulsion fractures secondary to the attachments of the tendons [7]. The treatment is very frequently symptomatic. Rest with unloading of the limb and bed rest for a period of 7 to 10 days is recommended. The patient's position during this time should reduce the strain of the tendon in question (e.g. flex hip for injuries of the anteroinferior iliac spine caused by traction of the rectus femoris).

Weight bearing is returned progressively. Potential muscle tightness must be assessed and treated by stretching as soon as the pain subsides. Return to sporting activities is generally allowed after the second month. Repositioning and surgical fixation should be discussed but does not allow a faster return to sports. Nevertheless, a recent meta-analysis showed a return to sports at a higher level after surgery compared to conservative treatment and recommends that fragments with a displacement superior to 15mm be treated with surgical fixation, especially in high-level athletes [8].

### Knee

Apophyseal injuries of the knees are essentially chronic injuries, such as Osgood-Schlatter disease of the tibial tuberosity and Sinding-Larsen-Johansson syndrome of the tip of the patella. Treatment consists primarily of relative rest. Cessation of all sports that are at the origin of the increased strain on the apophysis will lead to progressive pain relief. Return to sports is achieved progressively with an adjustment of the volume and intensity of the training and is primarily guided by the persistent symptoms. Painkillers and local treatments such as icing are also useful.

Immobilization with a knee brace may be considered if symptoms are severe or resistant to symptomatic treatment. Once a pain-free return to sports has been achieved, patients with Osgood-Schlatter disease or Sinding-Larsen-Johansson syndrome must maintain adequate flexibility of the quadriceps and hamstrings in order to limit strain and traction on the apophysis and decrease the risk of injury recurrence [7,9]. In fact, a direct link has been found between symptomatic Osgood-Schlatter disease and muscle tightness [10].

Intratendinous heterotopic ossifications may be a potential complication of apophysitis and require surgical excision if pain is experienced on the long term despite physal closure [3,7,11].

### Ankle and foot

Apophysitis of the greater tuberosity of the calcaneus (Sever disease) is frequently encountered in children between 8 and 12 years of age [7] and treatment is always symptomatic. Initially, physical exercise must be limited until the pain subsides. A triceps surae stretching program must then be initiated since posterior chain tightness is a fundamental element of Sever disease [9,11]. Commercial elastomer insoles may be

prescribed decreasing ground reaction forces during gait. Nevertheless, these insoles must reduce the shock all the while avoiding concealing any equinus that may be due to a tight heel cord. In fact, such equinus would only exacerbate the traction forces on the calcaneal apophysis.

More severe pain may require offloading the affected limb with crutches or a wheelchair. Immobilization with a short leg cast or a walking boot for a period of 3 to 4 weeks may sometimes also be useful in more severely painful episodes [9].

Other osteochondrites of the foot are rarely encountered. Navicular injuries are managed with symptomatic treatment. The bone is physiologically reconstructed over a period of months, during which intense sporting activities must be suspended and plantar orthoses supporting the medial arch, or even short periods of immobilization, may be utilized depending on the severity of the pain [7].

The treatment of Iselin disease (base of the 5<sup>th</sup> metatarsal), Renander disease (sesamoid of the greater toe), and Freiberg disease (head of 2<sup>nd</sup> metatarsal) is identical to Sever disease. Rarely, in some patients with Freiberg's disease, an osteotomy of the head of the 2<sup>nd</sup> metatarsal may be necessary [7].

## **Treatment of epiphyseal injuries**

### Elbow

Osteochondritis of the lateral condyle of the elbow, or Panner disease, occurs in very young children and presents primarily as painful and limited elbow extension without locking sensations or intra-articular loose bodies. The natural history of the disease always progresses toward healing.

Treatment is therefore only symptomatic with painkillers, restriction of sporting activities, and exceptionally immobilization until symptomatic relief over 12 to 18 months [7,12]. Osteochondritis dissecans of the capitellum, occurring essentially in adolescents, presents a similar clinical scenario with sometimes locking and clicking due to loosening of an osteochondral fragment within the joint.

Treatment depends on the stability of the osteochondral fragment. Stability is generally evaluated using MRI: instability is evidenced by an increased signal intensity surrounding the osteochondral fragment on T2-weighted images [12].

In patients with stable lesions, rest and restriction of all physical exercise causing excessive valgus load on the elbow, such as gymnastics, for a period of at least 6 months is recommended. Patients with persistent signs of instability or with an intra-articular loose body after a period of conservative management may benefit from surgery [11,12], preferably by arthroscopy. Arthroscopic management consists of debridement of the osteochondral area and removal of the loose osteochondral fragment from the joint space. Perforating the lesion area may relieve the pain and facilitate healing, especially in skeletally mature patients [13]. For lesions larger than 10mm, osseocartilaginous grafts are generally used [14].

## Knee

Juvenile osteochondritis dissecans of the knee often only requires symptomatic treatment [9]. The lesions are generally stable with an intact joint line. Load from physical exercise should be reduced for a minimum of 6 months, although weight bearing is authorized. A short non-weight-bearing period may be indicated if pain is the primary complaint [7]. MRI determines the presence of associated cartilaginous lesions and guides management. Unlike in adults, an increased signal intensity on T2 images in children does not indicate instability of the osteochondral fragment [15]. If the osteochondral fragment is smaller than 2.5 cm<sup>2</sup>, half of patients achieve healing over a period of 6 months, with the rest almost always within 18 months [7]. In patients in whom healing is delayed, extra-articular perforations of the osteochondral area through the epiphysis may be considered in order to increase vascularization and facilitate consolidation, all the while preserving the articular surface [9,16]. If the cartilage is injured, arthroscopy may be used to assess and then perforate the damaged area through the joint line in order to accelerate healing. Loose osteochondral fragments within the joint may also be removed via arthroscopy. If the fragment is large enough, fixation must be attempted even though healing is not guaranteed. However, if the same fragment is on a weight-bearing portion, then osteochondral mosaicplasty may be attempted [17].

## Talus

Osteochondral lesions of the talus may be either anteromedial (primarily as a complication of a sprain) or posteromedial, with the latter being the most frequent location of osteochondrosis [7]. Treatment consists primarily of complete sporting restriction. Radiographic and MRI surveillance are used to guide the remainder of the treatment. These lesions may heal spontaneously over several months, although chondral lesions may require perforation either by arthroscopy or arthrotomy with sometimes a trans-malleolar approach to better expose the injured area.

## **Treatment of physeal injuries**

### General principles

Physeal injuries may be due to acute trauma, the treatment of which is within the realm of general trauma and will not be detailed in this chapter. Overuse injuries of the growth plate may occur in young athletes and at different anatomical locations, and be due to different types of sports. The majority of these lesions heal without complications by simply limiting physical exercise. However, epiphysiodesis has been described in rare instances sometimes resulting in a varus deformity of the knee secondary to injury to the distal femoral or proximal tibial growth plate in rugby and tennis players [18].

These injuries can be prevented; Training intensity and load on the physes during growth spurts should be limited. Exercise should be varied during training in order to avoid overly straining a single joint. Proper physical preparation with appropriate warm-ups, proprioception training and stretching must be compulsory. Trainers must further be educated on the actuality of these pathologies. Finally, in order to properly rest the physes,

the periodic nature of training sessions must be respected, and resting periods, particularly summer breaks, must be imposed [18].

### Shoulder

At the level of the shoulder, overuse of the proximal humeral physis (leading to widening of the physis and epiphysiolysis of the humerus) is treated by suspension of sporting activities for a period of 3 months (often baseball pitchers, gymnasts, tennis players, volleyball players, or swimmers) [7,11]. A program based on strengthening, better pitching mechanics, and physiotherapy is suggested for high-level athletes [6]. A preexisting deficit in internal rotation of the glenohumeral joint is a predisposing factor for these sorts of injuries in children. Recurrence of injury is common (7% at 7 months) and 3 times more frequent in children with a preexisting deficit in internal rotation [19].

### Wrist

At the level of the wrist, physeal injuries are frequently encountered in gymnasts, especially in patients in whom the ulna is shorter than the radius [7,20]. Treatment is based on interruption of sporting activities and immobilization in an attempt to avoid premature closure of the physis [7]. Cast immobilization is preferred over splinting in order to discourage premature mobilization by children who may be pressured by their trainers or parents. More severe injuries found on imaging may require longer recuperation periods. As a result, some authors prefer early screening of radial physis injuries in gymnasts [20]. The complete fusion of the distal radius is one complication of these types of sports injuries and may require distal ulnar epiphysiodesis or an ulnar shortening osteotomy [21].

### Knee

Overuse injuries of the physes of the distal femur may be visualized on conventional radiographs as a widening of the physis, although a more adequate diagnosis may be made on MRI. Healing is always achieved with a return to normal growth by simply limiting physical exercise, and the use of a knee brace may accelerate recuperation. Return to normal activity in over 3 months is determined both clinically and after normalization of imaging. Rest is an essential component of treatment since non-compliance may lead to axial deformities of the knee [9,22].

## **Treatment of stress fractures**

Overuse leading to stress fractures are common and may be seen in 13 to 50% of young athletes, depending on the practiced sport [6]. Conventional radiographs have a sensitivity of only 10% in the acute setting. The diagnosis is usually made on MRI. Findings in the acute setting may be limited to simple periosteal edema without a clear fracture line, whereas more advanced stages tend to show a complete fracture line [23].

Management is preferentially undertaken on an individual basis depending on the site of injury, age of the child, and practiced sport. Nevertheless, the general principles of treatment include reduction of the load to allow proper healing. Immobilization, protected weight-

bearing (e.g. using a walking boot or a long pneumatic splint) or full-weight-bearing using crutches may further reduce the load and control the pain. It is recommended to alternate training with activities sparing the injured limb, such as biking or swimming, in order to maintain cardiovascular conditioning [6]. Return to sports must be done progressively after complete resolution of the pain and radiographic signs of healing are evident, a process requiring around 3 to 4 months in periosteal forms, and 6 months in patients with an actual fracture [6,23]. Management is exceptionally surgical but may be indicated in certain rare cases, such as stress fractures of the femoral neck with progressive displacement or a non-union. These patients are generally treated by osteosynthesis similar to conventional fractures [24].

## **Prevention**

The best treatment of overuse injuries is prevention by acting early and addressing the multiple pathophysiological elements that are at the origin of these injuries.

### Early specialization

Early sports specialization is one of the primary causes of overuse injuries [25,26]. This generally entails intense training throughout the year for a single type of sport while excluding other types of physical exercise [27]. In fact, there has been an increase in training intensity in young children, the majority of which are already specialized before the age of 7 years. Participating in multiple types of activities allows skill transfer from one sport to another and a better overall development of young athletes [28]. This variety in sporting activities also allows a more balanced neuromuscular development and a decrease of the repetitive stresses over the same joints [27]. As a result, preventing overuse injuries must imperatively include a varied approach to the volume and quality of physical exercise in these young athletes.

### Lifestyle

Similar to adults, high-level athletic children must maintain a healthy lifestyle. This allows the proper conditioning for the body to endure the repetitive load it is subjected to during physical exercise. An adapted diet, proper hydration, and sufficient sleep (more than 8 hours per night) all play a major role in limiting these overuse injuries [7,27,29]. Finally, the equipment must be adapted to the type of strain caused during exercise (proper socks, softer balls in children...) [7].

### Adapting training frequency

The volume and quality of training must be evaluated. There is a broad consensus on the rule of 10%, which states that the work done must not increase more than 10% per week in order to allow better recuperation. Depending on the type of sport, this signifies that training time, weight, distance, and speed must not increase by more than 10% [7]. Trainers must also be included while adapting the quality of the training program. This allows for a better analysis of the child's athletic techniques and performance errors. The total weekly time spent training is also important: if this time is superior to 16 hours per week, the risks of sustaining an injury

increase significantly [6]. Moreover, a minimum of 1 day per week and 3 months per year must be afforded for adequate rest and recuperation.

### Flexibility

Musculotendinous tightness is an essential element of overuse injuries [10]. Flexibility both improves the child's performance and avoids increased strain on the apophyses. Regular stretching sessions (quadriceps, hamstrings, triceps surae) should be mandatory. However, these stretching sessions should preferentially take place at a distance from the training session, since they can aggravate the muscular microlesions that have been sustained during exercise. These sessions are preferentially held during dedicated periods after apophyseal pain has subsided and follow a thorough program [7].

### Adapting training during puberty

Trainers and parents must be continually aware of the child's pubertal stage. In children younger than 12 years old, growth plate fragility makes the occurrence of an overuse injury more likely if both training intensity and volume are not adapted.

The pubertal status must be favoured over chronological age, since, in a similar age group, there are evidently differences in stages of pubertal development between children. In some children, certain movements risk placing too much stress on certain joints, and training must be adapted on an individual basis [7].

## **Conclusion**

The majority of overuse injuries are benign, and their treatment is often simple and solely symptomatic, relying primarily on common sense and adaptation of the sporting activity based on the athlete's age. The means of prevention must be well known by children, parents, and trainers.

Cessation of sports is always the initial management strategy, and after returning to sports, children must be aware of early signs of recurrence.

Even though immobilization, or even surgery, may sometimes be necessary, adapting the activity based on the child's age and pubertal status is the primary element in management and dictates treatment outcomes.

## **References**

1. Caine D, Maffulli N, Caine C. Epidemiology of injury in child and adolescent sports: injury rates, risk factors, and prevention. *Clin Sports Med* 2008;27:19– 50.
2. Bedoya MA, Jaramillo D, Chauvin NA. Overuse injuries in children. *Top Magn Reson Imaging* 2015;24:67–81.
3. Hoang QB, Mortazavi M. Pediatric overuse injuries in sports. *Adv Pediatr* 2012;59:359–83.
4. Reider B. Too Much? Too Soon? *Am J Sports Med* 2017;45:1249–51.

5. Journeau P, Haumont T, Métaizeau JD, Lascombes P. [Overuse injuries in the young athletes]. *Arch Pediatr* 2006;13:545–8.
6. DiFiori JP, Brenner JS, Jayanthi N. Overuse injuries of the extremities in pediatric and adolescent sports. *Inj. Pediatr. Adolesc. Sports Contemporary Pediatr. Adolesc. Sports Med.* Springer International Publishing, Switzerland: D. Caine, L. Purcell; 2016, p. 93–105.
7. Launay F. Sports-related overuse injuries in children. *Orthop Traumatol Surg Res* 2015;101:S139- 147.
8. Eberbach H, Hohloch L, Feucht MJ, Konstantinidis L, Südkamp NP, Zwingmann J. Operative versus conservative treatment of apophyseal avulsion fractures of the pelvis in the adolescents: a systematical review with meta-analysis of clinical outcome and return to sports. *BMC Musculoskelet Disord* 2017;18.
9. Chang GH, Paz DA, Dwek JR, Chung CB. Lower extremity overuse injuries in pediatric athletes: clinical presentation, imaging findings, and treatment. *Clin Imaging* 2013;37:836–46.
10. Omodaka T, Ohsawa T, Tajika T, Shiozawa H, Hashimoto S, Ohmae H, et al. Relationship Between Lower Limb Tightness and Practice Time Among Adolescent Baseball Players With Symptomatic Osgood-Schlatter Disease. *Orthop J Sports Med* 2019;7:2325967119847978.
11. Wu M, Fallon R, Heyworth BE. Overuse Injuries in the Pediatric Population. *Sports Med Arthrosc Rev* 2016;24:150–8.
12. Tisano BK, Estes AR. Overuse Injuries of the Pediatric and Adolescent Throwing Athlete. *Med Sci Sports Exerc* 2016;48:1898–905.
13. Bradley JP, Petrie RS. Osteochondritis dissecans of the humeral capitellum. Diagnosis and treatment. *Clin Sports Med* 2001;20:565–90.
14. Mihara K, Suzuki K, Makiuchi D, Nishinaka N, Yamaguchi K, Tsutsui H. Surgical treatment for osteochondritis dissecans of the humeral capitellum. *J Shoulder Elbow Surg* 2010;19:31–7.
15. Haeri Hendy S, de Sa D, Ainsworth K, Ayeni OR, Simunovic N, Peterson D. Juvenile Osteochondritis Dissecans of the Knee: Does Magnetic Resonance Imaging Instability Correlate With the Need for Surgical Intervention? *Orthop J Sports Med* 2017;5:2325967117738516.
16. Heyworth BE, Edmonds EW, Murnaghan ML, Kocher MS. Drilling techniques for osteochondritis dissecans. *Clin Sports Med* 2014;33:305–12.
17. Kocher MS, Tucker R, Ganley TJ, Flynn JM. Management of osteochondritis dissecans of the knee: current concepts review. *Am J Sports Med* 2006;34:1181–91.
18. Caine D, DiFiori J, Maffulli N. Physeal injuries in children's and youth sports: reasons for concern? *Br J Sports Med* 2006;40:749–60.
19. Heyworth BE, Kramer DE, Martin DJ, Micheli LJ, Kocher MS, Bae DS. Trends in the Presentation, Management, and Outcomes of Little League Shoulder. *Am J Sports Med* 2016;44:1431–8.
20. Paz DA, Chang GH, Yetto JM, Dwek JR, Chung CB. Upper extremity overuse injuries in pediatric athletes: clinical presentation, imaging findings, and treatment. *Clin Imaging* 2015;39:954–64.
21. Cornwall R. The Painful Wrist in the Pediatric Athlete. *J Pediatr Orthop* 2010;30:S13.
22. Laor T, Wall EJ, Vu LP. Physeal Widening in the Knee Due to Stress Injury in Child Athletes. *Am J Roentgenol* 2006;186:1260–4.

23. Nattiv A, Kennedy G, Barrack MT, Abdelkerim A, Goolsby MA, Arends JC, et al. Correlation of MRI Grading of Bone Stress Injuries With Clinical Risk Factors and Return to Play: A 5-Year Prospective Study in Collegiate Track and Field Athletes. *Am J Sports Med* 2013;41:1930–41.
24. Goolsby MA, Barrack MT, Nattiv A. A displaced femoral neck stress fracture in an amenorrheic adolescent female runner. *Sports Health* 2012;4:352–6.
25. Jayanthi NA, LaBella CR, Fischer D, Pasulka J, Dugas LR. Sports-specialized intensive training and the risk of injury in young athletes: a clinical case-control study. *Am J Sports Med* 2015;43:794–801.
26. Bell DR, Post EG, Biese K, Bay C, Valovich McLeod T. Sport Specialization and Risk of Overuse Injuries: A Systematic Review With Meta-analysis. *Pediatrics* 2018;142.
27. Straccolini A, Sugimoto D, Howell DR. Injury Prevention in Youth Sports. *Pediatr Ann* 2017;46:e99–105.
28. Myer GD, Jayanthi N, Difiori JP, Faigenbaum AD, Kiefer AW, Logerstedt D, et al. Sport Specialization, Part I: Does Early Sports Specialization Increase Negative Outcomes and Reduce the Opportunity for Success in Young Athletes? *Sports Health* 2015;7:437–42.
29. Brenner JS, American Academy of Pediatrics Council on Sports Medicine and Fitness. Overuse injuries, overtraining, and burnout in child and adolescent athletes. *Pediatrics* 2007;119:1242–5.

# Medical eligibility to participate in high-performance sports in children and adolescents

Edem Allado<sup>1,2</sup> and Bruno Chenuel<sup>1,2</sup>

1. University Center of Sports Medicine and Adapted Physical Activity, Nancy University Hospital. Allée du Morvan. 54511 Vandœuvre-lès-Nancy
2. EA DevAH – Department of Physiology – Nancy Faculty of Medicine – Lorraine University. 9 Avenue de la Forêt de Haye. 54505 Vandœuvre-lès-Nancy

Even though regular physical activity is one of the most effective ways to stay healthy [1], high-performance sports, due to the high intensity and frequency of training, may have significant detrimental consequences on a person's health, requiring a sports physician's expertise. The primary role of a sports physician is to ensure an athlete's medical eligibility and physical condition to participate in sports, followed by avoiding exacerbating any preexisting sports injuries and accompanying the athlete in further preventing new injuries and sport-specific accidents. Finally, sports injuries should be properly cared for and a return to sports must be organized in a manner as to conserve the patient's athletic future, be safe, and allow a progressive return to activity. In children and adolescents who aim at elite or high-performance athleticism, schedules including training for multiple hours per week as well as schooling leave little space for rest and physical recuperation. In addition to an initial medical eligibility assessment, sports physicians must accompany these athletes in order to avoid any exercise-related consequences on growth, bone development, metabolism and puberty, and overuse injuries [2-4].

## **I. Definition and organization of high-performance sports in France:**

Common use of the term "high-performance" relates to intense and competitive engagement in a given sport. In fact, this term refers to a very specific and thoroughly codified statute (Articles R221-1 to T221-2 of the Sports Code, Decree no. 2016-1286 from September 29, 2016 relating to high-intensity sports). The status of high-performance athlete is obtained after registering to the high-performance athlete list under the jurisdiction of the ministry responsible for sporting affairs. This registration requires a recommendation by the corresponding sporting federation and approval of the deputy national technical director. Furthermore, high-performance athletes may only apply to the high-performance athlete list if they have previously participated in international competitions in a given sport (of which the high-performance nature is recognized), if they qualify for a high enough performance level in a given sport, and if they were 12 years of age or older during the year in which they had applied to the ministerial list. Four different categories exist on the high-performance list: Elite, Senior, Youth, and Reconversion.

In addition to the high-performance athlete list, two additional categories exist:

- National collective athlete list: Includes athletes who did not meet the above criteria but whose integration in a national collective was deemed necessary. These may include training partners who were previously listed as high-performance athletes themselves who suffer from health-related issues (sports injuries), or even athletes with potential for future high-performance status.
- Hope athlete list: Includes athletes who have shown proficiencies in certain sports that are considered high-performance and who are testified by the deputy national technical director of the concerned sporting federation, but do not yet meet the criteria for registering to the high-performance athlete list.

Children and adolescents are mostly included in the Elite and Youth categories of the ministerial list and the Hope athlete list. An athlete can apply to the “Elite” list if they undertake a significant performance or obtain a significant rank during international benchmark tests (R221-4 Sports Code). Performance, rank and evaluations are identified in the Federal Performance Project of each sporting federation. Registration is valid for two years. Registration in the “Youth” category, the category concerning athletes selected for the French national team for an international competition, is valid for one year (R221-6 Sports Code).

Once registered on the ministerial high-performance or Hope lists, athletes have certain rights (e.g. access to national training facilities) and duties. A convention provided for in Article L. 221-2-1 of the French Sports Code determines the reciprocal rights and obligations of the sporting federations and elite athletes. These include directives regarding the medical follow-up of these high-performance athletes in the form of regulatory longitudinal medical monitoring.

## **II. Certificate of medical non-contraindication to the practice of a sport, reglementary aspects and concept of “playing-up”:**

Medical assessment for the eligibility to participate in sports has the ultimate goal of delivering the certificate of medical non-contraindication (CMNCI) to the practice of a sport. This certificate may be delivered after all clinical anomalies or pathologies susceptible of being exacerbated during physical activity have been ruled out. The delivery of such a certificate commits the professional responsibility of the physician and must therefore be based on a minute physical exam. The Sports Code clauses relative to this medical certificate have been modified by the law no. 2016-41 of January 26, 2016 for the modernization of France’s healthcare system, as well as that of August 24, 2016 that pertains to the medical certificate testifying the absence of contraindications to sports. One of the primary modifications concerns the intervals at which a new CMNCI to the practice of a sport must be issued in order to renew a sports license, which increased from 1 to 3 years: Health questionnaires (QS-Sport) must be administered on a yearly basis; in case of a change in the athlete’s health, the responses to this questionnaire will determine the delivery of a new CMNCI.

Obtaining a CMNCI is mandatory in order to receive an initial license allowing participation in an organized competition by the relevant sporting federation (said CMNCI must be no older

than 1 year). The renewal of a license depends on presenting every 3 years a CMNCI to participate in a sporting competition dating less than a year old, and a confirmation by the athlete that they have would have responded as negative on each section of the QS-Sport questionnaire, every year, even when a certificate was not required (CMNCI every year for high-risk athletes, detailed below). A CMNCI dating less than a year old is also necessary for participation in organized sports competitions by the authorized sporting federation (competitions such as select organized running competitions, including marathons, trails, and ultra-trails).

Some sports are known to induce excessive load on certain body parts and are thus deemed as high-risk sports (Sports Code: law no. 2016-41 of January 26, 2016 – article 219, D. 231-1-5 of the Sports Code):

1. Sports that require a specialized facility
  - a. Mountaineering
  - b. Underwater diving
  - c. Speleology
2. Competition combat sports in which knockouts are authorized
3. Sports including the use of firearms or pneumatic weapons
4. Sports requiring the use of motor vehicles in a competition setting, not including radio-controlled model vehicles
5. Sports requiring the use of aircrafts, not including model aircrafts
6. Rugby XV, rugby XIII and rugby VII.

Delivering an initial license to participate in sports with a high risk on the security or health of the athlete implies an annual medical follow-up specific to the type of sport. As a result, receiving or renewing a license are dependent on obtaining a CMNCI dating less than one year old, thus indicating the absence of contraindications to the participation in the specific sport (notably in competition).

Such certificates are delivered after a specific medical assessment detailed by the ministry responsible for health and sports (decree of July 24, 2017 pertaining to the characteristics of the specific medical assessment required to deliver the CMNCI to practice high-risk sports).

The medical assessment can be done by all medical doctors according to the recommendations of the French Society of Exercise and Sports Medicine (SFMES) and presents the following characteristics (Article A231-1 of the Sports Code):

1. For mountaineering below 2,500 meters in altitude:
  - a. Cardiovascular assessment;
  - b. History of or risk factors related to high-altitude-hypoxia justifying the need for a specialized or mountain medicine consultation;
2. For underwater diving: ENT (eardrum, Eustachian tube equilibration/permeability, vestibular examination, auditory acuity) and dental assessment;
3. For speleology: cardiorespiratory assessment; For cave diving, ENT (eardrum, Eustachian tube equilibration/permeability, vestibular exam, auditory acuity) and dental assessment;

4. For competition combat sports in which a fight might end, especially or exclusively, due to a knockout:
  - a. Neurological exam and mental health;
  - b. Ophthalmic assessment: visual acuity, visual fields, intraocular pressure and fundoscopy;
5. For sports including the use of firearms or pneumatic weapons:
  - a. Neurological exam and mental health;
  - b. Auditory acuity and assessment of the dominant upper limb for the biathlon;
  - c. Spinal exam in minors participating in upright shooting;
6. For competition sports including the use of motor vehicles:
  - a. Neurological exam and mental health;
  - b. Ophthalmic assessment (visual acuity, visual field, color vision);
7. For sports requiring the use of aircrafts:
  - a. Neurological exam and mental health;
  - b. Ophthalmic assessment (visual acuity, color vision);
  - c. ENT (eardrum, Eustachian tube equilibration/permeability, auditory acuity, vestibular exam) and dental assessment;
  - d. Shoulder exam for parachuting and paragliding;
  - e. Spinal exam for class 1 ultralight aviation pilots;
8. For Rugby XV and VII aged 12 to 39 years old, during and out of competition:
  - a. Cardiovascular assessment;
  - b. Spinal exam;
9. For Rugby XIII: orthopedic exam;

The concept of playing-up:

Playing-up corresponds to a special authorization given to a child allowing competition in a higher age category, including the adult category. Playing-up is managed by regulations and special forms specific to each sporting federation. Depending on the federation and each case, physicians may be faced with demands for playing-up at one, two or even three categories. Although a demand for a one-category advancement may sometimes be undertaken by general practitioners, two or three-category advancements require the expertise of a certified sports physician or a physician authorized by the corresponding sporting federation. The requests should then be validated by a federal (regional or general) physician and approved by the technical supervisory staff.

There is a lack in objective criteria for the appraisal of playing-up demands and the decision is generally left to either the examining or federal physician.

Nevertheless, the criteria for a playing-up request to be granted must ensure a physiological response by the athlete that is higher than that of the normative values for their age category. As a result, playing-up should not be granted to athletes with growth delay or if the intense physical training risks inducing a negative effect on growth. Playing-up demands must therefore be granted to athletes whose capacity to adapt to physical stress at least equals or surpasses that of their age category (maximal strength and recuperation during a maximal stress test).

### **III. Goals of the evaluation for the eligibility to participate in sports in children and adolescents**

Medical assessment for eligibility to participate in sports must answer the following principal questions:

- Is there any temporary or permanent contraindication to participate in sports (infectious, cardiac, respiratory, osteoarticular, muscular, psychological or addictive)?
- Are there risk factors for sports-related injuries (clinical or osteoarticular pathologies, working conditions)?
- Is there a history of or present illness that may increase the risk of injury when engaging in a given type of sport?
- Does the sport in question entail intense physical training that may require a specific medical assessment and screening of risk factors (overuse injuries, overtraining, burn-out, effect on growth, malnutrition...)? Such is the case of high-performance athletes.
- Have I properly adapted my physical examination according to age, practiced sport, type of practice, and level of the athlete (hobby, high-performance or playing-up)?

Intensive training is defined as a total training time superior to 10 hours per week in children older than 10 years or 6 hours per week in children younger than 10 years [5]. Only supervised training sessions are included in this definition, thereby allowing spontaneous recreational play for the child during their free time. As such, intensive training is defined by a threshold value that does not consider the intensity of the training sessions, which is generally the cause of overuse injuries [2]. Practically, the training sessions of young high-performance athletes surpasses 15 hours per week and may even reach 20 to 25 hours per week.

In order to reduce the risk of injury, it is recommended that children be allowed resting periods of one or two days per week and at least three months per year in one-month intervals [6].

The primary risks of intensive training include:

- Growth disturbance: delayed growth, deviation in the growth chart, delayed puberty and primary or secondary amenorrhea;
- Overuse injuries: microtraumatic damage to bone, muscle or tendon caused by repetitive biomechanical stress. The American Academy of Pediatrics has described 4 stages for these types of injuries [6]: 1) pain in the affected area after physical activity; 2) pain that occurs during the activity but does not restrict performance; 3) pain that occurs during the activity and restricts performance; and 4) chronic, unremitting pain, even at rest;
- Osteochondral lesions: see specifics in athletic children in paragraph 5;
- Loose body complicating a neglected osteochondral lesion;
- Stress fractures and muscle injury: due to abusive repetitions of certain exercises that may be more traumatic than others;
- Overtraining syndrome;
- Psychological risks: ill-systematized in a global context of athletic, academic and personal stressors. The stress of training, management of competitions, constant traveling, separation from family, stress of group living, and difficulties finding personal time in an academically and athletically full schedule. The expectation of a certain performance or the maintenance of said performance significantly adds to the multiple psychological risk factors and may induce a variety of psychological disorders,

ranging from anxiety to depression or substance abuse with the specific risk of doping. Specific questionnaires for the screening of stress, anxiety, depression, unhealthy eating habits, low self-esteem, and burnout may be used.

#### **IV. Medical assessment for eligibility to participate in sports: history and physical exam**

A thorough medical assessment is essential in order to collect all the required data for the delivery of a CMNCI to participate in sports, in the absence of findings requiring further investigation. In the setting of a presentation for any other complaint (symptoms during activity or at rest), a rigorous approach must be maintained in order to undertake an assessment that is as comprehensive as possible. The French Society of Exercise and Sports Medicine suggests the following [7]:

- Vital record and administrative information
- The child's environment:
  - o Collection of data specific to the sport: the practiced sport, role, level of practice (from amateur to international), age at onset of training, age at onset of competition, and the motivations for the choice of the practiced sport (child's own, family or other). It is also essential to assess the volume and intensity of the practice (volume and duration of training sessions), the conditions of practice (terrain, equipment, engagement in a weight-category sport, climatic conditions...)
  - o Collection of data specific to the environment: the home environment (divorce, siblings, family's perspective on the child's sporting practice), the number of trainers, group or individual sport
  - o Collection of data on the academic environment: academic level, plans of academic future, presence of sports/study supervision
- History: Child athletes are still children and it is essential to include all of the elements of a pediatric physical examination:
  - o Assessment of the child's health records
    - Verification of vaccination records depending on the latest recommendations;
    - History of childhood illnesses, allergies and drug contraindications, age of adiposity rebound: red flag if earlier than 6 years old (risk of obesity).
  - o Past history
    - Medical and surgical history: Personal cardiovascular risk factors, history of or active or passive smoking (pack-year), alcohol consumption, cannabis use and/or other drugs. The presence of type I diabetes, overweight or obesity (BMI and control of the adiposity rebound age), cardiac pathologies (last resting electrocardiogram and/or echocardiogram), asthma (last pulmonary function testing), epilepsy, near-sightedness (last corrections), traumatic brain injury (with or without loss of consciousness), muscle pathology, neurological pathology, coagulation disorders, loss of function of a paired organ, genetic disorders, and history of trauma (previous injuries, recurring sprains);
    - First-degree family history: congenital diseases, diabetes, muscle pathology, genetic disorders, and coagulation disorders;

- Gynecological history: Age of menarche, regularity of menstrual cycle, use of contraceptives, HPV and Hepatitis B vaccines
- Treatments:
  - Information on the totality of medical treatments and nutritional supplementation of the athlete, absence of illegal substance use as per the World Antidoping Agency (WADA), follow-up on Therapeutic Use Exemption (TUE) in patients requiring a treatment that is included in the list of prohibited substances, reconciliation of the totality of non-medical treatments: physiotherapy, foot orthoses (orthotic insoles), other orthoses (knee or ankle braces), or prostheses in parasports; evaluation of eyeglasses in patients with vision disorders.
- Lifestyle
  - Complete nutritional assessment: the number of meals and snacks per day, the quality of the food and/or adherence to any particular diet
  - Sleep: time of sleep and waking, quality of sleep, presence of nightmares (may be anxiety-related, but beware oxyurosis), enuresis.
  - Sedentary behavior: screen time (TV and/or video games, computer time, mobile phone).
- Functional symptoms leading to consultation: symptoms at rest and/or on exertion: musculoskeletal symptoms (occurrence, location and type of pain), cardiovascular and pulmonary symptoms, weakness, etc.

#### Physical exam

- Morphology and development: weight, height, body mass index, estimated adipose mass, wingspan, evaluation of growth, psychomotor development, pubertal stage (Tanner stage).
- Physical exam undertaken systematically with a particular attention to the patient's current concerns:
  - Cardiovascular and pulmonary exam: resting heart rate, bilateral blood pressure, peripheral pulses, cardiac auscultation to eliminate any bruits or murmurs, and pulmonary auscultation and cyanosis. The French and European societies of cardiology recommend systematically obtaining a resting ECG in patients aged 12 years or older, repeated every 3 years until 20 years of age and, in case of any anomalous findings, a stress-test may be suggested.
  - Orthopedic exam: spinal alignment disorders (nonstructural or structural scoliosis with an assessment of gibbosity on Adams forward bending test), lower limb deformities (genu valgum or varum), joint range of motion, muscle strength assessment, apophyseal tenderness, foot exam.
  - Digestive system, lymph node palpation, ENT (eardrum, sinuses, auditory acuity...), and dental assessment: dental follow-up, wisdom teeth, tooth alignment, oral parafunctional activities (e.g. bruxism).

#### **V. Specifics of child and adolescent high-performance athletes**

1. Microtraumatic pathologies [8]:
  - Osteochondral lesions: intense physical exercise leads to bony repercussions, especially at the ossification centers, leading to repetitive microtrauma and chronic inflammation: Osteochondritis (osteochondrosis). The diagnosis relies

on the presence of both mechanical pain and radiographic findings. Depending on the location of osteochondral lesion:

- Knee:
  - Osteochondral lesions of the tibial tuberosity (Osgood Schlatter disease): This pathology appears primarily in boys aged 11 to 15 years old and occurs mostly in sports requiring repetitive jumping (football, basketball, handball, etc.).
  - Osteochondral lesions of the tip of the patella (Sinding-Larsen-Johansson syndrome): Less frequently encountered than Osgood-Schlatter disease, Sinding-Larsen-Johansson syndrome affects children aged 11 to 13 years old who participate in sports with trauma to the knees (Figure skating, handball, etc.).
- Foot and ankle:
  - Osteochondral lesions of the calcaneal apophysis (Sever's disease): This pathology preferentially occurs in boys aged between 10 and 13 years old and is more frequently found in practitioners of sports including sudden changes of direction and significant traction on the heel (football, gymnastics, etc.). The pain is usually localized at the level of the attachment of the Achilles tendon and is manifested by mechanical talalgia.
  - Osteochondral injuries of an accessory navicular bone (Köhler disease)
  - Osteochondral injuries of the growth plate of the base of the fifth metatarsal (Iselin disease).
  - Osteochondral injuries of the heads of the 2<sup>nd</sup> or 3<sup>rd</sup> metatarsals (Freiberg disease)
  - Osteochondral injuries of the sesamoid bones (Renander's disease)
- Spine:
  - Osteochondral injuries of the spine (Scheuermann's disease): this pathology is equally called osteochondritis deformans juvenilis dorsi and causes sagittal deformities of the spine with loss of disc height. The following radiographic findings characterize Scheuermann's disease:
    - ◆ Vertebral endplate irregularities
    - ◆ Wedging of at least 3 adjacent vertebrae
    - ◆ Schmorl nodes or intravertebral disc herniations
    - ◆ Intervertebral disc narrowing
    - ◆ Limbus vertebrae
- Tibial periostitis: These injuries are induced by microtraumatic events at the level of the anteromedial aspect of the tibia. They are very frequently found in long distance runners and jumpers.  
Other overuse injuries may also be found in children: stress fractures, bone fragility, and tendinopathies.
- Spondylolysis: defined by an acquired defect of the isthmus of the posterior vertebral arch and may be isolated or associated with spondylolisthesis. Patients with spondylolysis often present with tenderness during physical

activities requiring hyperlordosis and other significant mechanical stresses (diving, gymnastics, judo, Olympic style weightlifting, skiing, football, etc.), although they may also be asymptomatic. Even though spondylolysis essentially affects both isthmuses, unilateral affections are usually found in sports requiring repetitive unilateral torsion (tennis, Javelin throw). Symptomatic patients usually complain of pain (low back pain, sciatica and/or lumbosciatica), intermittent claudication and/or loss of lumbar lordosis.

2. Acute traumatic injuries [8]:

- Apophyseal avulsions: Often secondary to intense physical exercise, apophyseal avulsions occur primarily at the level of the pelvis (ischium, iliac spines, iliac crest, lesser trochanter, ischial tuberosity, and greater trochanter). Sports increasing the risk of these types of avulsions include football (soccer), athletics and gymnastics. This diagnosis must be considered in skeletally immature patients presenting with traumatic injuries that appear to be of muscular origin.
- Classic traumas: Ankle sprain (anterior talofibular and calcaneofibular ligaments), knee sprain and trauma (anterior cruciate ligament rupture), muscle injury after growth plate fusion (muscle tears, tears at the myoaponeurotic junction, etc.) and fractures.

3. Growth defects [9]:

Physical exercise may lead to failure to thrive. As a result, growth chart monitoring is essential: early adipose rebound before 6 years of age suggests obesity, and a deviation from the growth chart suggests hormonal disorders and/or unhealthy nutritional habits. In both cases, a more comprehensive assessment is required. In patients presenting with failure to thrive, gymnastics is often identified as a cause. In fact, intensive training in children is the essential element leading to growth defects by delaying growth spurt. Nevertheless, regular follow-ups are necessary in order to monitor the child's growth.

4. Child abuse:

Although sports are meant to be motivated the child's own ambitions, child athletes are often influenced by external pressures including the sporting federations and families. The former only work towards international recognition, which, in some countries, may lead to the detriment of the child's development. Furthermore, the family's motivations for athletic success may be influenced by secondary financial gains and social recognition (mediatized children, increased incomes). This set of motivations may lead to overtraining, psychological consequences (anxiety and depression, conduct disorders) and eating disorders (malnutrition, specific diets for weight-specific sports).

## **VI. Longitudinal medical follow-up of high-performance athletes:**

Adopted policies consist of placing athletes that are registered on the ministerial high-performance athlete lists, especially potential candidates for registration to these lists and athletes who are integrating the path of athletic excellence, on a specific medical surveillance.

This medical surveillance allows screening, preventing and limiting the risks that are related to high-intensity physical exercise.

The decree of June 13, 2016 concerned with the medical surveillance of high-performance athletes, Hope athletes and national collectives modernized the Sports Code (A231-3) in regard to the medical surveillance of athletes.

In the two months following their first registration to the ministerial list of high-performance athletes and annually afterwards, high-performance athletes must be submitted to:

1. A medical assessment by a sports physician:
  - a. A physical exam and history as per the recommendations of the French Society of Exercise and Sports Medicine (SFMES);
  - b. A dietary assessment and nutritional recommendations;
  - c. A psychological assessment in order to screen for psychopathological complications related to intense physical exercise;
  - d. Indirectly screening for overtraining syndrome through an elaborate questionnaire as per the recommendations of the SFMES;
2. Resting electrocardiogram

At the recommendation of the sports physician and under their responsibility, the psychological and nutritional assessment discussed above may be undertaken by a clinical psychologist and dietitian, respectively.

On this common basis, sporting federations may also include specific complementary exams. For athletes registered on the Hopes athletes list or included in the national collective, the contents and implementation of medical surveillance must consider the age, burden of training, specific physical stressors of the specific sport, morbidity and inherent risks of the type of sport.

The delegate sporting federations ensure the organization of the medical surveillance of their licensed athletes and may require complementary medical evaluations specific to the type of sport.

The French Federation of Rugby (FFR) serves as an interesting example since high-performance Rugby players require specific medical evaluations due to the traumatic nature of the sport. The longitudinal medical follow-up of these athletes is detailed in Annex no. XIV (medical regulation) of the General Regulations that dictate the regulatory environment relative to the management of Rugby in France [10].

As a result, the FFR has established specific evaluations in addition to the regulated medical exam for all athletes registered on the ministerial high-performance or Hope athlete lists.

As a result, rugby players must be subjected to the following medical exams:

1. Evaluation for proteinuria, glycosuria, hematuria, and urinary nitrites on a urine dipstick;
2. Resting transthoracic echocardiogram with a medical report;
3. In the absence of apparent anomalies on resting cardiovascular physical exam and the two preceding exams, a stress test at maximal intensity (coupled, if required, to the measurement of gaseous exchanges and to pulmonary function testing) realized by a physician, according to the most recent scientific data.

This stress test aims at screening possible abnormalities or lack of adjustment during exertion, which impose a specialist's opinion;

4. A dental assessment by a certified specialist;
5. An MRI of the cervical spine for the screening of cervical spinal stenosis in loose-head prop, hooker and tight-head prop positions after the age of 16 years;

The medical surveillance of these athletes includes:

1. Twice a year:  
A medical exam realized by a medical practitioner licensed in sports medicine including:
  - a. Medical interview;
  - b. Physical exam;
  - c. Anthropometric measurements;
  - d. Nutritional assessment and recommendations that may be aided by a specialist;
  - e. Search for proteinuria, glycosuria, hematuria, and urinary nitrites on urine dipstick;
2. Once a year:
  - a. Dental assessment by a certified specialist;
  - b. Standardized resting electrocardiogram with medical report;
  - c. After parental authorization for minors, a biological exam for athletes aged 15 years or more including a
    - i. complete blood count;
    - ii. reticulocytes;
3. A psychological assessment is conducted twice a year during a specific interview either by a physician or by a psychologist with medical supervision. This psychological assessment aims at:
  - a. Detecting psychopathological difficulties and personal and family factors of vulnerability or protection;
  - b. Preventing difficulties related to intense training;
  - c. Orienting toward management;
4. Once every four years:  
A maximal effort stress test
5. Candidates for registration to the high-performance athlete lists or Hope athletes who have already undergone an ECG before the age of 15 years must have another ECG between the ages of 18 and 20 years. For players in sports academies, varsity teams, France national teams or professional Rugby teams, in addition to the ones already planned for, complementary medical surveillance is ensured by the FFR.

Each concerned player must undergo the following exams:

- Cardiological assessment:
  - o Resting ECG every 2 years
  - o Stress test every 2 years
  - o Echocardiography every 4 years or at changing of status.
  - o An MRI of the cervical spine for players of all positions, to be reassessed in the case of new pathology or a change of status depending on the intervals set by the regulations.

- Preseason neurological assessment (concussion follow-up).
- Biological follow-up.

## Conclusion

Participation in sports, even at high-performance, must remain a vector for good health. It is thus imperative to ensure that the risks specific to each type of sport be maximally reduced. This requires a rigorous evaluation of the initial medical eligibility and a meticulous and regular medical follow-up. The primary objectives of this management are to accompany the child's performance, normal development and self-fulfillment.

## References

1. World Health Organization. Global action plan on physical activity 2018–2030: more active people for a healthier world. Geneva: WHO; 2018. <http://apps.who.int/iris/bitstream/handle/10665/272722/9789241514187-eng.pdf>.
2. Brenner, J.S., M. American Academy of Pediatrics Council on Sports, and Fitness, Overuse injuries, overtraining, and burnout in child and adolescent athletes. *Pediatrics*, 2007. 119(6): p. 1242-5.
3. Duclos, M., P. Barat, and Y. Lebouc, [Growth and elite sports practice in children]. *Arch Pediatr*, 2003. 10 Suppl 1: p. 207s-209s.
4. Georgopoulos, N.A., et al., The influence of intensive physical training on growth and pubertal development in athletes. *Ann N Y Acad Sci*, 2010. 1205: p. 39-44.
5. Intensive training and sports specialization in young athletes. American Academy of Pediatrics. Committee on Sports Medicine and Fitness. *Pediatrics*, 2000. 106(1 Pt 1): p. 154-7 Reaffirmed October 2014.
6. Brenner, J.S., M. Council On Sports, and Fitness, Sports Specialization and Intensive Training in Young Athletes. *Pediatrics*, 2016. 138(3).
7. Document téléchargeable aux adresses suivantes : <http://www.sfmes.org/sfmes/textes-utiles> ou [https://www.sfmes.org/images/sfmes/pdf/Visite\\_NCI.pdf](https://www.sfmes.org/images/sfmes/pdf/Visite_NCI.pdf)
8. Brunet-Guedj, E., Pathologies de l'appareil locomoteur chez l'enfant sportif, in *Traité de Médecine AKOS, E. Médico-Chirurgicale*, Editor. 2013. p. 1-8.
9. Grélot, L., Activités physiques et sportives de l'enfant et de l'adolescent : des croyances aux recommandations sanitaires. *Journal de Pédiatrie et de Puériculture*, 2016. 29: p. 57-68.
10. Annexe XIV: Règlement Médical de la Fédération Française de Rugby. <https://api.ligueidf.ffr.fr/wp-content/uploads/2019/07/RG-2019-20-Annexe-XIV.pdf>. 2019.

# Disability and sports

Jérôme Porterie<sup>1</sup>, Anne Safi<sup>1</sup>, Claire Duran Joya<sup>1</sup>, Jérôme Sales De Gauzy<sup>2</sup>

1. Centre Pédiatrique de Médecine Physique et de Réadaptation Roquetaillade. 32550 Montegut
2. Toulouse University Children's Hospital

For children with disabilities, partaking in sports presents additional challenges that must be overcome. Any limitations caused by their disabilities, especially in patients with more severe cases, may be a source of discouragement. As a result, disabilities are often responsible for a decrease or even an arrest in sports, which hinders their well-being of these children. In fact, sports allow an improvement in physical and mental health, and particularly social skills and self-esteem in these children.

In order for benefit rather than harm these children with disabilities, physical activity must be supervised and adapted depending on the child's specific disability and degree of severity. Meticulously detailed recommendations may be found on the website of the French Federation for Handicapped Sport.

In this presentation, recommendations for the practice of sports in children with certain orthopedic pathologies will be discussed, followed by a portrayal of the experience of the parasports division of the Roquetaillade children's rehabilitation center.

## Orthopedic pathologies and sports

### 1. Cerebral palsy

The proposed sporting activity depends on the level of the child's disability. Sports allow an improvement in motor function, especially in skill and strength, and an increase in bone density [6]. In counterpart, physical activity has little effect on the improvement of gait [2]. Sports teams also allow social interaction.

All activity must be supervised, since abnormal muscle control in these children can lead to both muscle and bone injuries [5,11]; wheelchair-based exercises may lead to injuries of the upper limbs, especially the shoulders. If there is a history of convulsions, certain activities must be avoided, such as aquatic sports and activities requiring high-speed movement or ballistic equipment (shooting, archery). The choice of sport must be made after an initial evaluation of the child's capabilities [5]. The type of activity, its duration, and its intensity must be personalized for each individual child [4,14].

### 2. Neuromuscular diseases

In children with myopathies or infantile spinal muscular atrophy, sports allow an improvement in quality of life and both gait and wheelchair use and slows the progression of the disease by slowing muscular deterioration [1,3,8]. However, if the level of exercise is too intense, this may cause an elevation in creatine phosphokinase and lead to the detriment of muscle [12]. Resistance or eccentric training risk exacerbating the dystrophic process [7].

Physical exercise is very beneficial for patients with neuromuscular diseases, as long as they are moderate in intensity [6]. In case of fatigue or pain, activity should be limited in terms of duration, intensity, and frequency [7,12].

### **3. Juvenile chronic arthritis**

Sports allow muscular reinforcement and increased endurance and bone density [13]. Non-weight-bearing activities are preferable, especially aquatic sports [9,10]. In patients with progressive disease, and depending on the anatomical location of the affliction, the sport may increase the risk of articular damage, or may even lead to fractures or neurological complications [6]. It is therefore imperative to adapt the level of activity depending on each child's abilities.

### **Parasports division of the Roquetaillade children's rehabilitation center.**

The Roquetaillade center is a pediatric follow-up care and rehabilitation center managed by the Order of Malta. It is situated next to the Auch commune in the Gers department in France. The center is specialized in orthopedics, burns, and neuromuscular diseases. Children who are admitted are provided rehabilitation, schooling and readaptation.

In 2004, an athletic study center for children with disabilities was founded at the Roquetaillade center. This was the first of its kind in France and was realized in association with the French chapter of order of Malta and the ministry of national education (Carnot College in Auch). The goals of this athletic section were to provide young people with motor disabilities of the high-school or equivalent age, a profound sporting experience in the setting of an athletic study section, at the same level as in able-bodied children. Multiple levels of activity were proposed: Discovery of a sporting activity, perfecting capacities, competition, and insertion with able-bodied children. Sporting coordination was ensured by a physical and sports education professor who was at the behest of the Carnot school for a period of 4 hours a week. Children were admitted after examination of their academic files and a medical assessment. The athletic section offered multiple sporting activities. The type of sport was chosen as a function of the child's capacity and was always adapted to their level of disability. This included sports that were agreed upon by the French Federation for Handicapped Sport, represented at the Paralympic games or in national competitions: horse-back riding, table tennis, sport blowgun, swimming, shooting, athletics, judo, archery, boccia, basketball, tennis, Olympic-style weightlifting, and handbike (figures 1-6).



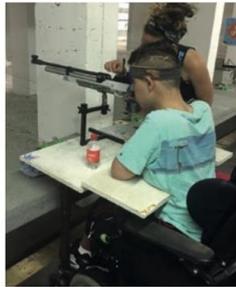
Blowdart



Tricycling



Archery



Bullseye shooting



Boccia



Fencing



Boccia

The primary results were very encouraging, with the integration of 12 children in the athletic division. The sports in question included horseback riding, swimming, football (soccer), table tennis, shooting, boccia, and blowgun.

However, partnership with the ministry of national education was cut short, which put an end to the athletic study section. This was later replaced by the Saint-Jacques Roquetaillade parasports organization, which works in partnership with the departmental and regional parasports comities. Sports tournaments are regularly arranged with its culmination being the “National Games of the Future Parasports” which take place every 2 years and which welcome, depending on the edition, 450 to 650 young people. The official program is

composed of 12 sporting activities: Athletics, basketball, boccia, fencing, 5-man football (soccer), swimming, table tennis, blowgun, archery, shooting, and tricycling. The parasports division of Roquetaillade includes 10 to 15 participants each year. Some have undertaken grand performances: participating in the Paralympic games, French blowgun championship, world and European champions in wheelchair football (soccer). The evaluation of athletes in the Roquetaillade center confirms the importance of sports in these children which has a major impact on motor function, quality of life, and well-being.

## Conclusion

The importance of sports for the physical and mental well-being of disabled children should no longer be debated. Nevertheless, the level of physical activity must be adapted to the disability of each child in order to avoid injury.

The role of the parasports division renders sports accessible to all children with motor disabilities and allows better social integration.

## References

1. Alemdaroglu I, Karaduman A, Yilmaz OT, Topaloglu H. Different types of upper extremity exercise training in Duchenne muscular dystrophy: effects on functional performance, strength, endurance, and ambulation. *Muscle Nerve*. 2015; 51:697-705.
2. Bania TA, Dodd J, Baker RJ, et al. The effects of progressive resistance on daily physical activity in young people with cerebral palsy: a randomized control trial. *Disabil. Rehabil.* 2016; 38:620-6.
3. Bora G, Subasi-Yildiz S, Yesbek-Kaymaz A, et al. Effects of arm cycling exercise in spinal muscular atrophy type II patients: a pilot study. *J. Child Neurol.* 2018; 33:209-15.
4. Butler JM, Scianni A, Ada L. Effect of cardiorespiratory training on aerobic fitness and carryover to activity in children with cerebral palsy: a systematic review. *Int. J. Rehabil. Res.* 2010; 33:97-103.
5. Carroll KL, Leiser J, Paisley TS. Cerebral palsy: physical activity and sport. *Curr. Sports Med. Rep.* 2006; 5:319-22.
6. Coleman N1, Nemeth BA, LeBlanc CMA. Increasing Wellness Through Physical Activity in Children With Chronic Disease and Disability. *Curr Sports Med Rep.* 2018 ; 17 :425-432.
- 7 . Grange RW, Jarrod A. Recommendations to define exercise prescription for Duchenne muscular dystrophy. *Exerc. Sport Sci. Rev.* 2007; 35:12-7.
8. Jansen M, van Alfen N, Geurts AC, de Groot IJ. Assisted bicycle training delays functional deterioration in boys with Duchenne muscular dystrophy: the randomized controlled trial «no use is disuse». *Neurorehabil. Neural Repair.* 2013; 27:816-27.
9. Kirchheimer JC, Wanivenhaus A, Engel A. Does sport negatively influence joint scores in patients with juvenile rheumatoid arthritis. An 8-year prospective study. *Rheumatol. Int.* 1993; 12:239Y42.

10. Long AR, Rouster-Stevens KA. Efficacy of a land-based home exercise programme for patients with juvenile idiopathic arthritis: a randomized, controlled, single-blind study. *Curr. Opin. Rheumatol.* 2010; 22:213-7.
11. Morris PJ. Physical activity recommendations for children and adolescents with chronic disease. *Curr. Sports Med. Rep.* 2008; 7:353-8.
12. Spaulding HR, Selsby JT. Is exercise the right medicine for dystrophic muscle? *Med. Sci. Sports Exerc.* 2018; 50:1723-32.
13. Van Oort C, Tupper SM, Rosenberg AM, et al. Safety and feasibility of a home-based six-week resistance-training program in juvenile idiopathic arthritis. *Pediatr. Rheumatol. Online J.* 2013; 11:46.
14. Verschuren O, Ada L, Maltais DB, et al. Muscle strengthening in children and adolescents with spastic cerebral palsy: considerations for future resistance training protocols. *Phys. Ther.* 2011; 91:1130-9.

# Surgical management of osteochondritis dissecans of the knee

Yan Lefèvre\*, Abdelfetah Lalioui\*, Franck Accadbled\*\*

\* Department of Pediatric Orthopaedic Surgery, Bordeaux University Children's Hospital

\*\* Department of Pediatric Orthopaedic Surgery, Purpan Toulouse University Hospital

Osteochondritis (OCD) of the knee is a pathology of the joint cartilage and underlying subchondral bone. OCD in skeletally immature patients is designated as juvenile OCD. Its preferred location is the lateral aspect of the medial femoral condyle. These types of lesions are generally classified based on their radiographic [1,2], MRI [3,4], and arthroscopic appearances [5,6]. The risk of progression is predicted primarily by the age of onset [7] and stability of the lesion [8,9].

The goal of treatment, in addition to pain relief, is to reconstitute the subchondral bone and prevent articular surface degeneration, ultimately preventing early secondary arthritis, especially in patients with a loose osteochondral fragment.

Surgical management of OCD may be considered after conservative treatment has failed. These modalities depend on the patient's symptoms and skeletal maturity, as well as the characteristics of the lesion (nature, location, size, and stability). Nevertheless, surgical management remains controversial without any real consensus on the matter. Nonetheless, different therapeutic options will be discussed, and a management algorithm based on the results in the literature will be suggested.

If surgical management of an osteochondral lesion is considered, associated favoring mechanical factors, such as axial deformities of the lower limbs, especially frequently encountered in more severe cases, must also be addressed.

## I – Surgical techniques

### 1. Arthroscopic exploration and assessment

Surgical management in patients presenting with OCD begins with an arthroscopic exploration of the knee, including inspection and testing of the osteochondral lesion with a hook. The lesion is thus characterized based on its appearance, size, and stability, allowing to either confirm or adjust the considered surgical technique [6] (figure 1).

Palpation		Inspection		
Stable lesions		<b>Cue ball</b> No abnormality detectable		
		<b>Shadow</b> Cartilage intact but subtly demarcated		
	Unstable lesions		<b>Wrinkle in the rug</b> Cartilage is demarcated with a fissure, buckle and/or wrinkle	
			<b>Locked door</b> Cartilage fissuring, <i>unable</i> to hinge open	
			<b>Trap door</b> Cartilage fissuring, <i>able</i> to hinge open	
			<b>Crater</b> Exposed subchondral bone defect	

**Figure 1:** Arthroscopic classification of osteochondral lesions of the knee.

Furthermore, certain arthroscopic appearances may correspond to both a stable and unstable lesion on MRI. As a result, considering the progress made in the field of magnetic resonance imaging, especially its ability to diagnose early lesions and its high sensitivity in evaluating signs of secondary instability [10], in cases where MRI and arthroscopy confer conflicting results, the surgical strategy should preferentially be based on the appearance on MRI.

The patients and their families should be informed of the possibility of such discordant findings during arthroscopic exploration compared to preoperative imaging, with a possible intraoperative change in surgical strategy and technique. These possibilities should be anticipated, and the appropriate instruments and materials should be readily available. In the case where a more severe lesion than previously thought is encountered and a change in surgical technique is required, the surgeon should not hesitate to delay the surgical reconstruction if the required tools are lacking until the appropriate instruments are available.

## 2. Microfractures

The surgical creation of microfractures in the treatment of OCD is considered as the standard surgical technique by many authors. It is less frequently used in France, where the perforation (Pridie) technique is more common.

The principle of this techniques is to mobilize the subchondral mesenchymal stem cells which colonize a post traumatic blood clot by punching a hole around the osteochondral lesion via a direct transchondral approach. This technique generally undertaken by an arthroscopic approach. After debridement of the lesion and removal of the calcified plaque (Tidemark), a hole is made through the residual cartilaginous tissue using a fine awl or an angulated punch, at 3 to 4mm intervals. The areas where the microfractures were created must be checked for bleeding at the end of the intervention [11].

The healing process is achieved through a relatively quick formation of fibrocartilaginous tissue ensuring the fusion of the osteochondral lesion. However, these fibrocartilaginous

formations seem to lose stability over the long term, with progressive degradation taking place [12].

### **3. Autologous matrix-induced chondrogenesis**

Autologous matrix-induced chondrogenesis is an evolution of the previously described surgical technique and consists of enclosing the clot induced by the microfractures by trapping it in a protective membrane. This membrane is generally fashioned out of periosteum or collagen (e.g. Chondro-gide®) that is either glued or sutured over the osteochondral loss of substance [13]. This membrane plays the role of a matrix securing the proteoglycans in place and inducing chondrogenic differentiation.

Early studies were very promising, with other authors later questioning the real efficacy of this technique [14].

### **4. Drilling**

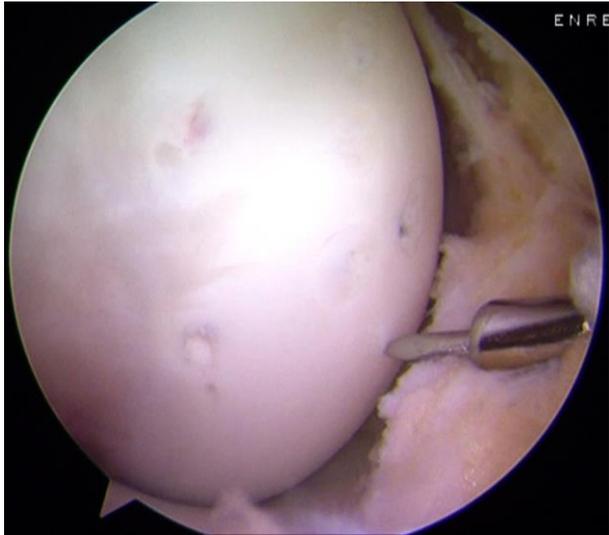
The drilling technique is much more frequently used in France compared to the microfractures technique. Pridie introduced the essential principles of drilling the exposed subchondral bone with the aim of achieving fibrocartilaginous healing, even before Smillie developed the drilling technique for osteochondritis dissecans [15].

This surgical technique is indicated after failure of conservative treatment of a stable lesion with a preserved cartilaginous surface.

The objective of this technique is to permeabilize the area of sclerotic bone surrounding the osteochondral lesion in order to induce bony healing through a local secretion of growth factors, revascularization of the osteochondral area, and migration and proliferation of osteochondral cells. This consists of perforating the floor of the lesion, either through a trans-chondral approach through the joint cartilage, or by a retroarticular approach. Both provide similar clinical and radiographic results (95% after 15 months of follow-up in patients between 10 and 16 years old) [16].

#### **4.a. Trans-chondral drilling**

Trans-chondral drilling is performed on stable lesions via knee arthroscopy, even though it was initially described by Smillie via open arthrotomy [15]. The osteochondral lesion is drilled by a retrograde approach directly through the cartilaginous surface by using fine K-wires (1.2mm) at a depth of 15 to 20mm (figure 2).

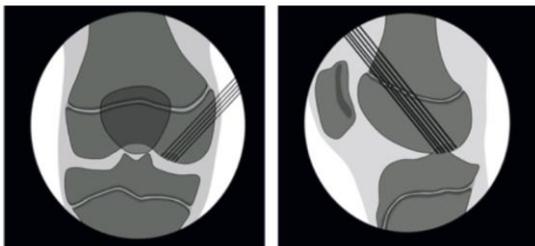


**Figure 2:** Arthroscopic view of trans-chondral drilling to treat stable osteochondritis dissecans.

Afterwards, a 1-month period of strict bed rest with complete unloading of the limb is imperative. Results are generally satisfactory, with younger subjects showing better results.

#### **4.b. Retroarticular drilling**

Retroarticular drilling implies the use of fluoroscopy. This technique respects the cartilaginous surface as well as the physis by utilizing a retrolesionnal and transepiphyseal approach. The K-wires are introduced under fluoroscopic guidance through the femoral condyle, on both anteroposterior and lateral images, until reaching the cartilage. A maximal number of perforations must be performed, covering the entire surface of the lesion [17,18] (figure 3).



**Figure 3:** *Depiction of the fluoroscopic anteroposterior and lateral views of the retroarticular perforations technique.*

#### **5. Fixation of an osteochondral fragment**

When MRI confirms the unstable nature of an osteochondral fragment, fixation of said fragment is the preferred method, and is ideally undertaken via fluoroscopic guidance. However, in order to achieve proper fixation, a sufficiently large fragment is required.

The bed of the osteochondral fragment is first debrided and prepared, then perforated by K-wires as previously described. If the lesion is too deep relative to the thickness of the loose

fragment thereby leaving a dead space after restoring the fragment, a cancellous bone graft from either the iliac crest or the proximal tibial metaphysis should be used.

In sum, the fragment is restored and maintained in place by a thin temporary K-wire. Osteosynthesis is then realized by 1 or 2, ideally cannulated, or even doubly threaded, screws. The head of the screws must be countersunk within the cartilaginous surface in order to avoid any local retractions or any friction between tibial plateau and the screw head (figure 4).



**Figure 4:** Fixation of an unstable osteochondral fragment. *a: fixation by 1 metallic screw. b: absorbable “low-profile” SmartNail® ConMed® implants presenting alternatives to screws.*

Both standard and absorbable screws may be used. In fact, metallic screws present the inconvenience of causing artefacts on a future MRI and are frequently the source of significant local friction, thus requiring revision surgery for the removal of the screw [5]. As such, absorbable screws essentially made out of two materials are preferred: polyglycolic acid (PGA) and Polylactic acid (PLA). The former has the advantage of being relatively fast absorbing (around 3 months) but generates a significant amount of local inflammation [19], whereas the latter has a slower absorption time (up to 6 years) which may lead to similar complications as non-absorbable screws [20].

Another alternative is an association of the 2 polymers. These implants are not screws, but rather jagged nails which avoid cut-out and allow compression of the fragment with a shallow head (low profile) [21] (figure 4).

The outcomes of fragment fixation are globally satisfactory in children with better results than in adults [22].

## **6. Autologous osteochondral graft**

The principle of autologous osteochondral grafting consists of packing the prepared defect zone with osseous tissue covered by a cartilaginous surface. The graft is harvested from a non-weight-bearing area in order to limit morbidity at the donor site.

The graft may consist of a single osteochondral block: this technique is limited in terms of grafting surface, since morbidity increases with the size of the harvested area and with the incongruity of the shape of the grafting area relative to that of the bone graft (radius of the curve). This technique is indicated in less extensive lesions.

Alternatives consist of grafting one or multiple cylinders (mosaic) allowing to globally model the shape of the grafting volume while playing on the lengths of different implanted grafts (plasty).

### 6.1. Mosaicplasty

This technique of osteochondral grafting allows the reconstruction of surfaces presenting a relatively extended loss of substance. Initially attempted by Matusue in 1988 [23], it was later developed by Hangody in the early 1990s, who based their findings on experimental animal studies and backed by a large number of case series with good global outcomes (around 90% depending on the series and outcome criteria) [24,25]. Outcomes are generally better in younger subjects [26].

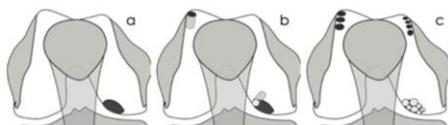
In fact, histological studies of grafted areas show that, 10 weeks after grafting, 60 to 70% of the grafted surface is formed by hyaline cartilage and 30 to 40% fibrocartilage. Similar results have been found in studies on humans who were biopsied 5 years after grafting.

Mosaicplasty presents, compared to autologous chondrocyte grafting – its modern alternative – the advantages of being easy-to-use since it does not require cellular cultures (and the means that this imposes), and being undertaken as a single event.

#### Objectives:

The objectives of mosaicplasty include:

- After debridement and preparation, completely fill the volume of the osteochondral lesion until reaching a healthy bony bed. The bony part of the graft must be in contact with the healthy bone.
- The surface of the grafted cartilage must constitute 70% of the surface of the defect (80% for some authors).
- Respect the physis which is deep to the lesion site. Figure 5 illustrates the procedure.



**Figure 5 :** Illustration of an autologous osteochondral graft by mosaicplasty. a: initial lesion. b: harvesting of the first graft and preparation of the grafting area. c: final grafting with 7 grafts harvested from both edges of the trochlea.

**Approach:**

Mosaicplasty is undertaken either via open arthrotomy (medial or lateral parapatellar approach depending on the location of the lesion) or by arthroscopy, depending on the surgeon's experience with arthroscopy, the accessibility of the osteochondral lesion, and the available instruments.

**Materials and instruments:**

Specific instruments are required, including drill bits for the preparation of the grafting site (with possibly an adjustable depth stop) of different diameters (2, 7 and 8mm), chisels for graft harvesting with diameters in accordance with those of the preparation drill bits, an adapted bone impactor, and a tube with a depth gage for the easy positioning of the grafts.

**Donor sites:**

The harvesting areas include, in descending order of preference, the edges of the femoral trochlea, preferably the medial side (especially superomedial), then lateral, and finally the periphery of the femoral intercondylar notch [27]. Some surgeons prefer harvesting from the edge of the contralateral femoral condyle in order to identify the origins of any possible future pain [28].

The lateral edge of the trochlea allows harvesting of larger grafts (up to 3 grafts of 10 to 11mm each), a more delicate procedure on the medial side (grafts of 7 to 8mm each) and at the intercondylar notch (grafts of 6mm each).

Note that the areas of bone and cartilage loss secondary to the harvesting of the grafts may bleed and may be responsible for a postoperative hematoma. As a result, some authors suggest packing these defects with, for example, collagen gauzes which allow, in addition to their powerful hemostatic abilities, proper reconstitution of a fibrocartilaginous surface [29].

**Preparation of the grafting site:**

Preparation consists of debriding any fibrous tissue and questionable edges around the lesion area using a shaver and/or a curette until reaching healthy cartilaginous edges that are regular and perpendicular to the subchondral bone. The depth of the lesion is debrided using a curette or a burr until arriving at healthy bone. Although contact with healthy tissue, either cartilaginous or bony, is essential for the graft to take properly, graft harvesting is nevertheless realized with moderation in order to avoid unnecessary damage.

The grafting area which will be receiving the cylindrical grafts is prepared with a drill bit, the diameter of which corresponds to the diameter of the cylinders. The required drilling depth should be previously set on the drill bit. Preparation of the osteochondral lesion must be done in a perfectly perpendicular fashion which implies a very precise and delicate positioning of the drill bit. The different cylinders should be either spaced at intervals of 1mm, or in contact with one another. This implies meticulous placement of the drill bit in order to achieve perfectly parallel tubes.

In practice, the grafting area is drilled after the osteochondral grafts have been harvested in order to ensure compatibility of size (height of graft and depth of the grafting area), a process which is easier to control while drilling rather than harvesting.

### **Size and amount of graft:**

The sizes of the grafts depend on the depth of the osteochondral lesion, the defect that must be filled, and the distance to the physis (which must be respected). Practically, the graft often has a length of around 15 to 25mm. The diameters of the grafts depend on both the size and shape of the defect and surgeon preference. Nevertheless, recent publications tend toward harvesting larger grafts, which present with the advantage of increased stability, generating less fibrous interposition between the fragments, providing a wider cartilaginous surface, and minimizing the risk of causing a fracture of the donor site. An alternative would be to harvest cylinders of varying diameters in order to optimize it to the shape of the lesion (>80%). The number of grafts required should be determined at the beginning of the intervention after the osteochondral lesion has been evaluated by comparing its surface to bone impactors of different diameter.

### **Graft harvesting:**

Specific instruments are required, including drill bits of different diameters (2, 7 and 8mm) for the preparation of the grafting site (with possibly an adjustable depth stop), chisels for graft harvesting with diameters in accordance with those of the preparation drill bits, an adapted bone impactor, and a tube for easy positioning of the graft with a depth gage.

Harvesting is undergone with dedicated tubular chisels. Care must be taken to properly place the chisels perpendicularly to the articular surface (figure 6).



**Figure 6 :** *Illustration of the arthroscopic view of the chisel during graft harvesting placed perpendicular to the lateral edges (a) of the trochlea far from the patellar tendon (b). Note the gradations (arrow) on the chisel for gaging the length of the graft.*

The chisel is then slowly advanced as to avoid excessively heating the surrounding tissues. Once the proper depth is reached, determined beforehand on the chisel, the graft is detached from its base by repetitive varus-valgus movements around the axis of the chisel. Care must be taken during this process in order to avoid fracturing the harvesting site.

A distance of 3mm should be respected between the different fragments.

### Implantation of the graft:

Various methods exist depending on whether a non-press-fit effect (cylinders at the grafting area are slightly dilated at the surface by using a slightly conic dilator, in order to allow a non-traumatic implantation) or a press-fit effect (cylinders of 1mm smaller diameter than the bone graft) is desired. Animal studies have shown that a press-fit effect is preferable for graft taking and is thus recommended by many authors [30].

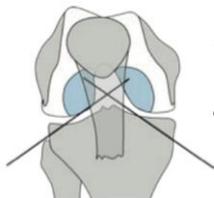
The graft is introduced through a tubular introducer of proper diameter and is then implanted with a bone impactor passed through the introducer, thus pushing the graft to the far end of the defect area. The graft must be introduced until the cartilaginous surface becomes level with the contiguous healthy or grafted cartilage (ideally, the height would be assessed using the bone impactor).

The cartilaginous surfaces must invariably be level with each other. In fact, it was previously established that a step off of 2mm or more would lead to involution of the cartilaginous layer [31]. As a result, it is important to harvest the grafts perfectly perpendicular to the articular surface, since a mere angulation of 10° while harvesting a 10mm diameter graft leads to a height difference of 1mm between the opposing edges.

### Specifics of arthroscopy:

This technique is reserved for surgeons with experience in arthroscopy due to its high level of difficulty. These difficulties are essentially twofold:

- Location of the lesion which must allow perpendicular access (figure 7).



- Size of the lesion as it is more difficult to pack a lesion superior to 2cm (a maximum of 6 cylinders).

Patient positioning must allow knee flexion up to 120°. Entry points are more central than usual since the lesions are often centered around the intercondylar notch (figure 7). A needle can be used to determine the ideal entry point allowing for perpendicular access to the grafting surface. If necessary, multiple entry points may be created. Contrarily, in order to decrease the number of required grafts, a single, large fragment may be used to fill the defect (figure 8).



**Figure 8:** Arthroscopic view of an osteochondral lesion (a) treated by osteochondral grafting using the mosaicplasty technique with a single, 10mm-wide graft (b).

All the instruments, including the drill bit, dilator, and chisel are graduated in order to assess the depth of the lesion on the instruments (figure 6).

The remainder of the procedure is undertaken according to the same principles as in open procedures.

#### **Postoperative management:**

The limb must be unloaded for an average of 6 weeks depending on the different authors (4 to 8 weeks), followed by progressive weight bearing. The knee is immediately mobilized in flexion and extension, at first only passively.

#### **Complications:**

The primary reported complications include postoperative hematomas, deep infections, deep vein thrombosis (adults), and rare, essentially painful, degenerative lesions around the donor site (3%) [32].

### **6.2. Massive osteochondral allograft**

This relatively old technique consists of using a fresh or frozen osteochondral allograft for voluminous losses of tissue and is more commonly used as a salvage procedure in adults.

### **6.3. Chondrocyte culture**

These techniques consist of grafting the previously prepared lesion using autologous chondrocytes which have been harvested during a prior surgery and have been placed in a culture medium in vitro for over 2 to 3 weeks [33]. First-generation grafts entailed autologous harvested chondrocytes, cultivated and amplified in a cellular culture medium, placed in the grafting area under a patch of periosteum removed from the tibia which is then sutured or glued to the healthy edges. Long-term results are good in patients with osteochondritis, with a hyaline-like tissue filling the gaps, as confirmed by histology [34]. Nevertheless, its superiority to mosaicplasty was questionable and a certain number of complications were reported, related especially to the periosteal patch (calcifications, ossifications, avulsions, leakage) [35,36].

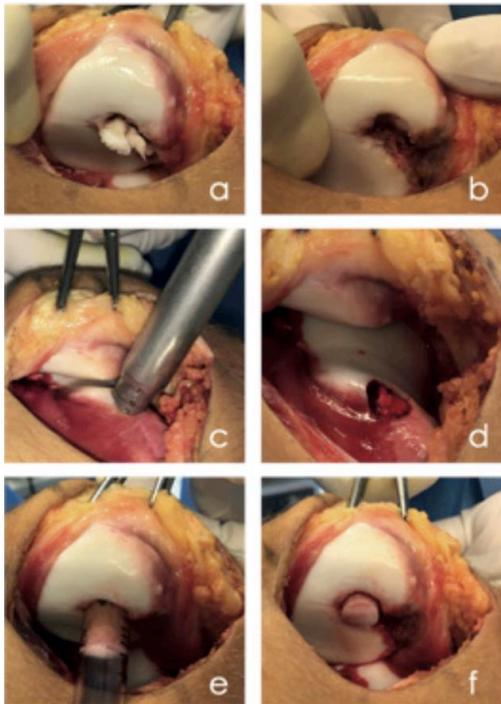
As such, in second-generation grafts, the patch was replaced by synthetic, protein or polysaccharide membranes, a technique known as autogenous matrix induced chondrogenesis (AMIC). These membranes contain interactive capacities with the grafted chondrocytes, favoring graft taking.

Finally, third-generation grafts consist of placing the chondrocytes in a culture medium in an implantable biological matrix, favorable for the promotion of cellular proliferation, conservation of phenotypical characteristics, and synthesis of extracellular matrix, all of which for a moderate cost [37]. In deeper lesions, multiple layers may be required, a technique

known as a “sandwich graft” (if depth of the defect >8mm). These third-generation grafts, such as those utilizing hyaluronic acid, are still being evaluated [38].

## II- The special case of osteochondral lesions of the patella

Osteochondral lesions of the patella are less frequently encountered and are treated with the same surgical techniques and therapeutic indications as was previously discussed, with the only exception that an open approach is preferred due to difficulties in accessibility by arthroscopy (figure 9) [39].

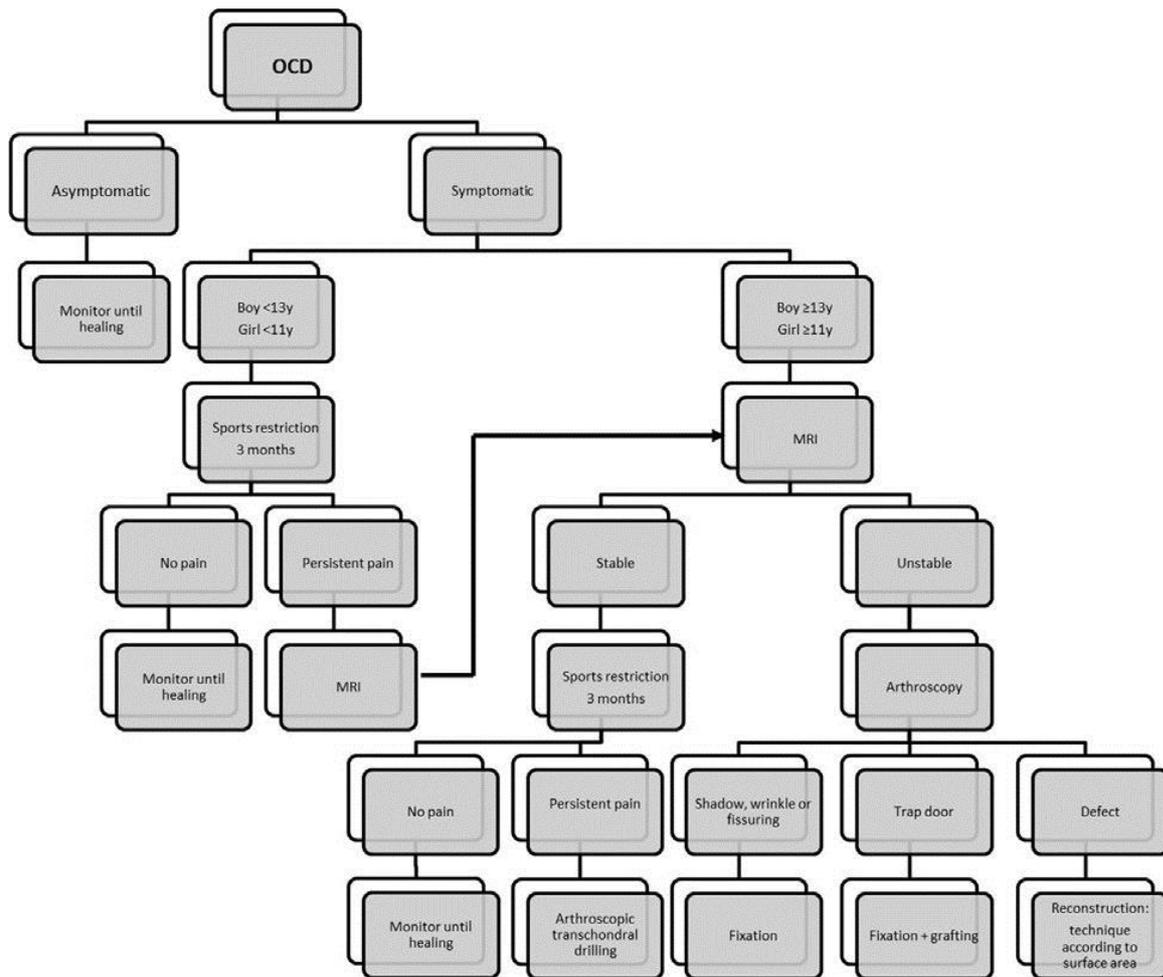


**Figure 9:** Treatment of osteochondritis dissecans of the patella by mosaicplasty by arthrotomy.

*a: unstable and eroded lesion. b: preparation of the lesion area to receive the graft. c: graft harvesting from the lateral edge of the trochlea with a graduated cylindrical chisel. d: appearance of the harvesting site after removal of the graft. e: insertion of the graft into the lesion. f: appearance at the end of the intervention with the osteochondral graft in place.*

## III- Indications

An algorithm, based on the patient’s age (major prognostic factor) and appearance of the lesion on MRI and arthroscopy (stability), as was proposed by Accadbled et al., with a modified version being later proposed based on the works on Carey and the American Association of Orthopedic Surgery (figure 10) [40].



**Figure 10:** Treatment algorithm for osteochondritis dissecans of the knee according to Accadbled et al. [6].

It is to note that, if there is discordance on the stability of the lesion between MRI and arthroscopy, MRI is privileged for the final decision (MRI signs are encountered earlier in the disease process).

#### IV- Conclusions

When operative treatment of osteochondritis dissecans is indicated, it must be preceded by an MRI. Most often, surgery is approached via arthroscopy allowing to firstly complete a macroscopic evaluation of the lesions, and to secondly treat the lesion by drilling the stable lesions (based on MRI and/or arthroscopy), or either fixing or grafting unstable lesions.

As for the clinical and radiographic outcomes of treatment, both drilling and mosaicplasty have shown good results and are relatively simple to achieve by surgeons experienced in arthroscopy.

Other solutions including autologous chondrocyte or matrix grafts are promising, even though they may be more difficult to achieve.

## References:

1. Bedouelle J. L'ostéochondrite disséquante des condyles fémoraux chez l'enfant et l'adolescent. In : Conférences d'enseignement 1988 (Cahiers d'Enseignement de SOFCOT) Expansion Scientifique Française ; 1988. p. 61–93.
2. Berndt AL, Harty M. Transchondral fractures (osteochondritis dissecans) of the talus. *J Bone Joint Surg [Am]* 1959. 41-A(6 ): p. 988–1020.
3. Dipaola JD, Nelson DW, Colville MR. Characterizing osteo- chondral lesions by magnetic resonance imaging. *Arthroscopy* 1991. 7: p. 101–4.
4. Kijowski R, Blankenbaker DG, Shinki K, et al. Juvenile versus adult osteochondritis dissecans of the knee : appropriate MR imaging criteria for instability. *Radiology* 2008. 248: p. 571–8.
5. Guhl Jf. Arthroscopic treatment of osteochondritis dissecans. *Clin Orthop Relat Res* 1982. (167): p. 65–74.
6. Accadbled F, Vial J, Sales de Gauzy J. Osteochondritis dissecans of the knee. *Orthop Traumatol Surg Res.* 2018. 104(1S): p. S97-S105.
7. Siegall E, Faust JR, Herzog MM, et al. Age predicts disruption of the articular surface of the femoral condyles in knee OCD: Can we reduce usage of arthroscopy and MRI? *J Pediatr Orthop.* 2018. 38(3): p. 176-180.
8. Masquijo J, Kothari A. Juvenile osteochondritis dissecans (JOCD) of the knee: current concepts review. *EFORT Open Rev*, 2019. 17; 4(5): p. 201-212.
9. Versier G, Dubrana F; French Arthroscopy Society. Treatment of knee cartilage defect in 2010. *Orthop Traumatol Surg Res*, 2011. 97(8 Suppl): p. S140-53.
10. Kijowski R, Blankenbaker DG, Shinki K, et al. Juvenile versus adult osteochondritis dissecans of the knee: appropriate MR imaging criteria for instability. *Radiology*, 2008. 248: p. 571–8.
11. Steadman JR, Rodkey WG, Singleton SB, et al. Microfracture technique for full-thickness chondral defects: technique and clinical results. *Oper Tech Orthop*, 1997. 7: p. 300-4.
12. Mithoefer K, McAdams T, Williams RJ, et al. Clinical efficacy of the microfracture technique for articular cartilage repair in the knee. An evidence- based systematic analysis. *Am J Sports Med*, 2009. 37(10): p. 2053-63.
13. Behrens P, Bitter T, Kurz B, et al. Matrix- associated autologous chondrocyte transplantation/ implantation (MACT/MACI), 5-year follow-up. *Knee*, 2006. 13(3): p. 194-202.
14. Dhollander AM, De Neve F, Almqvist F, et al. Autologous matrix-induced chondrogenesis combined with platelet-rich plasma gel: technical description and a five pilot patients report. *Knee Surg Sports Traumatol Arthrosc*, 2011. 19: p. 536-42.
15. Smillie IS. Treatment of osteochondritis dissecans. *J Bone Joint Surg Br*, 1957. 39-B(2): p. 248-60.
16. Rammal H, Gicquel P, Schneider L, et al. Juvenile osteochondritis of femoral condyles: treatment with transchondral drilling. Analysis of 40 cases. *J Child Orthop*, 2010. 4(1): p. 39-44.
17. Edmonds EW, Polousky J. A review of knowledge in osteochondritis dissecans: 123 years of minimal evolution from König to the ROCK study group. *Clin Orthop Relat Res*, 2013. 471(4): p. 1118–26.
18. Edmonds EW, Albright J, Bastrom T, et al. Outcomes of extra- articular, intra-epiphyseal drilling for osteochondritis dissecans of the knee. *J Pediatr Orthop*, 2010. 30(8): p. 870–8.
19. Fridén T, Rydholm U. Severe aseptic synovitis of the knee after biodegradable internal fixation. A case report. *Acta Orthop Scand*, 1992. 63(1): p. 94–7.
20. Mainil-Varlet P, Rahn B, Gogolewski S. Long-term in vivo degradation and bone reaction to various

- polyactides. 1. One-year results. *Biomaterials*, 1997. 18(3): p. 257-66.
21. Tabaddor RR, Banffy MB, Andersen JS, et al. Fixation of juvenile osteochondritis dissecans lesions of the knee using poly 96L/4D-lactide copolymer bioabsorbable implants. *J Pediatr Orthop*, 2010. 30(1): p. 14–20.
22. Lefort G, Moyon B, Beaufils P, et al. Osteochondritis dissecans of the femoral condyles: report of 892 cases. *Rev Chir Orthop Reparatrice Appar Mot*, 2006. 92(5 Suppl): p. 2S97-2S141.
23. Matsusue Y, Yamamuro T, Hama H. Arthroscopic multiple osteochondral transplantation to the chondral defect in the knee associated with anterior cruciate ligament disruption. *Arthroscopy*, 1993. 9(3): p. 318-21.
24. Hangody L, Kish G, Kárpáti Z, et al. Mosaicplasty for the treatment of articular cartilage defects: application in clinical practice. *Orthopedics*, 1998. 21(7): p. 751-6.
25. Hangody L, Vásárhelyi G, Hangody LR, et al. Autologous osteochondral grafting technique and long-term results. *Injury*, 2008. 39(Suppl. 1): p. S32-9.
26. Gudas R, Simonaityte R, Cekanauskas E, et al. A prospective, randomized clinical study of osteochondral autologous transplantation versus microfracture for the treatment of osteochondritis dissecans in the knee joint in children. *J Pediatr Orthop*, 2009. 29(7): p. 741-8.
27. Garretson RB, Katolic LI, Beck PR, et al. Contact pressure at osteochondral donor sites in the patellofemoral joint. *Am J Sports Med*, 2004. 32 : p. 967-74.
28. Robert H. Technique de réparation du cartilage du genou par plastie en mosaïque. In: *Conférence d'enseignement 2010 (Cahiers d'enseignement de la SOFCOT n° 99)*. 2010, Elsevier- Masson: Paris. p. 368- 84.
29. Feczko P, Hangody L, Varga J, et al. Experimental results of donor site filling for autologous osteochondral mosaicplasty. *Arthroscopy*, 2003. 19(7): p. 755-61.
30. Makino T, Fujioka H, Terukina M, et al. The effect of graft sizing on osteochondral transplantation. *Arthroscopy*, 2004. 20: p. 837-40.
31. Huang FS, Simonean PT, Norman AG, et al. Effects of small incongruities in a sheep model of osteochondral grafting. *Am J Sports Med*, 2004. 32: p. 1842-8.
32. Hangody L, Feczko P, Bartha L, et al. Mosaicplasty for the treatment of articular defects of the knee and ankle. *Clin Orthop Relat Res*, 2001. (391 Suppl): p. S328-36.
33. Brittberg M, Lindahl A, Nilsson A, et al. Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. *N Engl J Med*, 1994. 331: p. 889-95.
34. Micheli LJ, Browne JE, Erggelet C, et al. Autologous chondrocyte implantation of the knee: multicenter experience and minimum 3-year follow-up. *Clin J Sport Med*, 2001. 11: p. 223-8.
35. Knutsen G, Drogset JO, Engebretsen L, et al. A randomized trial comparing autologous chondrocyte implantation with microfracture: findings at five years. *J Bone Joint Surg Am*, 2007. 89(10): p. 2105-12.
36. Bentley G, Biant LC, Carrington RW, et al. A prospective, randomized comparison of autologous chondrocyte implantation versus mosaicplasty for osteochondral defects in the knee. *J Bone Joint Surg Br*, 2003. 85: p. 223-30.
37. Kon E, Delcogliano M, Filardo G, et al. A novel nano-composite multi-layered biomaterial for treatment of osteochondral lesions: Technique note and an early stability pilot clinical trial. *Injury Int J Care Injured*, 2010. 41: p. 693-701.
38. Marcacci M, Zaffagnini S, Kon E, et al. Arthroscopic autologous chondrocyte transplantation: technical note. *Knee Surg Sports Traumatol Arthrosc*, 2002.10: p. 154-9.
39. Visonà E, Chouteau J, Aldegheri R, et al. Patella osteochondritis dissecans end stage: The osteochondral mosaicplasty option. *Orthop Traumatol Surg Res*, 2010. 96(5): p. 543-8.

40. Carey JL, Shea KG. AAOS Appropriate Use Criteria: Management of Osteochondritis Dissecans of the Femoral Condyle. *J Am Acad Orthop Surg*, 2016. 24(9): p. 105-11.