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Observations on Rotatory Instability of the Lateral Compartment of the Knee

Experimental Studies on the Functional Anatomy and the Pathomechanism of the True and the Reversed Pivot Shift Sign

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Helv. chir. Acta 43, 451–456.

JAKOB, R. P., NOESBERGER, B. & MUELLER, M. E. (1977):
The Diagnostic Value of the Pivot Shift Sign in Anterior Instability of the Knee and the Specific Lateral Repair; From Chapchal G.: Injuries of the Ligaments and their Repair.
G. Thieme Publishers Stuttgart.

JAKOB, R. P. (1977):
The «Giving way»-Test.
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JAKOB, R. P. (1981):
Die Untersuchung des Kniegelenkes bei Bandrupturen, Meniskusläsionen und Knorpelschäden.
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Das umgekehrte Pivot-Shift-Phänomen (Reversed Pivot-Shift) – ein neues Zeichen der posterolateralen Knieinstabilität.
Aus «Experimentelle Grundlagen der Diagnostik und Therapie der Kapselbandläsionen des Kniegelenkes». III. Münchner Symposium für experimentelle Orthopädie, G. Thieme Verlag Stuttgart (in press).

JAKOB, R. P., STAEUBLI, H. U. (1981):
The Reversed Pivot-Shift Sign. A New Diagnostic Aid For Posterolateral Rotatory Instability of the Knee.
Paper read at the Second Congress of the International Society of the Knee, New Orleans, U.S.A.

HASSLER, H. und JAKOB, R. P. (1981):
Ein Beitrag zur Ursache der anterolateralen Instabilität des Kniegelenkes.
Arch. Orthop. Traumat. Surg. 98: 45–50

HASSLER, H., JAKOB, R. P. (1981):
Ein Beitrag zur Ursache der antero-lateralen Instabilität des Kniegelenkes: Eine Studie an Leichenknien.
Aus «Experimentelle Grundlagen der Diagnostik und Therapie der Kapselbandläsionen des Kniegelenkes». III. Münchner Symposium für experimentelle Orthopädie, G. Thieme Verlag Stuttgart (in press).

STAUEBLI, H. U., NOESBERGER, B., JAKOB, R. P. (1981):
Experimentelle Grundlagen zur Diagnostik der posterolateralen Rotationsinstabilität.
Aus «Experimentelle Grundlagen der Diagnostik und Therapie der Kapselbandläsionen des Kniegelenkes». III. Münchner Symposium für experimentelle Orthopädie, G. Thieme Verlag Stuttgart (in press).

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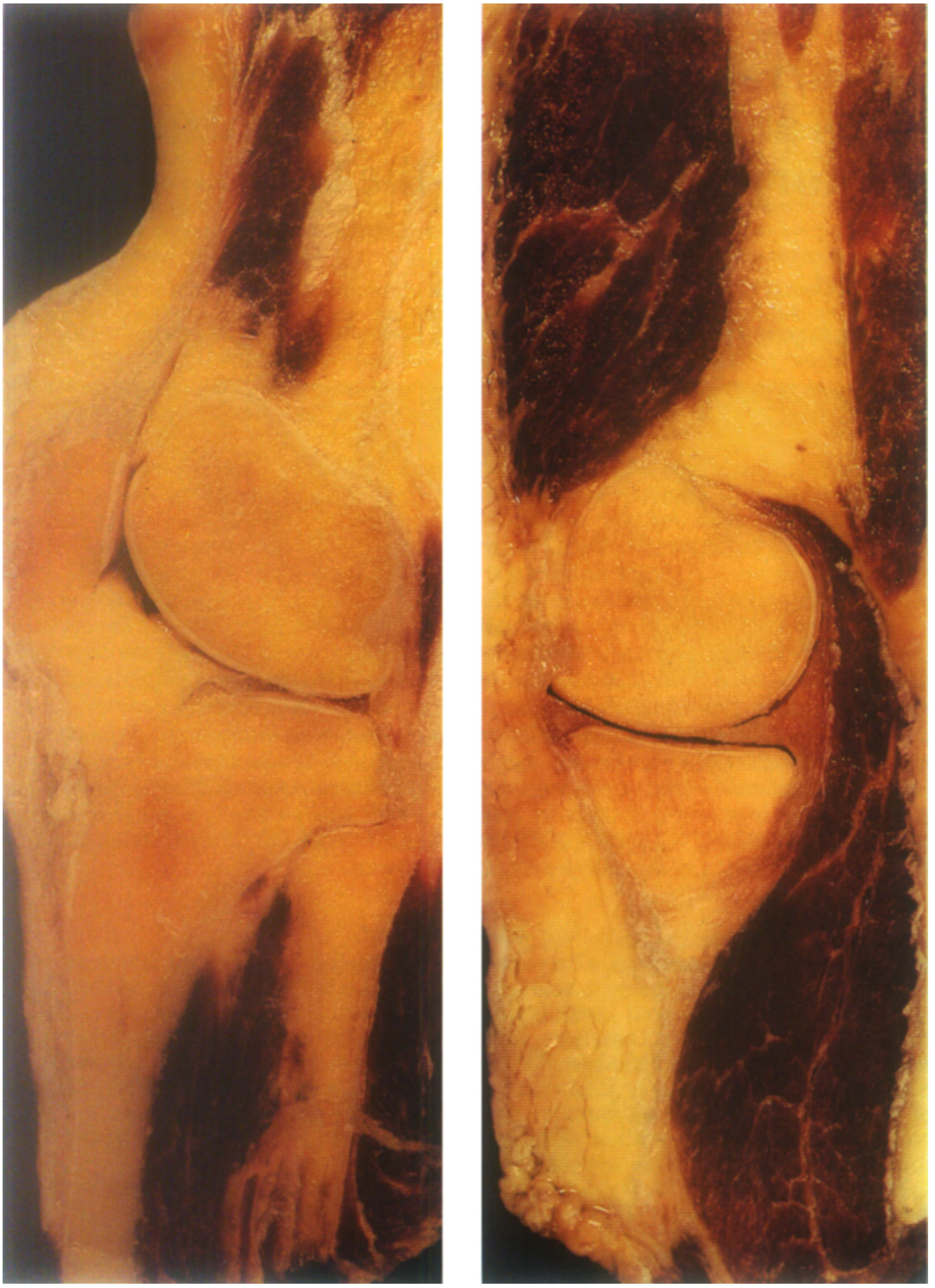

Fig. 1
The subluxation in the lateral compartment is enhanced by the convex shape of the lateral tibial plateau (left) as compared to the concave shape of the medial plateau (right).

Part I

Observations concerning the clinical signs and the etiology of anterolateral rotatory instability of the knee

Abstract

Cadaver experiments have been carried out to study the etiology of anterolateral instability and the production of a pivot shift sign. Anterior subluxation of the lateral tibial plateau, e.g. anterolateral instability is detectable with an anterior drawer sign in neutral or slight internal rotation and with a positive pivot shift sign. Anterolateral instability is found in the presence of a ruptured anterior cruciate ligament in conjunction with a disruption or attenuation of the lateral capsular ligament. In the experiments the pivot shift sign was only present when an anterior cruciate ligament lesion was produced. Conversly a true pivot shift sign was never seen associated with a lesion produced in the lateral capsular ligament alone. This latter lesion was only able to produce an inconstant snapping phenomenon due to subluxation and entrapment of the lateral meniscus. With a deficient anterior cruciate ligament the lateral capsular ligament may secondarily stretch thus enhancing chronic anterolateral instability. Dividing the three insertions of the iliotibial tract will increase anterolateral instability, i.e. the anterior drawer sign of the lateral tibial plateau in neutral rotation and the pivot shift sign in its first phase of anterior subluxation. The clinically relevant phase of painful reduction will however be less marked. Thus the pivot shift sign is intimately dependant upon the normal function of the iliotibial tract. The presence or absence of a pivot shift sign alone should not be interpreted as a reliable index of the presence of anterolateral rotatory instability since the pivot shift sign is observed in all other conditions with anterior cruciate insufficiency, i.e. marked anteromedial rotatory instability («Unhappy triad») or straight anterior instability (isolated rupture of the anterior cruciate ligament).

Posttraumatic anterolateral instability, often in conjunction with anteromedial rotatory instability, of the knee is the most common type of disabling ligamentous instability of the athlete and it severly interferes with normal knee function. This combined instability is caused by a partial or total rupture of the medial and the lateral capsular and collateral ligament in association with rupture or attenuation of the anterior cruciate ligament (*Palmer*, 1938; *Hallen & Lindahl*, 1965; *Slocum* et al., 1968, 1976, 1977; *Kennedy* et al., 1971, 1976, 1977, 1978; *Bousquet*, 1972; *Trillat* et al., 1972; *Menschik*, 1974; *Mueller*, 1975; *Eriksson*, 1976; *Hughston* et al., 1976; *Cabaud & Slocum*, 1977; *Jakob* et al., 1976, 1977, 1980; *Karpf*, 1977; *Losee* et al., 1978; *Jaeger & Wirth*, 1978; *Norwood* et al., 1979).

In the healthy knee anterior displacement of the tibia is less likely to occur on the medial side due to a more constrained articulation between the concave tibial plateau and the convex femoral condyle. In the lateral compartment however anterior subluxation is enhanced by a flat or even slightly convex shape of the plateau thus lacking the inherent stability of the medial side (Fig. 1). This increased tendency toward anteroposterior «shifting» of the tibia becomes clinically significant since it is associated with a painful giving way episode caused by anterior subluxation and termed the «pivot shift sign» (*Hey-Groves*, 1920; *Brantigan & Voshell*, 1941; *Galway* et al., 1972; *Wirth & Artmann*, 1974).

The anterior subluxation is initiated when the patient standing on the straight leg with the foot planted pivots away from the affected side causing slight flexion and a valgus force to be applied to the knee. As the knee is flexed over 20 degrees the anterior subluxation will reduce with a sudden painful jerk. Such a circumstance is seen dynamically in a patient who while running suddenly changes direction, the so called «cutting manœuvre».

This phenomenon can be demonstrated clinically with the patient lying on the examining table by various tests, the most popular being the «pivot shift sign» described by *Galway* et al. (1972) and *MacIntosh* (1974, 1976) (Fig. 2). The examiner lifts the foot of the extended leg with one hand, forces the knee under slight axial load into a valgus position with the other hand by pushing on the lateral side of the calf, and while maintaining this continuous valgus force, flexes the knee. If the anterior cruciate is torn the patient's painful symptoms and «giving way» feeling will be reproduced.

Similar signs depicting the same event were illustrated by other authors (*Hughston*, 1976; *Slocum* et al., 1976, *Losee* et al., 1978).

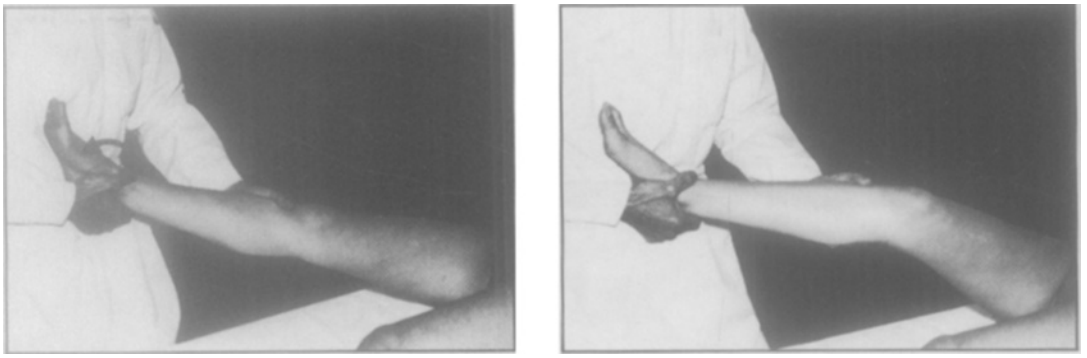

Fig. 2
Demonstration of the pivot shift sign. The foot of the extended knee is lifted with one hand and the knee is forced under slight axial load into a valgus position by pushing with the other hand on the lateral side of the calf. Flexing the knee the examiner will elicit a sudden jerk

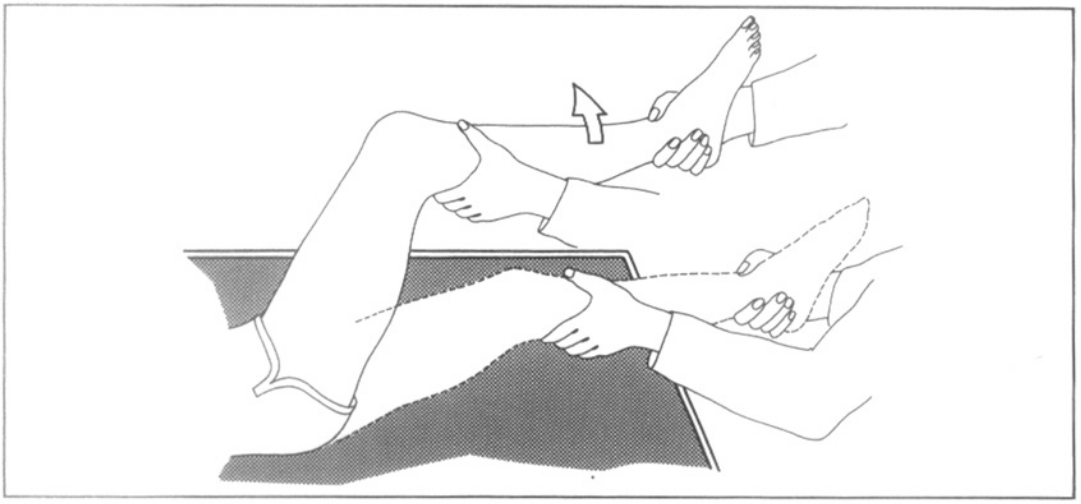

Fig. 3
Schematic drawing of the jerk test which represents the reversed mechanism of the pivot shift sign. While the pivot shift is tested from extension to flexion, the jerk test is elicited when moving the knee from flexion to extension

In the jerk test (*Hughston*, 1976) the patient lies supine, the knee is flexed to about 60 degrees, the distal tibia is grasped so that an internal rotation force can be applied. With the opposite hand a valgus stress is applied while the knee is extended and the tibial plateau is pushed forward. At about 20 degree of flexion a sudden rapid subluxation occurs as the lateral tibial plateau slides forward (Fig. 3).

When the patient is very apprehensive so that the tight hamstrings prevent an easy examination the Anterolateral Rotatory Instability-Test described by *Slocum* (1976) has a slight advantage. In this test the patient lies on his healthy side with the foot of the involved side supported on the table in slight internal rotation of the tibia. In this position the weight of the leg applies its own valgus stress. By grasping the distal femur with one hand and the proximal tibia with the other placing one thumb on the back of the fibula the straight knee is flexed allowing the examiner to palpate the subluxation and subsequent reduction of the tibia (Fig. 4).

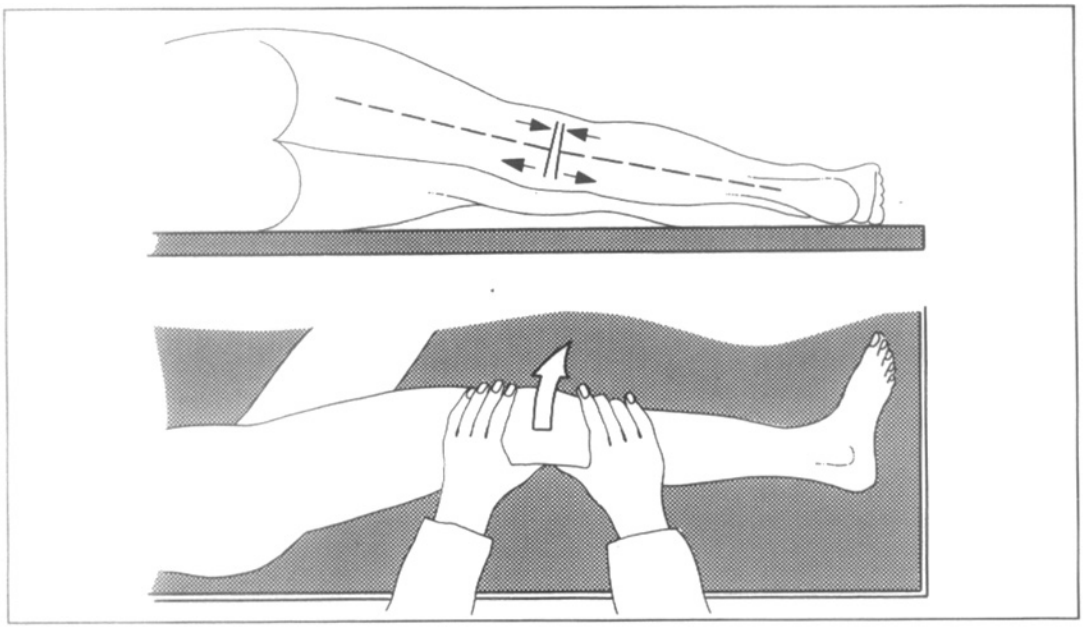

Fig. 4
In the anterolateral rotatory instability test the patient is placed on the side (see text). This test demonstrates anterolateral instability in a more careful way, thus it is more suitable for the apprehensive patient

In 1976 we introduced another test to reproduce anterior subluxation of the lateral tibial plateau (*Jakob* 1977). With the patient standing and leaning with his sound side against the wall, he is asked to distribute his weight equally on both feet. The examiner places his hands above and below the injured knee and exerts a valgus stress while initiating flexion. This will elicit the «jerk» and the knee will dramatically give way (Fig. 5).

These signs for the diagnosis of anterolateral instability should not be confused with another «jerking» phenomenon of the lateral compartment of the knee which is always observed in presence of posterolateral instability. It represents the alternating shift of the lateral tibial plateau in posterior subluxation and reduction as the knee is flexed and extended under a valgus stress. This sign we called the «reversed pivot shift sign», since the displacement of the tibia takes place in an opposite direction to the true pivot shift.

The strong shearing forces acting on the articular cartilage and the repetitive character of the giving way episodes lead to cartilage erosions in both compartments, and

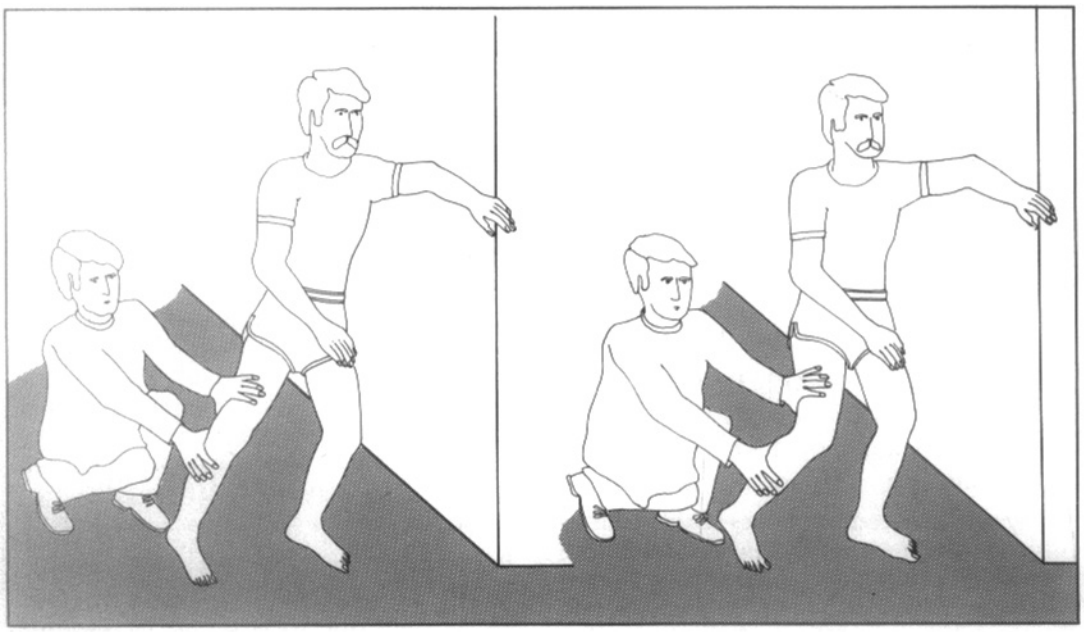

Fig. 5
Giving way test to reproduce anterior subluxation of the lateral tibial plateau with the patient standing (see text)

with time to secondary meniscal damage and osteoarthrosis – changes described by *MacIntosh* as the «anterior cruciate syndrome» (Fig. 6).

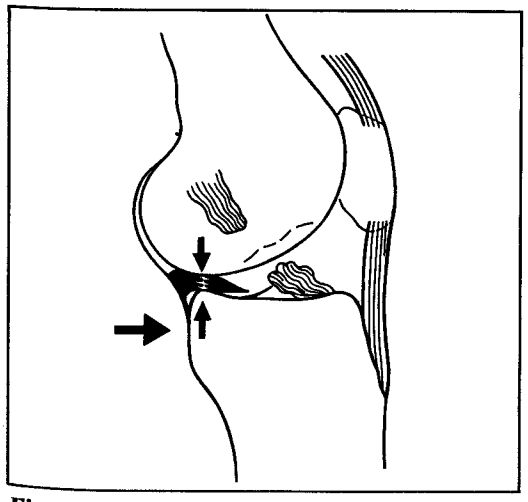

Fig. 6
The shearing forces acting on the articular cartilage produce cartilage erosions, menisceal damage and lead to osteoarthrosis, changes described by MacIntosh as the «anterior cruciate syndrome»

Since pathology of the lateral compartment has been recognised as being the cause of the prevalent symptoms in combined anteromedial-anterolateral instability the previous emphasis for surgical repairs on the medical side (*Nicholas*, 1973; *O'Donoghue*, 1973; *Noesberger* et al., 1977) has been replaced by an increasing interest in reconstructions on the lateral side. Over the past ten years various methods have been specifically described to eliminate the anterior subluxation of the lateral tibial plateau thus abolishing the pivot shift phenomenon (*MacIntosh*, 1974, 1976; *Ellison*, 1975; *Jakob* et al., 1977; *Kennedy* et al., 1978; *Losee* et al., 1978). The basic principle in these repairs is to reinforce the lateral capsular structures using the iliotibial band and to substitute for the torn anterior cruciate ligament. In combined techniques the anterior cruciate ligament is replaced and a lateral and medial repair are added (*Feagin* et al., 1980).

Thus *MacIntosh's* concept of the importance of the lateral compartment has redirected the philosophy of knee ligament reconstructions and his test for the diagnosis of anterolateral instability, or modifications of it, are of great value.

What then is the cause of a positive pivot shift sign?

The lesions discussed are the rupture of the anterior cruciate ligament, the disruption of the lateral capsular ligament and/or the fibular collateral ligament with the arcuate ligament and the rupture of the iliotibial tract (*Galway* et al., 1972; *Hughston* et al., 1976; *Jakob* et al., 1977; *Fetto & Marshall*, 1979; *Norwood* et al., 1979; *Woods* et al., 1979).

Anatomical investigation

Studies were carried out on fresh human cadavers to examine the pathomechanics of a pivot shift sign.

In twenty fresh cadaver knees without instability or degenerative arthrosis and before rigor mortis had started we produced a series of sequential lesions which resulted in specific instabilities. A short medial parapatellar arthrotomy was performed through which the anterior cruciate ligament could be sectioned. Utilizing a lateral arthrotomy sequential sectioning of the other structures thought to be involved in the production of a pivot shift sign were performed, i. e. the lateral capsular ligament, the fibular collateral ligament, with the arcuate ligament and the iliotibial tract.

Results

The results as listed in table 1) summarize the findings as reproduced in two to three knees for each step of the experiment.

Discussion

In our experiments a pivot shift sign could never be produced without first dividing the anterior cruciate ligament. The pivot shift sign was amplified by cutting the middle third of the lateral capsular ligament and became more marked when dividing the fibular collateral ligament and the arcuate ligament (Exp. 1).

In an earlier study we reported that for a pivot shift sign to occur the anterior cruciate ligament had to be ruptured or attenuated (*Jakob* et al., 1977). We also stated that the reason that not all acute tears of the anterior cruciate ligament in «Unhappy triad» lesions were associated with a positive pivot shift test was, because in some of them the medial collateral ligament was completely ruptured so that the applied valgus force could not build up enough contact pressure in the lateral compartment for the pivot shift phenomenon to take place.

Hughston in 1976 stated that in a series of six cases of fresh anterolateral instability he found a tear of the midlateral capsular ligament in five patients and a rupture of the anterior cruciate ligament in only two patients. *Norwood* et al. (1979) found in a series of thirtysix knees with acute anterolateral rotatory instability thirty cases with a tear of the anterior cruciate ligament usually combined with a tear of the lateral capsular ligament. In only six cases was the latter lesion found to be the only one involved.

In our experiment however, a complete dissection of the midthird of the lateral capsular ligament alone never led to a positive pivot shift sign (Exp. II). The only jerklike phenomenon which this lesion produced was a snapping sensation due to subluxation and entrapment of the lateral meniscus in one knee joint. Division of the fibular collateral ligament and the arcuate ligament added to the rotatory instability but it was only after adding section of the anterior cruciate ligament that the pivot shift became positive. These experiments therefore confirm our previous finding, that the anterior cruciate ligament is the key structure which prevents the lateral as well as the medial plateau from shifting forward enough for a pivot shift sign to occur.

This further agrees with *Larson* (1979) who feels that the capsular ligament sometimes is very thin and would not, therefore play an important role in the pathogenesis of the pivot shift phenomenon. The same finding was confirmed later by *Fetto* and *Marshall* (1979) who stated that in no case did the division of the lateral capsular ligament of the knee with the anterior cruciate ligament left intact produce a positive pivot shift.

On the other hand it has been observed by several authors that a varus and internal rotation injury of the knee may be associated with an avulsion of the lateral meniscotibial ligament with a fleck of bone where anterolateral instability was detected (*Hughston* et al., 1976, *Losee* et al., 1978, *Norwood* et al., 1979, *Woods* et al. 1979).

Table 1

Ligamentous lesions produced in 20 cadaver knees

Lesions	Anterior Drawer Sign in Neutral Rotation	Pivot Shift Sign
EXP. I SECTION OF: ACL plus LCm/3 plus FCL, AL	 – + ++	 + +(+) ++
EXP. II SECTION OF: LCm/3 plus FCL, AL plus ACL	 (+) + ++	 – – ++
EXP. III SECTION OF: ITT (K) plus LCm/3 plus ACL	 (+) + ++	 – – ++
EXP. IV SECTION OF: ACL plus ITT (P)	 – –	 + (+)
EXP. V SECTION OF: ITT (G) plus ACL	 +(+) ++	 – (+)
EXP. VI SECTION OF: ACL plus ITT (G)	 – ++	 + (+)

ACL = *anterior cruciate ligament*
FCL = *fibular collateral ligament*
ITT (K) = *iliotibial tract fibers blending in the intermuscular septum and the linea aspera proximal to the lateral femoral condyle (Kaplan's fibers)*
ITT (G) = *detachment of Gerdie's tubercle*
ITT (P) = *patellar expansion of iliotibial tract*
LCm/3 = *middle third of the lateral capsular ligament*
AL = *arcuate ligament*

Anterior drawer sign
\+ = *slight*
++ = *moderate*
+++ = *marked*

Pivot shift sign
\+ = *slight*
++ = *moderate*
+++ = *marked*

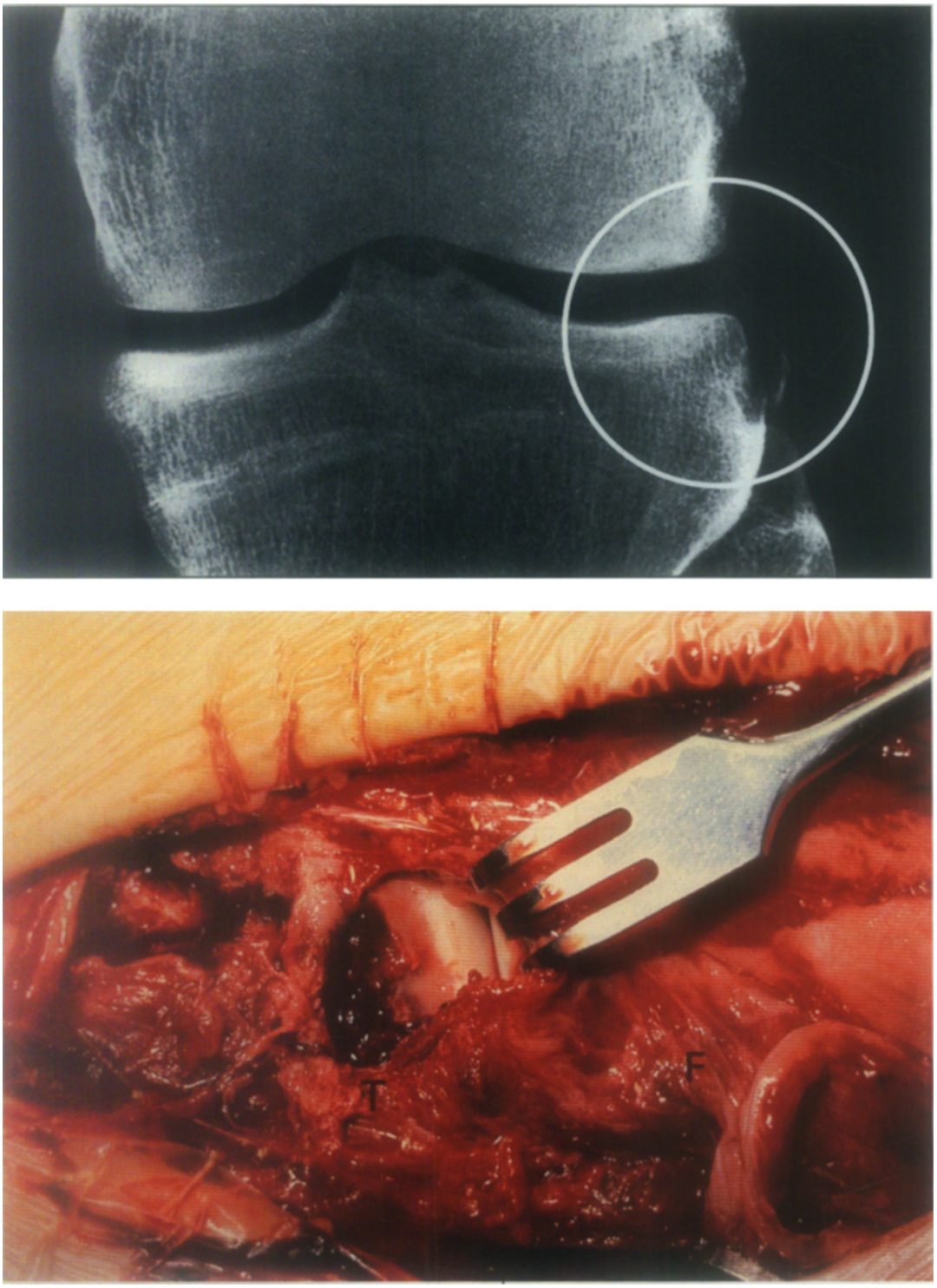

Fig. 7
Avulsion of the menisco-tibial ligament in a fresh varus and internal rotation injury. The anterior drawer sign of the lateral tibial plateau in neutral rotation was +, the pivot shift sign was negative with the anterior cruciate ligament being intact (Courtesy of B. Noesberger)

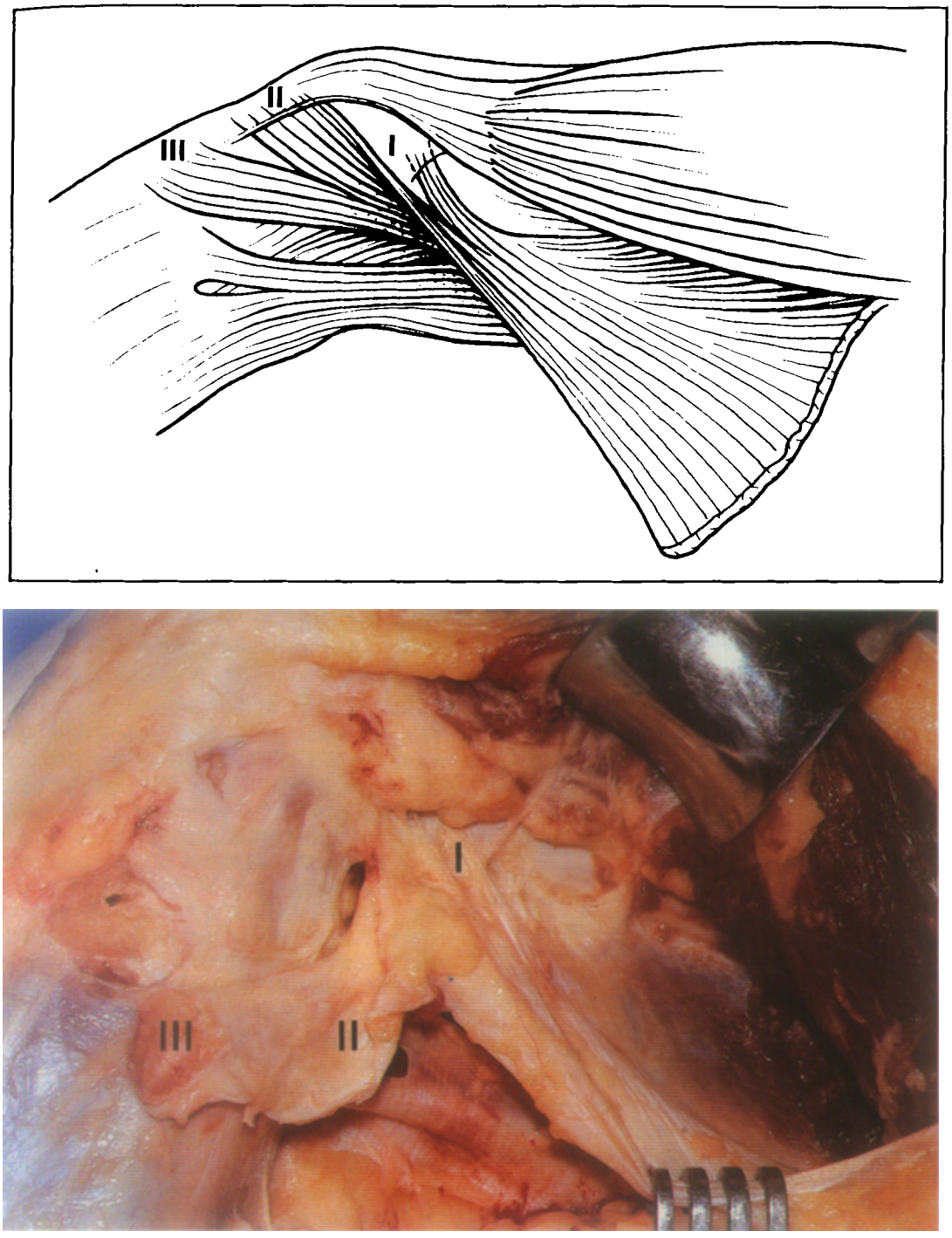

Fig. 8
The three insertions of the iliotibial tract (above) Part I of the iliotibial tract blending into the intermuscular septum and inserting on the supracondylar tubercle (Kaplan's fibers). The insertions at the patella and the patellar ligament (part II) as well as Gerdie's tubercle (part III) have been removed showing that there is no attachment on the lateral aspect of the femoral condyle (below)

We were able to confirm this in a fresh case with the same finding of a slight anterolateral drawer sign in which the anterior cruciate ligament was intact and the pivot shift sign was negative (Fig. 7). In these cases the midlateral capsule is substantial enough to withstand a strong pull, producing instead an avulsion fracture from the tibia. Such a lesion should be surgically repaired since if not it may lead to secondary stretching of the anterior cruciate ligament. We stress the importance of carefully examining the anterior cruciate ligament at the time of surgery to be sure it is not ruptured or attenuated.

In attempt to further elucidate the causative lesions involved in the pivot shift event, we next studied the influence of the iliotibial tract. How do the different parts of the iliotibial tract contribute to anterolateral instability?

Hughston et al. (1976) mentioned that a lesion of those fibers of the iliotibial tract inserting on the linea aspera proximal to the lateral femoral condyle are of some importance since they commonly found a hematoma at this area in lesions combined with fresh disruptions of the anterior cruciate ligament. We are convinced of the common finding of such a hematoma, sometimes extending as far down as the head of the fibula, in acute «unhappy triad» lesions. We were, however, only rarely able to demonstrate a true disruption of the fibers of the iliotibial tract.

The part of the iliotibial tract which blends into the intermuscular septum and inserts on the supracondylar tubercule was thoroughly described by *Kaplan* (1958, 1962) (Fig. 8). From studies of comparative anatomy and investigation with electrical stimulation he stated that the contraction of the tensor fasciae latae muscle is not transmitted to the lower most part of the structure and that the iliotibial tract acts as an accessory anterolateral ligament of the knee preserving stability in instances in which the fibular collateral ligament has been lost. This is explained by the fact that it is conjoined with the intermuscular septum, inserting together with it into the supracondylar tubercle of the lateral femoral condyle, and extending further down from there free of bony connection to the lateral tubercle of the tibia of Gerdie. It therefore becomes a tense ligament extending from the iliac crest to the upper portion of the lateral femoral condyle and to Gerdie's tubercle, moving forward in extension and backward in flexion.

In our study we observed that the part of this ligament between the supracondylar tubercle and Gerdie's tubercle becomes tight in flexion since the distance of the two points increases. Because of this fact there is, in flexion, a marked backward force acting on the tibia through the iliotibial tract. This force is directed toward external rotation. We also recognised that «Kaplan's fibers» are tight in internal rotation of the tibia and loose in external rotation.

The division of Kaplan's fibers alone did not result in a pivot shift and caused only a small amount of foreward displacement of the lateral tibial plateau on the anterolateral drawer test, when combined with the division of the lateral capsular ligament (Exp. III). In association with a transected anterior cruciate ligament one would have expected the pivot shift sign to be very marked because of the increased anterior laxity of the lateral plateau. The pivot shift became however less marked.

The explanation for this observation was readily found in the fact that after the section of «Kaplan's fibers» the iliotibial tract was not attached anymore to the femoral condyle. When initiating flexion the iliotibial tract immediately shifted posterior thus preventing anterior subluxation of the plateau sufficient for a pivot shift sign to occur. The detachment of «Kaplan's fibers» could easily be reversed when a Kirschner-wire was inserted into the femoral epicondyle posterior to the iliotibial tract (Fig. 9). This prevented it from sliding backward. Immediately the pivot shift became marked because the more anteriorly directed fibers of the iliotibial tract now permitted a sufficient anterior subluxation of the lateral tibial plateau to take place. This allowed a greater pressure in the lateral compartment to build up to a point, where a sudden reduction occured.

A similar observation was made in another cadaver when, after section of the anterior cruciate ligament, the anterior part of the iliotibial tract which blends into the patella and patellar ligament was cut (Exp. IV). In

this case the pivot shift sign expected because of the anterior cruciate lesion, became less marked since once more the tract was able to slide posteriorly earlier than normal.

In another series of specimens we osteotomized Gerdie's tubercle alone (Exp. V). We thought this would result in an immediate pivot shift. All that occured however was a + – ++ drawer sign in slight internal rotation of the tibia. When we cut the anterior cruciate ligament, we immediately got a ++ anterior drawer of the lateral tibial plateau in neutral and slight internal rotation, showing that with an absent anterior cruciate the main structure preventing anterior subluxation in internal rotation is not as was thought the intact posterior cruciate but rather the iliotibial tract.

This also held true, when the iliotibial tract was sectioned at a midfemoral level after division of the anterior cruciate ligament. As before the anterior drawer became ++ in neutral rotation. This reverted to a + when the distal part of the tract was fixed proximally with a sharp hook (Fig. 10).

In another series the anterior cruciate ligament was sectioned producing a positive pivot shift sign (Exp. VI). When the additional detachment of Gerdie's tubercle was carried out and the pivot shift sign was tested, the tibia shifted into marked anterior subluxation and internal rotation as was the case in Exp. V. Instead of reducing at 30 degrees of flexion it became trapped in such a way that the femoral condyle was caught behind the convexity of the lateral plateau even as far as to flexion of 60 degrees. When the biceps tendon was detached as well this became even more marked. It was only by pushing the tibia manually backward that the subluxation could be reduced. Immediately after having fixed Gerdie's tubercle with a Kirschner-wire the anterior subluxation and internal rotation became less marked and a marked pivot shift test reappeared again.

In an additional experiment we inserted a Kirschner-wire in front of the iliotibial tract at the femoral epicondyle with the knee in flexion (Fig. 11). When the knee was extended the fibers of the iliotibial tract now displaced posteriorly, became tight as well as assuming a position paralleling the direction of the anterior cruciate ligament. This kept the tibia back in reduction preventing any anterior displacement of the lateral tibial plateau and thus the pivot shift sign. This represents a repetition of the classical experiment of *MacIntosh*, which demonstrated the principal of his lateral repair. This repair consists of rerouting a strip of iliotibial tract, left attached at Gerdie's tubercle, under the femoral insertion of the fibular collateral ligament to decrease anterolateral instability.

Conclusion

From our experiments we conclude that the presence of a pivot shift sign points to anterior cruciate insufficiency. Anterolateral instability, i. e. anterior subluxation of the lateral tibial plateau occurs with an anterior cruciate rupture in combination with a primary tear or secondary stretching of the midlateral capsule. These lesions will only result in a positive pivot shift sign – with its phases of anterior subluxation followed by jerklike reduction – as long as the functional anatomy of the iliotibial tract is unimpaired. The pivot shift sign will become weaker when we detach the part of the iliotibial tract inserting at the supracondylar area (Kaplan's fibers) and when we divide the fibers blending into the patellar ligament. It will become even less prominent when we detach Gerdie's tubercle although its first phase of anterior subluxation and the anterior drawer sign in neutral rotation will get worse. However the phase of the jerklike reduction due to the backward pull of the iliotibial tract is missing. Absence of the pivot shift does not equal absence of anterolateral instability since the anterolateral drawer sign is still grossly positive in this situation. Presence therefore of a pivot shift sign is intimately connected with the deficiency of the anterior cruciate ligament and the functional integrity of the iliotibial tract.

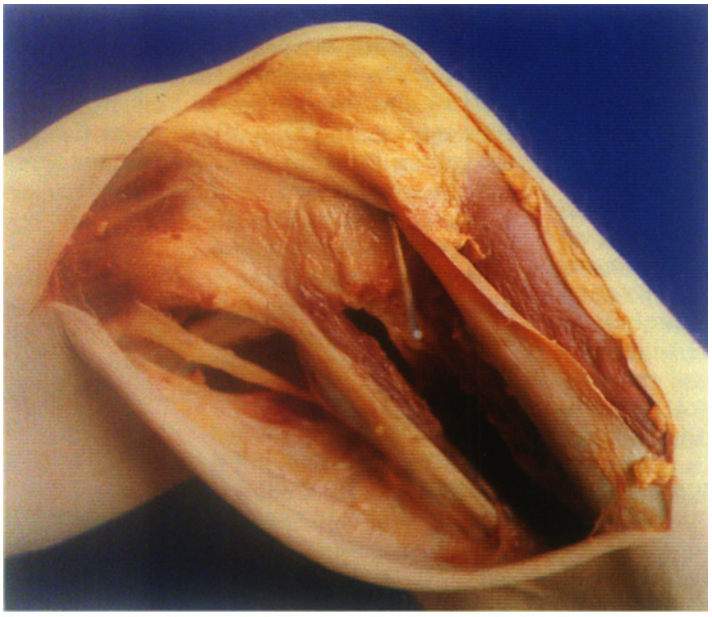

Fig. 9
The detachment of Kaplan's fibers could easily be reversed when a Kirschner-wire was inserted into the femoral epicondyle posterior to the iliotibial tract which prevented it from sliding backward

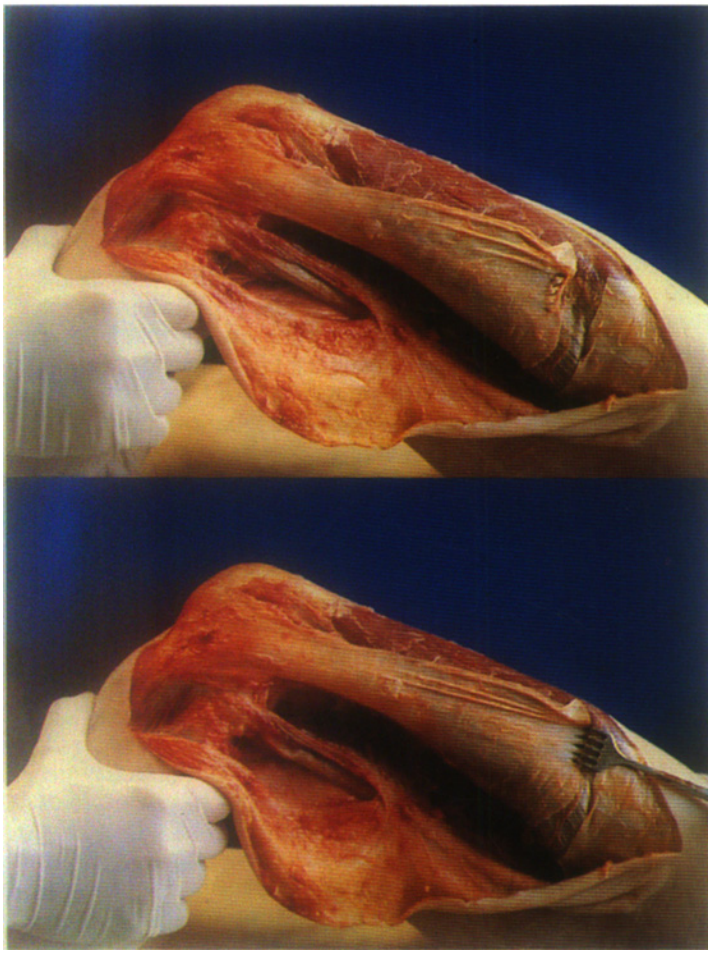

Fig. 10
Section of the iliotibial tract at midfemoral level with a ++ anterior drawer sign after section of the anterior cruciate ligament. This reverted to + when the sectioned distal part of the tract was fixed proximally with a sharp hook

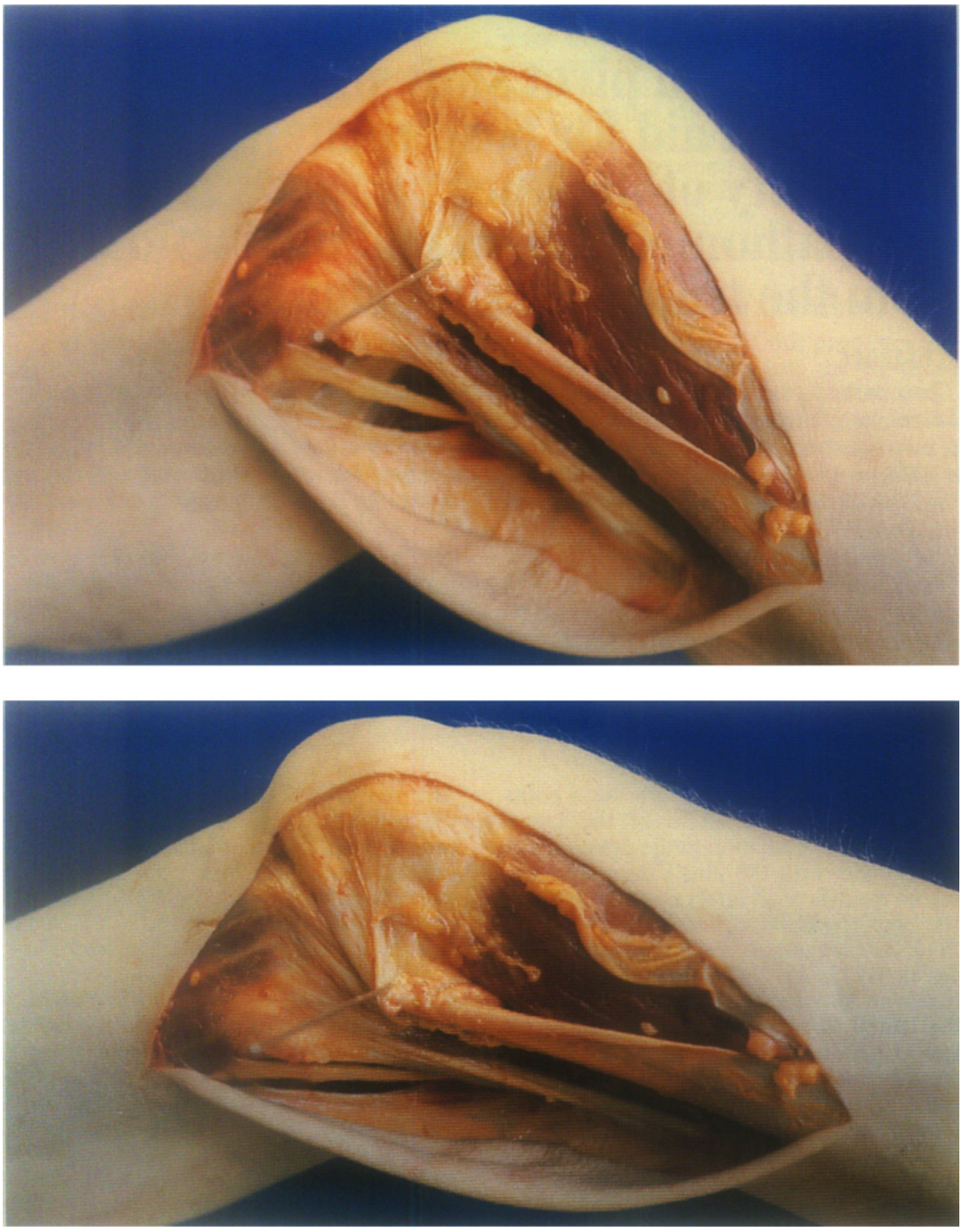

Fig. 11 a, b
Insertion of a Kirschner-wire in front of the iliotibial tract at the femoral epicondyle which immediately prevented any pivot shift sign initially positive after division of the anterior cruciate ligament. The fibers of the intact iliotibial tract deviated posteriorly around the wire got very tight the more the flexed knee (Fig. 11 a, 80 degrees) was extended (Fig. 11 b, 40 degrees) running parallel to the anterior cruciate ligament and keeping the tibia back in reduction (experiment of MacIntosh)

Part II

The reversed pivot shift sign – a new diagnostic aid for posterolateral rotatory instability of the knee (the pathomechanism and distinction from the true pivot shift sign)

Abstract

The reversed pivot shift is a sign occuring in patients with acute and chronic posterolateral instability of the knee.

It consists of a shift of the lateral tibial plateau from a position of posterior subluxation to a position of reduction as the flexed knee is extended with valgus stress and with the foot held in external rotation. It subluxates again as the knee is flexed in the opposite manner.

The manœuvre reproduces the patient's discomfort and simulates his feeling of «the knee giving way». Although similar to the true pivot shift on first inspection it can be clearly distinguished from it by the position of the foot (i. e. external rotation) and by other definite signs of posterolateral instability.

Since it describes a shift of the lateral tibial plateau in the opposite direction from the true pivot shift sign we suggest it be called the «reversed pivot shift sign».

Posterolateral rotatory instability is a posterior rotational subluxation of the lateral tibial plateau in relation to the lateral femoral condyle, with the tibia externally rotating on the axis of an intact posterior cruciate ligament (*Dehaven*, 1978; *Hughston* et al., 1976; *Kennedy* et al., 1977; *Bousquet*, 1972; *Trillat*, 1972, 1977). Anterolateral instability is an anterior rotational subluxation of the lateral tibial plateau in relation to the lateral femoral condyle, with the tibia internally rotating on the axis of an intact posterior cruciate ligament. This last type of instability is one of the most common types of disabling ligamentous instabilities in athletes and it must be distinguished from posterolateral instability (*Losee* et al., 1978; *Cabaud & Slocum*, 1977; *Ellison*, 1975, *Fetto & Marshall*, 1979; *Jakob*, 1980; *Kennedy* et al., 1978). In detecting anterolateral instability there exists, apart from a slight anterior drawer sign of the lateral tibial plateau in neutral rotation of the tibia, a reliable diagnostic sign – the «pivot shift sign». This phenomenon described by *Galway* and *MacIntosh* (1972) not only demonstrates to the examiner the pathomechanics of an excessive anterolateral subluxation but it also reproduces the patient's symptoms of his knee giving way. Similar tests depicting this pathology have been described by *Slocum* et al. (1976), *Hughston* et al. (1976) and *Losee* et al. (1976).

When discussing the etiology of anterolateral instability and the specific ligamentous lesions involved, there is general agreement that for a pivot shift sign to occur the anterior cruciate ligament must be torn or stretched (*Fetto & Marshall*, 1979; *Galway* et al., 1972; *Jakob* et al., 1978; *Losee* et al., 1978; *Norwood* et al., 1979). Furthermore the lateral capsular ligament may tear or be avulsed at the time of the anterior cruciate injury or it may stretch secondarily thus enhancing the instability and the pivot shift sign (*Hughston* et al., 1976; *Woods* et al., 1979). A jerking or snapping sensation is sometimes felt in the lateral compartment in presence of an isolated lesion of the lateral capsular ligament and of the lateral meniscus.

This may be misinterpreted as a positive pivot shift sign.

There is however an important phenomenon which has come to our attention. This has been mentioned independently by others *(Losee* et al., 1978; *Slocum and James*, 1979) but as yet has not been defined pathologically. This abnormality may be difficult to distinguish from a true pivot shift sign and may confuse the examiner. It is a sign imitating the pivot shift phenomenon that must be looked for carefully because its diagnostic meaning and its therapeutic implications are opposite to those of the true pivot shift test. While the true pivot shift sign is diagnostic for anterolateral instability the new phenomenon described here as the «Reversed Pivot Shift Sign» is present in posterolateral instability.

Clinical test for posterolateral rotatory subluxation of the tibia

The patient is placed supine on the examining table. To test the right knee the examiner, facing the patient, lifts the foot and ankle with his right hand, resting it on the right side of his pelvis (Fig. 1). The left hand supports the lateral side of the calf with the palm on the proximal fibula. The knee is moved several times through a full range of motion to decrease muscular resistance. Now the examiner bends the knee to 70 to 80 degrees of flexion. At this position external rotation of the foot and the leg causes the lateral tibial plateau to subluxate posteriorly in relation to the lateral femoral condyle. This is visualized as a posterior sag of the proximal tibia. The knee is now allowed to straighten, using nothing more than the weight of the leg. With the examiner leaning slightly against the foot, an axial load is transmitted through the leg and a valgus

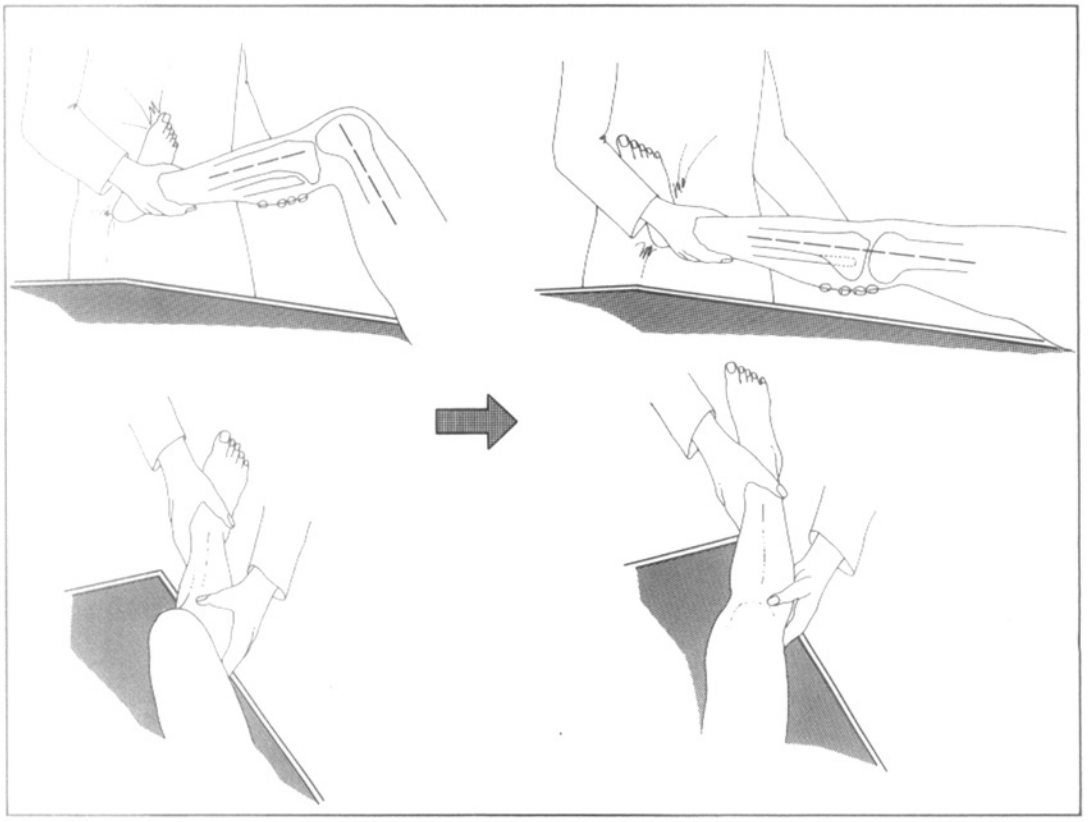

Fig. 1
Demostration of the reversed pivot shift sign (for details see text)

stress is applied to the knee, using the iliac crest as a fulcrum. As the knee approaches 20 degrees lacking full extension one can feel and observe the lateral tibial plateau moving anteriorly with a jerklike shift from a position of posterior subluxation and external rotation into a position of reduction and neutral rotation.

The test may be also carried out by beginning it with the tibia in the reduced position of full extension. In neutral rotation the knee is quickly bent under continuous valgus stress allowing the foot to rotate externally. At about 10 degrees of flexion the same jolt will occur as the tibia falls into posterior subluxation. In this way the two phases of the sign, posterior subluxation and reduction, can be repeated on and off from flexion to extension and back to flexion.

The test is even more pertinent if it reproduces the patient's symptoms. The intensity of the finding of subluxation and reduction and the patient's reaction to it depend on the degree of instability, the skill with which the examiner performs the test, and the patient's ability to relax his muscles.

As is the case with most descriptive findings in the evaluation of rotational instability of the knee, the preceding sequence of events is discussed in terms of the abnormal mobility of the tibia about the relatively fixed femur. In the actual situation it is most likely that the femur exhibits the reciprocal abnormal rotation about the tibia which is fixed by contact of the foot with the ground (Fig. 2).

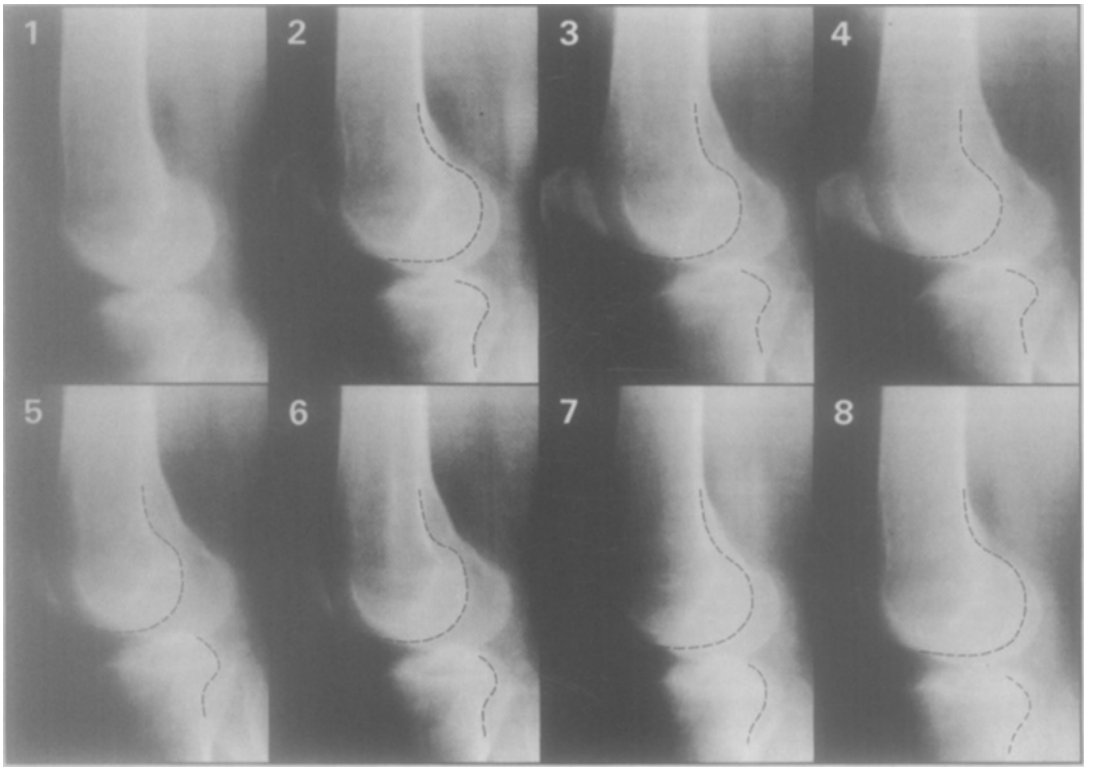

Fig. 2

Sequential pictures of a cineradiogram (frequency twentyfour pictures per second) taken during the reversed pivot shift manoeuvre, elicited from a position of extension to slight flexion and immediately back to extension. For technical purposes the tibia had to be held in neutral rotation. The rotational phenomenon is therefore occuring at the femur, which is probably the case when the patient feels his knee «give way». The lateral femoral condyle and the lateral tibial plateau are marked with dotted lines. Within 1/3 of a second marked anterior displacement of the lateral femoral condyle on the lateral tibial plateau is seen in 10 – 20 degrees of flexion (frames 4 and 5), reducing in extension (frames 1 and 8)

Anatomical and clinical investigation

In an attempt to learn more about the clinical significance and the diagnostic value of this sign, we performed a set of anatomical studies.

Anatomical study

Studies were carried out on fresh human cadavers to examine the pathomechanics necessary to produce a reversed pivot shift sign. Twenty knees from fresh cadaver specimens were dissected one to six hours post mortem. All knees had a full range of motion and none had degenerative arthritis or any suggestion of ligamentous instability. Serial sectioning of the posterolateral structures was performed in various sequences and combinations. The results, as listed in Table 2), summarize the findings as reproduced in two to four knees for each experiment. For purposes of description of the experimental findings «reversed pivot shift» will be refered to as RPS, posterior drawer sign as PDS, internal rotation as IR, neutral rotation as NR and external rotation as ER. In assessing the degree of positivity of the above signs, the number +, ++ and +++ are used to refer to displacements of mild, moderate and marked degrees respectively.

In the first experiment the division of the middle and posterior part of the lateral capsular ligament did not affect the PDS nor the RPS. When sectioning the fibular collateral ligament both the PDS in ER and the RPS became just detectable. Adding section of popliteus tendon and the arcuate ligament both the PDS in ER and RPS immediately increased to ++. The PDS in neutral rotation remained negative. Further section of the lateral gastrocnemius origin did not affect the degree of rotatory instability. Cutting the posterior cruciate ligament made the PDS increase to ++ in NR and +++ in ER while it remained negative in forced IR. The RPS increased to +++.

In the second series section of the fibular collateral ligament created a just detectable PDS in ER and RPS. Adding section of popliteus tendon and arcuate ligament at the joint level, both increased to ++. Section of the lateral capsular ligament or the origin of the lateral gastrocnemius did not add to the instability. Again, upon addition of section of the posterior cruciate ligament the PDS was ++ in NR and +++ in ER; the RPS was +++.

In the third experiment, serial sections were started with the popliteus tendon and the arcuate ligament resulting in a PDS in ER and RPS of +. Adding division of the fibular collateral ligament the PDS in ER and the RPS became ++. Further section of the lateral gastrocnemius origin did not alter the instability. Section of the posterior cruciate ligament however was again associated with an increasing instability as in the previous knees.

In the fourth experiment the posterior cruciate ligament was cut first which resulted in a (+) PDS in NR. Additional section of the lateral capsular ligament did not add to the instability. When sectioning the fibular collateral ligament the PDS in ER and RPS became just detectable. Only after cutting the popliteus tendon and the arcuate ligament did the PDS become ++ in NR and +++ in ER and the RPS +++. Further section of the lateral gastrocnemius origin did not increase the instability.

Finally in the fifth series the section of the posterior cruciate ligament and the lateral capsular ligament created a + PDS in NR while the PDS remained negative in ER. This was followed by section of the popliteus tendon and arcuate ligament resulting in a PDS in ER and RPS of +(+). Section of the fibular collateral ligament made the RPS and PDS in ER become ++. Only upon section of the gastrocnemius origin which was strong, tendinous in two cadavers and adherent to the arcuate ligament did the RPS and PDS in ER increase to +++.

From the experiments we conclude that in the normal knee the reversed pivot shift sign is prevented by the integrity of the combined function of the popliteus tendon, the arcuate ligament and the fibular collateral ligament. The most important single structure to inhibit posterolateral instability is definitely the popliteus muscle and tendon with its expansion to the arcuate ligament. The popliteus tendon and arcuate ligament form a tight functional unit so that during dissection, one cannot be divided without substantial weakening of the other (*Wang &*

Table 2

Ligamentous lesions produced in 20 cadaver knees

Lesions	Posterior Drawer Sign		Reversed Pivot Shift Sign
	Neutral	External	
EXP. I			
SECTION OF:			
LCm/3	–	–	–
plus FCL	–	(+)	(+)
plus PT, AL	–	++	++
plus LG	–	++	++
plus PCL	++	+++	+++
EXP. II			
SECTION OF:			
FCL	–	(+)	(+)
plus PT, AL	–	++	++
plus LCm/3	–	++	++
plus LG	–	++	++
plus PCL	++	+++	+++
EXP. III			
SECTION OF:			
PT, AL	–	+	+
plus FCL	–	++	++
plus LG	–	++	++
plus PCL	++	+++	+++
EXP. IV			
SECTION OF:			
PCL	(+)	–	–
plus LCm/3	(+)	–	–
plus FCL	+	(+)	(+)
plus PT, AL	++	+++	+++
plus LG	++	+++	+++
EXP. V			
SECTION OF:			
PCL	+	–	–
plus LCm/3	+	–	–
plus PT, AL	+	+(+)	+(+)
plus FCL	++	++	++
plus LG	++	+++	+++

PCL = *posterior cruciate ligament*
FCL = *fibular collateral ligament*
PT, AL = *popliteus tendon, arcuate ligament*
LCm/3 = *middle third of the lateral capsular ligament*
LG = *origin of the lateral gastrocnemius tendon*

Posterior Drawer Sign
\+ = *slight*
++ = *moderate*
+++ = *marked*

Reversed Pivot Shift Sign
\+ = *slight*
++ = *moderate*
+++ = *marked*

Marshall, 1977; *Basmajian & Lovejoy*, 1971). Comparing the dynamic strength of the popliteus tendon and the fibular collateral ligament it became obvious that the popliteus tendon is the more important structure. Due to its oblique direction in relation to the posterior side of the knee, it is capable of preventing posterior subluxation of the lateral plateau because it is opposed to the direction of this instability. The fibular collateral ligament however is directed more vertically; thus it is able to prevent varus instability more effectively.

Section of the gastrocnemius origin or of the lateral capsular ligament did not substantially add to posterolateral instability in most of the knees examined. In two cadavers however, the gastrocnemius origin was very strong in both knees and tendinous over the posterior aspect of the joint (as it is the case for the medial head of the gastrocnemius). In these knees it could also not be separated from the arcuate ligament. It became obvious that in these cases section of the gastrocnemius tendon and origin added definitely to the instability. A marked rotational subluxation of the lateral tibial plateau with an obvious reversed pivot shift sign were not detected until the structures of the arcuate complex were divided. When this was combined with section of the posterior cruciate ligament, as in the case of combined lesions, the symptoms became even more markedly positive.

Clinical observation

The reversed pivot shift has been recognised as an indicator of posterolateral rotatory instability in our clinic for two years. The sign has been observed in eleven fresh injuries with lesions in the posterolateral corner, including rupture of the popliteus tendon.

During this period we have observed 14 patients with chronic posterolateral instability, with various complaints. All these patients had a posterior drawer sign of ++ to +++ in ER of the tibia which was definitely less prominent in neutral rotation and which disappeared in marked internal rotation.

Among these were six patients with a positive external rotation recurvatum sign. Five patients were walking with the foot in internal rotation on the involved side.

In all 14 patients we were able to confirm the posterolateral instability with a definite reversed pivot shift sign.

In patients with straight posterior instability of the knee we were never able to elicit a reversed pivot shift since the degree of posterolateral subluxation in this non-rotatory type of instability was always less marked in external rotation.

The reversed pivot shift sign was observed as well in routine knee examinations as in a patient with Ehlers-Danlos-disease and a twelve years old girl with generalised ligamentous laxity. We have also found a positive reversed pivot shift sign in certain rare orthopedic conditions, i.e. femoral hypoplasia and coxa vara congenita. Because of these positive findings in atraumatic conditions, we performed a control study.

Control study

One hundred soldiers of a mountain infantry regiment were examined to find signs of posterolateral laxity and to look for the frequency of a reversed pivot shift sign in otherwise healthy knees. A bilateral reversed pivot shift sign was found in three persons. Two of them had some constitutional ligamentous laxity and some degree of recurvatum. None of them complained of knee problems. In eight other persons we were able to elicit just a slightly positive reversed pivot shift bilaterally. Five of them showed a ligamentous laxity, none of them had definite recurvatum or other pathology. In none of the two hundred knees examined was a true pivot shift present.
The occurence of a reversed pivot shift is therefore only of clinical significance if the sign is observed in one knee only, if the monoeuvre is painful and reproduces the patient's symptoms, if there are other signs of posterolateral instability and there is appropriate history of trauma.

Discussion

The reversed pivot shift sign is a sign which we identified only after we had considerable experience with the pathology of the true pivot shift. The knowledge of the existence of this «reversed» sign is important, because

it brings to the examiners attention that not all jerking movement elicited in the lateral compartment of the knee is indicative of a true pivot shift and represents only anterolateral rotatory instability.

How then does one distinguish between a reversed and a true pivot shift sign?

The pivot shift sign and related tests (*Hughston* et al., 1976; *Losee* et al., 1978; *Slocum* et al., 1976; *Del Pizzo* et al., 1977) indicate anterolateral instability, i.e. an abnormal internal tibial rotation with the lateral tibial plateau subluxating anteriorly in relation to the lateral femoral condyle, the vertical axis of rotation being displaced medially and anteriorly.

The true pivot shift may be divided in three phases (Fig. 3 a). During the first phase of full extension the tibia stays reduced. During the second phase between 5 and 20 degree of flexion the tibia is rotated internally and loaded with a valgus stress and maximal anterior subluxation of the lateral tibial plateau occurs. In the third phase the actual jerk is taking place, as the tibia shifts back into the reduced position with a painful jolt.

If the test is initiated as described above, the actual shift of the tibia will be more accentuated since internal rotation increases the anterior subluxation and therefore the reduction phenomenon. If the test on the other hand is initiated in marked external rotation the true pivot shift may be abolished in presence of pure anterolateral instability or attenuated to greater or lesser degrees in combined anteromedial/anterolateral instability.

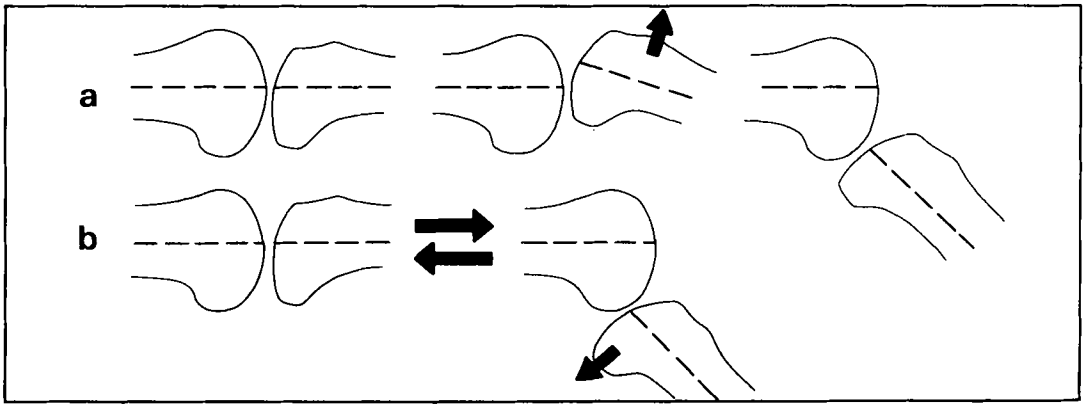

Fig. 3
The displacement of the lateral tibial plateau during the true pivot shift test (a) and the reversed pivot shift test (b). In the true pivot shift test the lateral tibial plateau shifts from a reduced position in extension into anterior subluxation in slight flexion and reduces again at 30 degrees of flexion.
In the reversed pivot shift test the lateral tibial plateau falls from a reduced position in extension into posterior subluxation and flexion.

In the reversed pivot shift test described here, the shift takes place in a reversed fashion (Fig. 3 b). Beginning in the first phase the knee is in extension and the tibia reduced. As one moves the knee into flexion the second phase is initiated – the lateral tibial plateau shifts abnormally into a position of posterior subluxation, producing a visible and palpable jerk. When repeating the manoeuvre in reverse the tibia again shifts in front from posterior subluxation into neutral.

The position of the foot significantly influences the test. Internal rotation of the leg reduces the posterior subluxation of the lateral tibial plateau in presence of true posterolateral instability, the tibial plateau being rotated around a vertical axis which is displaced posteriorly and medially in this condition. External rotation of the tibia however facilitates the occurrence of posterior subluxation and is a prerequisite for the test (Fig. 4). If there is difficulty as to the analysis of the test in terms of the displacement

occuring, there is no doubt that a shift which is more prominent in internal rotation is likely to be a true pivot shift, while a shift getting more marked in external rotation is definitely a reversed pivot shift due to posterolateral instability.

The differentiation between the two signs is therefore extremely important. It is relatively easy when only one type of instability is present in a given knee. Over the last years however it has become increasingly apparent that major injuries to the knee cause combined lesions (*Del Pizzo* et al., 1977; *Dehaven*, 1978; *Hughston*, 1969; *Hughston* et al., 1976). One quite commonly encountered is combined anterolateral and posterolateral instability. It was in such a knee in fact that the reversed pivot shift phenomenon was discovered. That particular patient had a marked anterior drawer test, a slight posterior sag of the proximal tibia and an obvious pivot shift test. He was treated with anterior cruciate ligament reconstruction and a modified *MacIntosh* repair. Six months after surgery his anterior drawer sign was negative, but there still was a definite shift in the lateral compartment. This we could not understand, having been confident in our lateral repair. Only after careful analysis of this instability including additional X-ray studies did we comprehend that we were dealing with a combined instability, in which the posterolateral component continued to express itself and that the phenomenon observed really was a reversed pivot shift.

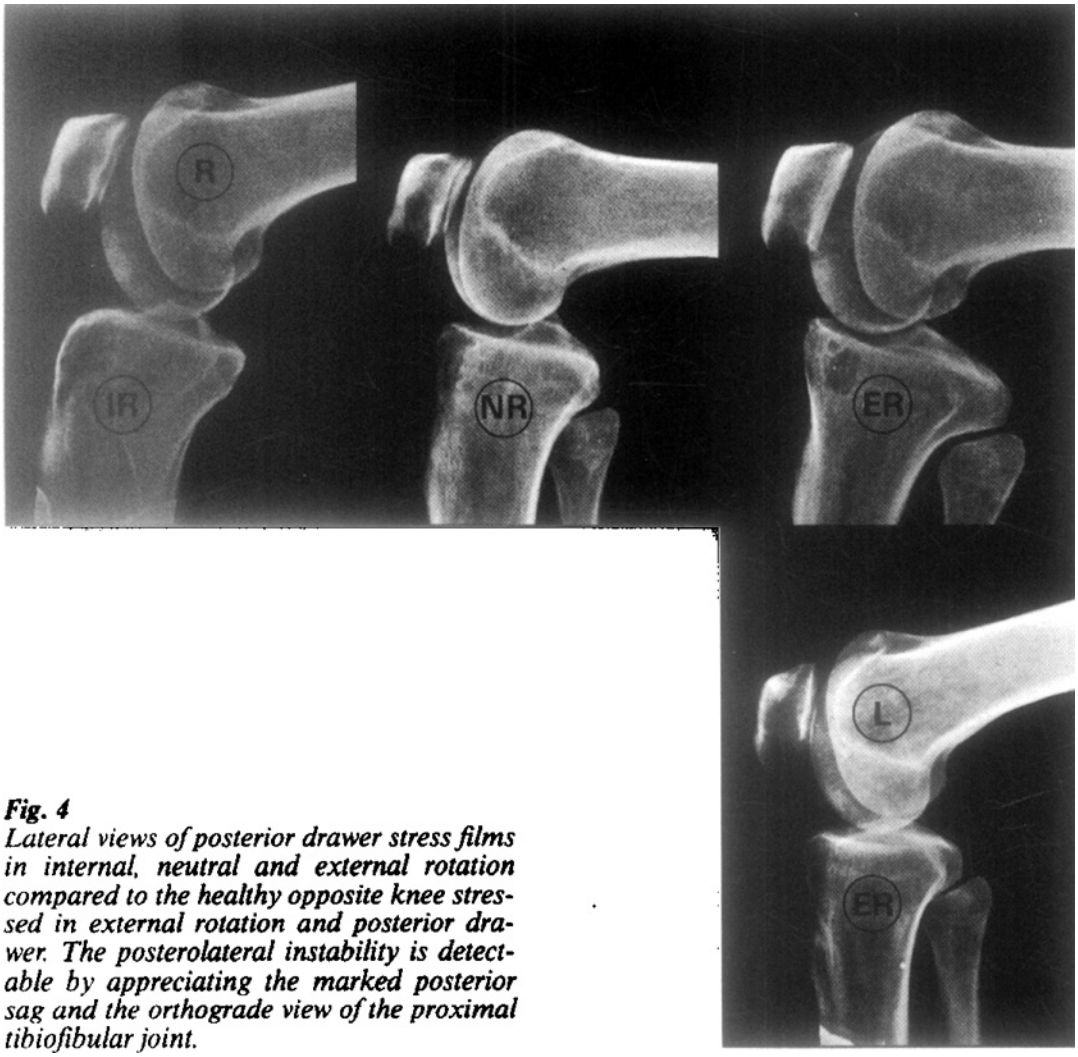

Fig. 4
Lateral views of posterior drawer stress films in internal, neutral and external rotation compared to the healthy opposite knee stressed in external rotation and posterior drawer. The posterolateral instability is detectable by appreciating the marked posterior sag and the orthograde view of the proximal tibiofibular joint.

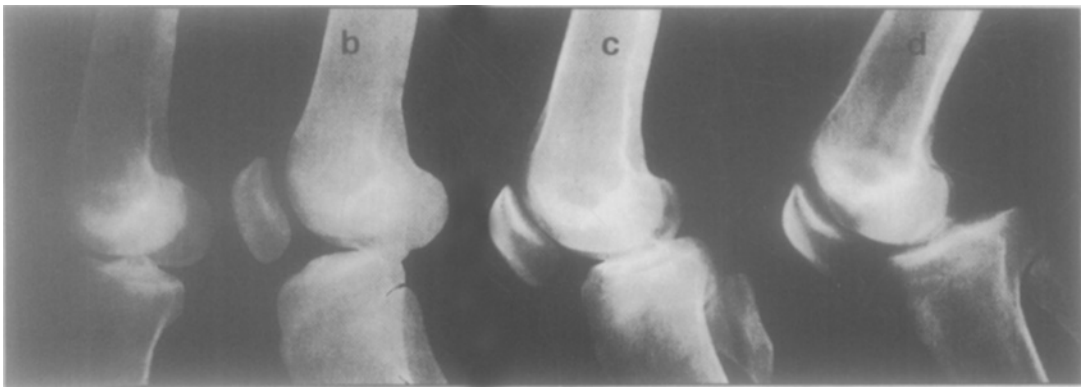

Fig. 5
X-ray study of a cadaver knee in which both cruciate ligaments and the arcuate complex were sectioned. When initiating the pivot shift test the tibia slides from a reduced position in extension (a) to a position of anterior subluxation and internal rotation (b). This is followed by the typical jerklike reduction of the true pivot shift. At 30 degrees there is a normal relationship between tibia and femur (c). When the manoeuvre is continued with more flexion a jerklike shift of the lateral tibial plateau into posterior subluxation will take place – the reversed pivot shift sign (d)

An indication as to the existence of a combined instability is the occurrence of a «double shift» in the lateral compartment (Fig. 5). When starting from a position of extension and internal rotation the knee is flexed a true pivot shift will occur as an expression of anterior subluxation and reduction. As we flex beyond this point, steering the foot in external rotation the tibia continues to shift posteriorly now into a subluxated posterior relationship, the reversed pivot shift has occured.

Cases in which the true and the reversed pivot shift can be clearly distinguished usually represent grossly unstable knees and are not common. In a less obvious, yet significant, combined instability it may be more difficult to keep the two entities separate.

In presence of such indefinite findings other diagnostic aids help to define the degree of posterolateral instability. The most important sign is the posterior drawer test which in the presence of posterolateral rotatory instability is negative in neutral rotation and + to ++ in external rotation of the tibia. In our observation this mild posterolateral instability with an intact posterior cruciate ligament is very rare. More common ist the posterolateral instability with a ruptured or attenuated posterior cruciate ligament which is diagnosed with a + to ++ posterior drawer in neutral rotation of the tibia and ++ to +++ in external rotation and which is negative in internal rotation (Fig. 6).

The relevant clinical finding supporting the suspicion of posterolateral instability is the observation that practically all patients suffering from severe instability show a constant tendency to walk with their involved tibia and foot in marked internal rotation. In this position the patient is at a decreased risk of taking an unguarded step with the occurrence of a reversed pivot shift and giving way of the knee.

Another important finding which substantiates the presence of chronic posterolateral rotatory instability is recurvatum of the involved knee when standing, and a positive external rotation-recurvatum test. In this test the knee will go into relative hyperextension at the lateral side when both legs are lifted up by the great toes, and the tibia will at the same time externally rotate, the tibial tuberosity moving to the lateral side. The medial side of the knee will reveal an apparent bow leg (*Hughston* et al., 1976) (Fig. 7).

In the cadaver knee, when the posterior cruciate and the arcuate complex are sectioned there is no resulting increased recurvatum as long as the anterior cruciate ligament and the posteromedial capsule which act as a

barrier against hyperextension are left intact. In fresh posterolateral injuries the recurvatum is therefore usually not detectable.

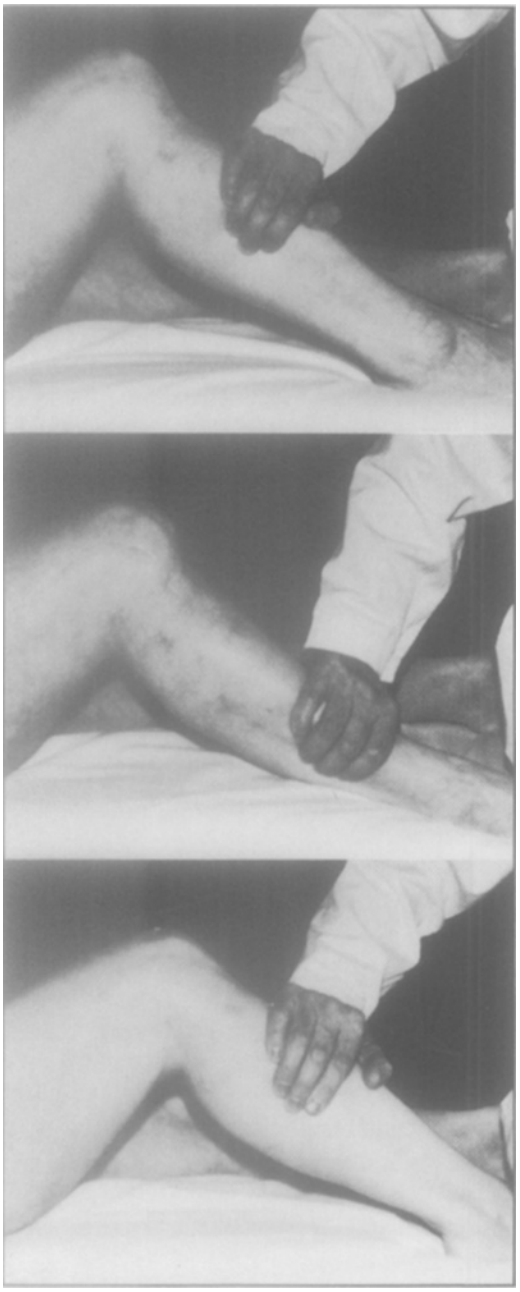

Fig. 6
A posterior drawer sign of ++ in neutral rotation, of +++ in external rotation which becomes negative in internal rotation of the tibia defines a severe posterolateral instability.

Finally there is always a certain degree of varus instability present when the knee is examined in 20 to 30 degrees of flexion. If the posterior cruciate ligament is insufficient as well, the varus instability will be present even in full extension.

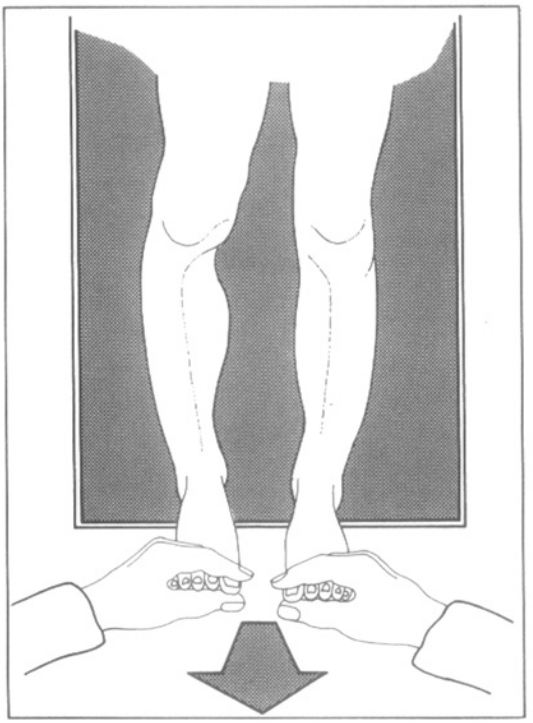

Fig. 7
External rotation recurvatum test to demonstrate posterolateral rotatory instability. The knee is falling into relative hyperextension at the lateral side when both legs are lifted up by the great toes. The tibia will at the same time externally rotate and the medial side will reveal an apparent bow leg (Hughston)

Conclusion

The reversed pivot shift sign is a constant finding in acute or chronic posterolateral instability. It represents the posterior subluxation of the lateral tibial plateau and its jerklike reduction. It only occurs when the tibia is held in external rotation. In the position of internal rotation, which reduces the tibial subluxation, the sign disappears. This allows its clear differentation from the true pivot shift.

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