Thesis

INFLUENCE OF HIGH-GRADE TROCHLEAR DYSPLASIA ON THE EXTENT OF PATELLAR TILT IN CHILDREN AND ADOLESCENTS

A Retrospective Analysis based on MR Images of the Knee

submitted by
Fabian Schmid

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under the supervision of
Sebastian Tschauner, MD PhD
and
René Schroedter, MD

Graz, 24/06/2025

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Zusammenfassung

Fragestellung: Das Ziel dieser Diplomarbeit war es herauszufinden, ob und in welchem Ausmaß eine hochgradige Trochleadysplasie den Patellatilt bei Kindern und Jugendlichen beeinflusst. Beide anatomischen Merkmale sind bekannte Risikofaktoren für die patellofemorale Instabilität, welche vor allem bei Kindern und Jugendlichen eine häufige Pathologie darstellt. Vor allem die Trochleadysplasie ist gut untersucht, kann anhand bildgebender Methoden klassifiziert werden und spielt eine wichtige Rolle in der Therapieentscheidung. Auch der Patellatilt gilt als einfach zu bestimmender und zuverlässiger Parameter für die Diagnostik einer patellofemoralen Instabilität. Er gibt die Neigung der Patella in der horizontalen Ebene in Relation zum Femur an und wird in einer axialen Aufnahme des Kniegelenks bestimmt. Eine vermehrte laterale Verkippung der Patella in der Horizontalebene – entsprechend einem erhöhten Patellatilt – gilt als Risikofaktor für eine patellofemorale Instabilität. Über das Ausmaß der Korrelation zwischen der Trochleadysplasie und dem Patellatilt ist derzeit noch wenig bekannt.

Methodik: In einer retrospektiven MRT-Analyse wurden anhand von definierten Parametern die Aufnahmen von 630 Kindern und Jugendlichen (Alterspanne 7.9 – 17.9 Jahre), die entweder aufgrund einer erstmaligen Patellaluxation, einer anderen Knieverletzung oder unklarem Knieschmerz eine Bildgebung erhielten, vermessen. Zur Beurteilung der Trochleadysplasie wurden die Trochleatiefe, der laterale Trochleainklinationswinkel und der Trochleasulkuswinkel herangezogen. Der Patellatilt wurde anhand des lateralen Patellainklinationswinkels, des Fulkerson-Winkels und des Laurin-Winkels bewertet. Zur Untersuchung der Korrelation zwischen den Parametern der Trochleadysplasie und den Parametern des Patellatilts wurden Korrelationsanalysen nach Pearson durchgeführt. Einfache lineare Regressionsanalysen wurden durchgeführt, um die prädiktive Aussagekraft einzelner Dysplasie-Parameter zu untersuchen. Die Werte der Trochleadysplasie wurden dabei als unabhängige Variablen und die Werte des Patellatilts als abhängige Variablen definiert. Darüber hinaus wurden die Patient*innen nach dem Schweregrad der Trochleadysplasie in zwei Gruppen eingeteilt, um die Parameter des Patellatilts zwischen Patient*innen mit und ohne hochgradiger Trochleadysplasie zu vergleichen.

Ergebnisse: In der Korrelationsanalyse korrelierten alle Parameter der Trochleadysplasie signifikant mit den Parametern des Patellatilts: LTIA und TD korrelierten positiv mit dem Fulkerson-Winkel ($r = 0.510$, $r = 0.467$) sowie dem Laurin-Winkel ($r = 0.406$, $r = 0.450$) und negativ mit dem Patellaneigungswinkel ($r = -0.431$, $r = -0.462$). TSA korrelierte negativ mit dem Fulkerson-Winkel ($r = -0.425$) sowie dem Laurin-Winkel ($r = -0.432$) und positiv mit dem Patellaneigungswinkel ($r = 0.300$). In der einfachen Regressionsanalyse war TD der stärkste Prädiktor für den Patellaneigungswinkel ($R^2 = 0.214$; $p < 0.001$), während LTIA am besten den Fulkerson-Winkel vorhersagte ($R^2 = 0.260$; $p < 0.001$).

Schlussfolgerung: Die Ergebnisse unserer retrospektiven Studie zeigen einen signifikanten Zusammenhang zwischen einer Zunahme der Trochleadysplasie und einem verstärkten lateralen Patellatilt. Dabei war die Trochleatiefe der stärkste Prädiktor für den Patellaneigungswinkel. Weitere Untersuchungen sind notwendig, um Auswirkungen auf die Diagnostik und Therapie der PFI bewerten zu können.

Abstract

Background: The aim of this diploma thesis was to find out whether and to what extent high-grade trochlear dysplasia influences patellar tilt in children and adolescents. Both anatomical features are known risk factors for patellofemoral instability, which is a common pathology, especially in children and adolescents. Trochlear dysplasia has been well studied, can be classified using imaging methods and plays an important role in treatment decisions. The patellar tilt is also considered an easy to determine and reliable parameter for diagnosing patellofemoral instability. It indicates the inclination of the patella in the horizontal plane in relation to the femur and is measured in an axial image of the knee joint. An increased lateral tilt of the patella in the horizontal plane - resulting in an increased patellar tilt - is considered a risk factor for patellofemoral instability. Little is currently known about the extent of the correlation between trochlear dysplasia and patellar tilt.

Methods: In a retrospective MRI analysis, defined parameters were used to measure the images of 630 children and adolescents (age range 7.9 - 17.9 years) who underwent imaging either due to a first-time patellar dislocation, a different knee injury or unclear knee pain. The trochlear depth (TD), lateral trochlear inclination angle (LTIA) and trochlear sulcus angle (TSA) were used to assess trochlear dysplasia. The patellar tilt was assessed using the Patellar Inclination Angle, the Fulkerson angle and the Laurin angle. Pearson correlation analyses were performed to investigate the correlation between the trochlear dysplasia parameters and the patellar tilt parameters. Simple linear regression analyses were performed to investigate the predictive value of individual dysplasia parameters. In addition, patients were divided into two groups according to the severity of trochlear dysplasia to compare patellar tilt parameters between patients with and without high-grade trochlear dysplasia. The trochlear dysplasia values were defined as independent variables and the patellar tilt values as dependent variables.

Results: In the correlation analysis, all parameters of trochlear dysplasia correlated significantly with the parameters of the patellar tilt: LTIA and TD correlated positively with the Fulkerson angle ($r = 0.510$, $r = 0.467$) and the Laurin angle ($r = 0.406$, $r = 0.450$) and negatively with the Patellar Inclination Angle ($r = -0.431$, $r = -0.462$). TSA correlated negatively with the Fulkerson angle ($r = -0.425$) and the Laurin angle ($r = -0.432$) and positively with the Patellar Inclination Angle ($r = 0.300$). In the simple regression analysis, TD was the strongest predictor of Patellar Inclination Angle ($R^2 = 0.214$; $p < 0.001$), while LTIA best predicted Fulkerson angle ($R^2 = 0.260$; $p < 0.001$).

Discussion: The results of our retrospective study show a significant correlation between an increase in trochlear dysplasia and an increased lateral patellar tilt. Trochlear depth was the strongest predictor of Patellar Inclination Angle. Further studies are necessary to evaluate the effects on the diagnosis and treatment of PFI.

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LIST OF ABBREVIATIONS

ACL .... *Anterior Cruciate Ligament*
CT .... *Computed Tomography*
ITB .... *Iliotibial Band*
LCL .... *Lateral Collateral Ligament*
LPFL .... *Lateral Patellofemoral Ligament*
LPTL .... *Lateral Patellotibial Ligament*
LTIA .... *Lateral Trochlear Inclination Angle*
MCL .... *Medial Collateral Ligament*
MPFL .... *Medial Patellofemoral Ligament*
MRI .... *Magnetic Resonance Imaging*
PCL .... *Posterior Cruciate Ligament*
PFI .... *Patellofemoral Instability*
PFJ .... *Patellofemoral Joint*
POL .... *Posterior Oblique Ligament*
TD .... *Trochlear Depth*
TSA .... *Trochlear Sulcus Angle*
TT-TG .... *Tibial tuberosity to Trochlear Groove Distance*
VMO .... *Vastus Medialis Obliquus Muscle*

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1. Introduction

With an incidence of 29-50/100,000 in the group of 10 to 17-year-olds, patellar dislocation is one of the most common pathologies of the knee joint in children and adolescents (1,2). In most cases, the patella dislocates laterally, and the most common cause is a combination of inadequate trauma and predisposing anatomical factors (3). A decisive anatomical factor is the morphology of the patellofemoral joint (PFJ) and particularly the morphology of the femoral trochlea. Up to 85% of patients with recurrent patellar instability have a dysplastic trochlea (4–6). However, the epidemiology and exact prevalence of trochlear dysplasia in the general population is still unclear. Another important anatomical factor for patellofemoral instability (PFI) is the lateral tilt of the patella. This parameter can be determined easily using axial X-ray of the patella, Magnetic Resonance Imaging (MRI) or Computed Tomography (CT) and is considered a good indicator for patellar instability (7).

1.1. Aim of the study

Trochlear dysplasia has already been the subject of numerous studies on classification, diagnosis and therapeutic options. Patellar tilt is also recognised in the literature as a risk factor for PFI. However, the relationship between these two parameters has not yet been sufficiently researched. Therefore, the main aim of this thesis is to analyse the correlation between trochlear dysplasia and the extent of patellar tilt using MRI scans of children and adolescents.

1.2. Hypothesis

The following two hypotheses were created to define the main objective of the thesis.

Null hypothesis H_0:

There is no significant correlation between high-grade trochlear dysplasia and the extent of patella tilt in children and adolescents.

Alternative hypothesis H_1:

There is a significant correlation between high-grade trochlear dysplasia and the extent of patella tilt in children and adolescents.

2. Overview

2.1. Anatomy and Biomechanics of the Patellofemoral Joint

The knee joint is the largest joint in the human body, connects the upper and lower leg and is made up of three bones: Femur, Tibia and Patella. The knee joint can be divided into the tibiofemoral joint and the patellofemoral joint. In addition to the bones involved, the following soft tissues are important for the stability and functioning of the joint: knee joint capsule, menisci and ligaments, bursae, tendons and muscles (8). These structures will be described in detail below.

2.1.1. Bones

The proximal part of the knee joint is formed by the femur. The femur (Figure 1) is the largest human bone and is divided into a proximal part (head and neck), a shaft and a distal part, which is relevant for the knee joint. At the distal part the cylindrical medial and lateral condyles of the femur are located. The epicondyles, which are the points of origin for the lateral gastrocnemius muscle and plantaris muscle (lateral epicondyle), as well as the medial gastrocnemius muscle (medial epicondyle), are located proximally. In the centre of the condyles, the femur deepens to the intercondylar fossa, where the two cruciate ligaments attach. The surface on the front of the condyles which articulates with the back of the patella is called the femoral trochlea (9).

The curvature of the condyles, which increases posteriorly, is crucial for knee joint movement, as it allows the knee to exhibit complex movements, including both rolling and sliding of the femur, which is essential for proper flexion and extension. The joint surface visible in the sagittal section resembles the mathematical function of an involute (Figure 5). The centres of curvature of all joint surface sections form the evolute, which is discussed in more detail in the chapter 2.1.3.

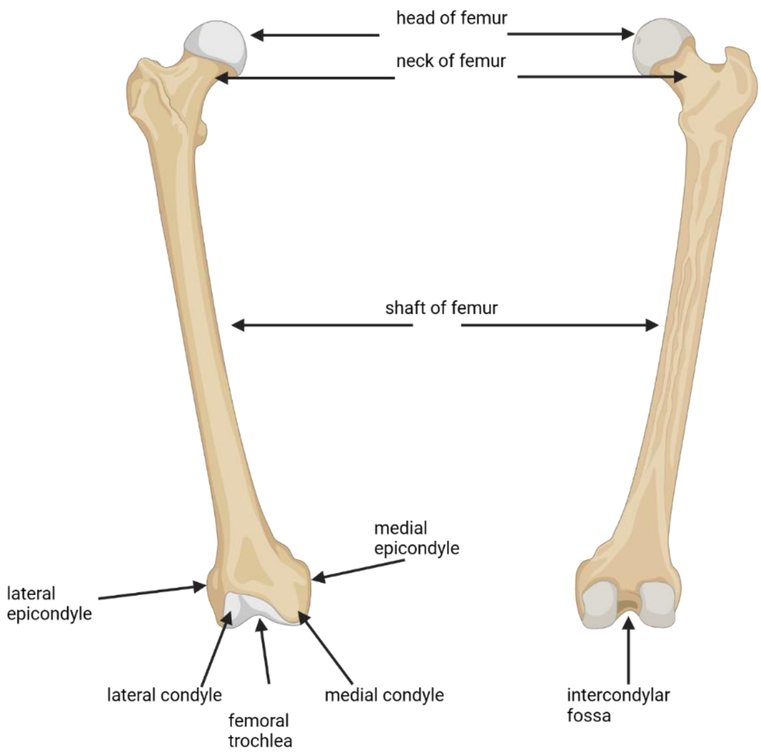


Figure 1: Femur bone

(created with BioRender.com)

Distal to the femur is the tibia (Figure 2). The tibia can also be divided into a proximal part, a shaft and a distal part, which together with the fibula and talus forms the ankle joint. The proximal end of the tibia is thickened into a medial and lateral condyle as a horizontal plateau for the femur. This plateau is covered by a layer of articular cartilage and is referred to as the superior articular surface. Between the two condyles is an area without cartilage, the intercondylar eminence, which serves as the tibial attachment point for the cruciate ligaments (8,10).

The third bone involved in the formation of the knee joint is the patella. The patella is a sesamoid bone, as it is embedded in the tendon of the quadriceps femoris muscle for better transmission of force. The patella is flat and shaped like an upside-down triangle from the ventral side. It narrows from the proximal base to the distal apex form where the patellar ligament extends to the tibial tuberosity. Apart from the apex, the joint surface of the patella is covered with a thick layer of articulate cartilage, which can be up to 7 mm, and articulates with the femoral trochlea. On the articular surface of the patella there is a vertical ridge separating a larger lateral from a smaller medial surface and the odd facet (8).

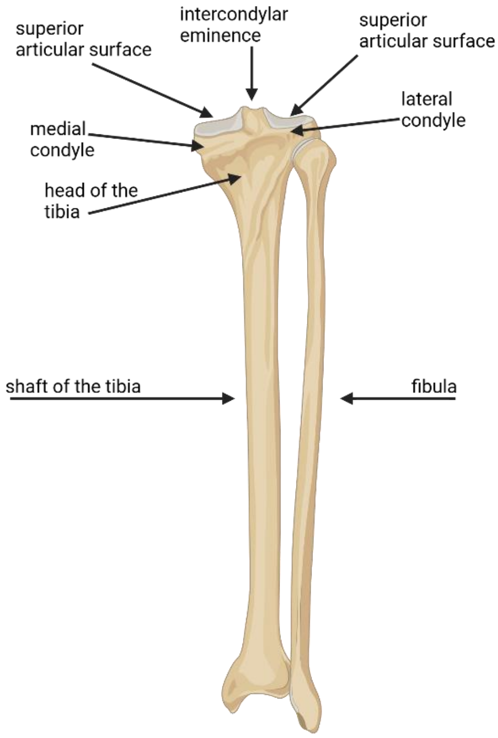


Figure 2: Right tibia and fibula

Seen from the dorsal side (created with BioRender.com)

2.1.2. Soft tissues

The knee joint is surrounded by a joint capsule (Figure 3) with the patella embedded in the capsule together with the quadriceps tendon and the patellar ligament. As with every joint, the capsule consists of an outer fibrous membrane, which is made up of dense connective tissue, and an inner synovial membrane, which produces the synovial fluid. Medially and laterally the fibrous membrane and synovial membrane are situated adjacent to each other. Distal to the patella, the infrapatellar fat pad divides the two layers of the knee joint capsule. Proximal to the patella, the capsule extends under the tendon of the quadriceps femoris muscle and forms the suprapatellar bursa, which allows the joint capsule to unfold during movement and enables the quadriceps tendon to glide almost without friction. On the dorsal side of the knee, the line of attachment of the synovial membrane on the tibial plateau runs anteriorly between the posterior cruciate ligament and the attachment point of the posterior horns of the medial and lateral meniscus. It encircles the anterior intercondylar area and thus the insertion of the anterior cruciate ligament. Therefore, the cruciate ligaments are located between the synovial membrane and the fibrous membrane. At the dorsal side the joint capsule is stabilised by ligaments such as the oblique popliteal ligament and arcuate ligament (8,9).

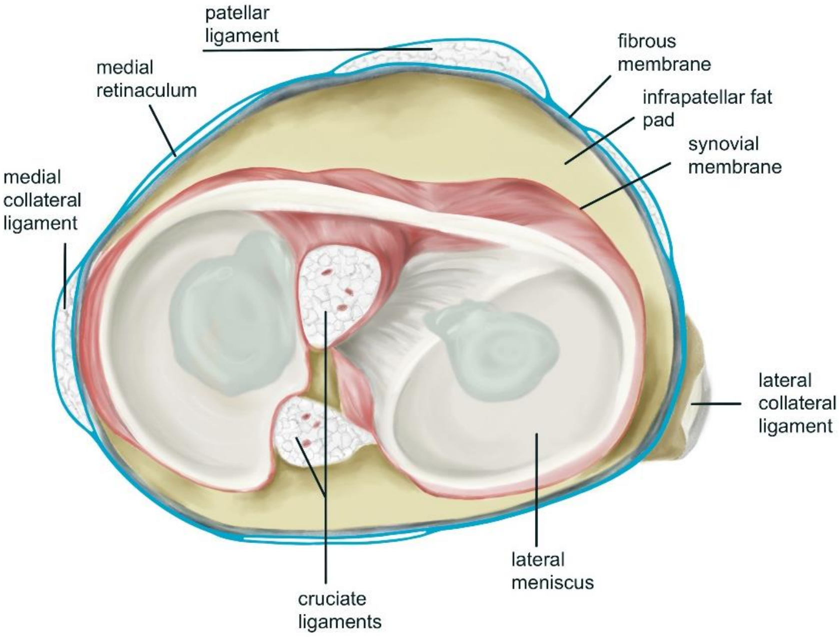


Figure 3: Joint capsule

Right knee after removal of the femur bone in an axial view. Graphic created by Viktoria Sorko upon request (2025), used with permission.

The two menisci are particularly important structures of the knee joint. They mainly consist of fibrocartilage and their primary function is to compensate for the incongruence in the joint surfaces of the femur and tibia. The medial meniscus is slightly narrower than the lateral meniscus and can be described as crescent-shaped or like a laterally open "C" in the horizontal plane. It is connected to the tibia by short taut ligaments that extend from the anterior and posterior horns of the meniscus to the anterior and posterior intercondylar area. In addition, the medial meniscus is connected to the deep portion of the medial collateral ligament, resulting in less freedom of movement than the lateral meniscus and is thus at increased risk of injury. The lateral meniscus, which is also attached to the intercondylar area by its horns, is more curved in top view, resulting in an almost closed circle. The two menisci can be connected at the anterior margins by the transverse meniscomeniscal ligament, which is reported in 55% of subjects (11). In cross-section, the menisci resemble a wedge, with the outer edge of the wedge fused to the joint capsule (12). The meniscus consists of dense connective tissue (fibrocartilage) and the outer edge is well supplied with blood by branches of the popliteal artery. In contrast, the inner 75-90% of the menisci are almost or fully avascular and are supplied by synovial fluid via diffusion (10). Due to the different blood supply, three different zones can be distinguished from the outside to the inside: red-red-zone, red-white-zone, white-white-zone (Figure 4). The red-red zone contains most of the blood vessels, nerves, and fibroblast-like cells. In the white-white zone there are no blood vessels or nerves, the cells are chondrocyte-like. The intermediate red-white zone is the transition from the outermost to the innermost zone, so structures of both areas can be found here (12).

Figure 4: Schematic blood supply of the menisci in a view from the front.

The outer edges of the menisci are well supplied with blood (red-red-zone). The inner parts are hardly supplied with blood (white-white-zone). The transition area between the above-mentioned zones is called the red-white zone (13).

The next important structures that play a large part in the stability of the knee joint are the ligaments. Due to the incongruence of femur and tibia, the bony guidance is not as pronounced as in other large joints in the body (e.g., hip joint). Therefore, the ligaments of the knee joint are very strong. Two major groups of ligaments can be distinguished: inner ligaments and outer ligaments. The inner ligaments include the anterior and posterior cruciate ligaments, as well as the variable transverse meniscomeniscal ligament and the posterior and anterior meniscofemoral ligaments. The external ligaments include the lateral collateral ligament, the medial collateral ligament, the oblique popliteal ligament and arcuate popliteal ligament on the posterior side, and the patellar ligament and retinacula on the anterior side (10). The anterior cruciate ligament (ACL) runs from the medial surface of the lateral femoral condyle to the anterior intercondylar area, following a spatial course from superior posterior lateral to inferior anterior medial. The posterior cruciate ligament (PCL) crosses the anterior cruciate ligament at right angles and runs from the lateral surface of the medial femoral condyle to the posterior intercondylar area, following a spatial course from superior anterior medial to inferior posterior lateral. The cruciate ligaments stabilize the knee joint throughout its range of movement by adjusting tension in different knee positions. Depending on whether the knee is extended or flexed, specific parts of the ligaments become tense, working together to prevent tibial translation (14,15). The stabilization of the knee joint in the sagittal plane is primarily achieved through the cruciate ligaments. This can be seen in the event of a cruciate ligament rupture. With a ruptured ACL, the anterior drawer phenomenon can occur: the tibia can be pulled anterior against the femur on clinical examination. Conversely, if the PCL is ruptured, a posterior drawer phenomenon can occur (8).

The two collateral ligaments prevent the joint from folding up in the frontal plane by tensioning in the extended position of the knee. The medial collateral ligament (MCL) extends from the medial epicondyle of the femur to the medial surface of the tibia below the tibial plateau and consists of both a superficial and a deep component (16). Although it was long thought that the superficial MCL was primarily responsible for knee stability, studies have shown that the deep part of the MCL together with the posterior oblique ligament (POL), which is also part of the medial collateral ligament complex and is located posterior to the MCL, play essential roles in medial knee stability as well (17). The superficial portion of the MCL primarily prevents medial opening of the knee during valgus stress at all knee flexion angles. The POL mainly protects against abnormal internal rotation at all knee flexion angles and serves as a stabilizer against valgus stress in extension and internal rotation. Meanwhile, the deep portion of the MCL helps to prevent external rotation of the tibia, anterior tibial translation and valgus motion, with the meniscofemoral part stabilizing against valgus opening at all flexion angles and the meniscotibial part focused on valgus restraint at 60° of flexion (17). The lateral collateral ligament (LCL) is located between the lateral epicondyle of the femur and the lateral aspect of the head of fibula. In contrast to the more broadly fanned out MCL, the LCL is cord-like and is not fused to the meniscus or capsule because the popliteus muscle and the inferior lateral genicular artery and vein pass between the capsule and the LCL (8,10). The oblique popliteal ligament and arcuate popliteal ligament reinforce the posterior part of the joint capsule and mainly prevent hyperextension of the knee. The strong patellar ligament is located at the front of the knee joint. This ligament is composed of superficial attachment tendons of the quadriceps femoris muscle that pass over the patella and deeper fibrous tracts that run from the apex of the patella to the tibial tuberosity (8). Since numerous muscles attach around the knee joint, multiple bursae are present (e.g., the suprapatellar bursa, the bursa semimembranosa, the infrapatellar bursa) to prevent friction between the tendons and the bone, serving as sliding bearings (9).

2.1.3. Biomechanics of the tibiofemoral joint

Movements around two axes are possible in the tibiofemoral joint: extension and flexion around a transverse axis, as well as internal and external rotation around a longitudinal axis, which is only possibly when the knee is flexed. Functionally, the tibiofemoral joint is therefore a modified hinge joint. The greatest freedom of movement is around the transverse axis with a maximum flexion of about 120°-150° and a maximum extension of about 5°-10° (9,10). Flexion can be significantly limited by various factors, such as soft tissue inhibition or active insufficiency of the hamstrings. External rotation of the knee is possible by a maximum of about 30°-40°, and internal rotation up to 10°. The rotational movements are restricted when the knee is fully extended (9,10).

All the movements in the tibiofemoral joint are possible by the interaction of the specially shaped joint partners, menisci, and the ligaments. First, we look at the movement around the transverse axis. Flexion of the knee is a combination of a rolling movement of the femoral condyles on the tibial plateau in the first 25° and a subsequent rotary-gliding movement. As mentioned above, the radius of curvature of the femoral condyles increases dorsally. This causes the axis of rotation to shift backwards in an arc when the knee is flexed. The imaginary line on which all rotation axes are located is called the evolute (10).

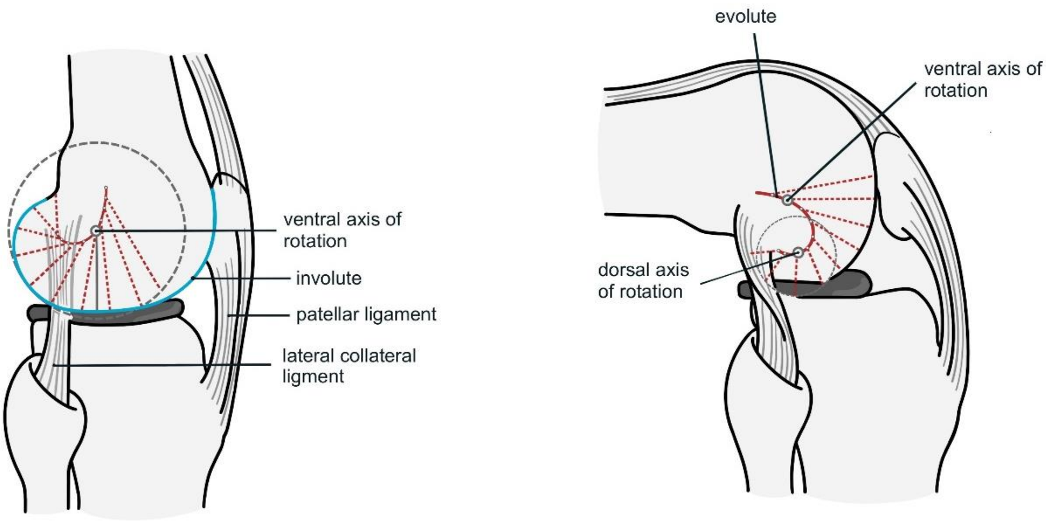


Figure 5: Sagittal section through the femoral condyle.

With increasing flexion, the current axis of movement moves backwards in an arc along a curve. The joint surface, whose curvature increases dorsally, corresponds to the mathematical function of an involute. Graphic created by Viktoria Sorko upon request (2025), used with permission.

The distance of the evolute and thus the distance of the momentary centre of rotation to the joint surface becomes smaller during flexion. In addition, the articular surface of the femur resting on the tibia becomes smaller due to the smaller radius of curvature with increasing flexion. Here, the menisci increase the pressure transmission surface and reduce the pressure acting on the joint surfaces. During the first 25° of flexion, they move backwards with the femoral condyles on the tibial plateau, whereby the lateral meniscus, with its greater ability to move, covers a farther distance. Further dorsal displacement of the femoral condyles is prevented by the anterior cruciate ligament. The collateral ligaments are predominantly relaxed in flexion and tense in extension. In conjunction with the dorsal capsule and the PCL, this prevents further extension of the knee joint, which would be possible due to the anatomy of the joint partners, as well as the opening of the knee in the frontal plane and rotational movements. The second possible movement in the knee joint is around a longitudinal axis. This axis passes through the lateral area of the medial tibial condyle, approximately through the medial intercondylar tuberosity. A rotational movement around this axis is only possible in flexion. During maximum extension, the pull of the ACL causes external rotation of 5-10° the tibia. This terminal rotation, the tense collateral ligaments, POL and the joint capsule prevent rotational movement in extension. In flexion external rotation is possible to a greater extent than internal rotation, as the two cruciate ligaments unwind during this movement. Further external rotation is prevented by the taut collateral ligaments and other structures such as the posterolateral and posteromedial capsule. Conversely, during internal rotation, the cruciate ligaments wrap around each other and thus prevent further rotation (9,10).

2.1.4. Biomechanics of the patellofemoral joint

In the PFJ, the extension movement is particularly important, but other (accompanying) movements of the patella are also possible. The patella is moved by the quadriceps tendon, which is found proximal to the patella as the union of the four quadriceps heads. In contrast to the tibiofemoral joint, the patellofemoral joint has an osseous guidance for the patella through the trochlear groove of the femur (8). The trochlear groove is located between the medial and lateral condyle and deepens posteriorly into the intercondylar fossa. Consistent with the articular surfaces of the patella, the lateral articular surface of the femur is larger as well (18). Given the anatomy, the patella is statically pulled laterally by the quadriceps femoris muscle. Therefore, the lateral condyle extends further ventrally to prevent the patella from dislocating into a lateral direction (19). Normally, the Trochlear Sulcus Angle (TSA), which is best determined in MRI (20), should be around 137 ± 8 degrees (21). If the angle is too large, this is referred to as trochlear dysplasia, which is discussed in a separate chapter. The position of the patella in relation to the trochlea changes during knee movement, which is called patellar tracking. There is no certain consensus on the exact movement of the patella. According to a review by Yu et al. (22), most studies assume that a medial patellar tilt, rotation, and shift occurs at the beginning of flexion and that a lateral tilt, rotation, and shift occurs with further flexion. The term patellar tilt refers to the lateral inclination or alignment of the patella around a longitudinal axis in relation to the femur, while shift refers to the transverse displacement of the patella medially/laterally in relation to the femur. Patellar rotation refers to the rotation of the patella around a sagittal axis. In addition, Yu et al. (22) found that during flexion of the knee joint, patellar flexion (rotation of the patella around the transverse axis) lags behind the tibiofemoral joint by about 30-40%. When moving the knee, the different structures have different influences on the stability of the patella. In an extended position it is mainly the soft tissues (quadriceps, patellar tendon, medial and lateral retinaculum, joint capsule) that hold the patella in position. Therefore, the patella is easiest moved laterally in the extended knee joint. As flexion increases it enters the deeper part of the trochlear groove (8). The stability of the position of the patella is strongly dependent on the structure of the trochlear groove and the patellar articular surface.

As the ligamentous parts of the PFJ are particularly significant for its biomechanics, they will be discussed separately here. The following structures have the most importance to the PFJ as ligamentous stabilizers (Figure 6): medial patellofemoral ligament (MPFL), medial meniscopatellar ligament, medial retinaculum, lateral patellofemoral ligament (LPFL), lateral patellotibial ligament (LPTL), joint capsule, and iliotibial band (ITB) (18,23).

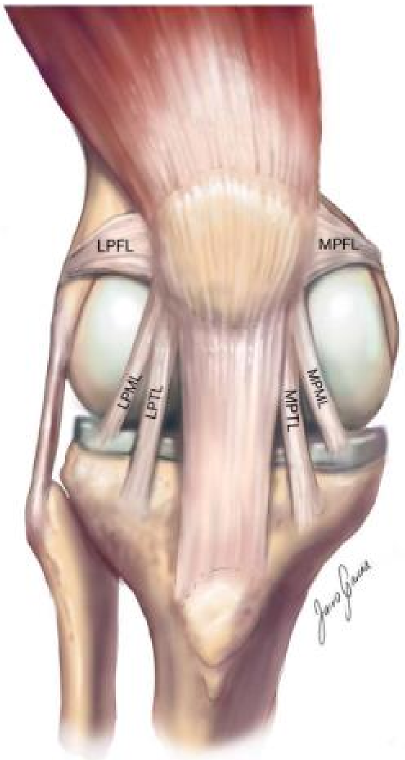


Figure 6: Important ligamentous stabilizers of the PFJ (23)

For a better overview, the medial and lateral structures will be discussed one after the other. According to Siljander et al. (23) the lateral soft structures of the PFJ can be divided into a superficial and deep retinaculum, and the overlying capsule. The layers are referred to as retinacula, as they stabilize the tendons of the extensor muscles of the knee joint. The superficial retinaculum consists of portions of the ITB, the quadriceps aponeurosis, and the vastus lateralis obliquus tendon. The fibre bundles within the superficial retinaculum are interwoven and attach mainly to the superficial and proximal regions of the lateral patella. The deep fibres of the ITB and the quadriceps aponeurosis also radiate into the deep layer of the retinaculum. The deep retinaculum additionally includes the LPFL, the lateral patellomeniscal ligament and the LPTL (23). On the medial side, the MPFL provides stability and is the most important ligamentous structure to prevent lateral displacement of the patella (24). Typically, the origin of the MPFL is between the adductor tubercle and the medial epicondyle of the femur, and the insertion is at the proximal medial half of the patella (25). Also involved in stabilisation are the medial meniscopatellar ligament and the medial retinaculum. The medial retinaculum consists of branches of the tendon of the quadriceps femoris muscle and can also be called the reserve extensor apparatus of the knee (8). It has superficial longitudinal fibres, that interdigitate with the medial collateral ligament and the patellar tendon (24), and deep transverse parts (9). Desio et al. (24) found that in addition to the MPFL, which is the most important structure preventing lateral translation of the patella at 20° of flexion with 60% of the total restraining force, the medial meniscopatellar ligament and the lateral retinaculum also prevent lateral translation of the patella with 13% and 10% of the total restraining force (Table *1*).

Table 1: Percentage contribution of soft tissues to the total restraining force during maximal patellar translation at 20° knee flexion

	Lateral retinaculum	MPFL	Medial retinaculum	Medial patellotibial ligament	Medial meniscopatellar ligament
Mean contribution	10%	60%	3%	3%	13%

Source: Adapted from Desio et al. (24)

In addition to the bony guidance by the trochlea and the ligaments, muscles (quadriceps muscle, pes anserine muscle group, biceps femoris muscle) also contribute to keeping the patella in position. In this context, the Q-angle must be mentioned. This angle is defined as follows: "The Q-angle is the angle between the line of pull of the quadriceps (anterior superior iliac spine to mid-patella) and a line connecting the centre of patella with tibial tuberosity (18)". An increased Q-angle (normal: males 10-13 degrees; females: 15-17 degrees (18)) leads to a stronger lateral pull on the patella due to the bowstring effect. Tightening the quadriceps muscle also creates a force vector that attempts to move the patella laterally (19). The vastus medialis obliquus (VMO) muscle is the only muscle that counteracts this lateral force vector. The VMO is a distally located, functionally separate part of the vastus medialis muscle (a head of the quadriceps femoris muscle). It originates from the tendon of the adductor magnus muscle and attaches to the medial side of the patella with oblique fibres (26).

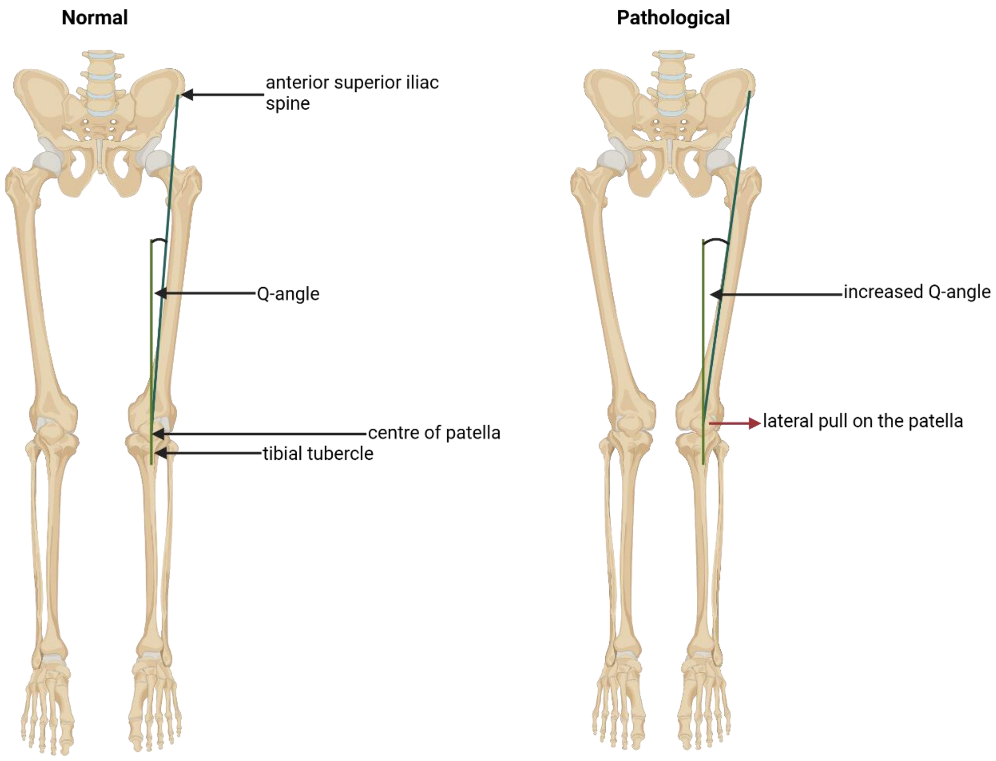


Figure 7: Q-angle

(created with BioRender.com)

As mentioned above, the patella is embedded in the attachment tendon of the quadriceps femoris muscle and thus fulfils several functions. On the one hand, it serves as a hypomochlion for the quadriceps tendon (Figure 8), thereby lengthening its distance from the transverse axis of the knee joint, which results in a significantly better transmission of force due to the larger virtual lever (9).

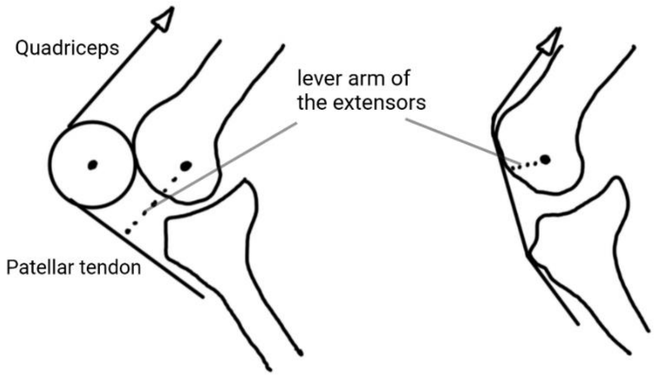


Figure 8: The patella as a hypomochlion

The patella lengthens the virtual lever arm of the quadriceps femoris muscle, almost doubling its torque (9)

Huberti et al. (27) highlight that the patella plays a critical role in the last 30 degrees of knee extension. The patella contributes 31% of the total knee extension torque when the knee is fully extended, but only 13% between 90 and 120 degrees of flexion. This means that when the patella is removed (patellectomy), significantly more tension must be produced by the muscles for the same torque in extension. According to a cadaver study by Kaufer (28), the quadriceps muscle would have to exert 1.3 times the force in this case to allow full extension in the knee joint. Mechanically, the quadriceps tendon and the patellar ligament can be seen as two levers in the movement of the knee joint (Figure 9). Depending on the position of the knee, the forces that must be applied by the two lever-arms change. This is due to the changing position of the patella during movement. In flexion, the patella is more distal and the contact surface with the femur shifts to the proximal pole of the patella. This lengthens the virtual lever arm of the patellar ligament, and it must apply less force. In extension, on the other hand, the lever arm of the quadriceps tendon lengthens because the contact surface of the patella is now more distal. This means that from 0 to 30 degrees of flexion, a greater force is applied in the patellar ligament (force ratio between force in the patellar ligament and force in the quadriceps tendon >1), and with further flexion, a greater force is applied in the quadriceps tendon (force ratio <1) (29). This consideration also leads to the assumption that a shorter patellar ligament is exposed to a higher load with the same applied force (30).

As a second important role, the patella protects the anterior surface of the distal head of the femur and reduces friction between the patellar tendon and the trochlea (18). The thick cartilage layer of the patella allows the patella to slide almost frictionlessly on the trochlea (29).

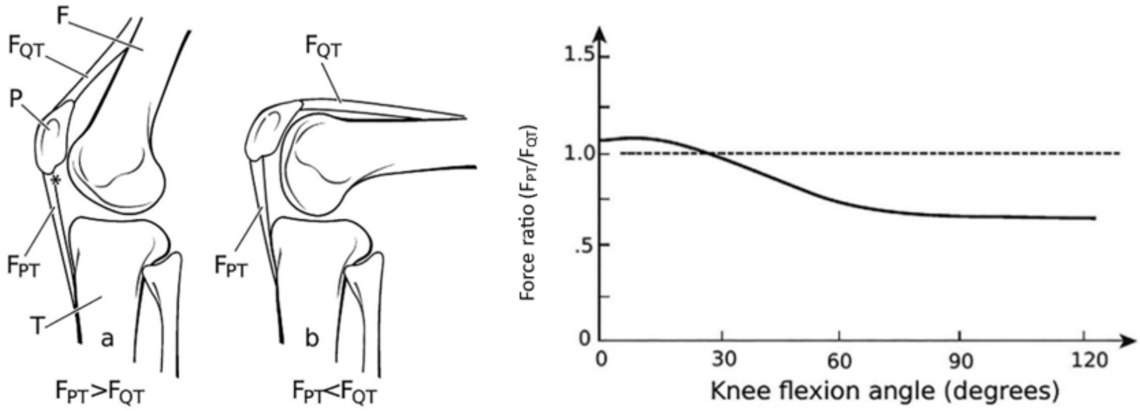


Figure 9: Patellar tendon to quadriceps tendon force ratio depending on extent of flexion.

The figure also shows the movement of the patella, (a) in extension the distal pole of the patella articulates with the femur and in (b) flexion the proximal articular surface of the patella articulates with the femur. F=Femur, P=Patella, T=Tibia, F_{QT}=force in quadriceps tendon, F_{PT}=force in patellar tendon (29)

2.2. Development of the Knee Joint

During the early embryonic development, the gastrula is formed during gastrulation in about the third and fourth week. The gastrula consists of three layers: endoderm, mesoderm and ectoderm. All structures of the human body are later derived from these three germ layers. The bones of the extremities and the skeletal muscles emerge from the mesoderm after further differentiation steps (31). These differentiation steps from a preliminary stage to a developed joint, which already resembles the adult joint, take place in a few days to weeks. The most important period for the development of the extremities is 3 to 6 weeks after fertilisation. The lower limb bud is first found 4 weeks after fertilisation. A few days later, chondrification of the skeleton of the lower limb begins. This occurs from proximal to distal, i.e. from femur to tibia and fibula. 7 weeks after fertilisation, the initially mostly blastemal femoral and tibial condyles can already be distinguished. At this stage, a blastemal homogeneous intervening zone is found between the two bones. The patella can also be recognised histologically as cellular condensation 7 weeks after fertilisation (32). Macroscopically, the quadriceps tendon, patellar ligament and patella can be seen as a continuous band of fibrous connective tissue up to this point (33). In some cases, chondrification of the later patella already begins at this stage and a hyaline cartilage mass can be visible. Simultaneously with the chondrification of the patella by hyaline cartilage, the patellar ligament is formed by a fibrocartilaginous transition distal to the patella. During the eighth postovulatory week, the knee joint is almost mature in shape and resembles the knee joint of an adult. Other structures such as the collateral ligaments, cruciate ligaments, popliteal tendon, menisci, and retinacula can also be identified (32). At 12 weeks post-fertilisation, the patella is entirely composed of cartilage and interrupts the continuity of the quadriceps band. At the beginning, the lateral and medial patellar surfaces are approximately the same size, because the patella and femur are still connected. From the 23rd week of pregnancy, after the patella has separated from the femur, a larger lateral facet can be seen, as in adults (33,34). As the patella develops later than the femur, it is initially small compared to the distal end of the femur. To balance this difference, until the sixth month of pregnancy, the patella grows relatively faster than the femur. From this point on, the size ratios of the bones remain constant until birth (34). Ossification of the patella normally begins at an age of 2 to 3 years; at this point the first ossification centres can be identified in the form of multiple loci (35). The transformation from cartilage to bone then proceeds from the centre to the edges of the patella. Periosteum subsequently develops on the ventral surface of the patella, while articular cartilage remains on the superior two-thirds of the articular surface. The lower one-third of the articular surface of the patella does not require hyaline cartilage as it is covered by a fat pad and does not articulate with the femur (35). Over time, the anterior, medial and lateral cortical bone fuses with fibres of the quadriceps tendon and a stable connection is formed between the subchondral bone and the tendon (33). Until further osseous maturation at the age of 10 to 12 years, the structure of the articulating surface of the patella is not yet determined by the subchondral plate, but by the cartilage itself (35).

Like most bones in the human body, the femur and tibia are formed by chondral ossification. This means that via the intermediate step of the initially cartilaginous skeleton made of mesenchyme, immature woven bone is formed, which is then secondarily remodelled into mature lamellar bone. The diaphyses of the long bones already begin to ossify during the embryonic period, while the epiphyses usually do not ossify until after birth. Exceptions are the distal femoral epiphysis (ossification prenatally) and the proximal tibial epiphysis (perinatally). The epiphyseal plate, which is located between the epiphysis and diaphysis, is important for length growth. This area does not ossify until length growth is complete, and cartilage cells remain present (36).

2.3. Trochlear Dysplasia

As mentioned above, the femoral trochlea is the surface which articulates with the patella. The shape of this osseous structure, along with functioning soft tissue structures, is therefore crucial for stability in the PFJ. An abnormal change in the morphology of the trochlea is called trochlear dysplasia and is a predisposing factor for patellar instability and the main risk factor for patellar dislocation (37). Several studies have shown that up to 85 % of patients with recurrent patellar instability have trochlear dysplasia (4–6). Trochlear dysplasia is morphologically characterised by a hypoplastic medial femoral condyle; therefore, the trochlear sulcus is displaced medially, and the slope of the lateral femoral condyle is flattened. The proximal trochlear sulcus can also be flat or even convex (38). In addition to the increased risk of patellar dislocation, trochlear dysplasia can lead to long-term degeneration of the PFJ, as a smaller patellofemoral contact surface results in increased pressure on the patellofemoral cartilage and damage to the articular cartilage (39).

Little is known about the epidemiology and the true prevalence of trochlear dysplasia in the general population. In an article by DeVries et al. (40), the prevalence of high-grade trochlear dysplasia in the general population is reported to be around 10 %. Onor Jr et al. (41) found patients with moderate to severe dysplasia in approximately 17 %. It also isn't fully cleared how trochlear dysplasia exactly develops; primary (genetic) and secondary (acquired) causes are discussed in the literature. A genetic cause is suggested by the fact that the morphology of the trochlea is already determined early in embryonic development and is almost maintained during further growth. In a retrospective analysis using MRI, Parikh et al. (4) showed that a dysplastic trochlea retains its abnormal shape during growth.

Trochlear dysplasia can be detected and evaluated using various imaging techniques, most notably a combination of X-ray and MRI or CT. According to a review by Saccomanno et al. (42) published in 2023, trochlear dysplasia can be reliably assessed using at least 3 parameters: Trochlear Depth (TD), Trochlear Sulcus Angle and Dejour's classification.

The TD can only be reliably determined by CT and MRI (Figure 10). It is determined by placing a tangent to the posterior condyles and then adding the arithmetic mean of the height of the lateral facet and the medial facet and subtracting the sulcus height from this (43). The trochlear depth therefore indicates the depth of the trochlear groove in relation to the femoral condyles.

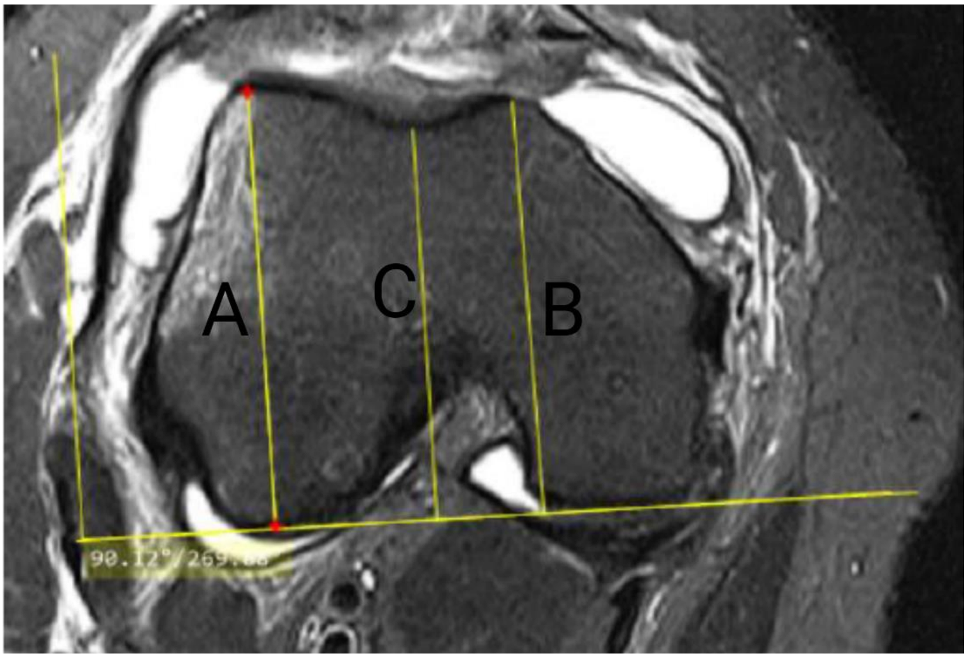


Figure 10: Axial MRI of the knee.

TD = (A+B)/2 – C; A = height of the lateral facet, B = height of the medial facet, C = sulcus height

The second parameter used to define trochlear dysplasia as mentioned above is the TSA (Figure 12), which is defined as the angle between the most anterior margins of the lateral and medial facet and the deepest point of the intercondylar groove measured on an axial slice at the level where the femoral epicondyles are most widely separated on the medial to lateral axis (44). It can be reliably measured on radiography, CT and MRI (42).

The Dejour classification is the most common classification of trochlear dysplasia. It is based on a lateral image (X-ray, CT, MRI) and an axial CT or MRI scan of the knee joint and categorises types A-D (45). Decisive for the classification into Dejour A, B, C and D is the presence of three radiographic parameters in a lateral image of the knee joint: crossing sign, supratrochlear spur and double contour sign (45). The crossing sign is seen when the line of the trochlear groove crosses the anterior border of one of the two condyles in a lateral radiograph and is thus indicative of a flat proximal trochlea (46). In a study from 2012, Donaldson et al. (47) showed that the crossing sign can be found in almost all patients with trochlear dysplasia with a sensitivity of 94% (specificity of 56%). A supratrochlear spur is a bony thickening on the anterior cortex of the distal femur above the trochlea, which also indicates a structural abnormality of the knee joint and is often associated with trochlear dysplasia (48). In the double contour sign, a second line of the hypoplastic medial femoral condyle can be seen posterior to the lateral facet instead of the single line normally visible due to the overlap of the anterior cortices of the two femoral condyles (45). Based on these criteria and an additive axial X-ray the Dejour classification is divided as follows:

- Type A: crossing sign, fairly shallow trochlea, >145°.
- Type B: crossing sign, supratrochlear spur, flat or convex trochlea.
- Type C: crossing sign, double contour.
- Type D: crossing sign, supratrochlear spur, double contour, asymmetry of trochlear facets, cliff pattern between medial and lateral facet (41).

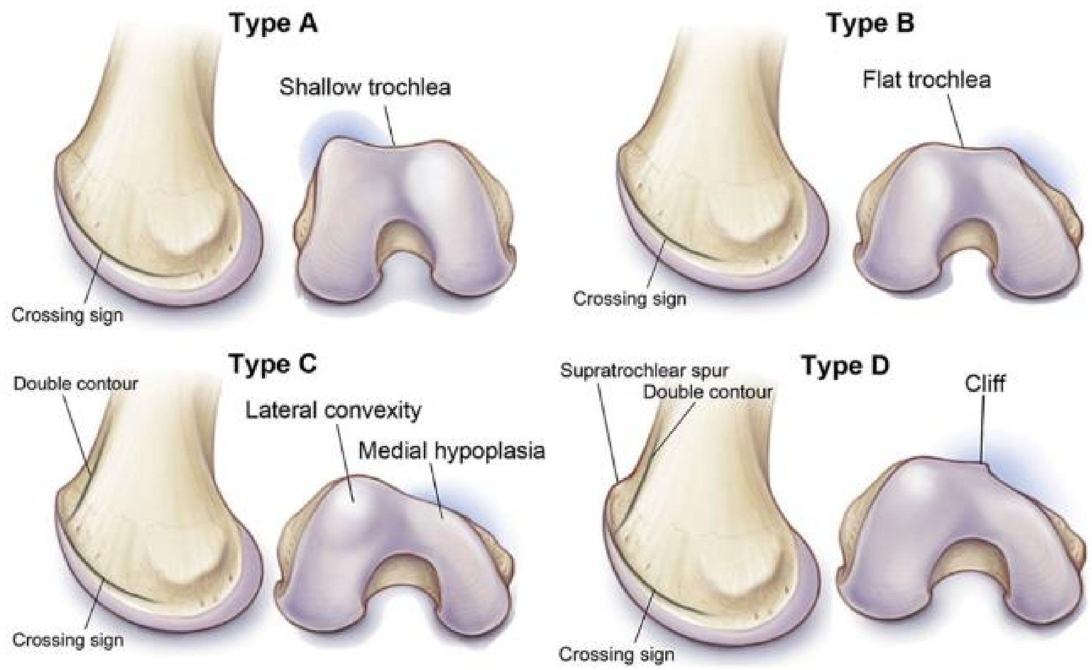


Figure 11: Dejour classification based on trochlear osseous morphology (41)

The Dejour classification from A to D does not necessarily mean an increasing severity of the extent of trochlear dysplasia and is controversially discussed in the literature. Some authors classify types B and D, where a supratrochlear spur is found, as high-grade and types A and C as low-grade dysplasia (49,50). This subdivision correlates with the therapy recommendations, as surgical treatment is recommended for Dejour types B and D with a supratrochlear bump >5mm and clinical symptoms. The Dejour classification is also criticised because it divides dysplasia purely according to morphological criteria and does not necessarily result in a therapeutic consequence, as the clinical aspect must also be considered. For example, trochlear dysplasia can often be difficult to categorise using the Dejour classification or can already be classified as high-grade dysplasia, even though there are no clinical abnormalities (38). Furthermore, there is disagreement as to whether the Dejour classification has sufficiently high inter- and intraobserver reliability and thus may not be a reliable method for quantifying trochlear dysplasia (51). According to the authors and a study by Roy et al. (52), the Oswestry-Bristol Classification recently proposed by Sharma et al. (53) should have a higher intraobserver reliability with similar interobserver reliability compared to the Dejour classification. Another recently proposed MRI-based classification system by Dejour et al. (54) for trochlear dysplasia aims to improve objectivity and reproducibility by relying solely on quantitative measurements. This classification defines trochlear dysplasia using cutoffs for TSA (≥157°) and LTIA (<14°), which were shown in the study to be optimal for discriminating between patellar instability and controls. The combination 'sulcus angle ≥157° OR LTIA <14°' showed a sensitivity of 87% for the diagnosis of PFI. The adapted Dejour classification proposed by the authors is:

- Type 0 = no dysplasia: (sulcus angle <157° AND LTI ≥ 14°)
- Type 1 = low-grade dysplasia: (sulcus angle ≥ 157° OR LTI < 14°) AND central bump <5mm
- Type 2 = moderate-grade dysplasia: (sulcus angle OR LTI are 'unmeasurable') AND central bump <5mm
- Type 3 = high-grade dysplasia: (sulcus angle ≥ 157° OR 'unmeasurable' OR LTI < 14° OR 'unmeasurable') AND central bump ≥5mm.

Due to the described disadvantages of the standard Dejour classification, the determination of the Lateral Trochlear Inclination Angle (LTIA) is described in the literature for a better assessment of trochlear morphology and as a reliable parameter for PFI (55). The LTIA is determined using an axial MRI. The level of the most proximal trochlear cartilage is determined in the sagittal section and then a parallel line to the lateral trochlear facet and a line to the posterior femoral condyles is drawn at this level in the axial MRI (Figure 12). The angle between these two lines is the LTIA (56). In the original paper on LTIA by Carrillon et al. (55), the authors state a 95% specificity for PFI due to trochlear dysplasia with an LTIA <11°.

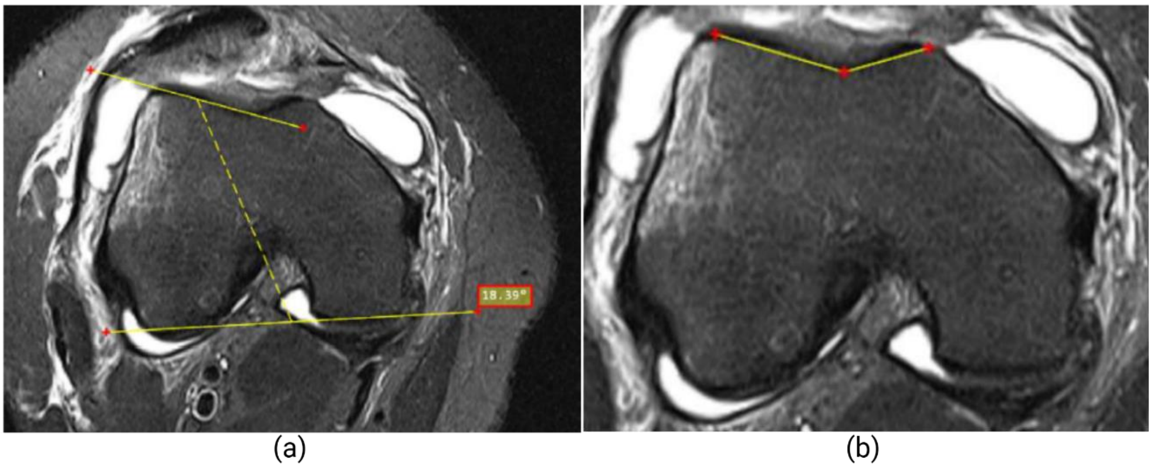


Figure 12: Axial MRI of a right knee.

(a) LTIA is measured between a line to the posterior femoral condyles and a parallel line to the lateral trochlear facet. (b) TSA is measured between the most anterior margins of the lateral and medial facet and the deepest point of the intercondylar groove

The treatment of choice for clinically relevant trochlear dysplasia is trochleoplasty. The indications for trochleoplasty are not entirely standardised, as not only the morphology of the trochlea is decisive, but a comprehensive picture of the patient including all risk factors for PFI (e.g. patella alta, increased TT-TG) is necessary. A patella alta or an elevated tibial tuberosity to trochlear groove distance (TT-TG) can also be corrected during trochleoplasty. Nevertheless, types B and D according to the Dejour classification with a supratrochlear bump of more than 5 mm and recurrent instability are recommended indications for surgery (38). In addition, a trochlear depth of less than 3 mm and the presence of a J-sign are mentioned as signs in favour of surgery (38,57). The J-sign can be detected in the clinical examination and is typical of significant trochlear dysplasia. It describes the path travelled by the maltracking patella during the transition from extension to flexion. This path resembles an upside-down J, as the patella is subluxated laterally in extension and suddenly moves medially in flexion (58). Three different trochleoplasty techniques are used: lateral facet elevation and lateral lengthening osteotomy, sulcus deepening techniques and recession wedge osteotomy (38).

According to studies, trochleoplasty can achieve good results with low dislocation rates when used for the right indication (59,60). It should be noted that the various procedures are challenging to perform and require an experienced surgeon.

2.4. Patellar Tilt

A second known risk factor for PFI, which is of importance for this diploma thesis is the patellar tilt angle. The patellar tilt angle indicates the tilt of the patella in the horizontal plane. The patellar tilt can be determined using two lines in an axial image of the knee: a tangential line to the posterior edges of the femoral condyles and a line through the transverse axis of the patella at its widest point (Figure 13). The angle between these two lines is the Patellar Tilt Angle or Patellar Inclination Angle (7).

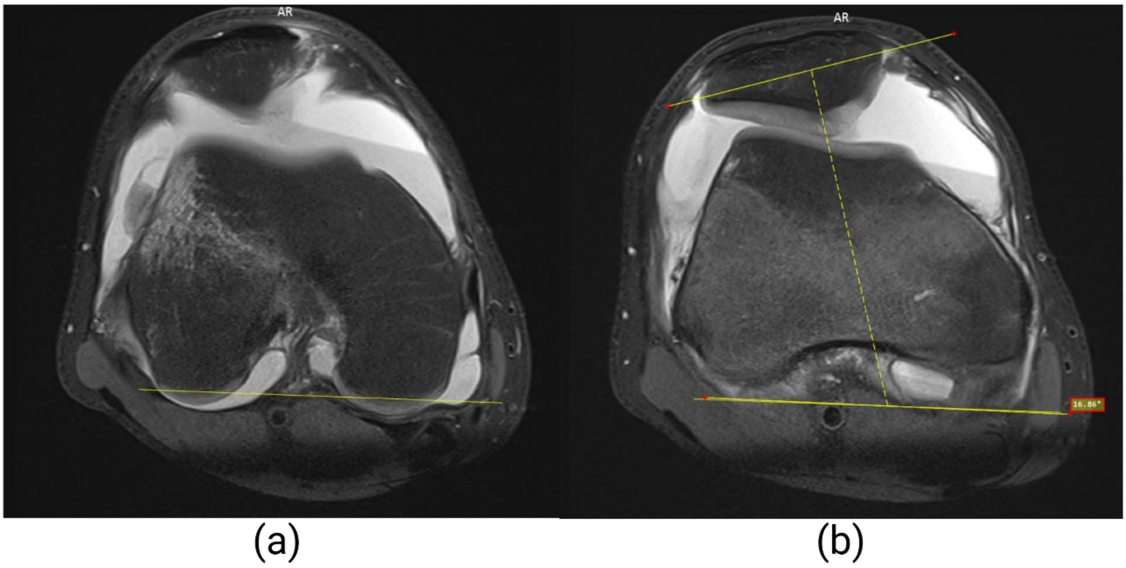


Figure 13: Patellar Inclination Angle

Measured in an axial MRI between a (a) tangential line to the posterior edges of the femoral condyles and (b) a line through the transverse axis of the patella. A positive angle opens up medially.

Whether a patellar tilt is present can also be assessed in a clinical examination. Grelsamer et al. (61) showed that if a tilt is present in the clinical examination of the knee joint, a patellar tilt of 10° or more can also be expected in an MRI. To determine whether a tilt is present in the clinical examination, the medial and lateral borders of the patella are palpated, and an imaginary line is drawn between these two points. Normally, this line should lie in the frontal plane. If the lateral edge of the patella is further dorsal than the medial edge, a patellar tilt can be assumed. This method is used for a quick and practical assessment of the patella alignment; imaging is required for a quantitative assessment of the tilt.

An increased patellar tilt is considered a risk factor for patellar instability. In 2013 Charles et al. (62) examined morphological differences between normal knees and those with PFI in MRI images and were able to show that the two groups differ significantly in terms of patellar tilt and that tilt is therefore an excellent measurement for distinguishing between control subjects and those with instability. With an interobserver reliability of 0,957 in a study by Gobbi (63) et al. and an intraobserver reliability of 0,953 in a study by Yu et al. (64), the patella tilt angle is a very good parameter for the assessment of PFI. Gobbi et al. (63) specified a cut-off value for the patella tilt angle of 15.9° for PFI, while Dejour et al. (65) suggested a cut-off value of 20°. In their study, Gobbi et al. found a mean patellar tilt of 22.9° in 142 patients with PFI and a mean of 10.4° in a control group of 181 people. These values also correspond with the results of other studies (62,64).

2.5. Patellar Instability

Patellar instability is abnormal lateral movement of the patella in the femoral groove during flexion or extension, either subluxation or complete dislocation. As described in chapter 2.1.4, normal movement of the patella requires an interplay of bony guidance by the trochlear groove and soft tissue structures. These factors must compensate for the displacing force exerted on the patella by the Q-angle of the extensor apparatus. If the osseous or ligamentous resistance is insufficient or the force exerted is too high, lateral patellar dislocation can occur. Patellar instability can be classified based on its underlying cause into two main categories: traumatic and atraumatic instability. Traumatic patellar instability typically results from direct injury or acute trauma and often results in complete dislocation of the patella, while atraumatic instability occurs without significant trauma and is often associated with congenital or acquired anatomical abnormalities. It typically only results in a subluxation (66).

2.5.1. Epidemiology

Patella dislocation accounts for 2-3% of all knee injuries. However, during maturation, patellar dislocation is the most common cause of acute knee injury, with an incidence of about 29-50 per 100,000 in the group of 10- to 17-year-olds (1,2).

Females in this age group have a higher risk for acute patellar dislocation than males (1). Individuals with a history of PFI are therefore more likely to be female and have a higher risk of further dislocation than patients who experience a dislocation for the first time. The literature describes a recurrent dislocation rate of up to 40% after initial dislocation (67). Numerous bilateral anatomical characteristics in the knees predispose the contralateral knee to similar instability as the initially affected knee. However, the contralateral knee dislocates at a considerably lower rate, estimated between 5% and 11% (68,69).

2.5.2. Classification and Aetiology

Patellar instability can be classified according to various aspects. The most clinically relevant classifications are based on aetiology and direction of dislocation. Aetiologically, acute traumatic, acute dispositional, recurrent, habitual, chronic, congenital and iatrogenic patellar dislocation can be distinguished. The patella most often dislocates laterally; a medial dislocation can occur due to an iatrogenic cause. Iatrogenic causes are rare, but can happen after treatment of lateral instability, e.g. after a lateral release. In this procedure, the lateral retinaculum is surgically split to allow better guidance of the patella in the trochlear groove if the capsule is too tight (70). Isolated use of this method, which used to be a standard procedure, is no longer recommended as the stability of the patella is negatively affected and it can result in a medial patellar dislocation (3). There are also vertical and intra-articular dislocations, both are very rare.

Acute instability (first dislocation) can be triggered by direct trauma, such as a blow or fall. However, acute dislocations caused by pathoanatomical factors are far more common in adolescents and have an increased risk of recurrent patellar dislocation (71). Predisposing factors are trochlear dysplasia, patella alta, increased patellar tilt, increased TT-TG (e.g. by a lateralised tibial tuberosity), genu valgum, increased tibial external torsion, increased femoral anteversion, hypoplasia of the vastus medialis muscle, chronic pathological laxity of the ligamentous guidance of the patella and tight lateral ligamentous apparatus (3). These risk factors are explained in more detail below. Femoral anteversion and increased tibial external torsion are collectively referred to as “miserable malalignment syndrome”, as they increase the Q-angle and thus contribute to patellar instability. Knee alignment is critical to PFJ stability (72,73). Physiologically, the centre of the knee joint should be exactly on the line between the centre of the hip joint and the ankle joint (=Mikulicz line) (10). The angle measured on the outside between the shaft axes of the thigh and lower leg should be around 174° (74). If this angle is smaller, it is a valgus deformity, the centre of the knee is medial to the Mikulicz line, and the patella is pulled laterally. A femoral anteversion of more than 20 degrees leads to an increased Q-angle and thus an increased force vector of the quadriceps muscle, which pulls the kneecap laterally and away from the physiological position when tensed (72). In trochlear dysplasia, as mentioned in chapter 2.3. the shallow trochlear groove leads to reduced osseous guidance and instability during flexion of the knee. A patella alta, an abnormally elevated patella in relation to the femur, results in late adequate contact of the patella with the intercondylar sulcus (75). A larger TT-TG means that the tuberosity is positioned more laterally in relation to the trochlea and is therefore associated with patellar instability (65,76,77). The TT-TG distance can be determined using axial CT or MRI images. As described in chapter 2.1.4, the vastus medialis obliquus muscle is the only muscle that counteracts the lateral force vector through the remaining quadriceps parts. Hypoplasia of the VMO can therefore also favour patellar instability.

2.5.3. Associated injuries

Complications or associated injuries in a patellar dislocation are very common and can occur acutely as part of the dislocation or develop over the course of the disease (78). Acute complications include injuries to the MPFL, chondral and osteochondral flake fractures, bone bruises of the medial patella and the lateral condyle of the femur and, more rarely, injuries to the ligamentous structures and menisci, although associated meniscal tears can be seen more often in females than in males (3,79). As lateral dislocation is most common, most associated injuries are to the medial ligamentous apparatus, the medial patellar margin and the lateral femoral condyle. Sillanpää et al. (80) showed that in a group of male military conscripts with first-time traumatic patellar dislocation, all presented with hemarthrosis and all patients who underwent MRI or surgery had a ruptured MPFL. In most cases of traumatic patellar dislocation, the MPFL tears at the patellar attachment (79). In a flake fracture, a chondral fragment is torn out or an osteochondral fragment is broken off from the joint surface, while the posterior surface of the patella slides over the lateral femoral condyle during lateral dislocation. This injury is found in around a quarter of cases as an associated injury in traumatic patellar dislocations (3). The more frequently the patella dislocates, the higher the prevalence of chondral lesions. Nomura et al. (81) found cartilage defects in 96% of patients with chronic lateral patellar dislocation during arthroscopy. In the presence of an osteochondral flake fracture, surgical treatment with removal or fixation of the fragment is always indicated to avoid further consequential damage (3).

Late complications include recurrent dislocations and chronic instability, cartilage damage and the development of osteoarthritis in the patellofemoral joint. Recurrent dislocations after an inaugural traumatic dislocation occur in up to 40% of patients and are promoted by the reduced medial stabilization after an injury to the medial ligament apparatus. Surgical treatment can reduce the risk of recurrent dislocation to 0-31% but entails the typical risks of surgery and does not necessarily lead to a better clinical outcome compared to conservative therapy (3,67,82). Therefore, the individual risk of recurrent dislocation should be assessed before indicating surgery (3). Patellofemoral osteoarthritis is a late complication after patellar dislocation, caused by chronic overload and cartilage damage in the patellofemoral joint because of repeated dislocations of the patella or inadequately healed injuries. In a study of 609 patients with initial patellar dislocation, Sanders et al. (83) found that after 25 years almost 50% of patients showed clinical and radiological signs of osteoarthritis, while this was only the case in 8.3% of patients in the control group. Salonen et al. (84) were able to show that not only repeated lateral patellar dislocations lead to degeneration of the cartilage in the knee joint, but also a first traumatic dislocation can lead to progressive degeneration of the patellofemoral cartilage and thus also leads to osteoarthritis of the knee joint. While the association between traumatic patellar dislocations and the development of osteoarthritis in adults is clear, the long-term effects of traumatic patellar dislocations in children have not yet been adequately addressed in the literature (85). Here, too, changes in the quality of the cartilage can occur, leading to premature degeneration of the joint. A study by Vollnberg et al. (86), which examined a patient group with a range of 11-56 years (average age 26 years), was able to show that osteoarthritis can be present at an early age. In MRI images of the group of patients, signs of mild to moderate osteoarthritis were found in more than half of them. They also compared the effects on the cartilage of a first-time dislocation, recurrent dislocations (2-9 dislocations) and chronic dislocations (more than 10 dislocations). In patients with first-time dislocation and recurrent dislocation, they mainly found osteochondral defects on the medial patellar facet and damage to the cartilage of the central dome of the patella, while in chronic dislocators, additional severe defects on the lateral patellar facet and on the trochlear cartilage were found. They were even able to establish a correlation between the extent of the cartilage defect and the severity of the osteoarthritis and the number of patellar dislocations. Nevertheless, further research is needed to understand the exact mechanisms of cartilage changes and long-term changes in the function of the PFJ in children, as this study also did not exclusively analyse a paediatric population.

Both conservative and surgical treatment of patellar dislocation increase the risk of patellofemoral osteoarthritis (3). However, the extent of the difference between the two treatment options in terms of the risk of osteoarthritis has not been fully clarified. A meta-analysis by Smith et al. (87) concluded that although surgical treatment seems to significantly increase the risk of osteoarthritis compared to conservative treatment, it significantly reduces the risk of re-dislocation. However, according to the authors of the meta-analysis these results should be viewed with caution due to potential biases and inconsistencies in the studies and various surgical methods used in the studies.

2.5.4. Diagnostics

In addition to a detailed medical history, the diagnosis of patellar dislocation always includes a clinical examination, an X-ray examination and magnetic resonance imaging. Optionally, in special cases and if a therapeutic consequence is planned, a diagnostic arthroscopy and, if a fracture is suspected, a CT scan can be performed (3). If there are indications of rotational deformities of the femur or tibia that are the cause of the PFI, a rotational CT/MRI can also be performed. In the clinical examination, acute patellar luxation is clearly recognisable on inspection due to the abnormal lateral position of the patella in relation to the knee joint. However, comparison with the opposite side is essential. Swelling of the knee joint and hemarthrosis are common. Visible atrophy of the vastus medialis muscle can be indicative of the aetiology, as this can lead to weaker dynamic medial stabilisation and therefore an increased risk of dislocation. Palpation should be used to assess the displacement of the patella. A positive patella tap test indicates an effusion or hemarthrosis, for which patellar dislocation is the second most common traumatic cause (88). There may also be palpable gaps medial or lateral to the patella, which are typically found in a rupture of the surrounding ligamentous structures. If there is pressure pain over the lateral femoral condyle and the medial patellar facet, a possible flake fracture must be considered. The apprehension test is a test used to detect a dislocation tendency in the event of a former patellar dislocation. It consists of carefully moving the patella laterally with the knee joint flexed to 20-30°. The apprehension test is positive if there is a protective tension in the quadriceps muscles or the patient shows a facial expression of fear. To obtain more precise information on the dislocation tendency of the patella, the apprehension test can also be carried out at 0°, 30°, 60° and 90°. A tendency to dislocate at 60° and 90° flexion indicates the presence of a bony cause, as in this position the patella slides deeper into the trochlear groove of the femur and should be held in position by it. Colatruglio et al. (89) found in a clinical study that 90% of the patients with persistent patellar apprehension over 60° flexion of the knee had significant patella alta and/or trochlear dysplasia. The patellar apprehension test is often not performed in an acute situation and should only be carried out with the utmost caution, as dislocation may occur. The J-sign is a further inspectorial sign of PFI. The patient is asked to fully extend the knee joint from a 90° flexed position. If a sudden lateralisation of the patella occurs during extension, the J sign is considered positive. In this case, the patella moves along an imaginary line that resembles an upside-down J.

In addition to these specific tests, which are indicative of PFI, a general examination of the knee joint should also be carried out to check mobility, ligament stability and possible hyperlaxity (3).

2.5.5. Treatment

In the case of an acute dislocation of the patella, the reposition should always be performed first after the clinical examination. Only after successfully reducing the dislocation further imaging should be performed (3). Treatment of a lateral patellar dislocation can be conservative or surgical. The question of whether a patellar dislocation should be treated conservatively or surgically has been the subject of controversial debates in the literature in recent years (82,90). A review by Smith et al. (67) published in the Cochrane Database in January 2023 was also unable to clearly clarify which procedure is preferable, as the certainty of the evidence from the studies was very low. However, a meta-analysis from 2015, which examined the outcome after acute patellar dislocation especially in children and adolescents, concluded that surgical treatment of a first-time patellar dislocation is associated with a lower risk of recurrent dislocation and a better functional outcome (91). At the same time, however, the risk of patellofemoral osteoarthritis may be increased compared to the conservative approach (87).

According to the current guideline of the German Society for Trauma Surgery (3) conservative treatment is indicated in the case of a traumatic initial dislocation without an osteochondral flake after weighing up the individual risk of a re-dislocation. A conservative approach should also be the primary aim in the event skeletally immature patients. The conservative procedure after a successful reposition consists of general acute measures such as immobilisation in a leg cylinder cast for a few days, elevation, cooling, use of crutches, analgesia and thrombosis prophylaxis. An orthosis with limited flexion is then fitted for 6 weeks. Functional follow-up treatment with physiotherapy and strengthening of the vastus medialis muscle should also be part of the conservative treatment plan (3).

Surgical treatment should always be carried out for osteochondral flake fractures, non-retrievable dislocations and fracture-dislocations. Other indications that should only be considered after careful assessment are initial dislocations with a high risk of recurrent dislocation, recurrent dislocations, a complete rupture of the MPFL or the medial retinaculum, a cartilage injury, a free joint body and failure of conservative treatment (3). Surgical treatment should always start with an arthroscopy to obtain an overview of the extent of the trochlear dysplasia and to identify any osteochondral fragments. The further surgical procedure should be individually tailored to the patient. A variety of different surgical procedures are available for paediatric patients. These include MPFL reconstruction, distal realignment, trochleoplasty, torsion corrections and the correction of a genu valgum based on temporary hemiepiphysiodesis (3). Trochleoplasty is very rarely performed in children with open growth plates, as the effects of this operation on the immature skeleton are unclear and there is concern about the risk of physeal arrest (92). Depending on the case, a combination of the individual techniques may also be necessary. The most common procedure is the reconstruction of the medial support structures of the patella. The consensus is that MPFL reconstruction is preferable to MPFL repair alone (93). A distal realignment may be indicated if the TT-TG distance is increased. Various techniques are used to medialise the tibial tuberosity (94–96). As already discussed in Chapter 2.3, trochlear dysplasia is a crucial risk factor for PFI and can be found in almost all patients with recurrent patellar luxation. In these cases, correction using sulcus-deepening trochleoplasty is a promising procedure that is associated with a low risk of recurrent dislocation (38). However, the current literature recommends that correction of dysplasia in children and adolescents should only be carried out after the growth plates have been closed (97).

3. Materials and Methods

3.1. Study design

The present monocentric study retrospectively examines the effect of trochlear dysplasia on the extent of patellar tilt in a paediatric and adolescent population, using knee MRI scans to analyse anatomical differences. The aim is to determine whether a higher grade of trochlear dysplasia contributes to increased patellar tilt. The data was retrospectively collected from March 2000 to March 2022 at the Department of Paediatric Radiology Graz. MRI scans of children and adolescents up to 18 years of age who underwent imaging due to either a first-time patellar dislocation, some other knee injury or unclear knee pain were used.

Patients with connective tissue disorders, such as Ehlers-Danlos syndrome or Marfan syndrome, were excluded to eliminate cases where generalized ligamentous laxity may impact patellar stability independently of trochlear morphology. Cases of habitual patellar dislocation were also excluded. Additionally, patients with prior surgical stabilization were not included to avoid alterations in knee morphology that might affect measurement accuracy. Patients with neurological disorders (e.g. infantile cerebral palsy) and patients with incomplete data sets and unclear histories were also excluded from the study.

The local institutional review board approved this study (EK Nr. 34-119 ex 21/22). There was no need for informed consent due to the retrospective nature of this study.

3.2. Data Collection

The MRI images were obtained from the local PACS server (Picture Archiving and Communication System). A total of 630 patients were included. There were 338 female and 292 male patients. Of the 630 knees used for the data collection 339 were left knees and 291 were right knees. The mean age was 14.5 years with an age range of 7.9 to 17.9 years. The MRI images were measured using defined parameters related to the PFI. Typical MRI protocols were used for the measurements, consisting of proton density-weighted (PDW) sequences with fat saturation in three planes, T1-weighted coronary sequences and sagittal 3D gradient echo sequences. Seven people with different levels of experience were involved in the measurements: two senior medical students, one PhD student in medical science, two residents for orthopaedics and traumatology, two attending physicians for paediatric orthopaedics and one attending physician for paediatric radiology. The MicroDicom DICOM (Digital Imaging and Communications in Medicine) viewer programme (MicroDicom Ltd., Sofia, Bulgaria) was used to carry out the measurements.

3.3. Radiological Parameters

To assess pathological parameters associated with an increased risk of PFI, MRI was used for qualitative evaluation. The parameters LTIA, TD and TSA were chosen for the quantification of trochlear dysplasia. They provide a reliable assessment of trochlear morphology and are closely associated with PFI. To measure the LTIA (Figure 12), we drew a line in the axial MRI in the slice in which the posterior condyles were clearly visible, touching both condyles at their posterior part. The angle that forms between this line and the lateral facet of the trochlear is the LTIA (43). To determine the TD (Figure 10), we carried out the measurements based on the bony structures using the method first described by Pfirrmann et al. (98). A line was placed on the posterior condyles in the axial plane with the greatest extension of the condyles and the distance from this line to the most anterior part of the medial and lateral joint facet was measured. The arithmetic mean was calculated from these two distances and the distance from the lowest point of the sulcus to the line at the posterior condyles was subtracted from this. The TSA (Figure 12) was also measured on the bone in the axial plane with the greatest distance between the medial and lateral femoral epicondyle. A protractor was used to measure from the deepest point of the sulcus to the most anterior parts of the medial and lateral trochlear (44).

To describe the patellar tilt, the following parameters were used: Angle of Fulkerson, Angle of Laurin, and Patellar Inclination Angle (Figure 13). The Angle of Fulkerson is measured in an axial image as the angle between a tangent to the posterior condyles and a tangent along the lateral patellar facet, while the Angle of Laurin is determined by placing a tangent on the anterior condyles. For both the Angle of Fulkerson and the Angle of Laurin, a positive angle opens laterally. The Patellar Inclination Angle is defined as the angle enclosed by a tangent to the posterior condyles and a transverse line through the longest diameter of the patella. In many publications, the term Patellar Inclination Angle is used synonymously with the term patellar tilt. In this study, the term Patellar Inclination Angle is always used explicitly, as the Angle of Laurin and Angle of Fulkerson were also used to assess the patellar tilt in addition to the Patellar Inclination Angle.

In various studies, all three parameters showed statistically significant differences between patients without PFI and patients with instability (62,65,99). Patients with PFI had an increased lateral inclination of the patella in the frontal plane. This is explained by the fact that patients with instability often have a weakness of the medial soft tissue structures, such as the MPFL (62).

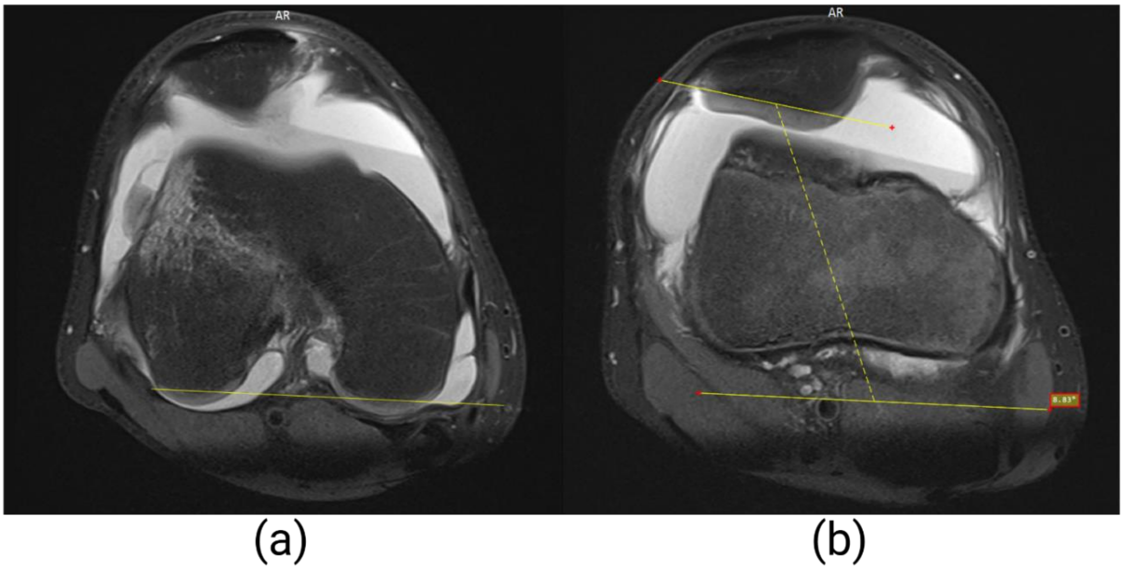
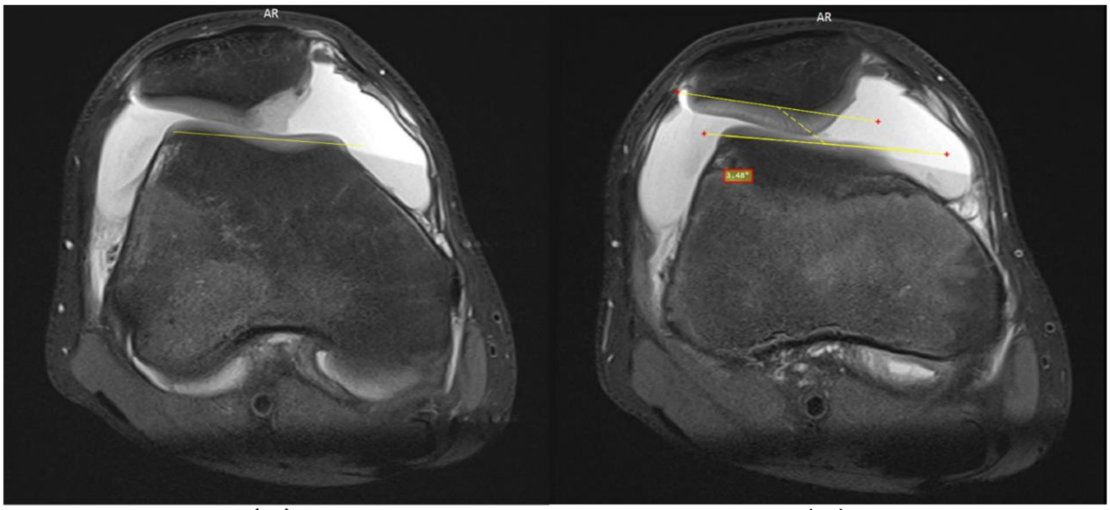


Figure 14: Angle of Fulkerson

Measured in an axial MRI between (a) a tangential line to the posterior condyles of the femur and (b) the lateral facet of the patella. A positive angle opens up laterally.



(a) (b)

Figure 15: Angle of Laurin

Measured in an axial MRI between (a) a tangential line to the anterior condyles of the femur and (b) the lateral facet of the patella. A positive angle opens up laterally.

3.4. Statistical Evaluation

The statistical analysis was conducted using SPSS Version 29.0 (IBM, Armonk, New York, USA). All values were assumed to be normally distributed according to the Q-Q-plots and histograms. Means and standard deviations were calculated for all variables studied. Pearson-correlation analysis was than performed to analyse the relationship between the trochlear dysplasia parameters and the tilt parameters. Simple linear regression analyses were performed to analyse the influence of trochlear dysplasia parameters (LTIA, TD, TSA) on patellar tilt. The Angle of Laurin, Angle of Fulkerson and Patellar Inclination Angle were considered as dependent and continuous variables, LTIA, TD and TSA as independent and continuous variables. Statistical significance was set at $p<0.05$.

Additionally, to explore the potential differences in tilt between patients with and without high-grade trochlear dysplasia, the patients were classified into two groups based on the LTIA and TSA, with an LTIA of less than 11° or a sulcus angle ≥157° serving as the cut-off for non-high-grade trochlear dysplasia. The independent sample t-test was applied to assess differences between these two groups. Statistical significance was set at $p<0.05$ for all analyses.

4. Results

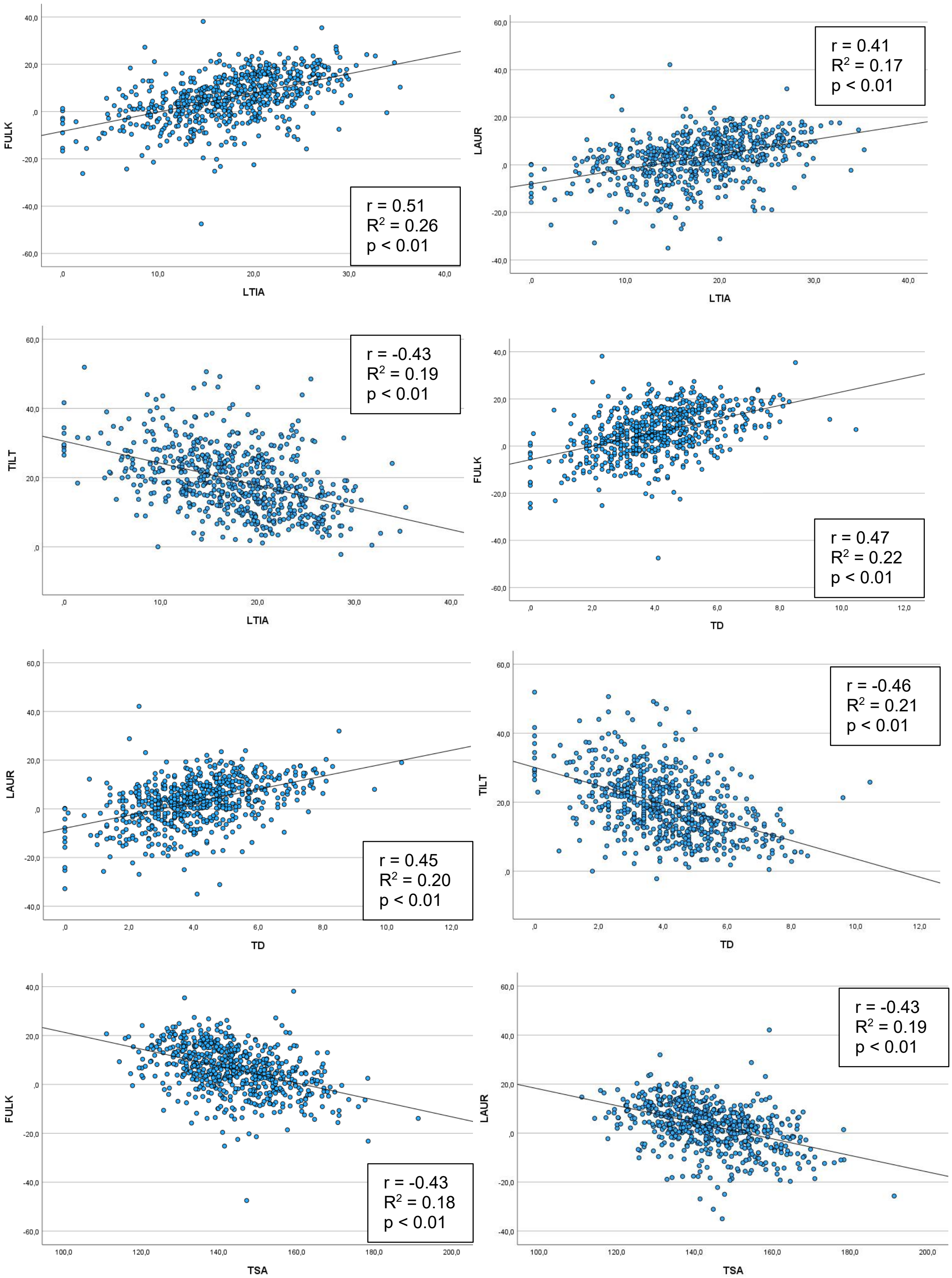
A total of 630 data sets were analysed as part of the descriptive statistics. The Patellar Inclination Angle showed a mean value of 19.29° with a standard deviation of 9.33°. For the Angle of Laurin and Angle of Fulkerson, the mean values were 2.91° (± 9.63°) and 5.92 (± 10.02°). TD showed a mean value of 4.06 mm (± 1.63 mm), TSA 143.73° (± 12.01°) and LTIA 17.57° (± 6.30).

Further correlation analysis shows a significant correlation between the parameters of trochlear dysplasia and patella tilt (Table 2). Reduced LTIA and TD correlate positively with an increased Angle of Fulkerson ($r = 0.510$ and $r = 0.467$, respectively, $p < 0.001$) and Angle of Laurin ($r = 0.406$ and 0.450, respectively, $p < 0.001$) and negatively with the Patellar Inclination Angle ($r = -0.431$ and $r = -0.462$, respectively, $p < 0.001$), indicating an increased lateral inclination of the patella with more severe trochlear dysplasia. In addition, the TSA showed a significant negative correlation with the Angle of Fulkerson ($r = -0.425$, $p < 0.001$) and the Angle of Laurin ($r = -0.432$, $p < 0.001$), and a positive correlation with the Patellar Inclination Angle ($r = 0.300$, $p < 0.001$), indicating that a flatter trochlea is associated with greater lateral patellar inclination.

Table 2: Correlations between trochlear dysplasia parameters and parameters of the patellar tilt

Variable	**Fulkerson**	**Laurin**	**Inclination**
LTIA	0.51**	0.41**	-0.43**
TD	0.47**	0.45**	-0.46**
TSA	-0.43**	-0.43**	0.30**

*Pearson correlations between the parameters of trochlear dysplasia (LTIA, TD, TSA) and the patellar angles (Fulkerson angle, Laurin angle, Patellar Inclination Angle). Positive values show a direct correlation, negative values an inverse relationship. **Significant correlations (p < 0.01) are marked with **.*


FULK
LTIA
r = 0.51
R^2 = 0.26
p < 0.01
LAUR
LTIA
r = 0.41
R^2 = 0.17
p < 0.01
TILT
LTIA
r = -0.43
R^2 = 0.19
p < 0.01
FULK
TD
r = 0.47
R^2 = 0.22
p < 0.01
LAUR
TD
r = 0.45
R^2 = 0.20
p < 0.01
TILT
TD
r = -0.46
R^2 = 0.21
p < 0.01
FULK
TSA
r = -0.43
R^2 = 0.18
p < 0.01
LAUR
TSA
r = -0.43
R^2 = 0.19
p < 0.01

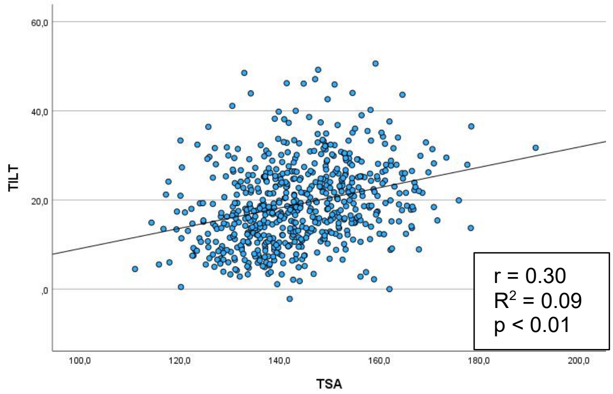


Figure 16: Scatter plot illustrating the correlation between the parameters of trochlear dysplasia (LTIA, TD, TSA) and the patellar tilt (Angle of Fulkerson = FULK, Angle of Laurin = LAUR, Patellar Inclination Angle = TILT).

A linear regression line is included to indicate the trend.

Simple linear regression analyses (Table 3) were also performed to quantify the respective predictive power of the individual dysplasia parameters on the patellar tilt angles. The strongest single prediction was found for LTIA in relation to Fulkerson angle ($R^2 = 0.260$; $p < 0.001$) and for TD in relation to the Patellar Inclination Angle ($R^2 = 0.214$; $p < 0.001$). The other regression models also showed consistently significant correlations with explained variances between 9.0 % and 26.0 %. TSA proved to be significant, although comparatively weaker predictor of Patellar Inclination Angle ($R^2 = 0.090$) and Fulkerson angle ($R^2 = 0.181$).

In addition to the correlation and regression analyses, a comparison of the tilt values between patients with and without high-grade trochlear dysplasia was also performed. For this purpose, the patients were divided into the following groups: Group 1 (no or mild trochlear dysplasia), Group 2 (high-grade dysplasia). The LTIA and TSA were used to differentiate between normal and dysplastic trochlea; an LTIA of less than 11° OR Sulcus Angle ≥157° was assumed as the cut-off for non-high-grade trochlear dysplasia (54). With this subdivision, there were 496 patients (mean age 14.48) in the group without high-grade trochlear dysplasia and 133 patients (mean age 14.50) in the group with high-grade trochlear dysplasia. The independent sample t-test revealed significant differences between patients with high-grade trochlear dysplasia and patients without high-grade trochlear dysplasia in the Angle of Laurin, the Angle of Fulkerson and the Patellar Inclination Angle (all $p < 0.001$). Patients with high-grade trochlear dysplasia had on average lower values for the

Laurin angle (-2.84° vs. 4.43°) and the Fulkerson angle (-0.69° vs 7.68°), while the Patellar Inclination Angle was significantly higher (24.66° vs. 17.89°).

Table 3: Results of simple linear regression analyses between trochlear dysplasia parameters (LTIA, TD, TSA) and patellar tilt measures (Patellar Inclination Angle, Laurin angle, Fulkerson angle)

Dependent variable	Independent variable	R^2	p-value
Patellar Inclination	LTIA	0.186	<0.001
Patellar Inclination	TD	0.214	<0.001
Patellar Inclination	TSA	0.090	<0.001
Laurin	LTIA	0.165	<0.001
Laurin	TD	0.203	<0.001
Laurin	TSA	0.187	<0.001
Fulkerson	LTIA	0.260	<0.001
Fulkerson	TD	0.218	<0.001
Fulkerson	TSA	0.181	<0.001

The explained variance (R^2) and p-values are shown.

5. Discussion

5.1. Interpretation of Findings

The results of our retrospective study show a significant correlation between the parameters of trochlear dysplasia and patellar tilt, confirming the hypothesis of increased patellar tilt with increasing trochlear dysplasia. The assumption that high-grade trochlear dysplasia is associated with significantly increased patellar tilt was also shown by dividing the patients into a group with high-grade dysplasia and a group without high-grade dysplasia according to the modified new Dejour classification (54).

To date, only a few studies have investigated the correlation between trochlear dysplasia parameters and patellar tilt parameters. Pace et al. (100) chose a similar study design to our study. In their retrospective study from 2020, they analysed MRIs of 65 patients between the age of 9 and 18. With a mean age of 14.2, this patient group corresponds to the patient group (mean age 14.5) that was measured for this thesis. While our study included patients with and patients without PFI, Pace et al. (100) only included patients who were treated for PFI. They analysed the correlation between lateral trochlear inclination and lateral patellar inclination, among other things. These two parameters are comparable with the LTIA and the Patellar Inclination Angle in our study, even though they were measured using other methods. For the measurement of lateral trochlear inclination, they used the modified measurement technique of Joseph et al. (101) and thus obtained a mean value of 4.2° ± 11.9°. For the measurement of the lateral patellar inclination Pace et al. (100) used a modified measurement technique proposed by Fucentese et al. (102) which resulted in a mean value of 19.6° ± 9.4°. The linear regression analysis by Pace et al. (100) showed a significant inverse correlation between lateral trochlear inclination and lateral patellar inclination ($r = -0.69$, $\beta = -0.54$). The authors thus conclude that an increase in trochlear dysplasia probably leads to an increase in patellar tilt, as the position of the patella adapts to the dysplastic trochlea.

Luczak et al. (102) also determined the lateral patellar inclination according to the method of Fucentese et al. (102) and the lateral trochlear inclination according to the method of Joseph et al (101). They found a significant correlation between these two parameters as well, although they emphasise that the correlation is not strong enough to conclude a close anatomical link. According to their study, patellar tilt is strongly influenced by trochlear dysplasia and patellar height.

A study by Frings et al. (103) investigated the influence of anatomical risk factors for PFI on patellar maltracking. Using 10 patients (mean age 19 years) with clinical patellar maltracking and 20 control patients (mean age 28 years), they were able to show, among other things, that the TSA correlates significantly with the dynamic patellar tilt. An enlarged TSA (higher level of trochlea dysplasia) was associated with an increased lateral tilt of the patella during knee movement. This study is not comparable with the results of the present study due to the dynamic measurement, but it gives an indication of the correlation between the TSA and a greater lateral tilt of the patella, which can also be seen in the results of our study.

In a study of 566 adult patients from the Multicentre Osteoarthritis Study (MOST-study), Stefanik et al. (104) also investigated the relationship between morphological criteria of the trochlea and PFJ alignment. For the measurements of TSA, LTIA and Patellar Inclination Angle in MRIs, the same measurement methods were used as in our study. In their study, LTIA was found to be the best predictor of patellar tilt among the trochlear parameters analysed, with a correlation of r = -0.32. TSA showed only a weak positive correlation with the Patellar Inclination Angle (r = 0.11). In our study, there were higher correlations between LTIA and Patellar Inclination Angle (r = -0.43) as well as between TSA and Patellar Inclination Angle (r = 0.30). However, we found that TD, which was not investigated by Stefanik et al. (104), was the best predictor of Patellar Inclination Angle ($R^2 = 0.214$) and the parameter with the strongest correlation coefficient in relation to Patellar Inclination Angle (r = -0.46). One of the possible reasons for the different results might be the different patient collective: as part of the MOST study, a prospective cohort study that included patients between 50 and 79 years of age who either had knee osteoarthritis or were at high risk of developing it. The underlying osteoarthritis as well as different cartilage to bone ratios during skeletal maturation may also have an influence on the biomechanics of the PFJ and therefore on the measurement results.

The correlation between TD and the lateral patellar tilt is even less studied in the literature than the relationship between the LTIA and patellar tilt. A study by Powers (105) was able to show a correlation between the depth of the trochlear groove and lateral patellar tilt using kinematic MRIs during knee extension with resistance. A second study assumes a correlation between a reduced trochlear depth and an increased lateral patellar tilt, however, in this study by Felicio et al. (106) the trochlear depth was not measured directly but a reduced trochlear depth was inferred based on an increased TSA. To better understand this relationship and to obtain a better comparison to the LTIA as a predictive value for patellar tilt, further investigations are necessary.

The above-mentioned studies consistently show that there is a significant inverse correlation between the degree of trochlear dysplasia - measured by LTIA - and the patellar tilt: the more severe the dysplasia, the greater the lateral tilt of the patella. This observation was also confirmed in our study by dividing the patient collective into a group with high-grade dysplasia and a group without high-grade dysplasia according to Dejour et al. (54): The group with high-grade dysplasia showed a significantly higher lateral patellar tilt than the group without or with mild trochlear dysplasia. Irrespective of this group division, we found significant moderate correlations between all parameters of trochlear dysplasia and the parameters of the patellar tilt. The strongest correlation exists between the LTIA and the Angle of

Fulkerson (r = 0.51), the weakest correlation between TSA and the Patellar Inclination Angle (r = 0.30). We were also able to show the inverse correlation between the LTIA and the Patellar Inclination Angle that was most frequently described in the studies (100,104,107).
In summary, our results show that a more pronounced dysplasia, characterised by a lower LTIA and TD, as well as a larger TSA, is associated with an increased lateral patellar tilt.

5.2. Strengths and Limitations

Our study has several strengths. Firstly, the number of patients is very high with a collective of 630 patients. By including patients with confirmed patellofemoral instability and patients without patellofemoral instability, the patient collective covers a broad spectrum of different anatomical and biomechanical variants in the knee joint area. Thus, different degrees of severity of trochlear dysplasia are represented and the relationship between trochlear parameters and the patellar tilt can be investigated across the entire clinical spectrum. In addition, all measurements were carried out using MRIs, which allows an accurate assessment of the PFJ.
Yet, the present study is not without limitations: due to its retrospective nature, a selection bias may have occurred. An attempt was made to minimise this problem by carefully reviewing the patients' histories. An observer bias cannot be ruled out due to the different people involved with different levels of experience despite being trained in the measurements. No conclusions can be drawn from our study for younger patients, as the youngest patients included were 8 years old.
Another possible limitation is the choice of measurement methodology. It should also be mentioned that many different measurement methods are used in the literature and a comparison is often difficult. Nevertheless, even with different measurement methods, the overall picture of the results in the literature is coherent with our results. However, other factors influencing the patellar tilt such as the patellar height described by Luczak et al. (107) were not considered.
Our study does not allow an assessment of whether there is a causal relationship between trochlear dysplasia and patellar tilt. This relationship is also unclear in terms of skeletal development, for which there are various theories. On the one hand, the patella could have a mechanical influence on the development of the

trochlea; on the other hand, the trochlear morphology could also be genetically determined and not influenced by the position of the patella (108–110).

5.3. Clinical Implications

We were able to show that there is a significant correlation between increasing trochlear dysplasia and increased lateral patellar tilt and that a comprehensive picture of patellofemoral morphology is therefore necessary in the diagnosis and treatment of patients with PFI. Since, for example, the patellar tilt can already be roughly estimated in the clinical examination, this could already provide an initial indication of possible trochlear dysplasia (61).

In addition, trochlear depth showed the highest predictive value and the greatest correlation with patellar tilt in our study and could therefore become more important in the assessment of PFI. Nevertheless, due to the small number of studies on this correlation, further investigations are necessary.

References

1. Fithian DC, Paxton EW, Stone M Lou, Silva P, Davis DK, Elias DA, et al. Epidemiology and Natural History of Acute Patellar Dislocation. Am J Sports Med [Internet]. 2004 Jul 1;32(5):1114–21. Available from: https://doi.org/10.1177/0363546503260788
2. Hasler CC, Studer D. Patella instability in children and adolescents. EFORT Open Rev. 2016 May 1;1(5):160–6.
3. Balcarek P, Jagodzinski M, Niemeyer P, Zeichen J. S2e-Leitlinie: Patellaluxation. In 2021.
4. Parikh SN, Rajdev N, Sun Q. The Growth of Trochlear Dysplasia during Adolescence. Journal of Pediatric Orthopaedics. 2018;38(6):e318–24.
5. Huntington LS, Webster KE, Devitt BM, Scanlon JP, Feller JA. Factors Associated With an Increased Risk of Recurrence After a First-Time Patellar Dislocation: A Systematic Review and Meta-analysis. Am J Sports Med. 2020 Aug;48(10):2552–62.
6. Sanders TL, Pareek A, Hewett TE, Stuart MJ, Dahm DL, Krych AJ. High rate of recurrent patellar dislocation in skeletally immature patients: a long-term population-based study. Knee Surg Sports Traumatol Arthrosc. 2018 Apr;26(4):1037–43.
7. Charles MD, Haloman S, Chen L, Ward SR, Fithian D, Afra R. Magnetic resonance imaging-based topographical differences between control and recurrent patellofemoral instability patients. American Journal of Sports Medicine. 2013 Feb;41(2):374–84.
8. Waldeyer A. Waldeyer-Anatomie des Menschen. 19th ed. Anderhuber F, Pera F, Streicher J, editors. Berlin/Boston: Walter de Gruyter GmbH & Co. KG; 2012.
9. Sobotta J. Anatomie das Lehrbuch. 2nd ed. Waschke J, Böckers T, Paulsen F, editors. München: München Elsevier, Urban & Fischer; 2019.
10. Schünke M, Schulte E, Schumacher U. PROMETHEUS LernAtlas - Allgemeine Anatomie und Bewegungssystem. 6th ed. Voll M, Wesker KH, editors. Stuttgart: Georg Thieme Verlag KG; 2022.

11. Tubbs RS, Michelson J, Loukas M, Shoja MM, Ardalan MR, Salter EG, et al. The transverse genicular ligament: Anatomical study and review of the literature. Vol. 30, Surgical and Radiologic Anatomy. 2008. p. 5–9.
12. Makris EA, Hadidi P, Athanasiou KA. The knee meniscus: Structure-function, pathophysiology, current repair techniques, and prospects for regeneration. Vol. 32, Biomaterials. 2011. p. 7411–31.
13. MedicalGraphics - Kostenlose medizinische Illustrationen. Licence: CC BY-ND 4.0 [Internet]. [cited 2025 Jun 4]. Available from: https://www.medicalgraphics.de/kostenlose-medizinische-illustrationen/
14. Morales-Avalos R, Torres-González EM, Padilla-Medina JR, Monllau JC. ACL anatomy: Is there still something to learn? Rev Esp Cir Ortop Traumatol. 2024;68(4):T422–7.
15. Śmigielski R, Zdanowicz U, Drwięga M, Ciszek B, Williams A. The anatomy of the anterior cruciate ligament and its relevance to the technique of reconstruction. Bone Joint J. 2016 Aug;98-B(8):1020–6.
16. Andrews K, Lu A, Mckean L, Ebraheim N. Review: Medial collateral ligament injuries. Vol. 14, Journal of Orthopaedics. Reed Elsevier India Pvt. Ltd.; 2017. p. 550–4.
17. Braaten JA, Banovetz MT, Rodriguez AN, Thomas P, LaPrade RF. From Anatomy to Complex Reconstruction: A Modern Review on the Medial Collateral Ligament of the Knee. Vol. 10, Archives of Bone and Joint Surgery. Mashhad University of Medical Sciences; 2022. p. 818–26.
18. Loudon JK. BIOMECHANICS AND PATHOMECHANICS OF THE PATELLOFEMORAL JOINT. Int J Sports Phys Ther. 2016 Dec;11(6):820–30.
19. Grelsamer RP, Klein JR. The Biomechanics of the Patellofemoral joint [Internet]. 1998. Available from: www.jospt.org
20. Toms AP, Cahir J, Swift L, Donell ST. Imaging the femoral sulcus with ultrasound, CT, and MRI: Reliability and generalizability in patients with patellar instability. Skeletal Radiol. 2009 Apr;38(4):329–38.
21. Feller JA, Amis AA, Andrish JT, Arendt EA, Erasmus PJ, Powers CM. Surgical Biomechanics of the Patellofemoral Joint. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2007 May 1;23(5):542–53.

22. Yu Z, Yao J, Wang X, Xin X, Zhang K, Cai H, et al. Research Methods and Progress of Patellofemoral Joint Kinematics: A Review. Vol. 2019, Journal of Healthcare Engineering. Hindawi Limited; 2019.
23. Siljander B, Tompkins M, Martinez-Cano JP. A Review of the Lateral Patellofemoral Joint: Anatomy, Biomechanics, and Surgical Procedures. Vol. 6, Journal of the American Academy of Orthopaedic Surgeons Global Research and Reviews. Wolters Kluwer Health; 2022.
24. Desio SM, Burks RT, Md §, Bachus KN. Soft Tissue Restraints to Lateral Patellar Translation in the Human Knee*. Vol. 26, THE AMERICAN JOURNAL OF SPORTS MEDICINE. 1998.
25. Patil S, Ahmad M, Patel M, Rathinam B, Pandita KK, Santoshi JA, et al. Cadaveric Study to Define the Anatomy of the Medial Patellofemoral Ligament (MPFL) and Its Variant Patterns. Cureus. 2023 May 22;
26. Rajput H, Rajani S, Vaniya VH. Variation in morphometry of vastus medialis muscle. Journal of Clinical and Diagnostic Research. 2017 Sep 1;11(9):AC01–4.
27. Huberti HH, Hayes WC. Patellofemoral contact pressures. The influence of q-angle and tendofemoral contact. J Bone Joint Surg Am. 1984 Jun;66(5):715–24.
28. Kaufer H. Mechanical function of the patella. J Bone Joint Surg Am. 1971 Dec;53(8):1551–60.
29. Dan M, Parr W, Broe D, Cross M, Walsh WR. Biomechanics of the knee extensor mechanism and its relationship to patella tendinopathy: A review. Vol. 36, Journal of Orthopaedic Research. John Wiley and Sons Inc.; 2018. p. 3105–12.
30. Basso O, Amis AA, Race A, Johnson DP. Patellar tendon fiber strains: their differential responses to quadriceps tension. Clin Orthop Relat Res. 2002 Jul;(400):246–53.
31. Sadler TW. Taschenlehrbuch Embryologie. 13th ed. Drews U, Brand-Saberi B, editors. Stuttgart New York: Georg Thieme Verlag; 2020.
32. Gardner E, Ronan A, Rahilly ’. The early development of the knee joint in staged human embryos. Vol. 102, J. Anat. 1968.

33. Fox AJS, Wanivenhaus F, Rodeo SA. The basic science of the patella: structure, composition, and function. Vol. 25, The journal of knee surgery. 2012. p. 127–41.
34. Walmsley R. The development of the patella. J Anat. 1940 Apr;74(Pt 3):360-368.3.
35. Ogden JA. Radiology of postnatal skeletal development. Skeletal Radiol. 1984 Apr;11(4):246–57.
36. Lüllmann-Rauch R. Taschenlehrbuch Histologie. 6th ed. Asan E, editor. Stuttgart: Georg Thieme Verlag; 2019.
37. Dejour DH, Deroche É. Trochleoplasty: Indications in patellar dislocation with high-grade dysplasia. Surgical technique. Orthopaedics & Traumatology: Surgery & Research. 2022 Feb 1;108(1):103160.
38. Schmeling A. Trochleadysplasie und ihre Therapie. Knie Journal. 2022 Jun;4(2):100–10.
39. Jungmann PM, Tham SC, Liebl H, Nevitt MC, McCulloch CE, Lynch J, et al. Association of trochlear dysplasia with degenerative abnormalities in the knee: data from the Osteoarthritis Initiative. Skeletal Radiol. 2013 Oct;42(10):1383–92.
40. Devries CA, Bomar JD, Pennock AT. Prevalence of Trochlear Dysplasia and Associations with Patellofemoral Pain and Instability in a Skeletally Mature Population. Journal of Bone and Joint Surgery. 2021 Nov 17;103(22):2126–32.
41. Onor GI, Yalcin S, Kaar SG, Pace JL, Ferrua P, Farrow LD. The Evaluation of Trochlear Osseous Morphology: An Epidemiologic Study. Orthop J Sports Med. 2021;9(4).
42. Saccomanno MF, Maggini E, Vaisitti N, Pianelli A, Grava G, Cattaneo S, et al. Sulcus Angle, Trochlear Depth, and Dejour's Classification Can Be Reliably Applied To Evaluate Trochlear Dysplasia: A Systematic Review of Radiological Measurements. Vol. 39, Arthroscopy - Journal of Arthroscopic and Related Surgery. W.B. Saunders; 2023. p. 549–68.
43. Kim HK, Shiraj S, Kang CH, Anton C, Kim DH, Horn PS. Patellofemoral instability in children: Correlation between risk factors, injury patterns, and

severity of cartilage damage. American Journal of Roentgenology. 2016 Jun 1;206(6):1321–8.

44. Toms AP, Cahir J, Swift L, Donell ST. Imaging the femoral sulcus with ultrasound, CT, and MRI: Reliability and generalizability in patients with patellar instability. Skeletal Radiol. 2009 Apr;38(4):329–38.
45. Kazley JM, Banerjee S. Classifications in Brief: The Dejour Classification of Trochlear Dysplasia. Clin Orthop Relat Res. 2019 Oct 1;477(10):2380–6.
46. Kappel P, Liebensteiner M. Imaging diagnostics of patellofemoral disorders. Vol. 33, Arthroskopie. Springer Medizin; 2020. p. 412–9.
47. Donaldson O, Heal J, Wakeley C, Eldridge J, Mulford J. DOES THE PLAIN RADIOGRAPH ACCURATELY PREDICT TROCHLEAR DYSPLASIA. J Bone Joint Surg Br. 2012 Jan 1;94-B:61.
48. Onor GI, Yalcin S, Kaar SG, Pace JL, Ferrua P, Farrow LD. The Evaluation of Trochlear Osseous Morphology: An Epidemiologic Study. Orthop J Sports Med. 2021 Apr;9(4):2325967121994548.
49. Fucentese SF, Zingg PO, Schmitt J, Pfirrmann CWA, Meyer DC, Koch PP. Classification of trochlear dysplasia as predictor of clinical outcome after trochleoplasty. Knee Surg Sports Traumatol Arthrosc. 2011 Oct;19(10):1655–61.
50. Giovannetti de Sanctis E, Mesnard G, Dejour DH. Trochlear Dysplasia: When and How to Correct. Clin Sports Med. 2022 Jan;41(1):77–88.
51. Rémy F, Chantelot C, Fontaine C, Demondion X, Migaud H, Gougeon F. Inter- and intraobserver reproducibility in radiographic diagnosis and classification of femoral trochlear dysplasia. Surg Radiol Anat. 1998;20(4):285–9.
52. Roy KD, Joshi P, Ali I, Shenoy PM, Malek I, Barlow D, et al. A comparative analysis of interobserver reliability and intraobserver reproducibility of the Oswestry-Bristol Classification and the Dejour Classification for trochlear dysplasia of the knee. Bone Jt Open. 2023 Jul 20;4(7):532–8.
53. Sharma N, Brown A, Bouras T, Kuiper JH, Eldridge J, Barnett A. The Oswestry-Bristol Classification. Bone Joint J. 2020 Jan;102-B(1):102–7.
54. Dejour DH, de Sanctis EG, Müller JH, Deroche E, Pineda T, Guarino A, et al. Adapting the Dejour classification of trochlear dysplasia from qualitative

radiograph- and CT-based assessments to quantitative MRI-based measurements. Knee Surgery, Sports Traumatology, Arthroscopy. 2024;

55. Carrillon Y, Abidi H, Dejour D, Fantino O, Moyen B, Tran-Minh VA. Patellar instability: assessment on MR images by measuring the lateral trochlear inclination-initial experience. Radiology. 2000 Aug;216(2):582–5.

56. Joseph SM, Cheng C, Solomito MJ, Pace JL. Lateral Trochlear Inclination Angle: Measurement via a 2-Image Technique to Reliably Characterize and Quantify Trochlear Dysplasia. Orthop J Sports Med. 2020;8(10).

57. Carstensen SE, Menzer HM, Diduch DR. Patellar Instability: When is Trochleoplasty Necessary? Sports Med Arthrosc Rev. 2017 Jun;25(2):92–9.

58. Hadidi O, Ellanti P, Lincoln M, Hogan N. The J-sign in patellar maltracking. BMJ Case Rep. 2018 Feb 14;2018.

59. Metcalfe AJ, Clark DA, Kemp MA, Eldridge JD. Trochleoplasty with a flexible osteochondral flap. Bone Joint J. 2017 Mar;99-B(3):344–50.

60. von Knoch F, Böhm T, Bürgi ML, von Knoch M, Bereiter H. Trochleaplasty for recurrent patellar dislocation in association with trochlear dysplasia. J Bone Joint Surg Br. 2006 Oct;88-B(10):1331–5.

61. Grelsamer RP, Weinstein CH, Gould J, Dubey A. Patellar tilt: The physical examination correlates with MR imaging. Knee. 2008 Jan;15(1):3–8.

62. Charles MD, Haloman S, Chen L, Ward SR, Fithian D, Afra R. Magnetic Resonance Imaging–Based Topographical Differences Between Control and Recurrent Patellofemoral Instability Patients. Am J Sports Med [Internet]. 2013 Jan 31;41(2):374–84. Available from: https://doi.org/10.1177/0363546512472441

63. Gobbi RG, Cavalheiro CM, Giglio PN, Hinckel BB, Camanho GL. Patellar Tilt and Patellar Tendon–Trochlear Groove Angle Present the Optimum Magnetic Resonance Imaging Diagnostic Reliability for Patients With Patellar Instability. Arthroscopy - Journal of Arthroscopic and Related Surgery. 2023 Nov 1;39(11):2339–51.

64. Ye Q, Yu T, Wu Y, Ding X, Gong X. Patellar instability: The reliability of magnetic resonance imaging measurement parameters. BMC Musculoskelet Disord. 2019 Jul 6;20(1).

65. Dejour H, Walch G, Nove-Josserand L, Guier C. Factors of patellar instability: an anatomic radiographic study. Knee Surg Sports Traumatol Arthrosc. 1994;2(1):19–26.
66. Abermann E, Adam F, Amis A, Anagnostakos K, Aßmann G, Benedetto KP, et al. Expertise Knie. In: Kohn D, editor. Expertise Knie. Stuttgart: Georg Thieme Verlag; 2016.
67. Smith TO, Gaukroger A, Metcalfe A, Hing CB. Surgical versus non-surgical interventions for treating patellar dislocation. Cochrane Database Syst Rev. 2023 Jan 24;1(1):CD008106.
68. Ricciuti A, Colosi K, Fitzsimmons K, Brown M. Patellofemoral Instability in the Pediatric and Adolescent Population: From Causes to Treatments. Vol. 11, Children. Multidisciplinary Digital Publishing Institute (MDPI); 2024.
69. Chen J, Li Q, Liu S, Fan L, Yin B, Yang X, et al. Prediction of Subsequent Contralateral Patellar Dislocation after First-Time Dislocation Based on Patellofemoral Morphologies. J Clin Med. 2023 Jan 1;12(1).
70. Lattermann C, Toth J, Bach BR. The role of lateral retinacular release in the treatment of patellar instability. Sports Med Arthrosc Rev. 2007 Jun;15(2):57–60.
71. Fithian DC, Paxton EW, Stone M Lou, Silva P, Davis DK, Elias DA, et al. Epidemiology and natural history of acute patellar dislocation. Am J Sports Med. 2004;32(5):1114–21.
72. Wolfe S, Varacallo M, Thomas JD, Carroll JJ, Kahwaji CI. Patellar Instability. 2023.
73. Bruce WD, Stevens PM. Surgical correction of miserable malalignment syndrome. J Pediatr Orthop. 2004;24(4):392–6.
74. Waldt Matthias; Wörtler Klaus SE, editor. Messverfahren und Klassifikationen in der muskuloskelettalen Radiologie [Internet]. 1. Auflage. Stuttgart: Georg Thieme Verlag KG; 2011. Available from: http://www.thieme-connect.de/products/ebooks/book/10.1055/b-004-134458
75. Singerman R, Davy DT, Goldberg VM. Effects of patella alta and patella infera on patellofemoral contact forces. J Biomech. 1994 Aug;27(8):1059–65.
76. Song EK, Seon JK, Kim MC, Seol YJ, Lee SH. Radiologic Measurement of Tibial Tuberosity-Trochlear Groove (TT-TG) Distance by Lower Extremity

Rotational Profile Computed Tomography in Koreans. Clin Orthop Surg. 2016 Mar;8(1):45–8.

77. Wittstein JR, Bartlett EC, Easterbrook J, Byrd JC. Magnetic resonance imaging evaluation of patellofemoral malalignment. Arthroscopy. 2006 Jun;22(6):643–9.
78. Elias DA, White LM, Fithian DC. Acute lateral patellar dislocation at MR imaging: injury patterns of medial patellar soft-tissue restraints and osteochondral injuries of the inferomedial patella. Radiology. 2002 Dec;225(3):736–43.
79. Guerrero P, Li X, Patel K, Brown M, Busconi B. Medial patellofemoral ligament injury patterns and associated pathology in lateral patella dislocation: an MRI study. BMC Sports Sci Med Rehabil. 2009 Dec;1(1).
80. Sillanpää P, Mattila VM, Iivonen T, Visuri T, Pihlajamäki H. Incidence and risk factors of acute traumatic primary patellar dislocation. Med Sci Sports Exerc. 2008 Apr;40(4):606–11.
81. Nomura E, Inoue M. Second-look arthroscopy of cartilage changes of the patellofemoral joint, especially the patella, following acute and recurrent patellar dislocation. Osteoarthritis Cartilage. 2005 Nov;13(11):1029–36.
82. Erickson BJ, Mascarenhas R, Sayegh ET, Saltzman B, Verma NN, Bush-Joseph CA, et al. Does Operative Treatment of First-Time Patellar Dislocations Lead to Increased Patellofemoral Stability? A Systematic Review of Overlapping Meta-analyses. Arthroscopy. 2015 Jun;31(6):1207–15.
83. Sanders TL, Pareek A, Johnson NR, Stuart MJ, Dahm DL, Krych AJ. Patellofemoral Arthritis After Lateral Patellar Dislocation: A Matched Population-Based Analysis. Am J Sports Med. 2017 Apr;45(5):1012–7.
84. Salonen EE, Magga T, Sillanpää PJ, Kiekara T, Mäenpää H, Mattila VM. Traumatic Patellar Dislocation and Cartilage Injury: A Follow-up Study of Long-Term Cartilage Deterioration. Am J Sports Med. 2017 May;45(6):1376–82.
85. Gao B, Shi Y, Zhang F. Pediatric patellar dislocation. Minerva Pediatr. 2020 Mar;72(1).

86. Vollnberg B, Koehlitz T, Jung T, Scheffler S, Hoburg A, Khandker D, et al. Prevalence of cartilage lesions and early osteoarthritis in patients with patellar dislocation. Eur Radiol. 2012 Nov;22(11):2347–56.
87. Smith TO, Song F, Donell ST, Hing CB. Operative versus non-operative management of patellar dislocation. A meta-analysis. Knee Surgery, Sports Traumatology, Arthroscopy. 2011 Jun;19(6):988–98.
88. Harilainen A, Myllynen P, Antila H, Seitsalo S. The significance of arthroscopy and examination under anaesthesia in the diagnosis of fresh injury haemarthrosis of the knee joint. Injury. 1988 Jan;19(1):21–4.
89. Colatruglio M, Flanigan DC, Harangody S, Duerr RA, Kaeding CC, Magnussen RA. Identifying Patients With Patella Alta and/or Severe Trochlear Dysplasia Through the Presence of Patellar Apprehension in Higher Degrees of Flexion. Orthop J Sports Med. 2020 Jun;8(6):2325967120925486.
90. Flores GW, de Oliveira DF, Ramos APS, Sanada LS, Migliorini F, Maffulli N, et al. Conservative management following patellar dislocation: a level I systematic review. J Orthop Surg Res. 2023 May 30;18(1):393.
91. Nwachukwu BU, So C, Schairer WW, Green DW, Dodwell ER. Surgical versus conservative management of acute patellar dislocation in children and adolescents: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2016 Mar;24(3):760–7.
92. Chaclas N, Orellana K, Huang K, Amato C, Williams BA. Trochleoplasty in Pediatric Patients - A Systematic Review. Iowa Orthop J. 2024;44(2):100–6.
93. Matic GT, Magnussen RA, Kolovich GP, Flanigan DC. Return to activity after medial patellofemoral ligament repair or reconstruction. Arthroscopy. 2014 Aug;30(8):1018–25.
94. Fulkerson JP, Becker GJ, Meaney JA, Miranda M, Folcik MA. Anteromedial tibial tubercle transfer without bone graft. Am J Sports Med. 1990;18(5):490–6; discussion 496-7.
95. Kraus T, Lidder S, Švehlík M, Rippel K, Schneider F, Eberl R, et al. Patella re-alignment in children with a modified Grammont technique. Acta Orthop. 2012 Oct;83(5):504–10.

96. Grammont PM, Latune D, Lammaire IP. [Treatment of subluxation and dislocation of the patella in the child. Elmslie technic with movable soft tissue pedicle (8 year review)]. Orthopade. 1985 Sep;14(4):229–38.
97. Vavken P, Wimmer MD, Camathias C, Quidde J, Valderrabano V, Pagenstert G. Treating patella instability in skeletally immature patients. Arthroscopy. 2013 Aug;29(8):1410–22.
98. Pfirrmann CW, Zanetti M, Romero J, Hodler J. Femoral trochlear dysplasia: MR findings. Radiology. 2000 Sep;216(3):858–64.
99. Escala JS, Mellado JM, Olona M, Giné J, Saurí A, Neyret P. Objective patellar instability: MR-based quantitative assessment of potentially associated anatomical features. Knee Surg Sports Traumatol Arthrosc. 2006 Mar;14(3):264–72.
100. Pace JL, Cheng C, Joseph SM, Solomito MJ. Effect of Trochlear Dysplasia on Commonly Used Radiographic Parameters to Assess Patellar Instability. Orthop J Sports Med. 2020 Jul 1;8(7).
101. Joseph SM, Cheng C, Solomito MJ, Pace JL. LATERAL TROCHLEAR INCLINATION IN CHILDREN AND ADOLESCENTS: MODIFIED MEASUREMENT TECHNIQUE TO CHARACTERIZE PATELLAR INSTABILITY. Orthop J Sports Med. 2019 Mar 1;7(3_suppl):2325967119S0014.
102. Fucentese SF, Schöttle PB, Pfirrmann CWA, Romero J. CT changes after trochleoplasty for symptomatic trochlear dysplasia. Knee Surg Sports Traumatol Arthrosc. 2007 Feb;15(2):168–74.
103. Frings J, Dust T, Krause M, Ohlmeier M, Frosch KH, Adam G, et al. Objective assessment of patellar maltracking with 3 T dynamic magnetic resonance imaging: feasibility of a robust and reliable measuring technique. Sci Rep. 2020 Oct 8;10(1):16770.
104. Stefanik JJ, Zumwalt AC, Segal NA, Lynch JA, Powers CM. Association between measures of patella height, morphologic features of the trochlea, and patellofemoral joint alignment: the MOST study. Clin Orthop Relat Res. 2013 Aug;471(8):2641–8.

105. Powers CM. Patellar kinematics, part II: the influence of the depth of the trochlear groove in subjects with and without patellofemoral pain. Phys Ther. 2000 Oct;80(10):965–78.
106. Felicio LR, Saad MC, Liporaci RF, Baffa A do P, dos Santos AC, Bevilaqua-Grossi D. Correlation between trochlear groove depth and patellar position during open and closed kinetic chain exercises in subjects with anterior knee pain. J Appl Biomech. 2012 Jul;28(3):335–42.
107. Luczak SB, Cheng C, Hedgecock JP, Brimacombe M, Pace JL. Patellar Height Correlates Modestly With Trochlear Dysplasia: A Magnetic Resonance Imaging Analysis. Arthrosc Sports Med Rehabil. 2022 Jun;4(3):e1031–7.
108. Huri G, Atay OA, Ergen B, Atesok K, Johnson DL, Doral MN. Development of femoral trochlear groove in growing rabbit after patellar instability. Knee Surg Sports Traumatol Arthrosc. 2012 Feb;20(2):232–8.
109. Yang G, Li F, Lu J, Niu Y, Dai Y, Zuo L, et al. The dysplastic trochlear sulcus due to the insufficient patellar stress in growing rats. BMC Musculoskelet Disord. 2019 Sep 5;20(1):411.
110. Glard Y, Jouve JL, Garron E, Adalian P, Tardieu C, Bollini G. Anatomic study of femoral patellar groove in fetus. J Pediatr Orthop. 2005;25(3):305–8.