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Principles and mechanisms of automatic rotation during terminal extension in the human knee joint

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ABSTRACT

The mechanisms solely responsible for automatic rotation during terminal extension were investigated by means of destruction experiments on knee joint structures. These are the posterior cruciate ligament (PCL), the anterior cruciate ligament (ACL) and the curvature of the medial femoral condyle. The first mechanism is based on the extraordinary obliquity of the constantly taut PCL guiding bundle, which produces torque in the final extension stage. The second mechanism depends on the fact that the ACL becomes 'too short' towards the final extension stage, which in turn is due to the shape of the articular surfaces. The third mechanism is caused by the deflection of the medial femoral condyle by the intercondylar eminence of the tibia. Each of the collateral ligaments can, in their own right, prevent excessive automatic rotation. On the one hand, the mechanism of the automatic rotation is reversed during initial flexion by the medial collateral ligament (this would be 'too short' in the case of nonreversal). On the other hand, it is reversed by the popliteus muscle. It is suggested that automatic rotation is not related to securing an amuscular stance (locking mechanism).

INTRODUCTION

This analysis of automatic rotation in the human knee joint arises out of a previously published paper on the cruciate ligaments (Fuss, 1989*a*). Compulsory inward rotation of the femur and outward rotation of the tibia accompanies terminal knee extension.

Three mechanisms are commonly held to be responsible for automatic knee rotation. (1) The anterior cruciate ligament becomes 'too short' during terminal extension (Fick, 1911) and the ligament is hence taut prior to maximal extension, causing rotation with further extension (Braus, 1921). This statement was limited to the anterior fibre bundles by Sieglbauer (1930). The articular surfaces were held responsible for the other 2 mechanisms. (2) The laterally oriented curvature of the (hence 'longer') medial femoral condyle (Langer & Toldt, 1902; Cunningham, 1939) causes automatic rotation in sliding past the medial tubercle of the intercondylar eminence (Gerrish, 1899). (3) The lateral 'shorter' femoral condyle was 'used up' before the 'longer' medial one, resulting in rotation (Barnett, 1953; Williams & Warwick, 1980).

As observed by Meyer (1853), to whom the discovery of automatic knee rotation is to be attributed (Fick, 1911), the purpose of the latter would be to lock the knee in an upright stance, i.e. to secure it (Benninghoff, 1985). This finally led to the expression 'locking the knee joint', referring to the mechanism responsible for the amuscular stance (Cunningham, 1939; Jones, 1946; Barnett, 1952, 1953).

The present investigation is based on findings from personal studies on the function of the cruciate ligaments (Fuss, 1989*a*, 1991*a*, *b*, *c*). The cruciate ligaments consist of 3 functionally different fibre groups which are differentiated according to the flexion–extension positions in which they are taut: permanently taut fibres ('guiding bundles'; Fuss, 1989*a*); fibres taut in intermediate positions; and fibres taut in extreme positions. The fibres taut in extreme positions restrict extension and/or flexion (Fig. 1*a*, *b*), and the others guide the movement of the joint.

The aim of this study is to clarify the mechanisms and causes of automatic rotation occurring involuntarily during terminal knee extension. Both the restriction of automatic rotation in maximal extension as well as its reversal on initiation of flexion will be

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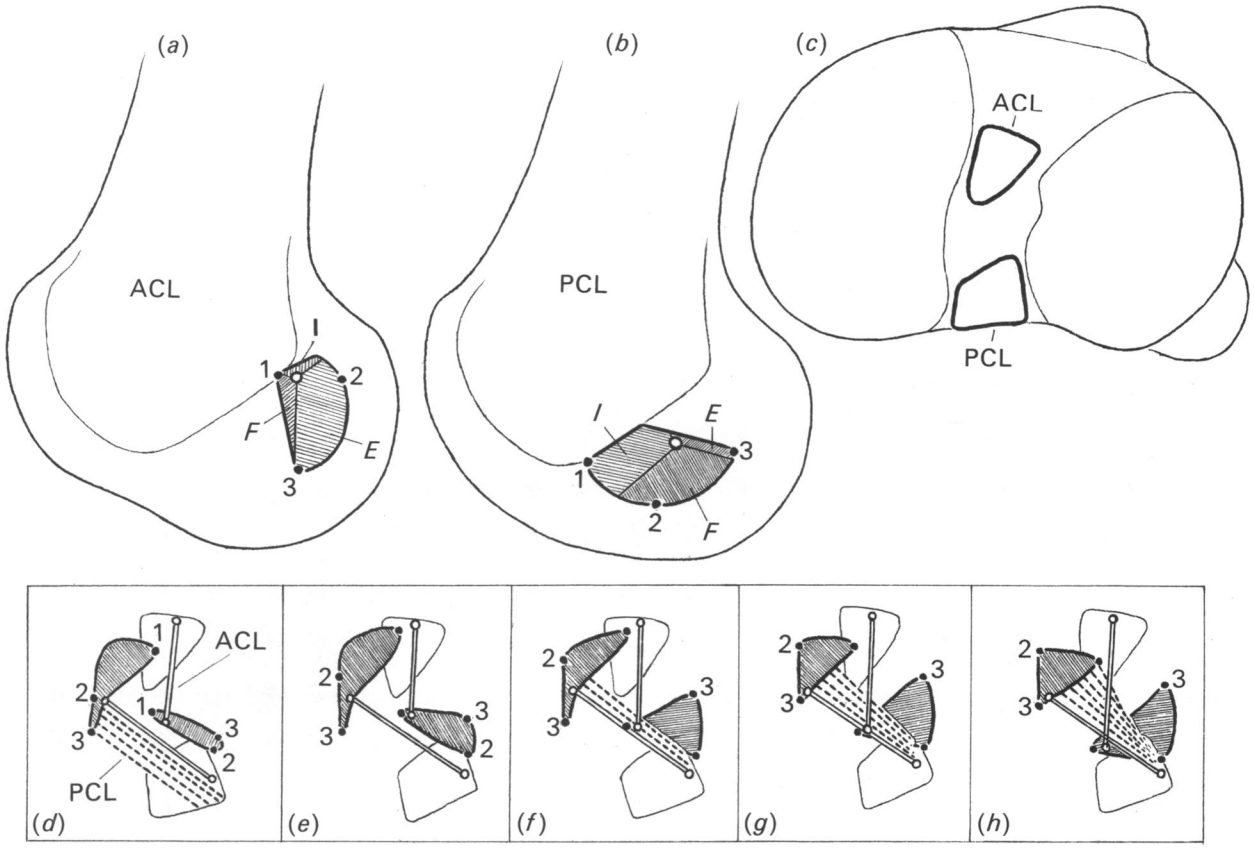

Fig. 1. (*a*, *b*) Femoral attachments of the cruciate ligaments. The cruciate ligament subunits are taut in extension (*E*), in maximal flexion (*F*), in intermediate positions (*I*), or permanently – 'guiding bundles', originating in the white circle (according to Fuss, 1991 *a*); the points marked with the numbers 1, 2 and 3, on the circumferences of the originating areas correspond to those in Figures (*d*)–(*h*), and Figures 4, 5 and 6. (*c*) Attachment locations of the cruciate ligaments to the tibial plateau. (*d–h*) Projection of the femoral cruciate ligament attachments, the guiding bundles and selected PCL fibres onto the tibial plateau in different positions. The permanently taut guiding bundles are doubly lined, the remaining taut PCL fibres are dotted. (*d*) Maximal extension; (*e*) 15° flexion; (*f*) 45° flexion; (*g*) 70° flexion; (*h*) 90° flexion.

considered. The applied method was based on the principle of an exclusion diagnosis; if the destruction of a knee structure does not change the joint mechanism, the structure in question cannot be responsible for it.

MATERIALS AND METHODS

Only fresh knee joints from the cadaver material available to the First Department of Anatomy were evaluated. The animal joints were provided by the University of Veterinary Medicine. The joints, after removal of the anterior and posterior portions of the articular capsule, were examined for intra-articular pathological changes. Only joints showing intact cartilage, menisci and cruciate ligaments were used. The collateral ligaments were also prepared and inspected for intactness. Fifty human knee joints (from individuals aged 38 to 59 y), 5 pig, and 2 dog knee joints were used for the following analyses.

The synovial layer of the cruciate ligaments was removed in 10 joints. The boundaries of the femoral and tibial attachments (as far as accessible) of the cruciate ligaments were marked by brass nails (0.8×11 mm), which were interconnected by thin copper wire. The tibial shaft was cut in a horizontal plane parallel to the tibial plateau, slightly distal to the attachment of the medial collateral ligament. Five sagittal and 5 horizontal (parallel to the tibial plateau) radiographs were taken of each joint, in positions varying from extreme flexion to extreme extension. The femur was fixed so that the axis of the tibial shaft was moved parallel to the sagittal film. The sagittal and horizontal diameters of the cruciate attachment areas were measured on the tibial plateau and projected onto the films, where the tibial attachments had already been partly marked by the shadows of the nails and wire. This procedure served to evaluate the variations in the 3-dimensional distance between the attachment areas of the various cruciate fibre bundles, as explained in detail in previous publications (Fuss, 1989 *a*, 1991 *a*, *b*, *c*). The radiographs taken in 2 planes also served to evaluate the special arrangement of the cruciate fibre bundles.

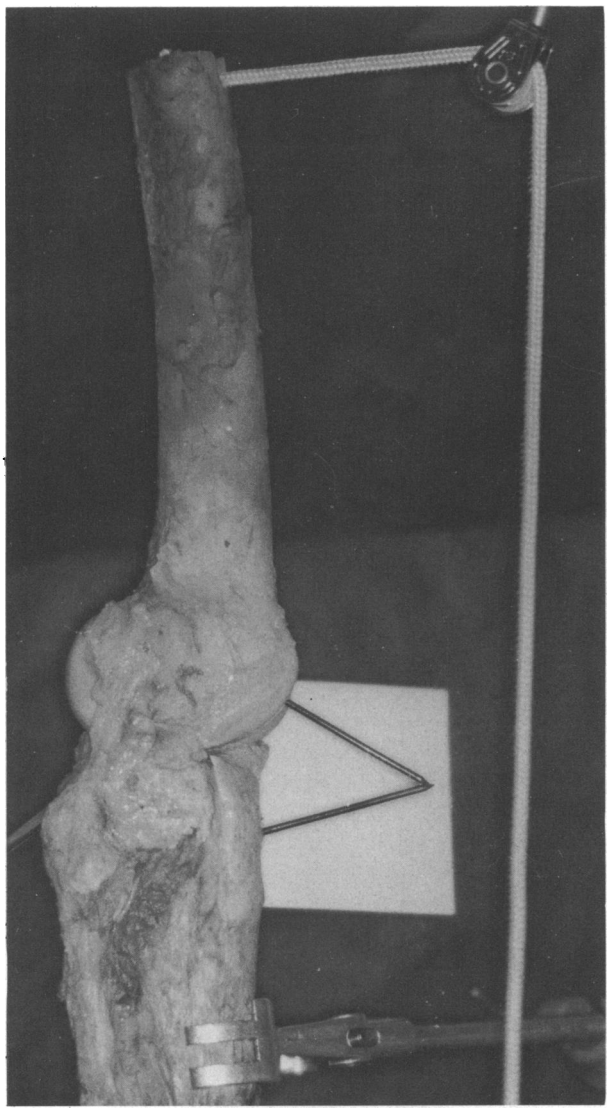

Fig. 2. Experimental arrangement: fixed tibia, nails inserted in femur and tibia (see Fig. 3), moment applied to the femur.

Forty knees were used for the destruction trials. The tibia was fixed in a clamping device. An extension moment of approximately 10 Nm was applied to the femur (distance between joint centre and application point of force = 0.2–0.25 m; force perpendicular to the latter distance = 40–50 N; see Fig. 2). Nails (2.5 mm × 10 cm) were driven into the front of the femur and tibia of each of the knees. Anteroposteriorly these nails converged so that their front ends touched in maximal extension (Figs 2, 3*b*). A nail deviation in the horizontal plane indicated either an augmentation (Fig. 3*a*) or a diminution (Fig. 3*c*) of the automatic rotation. The nails were positioned in the following way. The femoral nail was driven 4 cm into the facies patellaris whilst the tibial one was driven 4 cm into the tibial tuberosity, medial to the attachment of the patellar ligament. The distance between these 2 insertion points in maximal extension (i.e. extension at a moment of 10 Nm) was approximately 4.5 cm; the nails hence converged at an angle of approximately 45° (Fig. 2). This standardised procedure served to evaluate functional discrepancies (Fig. 3*a*, *c*). One structure was then destroyed, the knee was extended (i.e. the extension moment was applied) and automatic rotation was examined. This was repeated until the automatic rotation either failed to appear or was amplified. The last structure to be destroyed would then be the one to have kept the mechanism for automatic rotation intact. The following structures were destroyed: the anterior cruciate ligament (ACL), the posterior cruciate ligament (PCL), the medial collateral ligament, the lateral collateral ligament and the curvature, i.e. the anterolateral portion of the medial femoral condyle (Fig. 4), which articulates with the intercondylar eminence. Menisci and articular surfaces essential to the flexion–extension process were left intact. Any changes in automatic rotation were noted after each destruction step (Fig. 3*a*, *b*, *c*). If no alteration occurred, an attempt was made to neutralise automatic rotation manually in full extension and see if the automatically rotated position could be maintained when flexing the joint. These destructions were undertaken in varying sequences (see Table).

In 5 knees in which all structures that could maintain the automatic rotation had been destroyed (see Results) and in which the collateral ligaments were left intact, 2 PCL fibre bundles were reconstructed by fixing wet catgut strings (diameter 0.5 mm) to the femur by means of small screws (Fig. 6). One string corresponded to the guiding bundles (see Results), i.e. from the lateral surface of the medial femoral condyle running obliquely in a lateroposterior direction. The other string originated deep in the intercondylar fossa and ran purely sagittally in a posterodistal direction. It is referred to as the 'sagittal bundle' and belongs to a fibre group which, according to earlier findings (Fuss, 1989*a*), is taut in a particular intermediate position (between extreme extension and extreme flexion). For this experiment, however, it was necessary that this fibre group be permanently taut. This was attained by not anchoring the reconstructed unit to the tibia but pulling it through a drill hole, the axis of which corresponded to that of the tibial shaft. This hole was drilled into the PCL attachment area (Fig 6). The string corresponding to the guiding bundle would, according to its definition, be constantly taut if correctly attached to the tibia. It was nevertheless also forwarded to the same tibial drill hole. Each of the 2 filaments was then alternately stressed by means of 0.5 kg weights so as to ensure

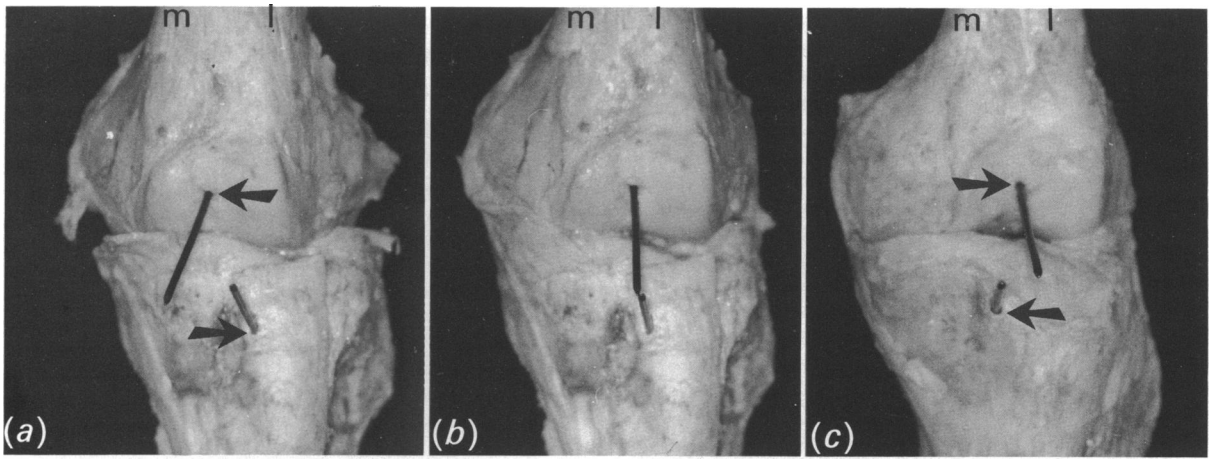

Fig. 3. Deviations of the nail ends in the horizontal plane; m, medial; l, lateral. (*a*) Amplified automatic rotation (increased outward rotation of the tibia); (*b*) normal rotation (unchanged outward rotation of the tibia); (*c*) diminished automatic rotation (decreased outward rotation of the tibia).

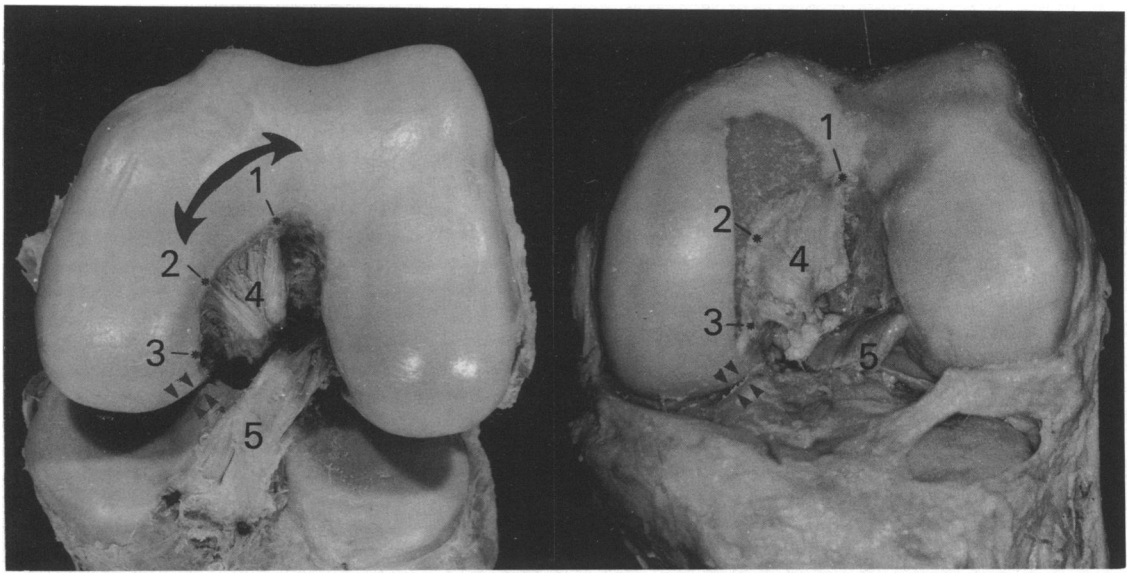

Fig. 4. Curvature of the medial femoral condyle; (*a*) intact, (*b*) destroyed (both cartilage and bone milled away). 1, 2, 3: border between the medial femoral condyle and the femoral PCL attachment; 4: PCL; 5: ACL; curved arrow: curvature of the medial femoral condyle. The arrowheads point to the corresponding articular surfaces of the medial femoral condyle and tibial intercondylar eminence.

permanent tension while the knees were extended in the manner described above. The positions of the nail ends relative to one another were noted.

The popliteus muscle was reconstructed by similar means in 5 knees. The catgut string was inserted at its femoral origin whilst the filament was drawn through a U-shaped nail at its tibial attachment and stressed by weights (see Results).

Five fresh pig and 2 dog knee joints were used to establish whether or not these animal knees also display automatic rotation.

RESULTS

The causes of automatic rotation

The destruction experiments showed that the ACL, the PCL and the curvature of the medial femoral condyle (Fig. 4) are all responsible for automatic rotation when the collateral ligaments are intact. Whether this counts as diminution or even as neutralisation of rotation (Fig. 3*c*) cannot be established, as a comparison with a knee joint without automatic rotation (nonexistent) would be necessary. The distance between the nail ends ranged from 0.5 to 1.5 cm. It should be noted that, after the destruction of all 3 structures, the medial femoral condyle was still longer than the lateral one.

The PCL mechanism. After destruction of ACL and the anterior curved portion of the medial femoral condyle, automatic rotation can be prevented by simultaneous manual outward rotation of the femur.

Table. *Destruction trials – sequences and results*

Sequences of destroyed structures	Behaviour of automatic rotation	Number of examined knees
ACL PCL Curv.	O O D	5
PCL ACL Curv.	O O D	5
PCL Curv. ACL	O O D	5
ACL Curv. PCL	O O D	5
Curv. PCL ACL	O O D	5
Curv. ACL PCL	O O D	5
Medial collateral ligament Lateral collateral ligament	O I	5
Lateral collateral ligament Medial collateral ligament	O I	5

Curv., destroyed curvature of the medial femoral condyle, see Fig. 4; O, unchanged automatic rotation, see Fig. 3*b*; I, increase, see Fig. 3*a*; D, decrease, see Fig. 3*c*.

The mechanism of the automatic rotation induced by the PCL is hence noncompulsory. Now, which torque actually causes the rotation?

The curvature of the femoral cruciate attachments and the guiding bundle origins in them lead to the greater obliquity of the PCL guiding bundle path in comparison with that of the ACL (Fig. 1*d–h*). This extraordinary obliquity of the PCL guiding bundle, which is not attached to the middle of the intercondylar fossa but to the lateral surface of the medial femoral condyle, is obviously responsible for the generated torque (Fig. 5*b*). Hence the following hypothesis results: an oblique PCL guiding bundle causes torque (Fig. 5*b*), a hypothetical 'sagittal bundle' does not (Fig. 5*a*). In order to test this hypothesis, the following model trial was executed. In knees with intact collateral ligaments and all 3 automatic rotation mechanisms destroyed (hence with neutralised automatic rotation), the actual PCL guiding bundle and the hypothetical 'sagittal bundle' were reconstructed (Fig. 6). With a constantly taut oblique fibre bundle (see Methods), the automatic rotation reappeared; with a constantly taut 'sagittal bundle' no rotation in the horizontal plane of the knee joint could be observed. Our hypothesis was therefore confimed.

The ACL mechanism. In contrast to the PCL mechanism, the automatic rotation of a knee joint equipped with the ACL only cannot be neutralised manually at any stage. This is hence compulsory. If one flexes a few degrees from maximal extension and attempts to counteract the automatic rotation, this will not be possible until the osseous attachment of the most anterior ACL fibres – the fibre group in which lies the guiding bundle – is torn out.

The mechanism of the curvature of the medial femoral condyle. The articular surface at the lateral surface of the medial femoral condyle glides on the

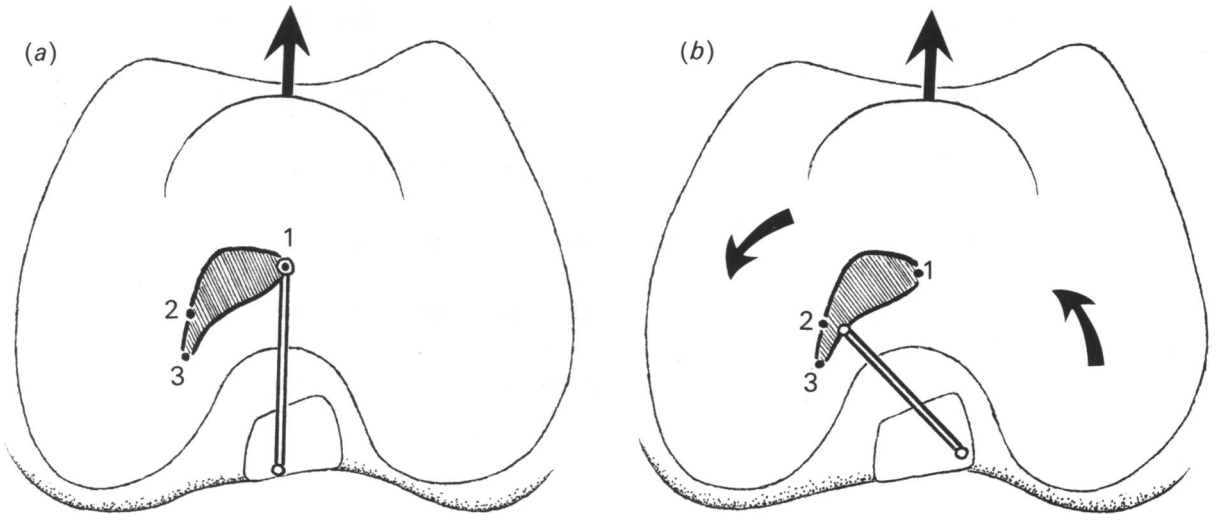

Fig. 5. Femur just prior to maximal extension; the forward oriented arrow shows the extension direction of the femoral shaft. (a) Position of the hypothetical 'sagittal' PCL guiding bundle. (b) Position of the real PCL guiding bundle. The curved arrows show the direction of rotation.

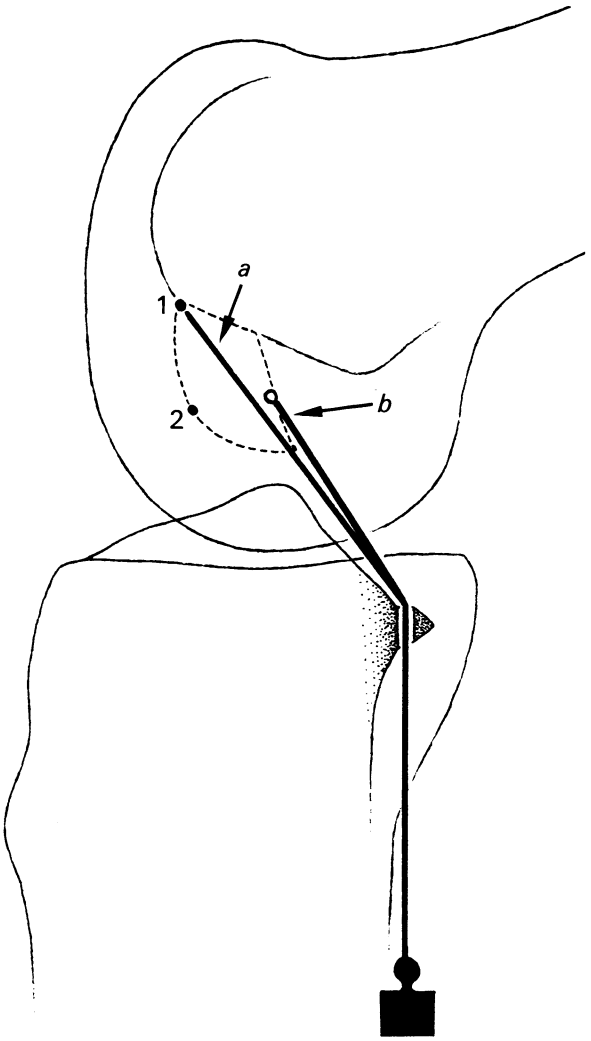

Fig. 6. Reconstruction of the hypothetical 'sagittal' (*a*) and of the real (*b*) PCL guiding bundles.

corresponding articular surface of the medial tubercle of the intercondylar eminence. This femoral articular surface is continued in a laterally oriented curvature (Fig. 4). When this curvature glides past the medial tubercle of the intercondylar eminence, a compulsory rotation must follow, as the femur (i.e. its curvature) would otherwise enter the eminence. This mechanism cannot be inhibited, nor can the resulting rotation be neutralised (either prior to or in maximal extension). The curvature of the medial femoral condyle corresponds to the curvature of the PCL originating area (Fig. 4).

The restriction of automatic rotation

It was not possible to increase the rotation in question manually in any period of the automatic rotation phase, irrespective of whether the knee was entirely intact or not. An increase in rotation (Fig. 3*a*) could only be obtained after destroying both collateral ligaments (Table). Removing one ligament was insufficient. The distance between the nail ends ranged from 1.5 to 3 cm. Thus the medial and lateral collateral ligaments both independently prevent excessive automatic rotation.

The reversal of automatic rotation in flexion

Attempts to maintain forcibly the automatically rotated position while flexing the knee joint manually from maximal extension will not succeed until the medial collateral ligament is severed. The maintenance of the rotated position leads to a clear increase in distance between the attachments of the medial collateral ligament. (The lateral one was left intact in this experiment and had no influence on the reversal of the automatic rotation.) Automatic rotation is hence reversed by the medial collateral ligament as it would prove 'too short' if the reversal of the automatic rotation is prevented manually. This compulsory mechanism is identical to the one causing automatic rotation by means of the ACL. Hence the ACL and the medial collateral ligament are *antagonists* with respect to compulsory rotation at the end of extension/beginning of flexion. When the popliteus muscle was replaced by a stressed catgut string, automatic rotation was reversed, even when the medial collateral ligament was severed. This could, however, be prevented by a manual counter-torque, the point being that this is not a compulsory mechanism. Thus the PCL guiding bundle and the popliteus muscle are *functional antagonists* with respect to automatic rotation only.

DISCUSSION

The causes of automatic rotation

The results show that several mechanisms are independently responsible for the automatic rotation that accompanies extension of the knee joint. In the publications cited in the Introduction, either the ACL or the curvature of the medial femoral condyle, or the longer medial femoral condyle were advanced as determining factors. However, no previous study portrayed these 3 mechanisms as acting simultaneously. The imbalance of the femoral articular surface (i.e. the longer medial femoral condyle), can no longer be considered to be a determining factor. This discrepancy cannot maintain the rotation mechanism upright after the destruction of the ACL and PCL, and the curvature of the medial femoral condyle. Hence the unequal length of the femoral condyles has only a minor influence on automatic rotation; but this

influence cannot be completely denied. This would be the case only if the rotation were completely neutralised after the destruction of the 3 above-mentioned structures, which, however, cannot be proved. Nevertheless, a 3rd determining factor of automatic rotation exists: the oblique PCL guiding bundle with its bearing on torque. This mechanism was neither observed nor reported on by previous investigators.

The PCL as a whole was drawn on for an explanation of the automatic rotation mechanism. One explanation was based on the overcrossing of the cruciates, pulling in opposite directions and hence 'screwing' the knee (Haines, cited by Barnett, 1953, and by Shaw & Murray, 1974). The same mechanism was advanced by Menschik (1975): 'in order to gain the full length of the cruciates' the tibial plateau would have to rotate so that the cruciates would run nearly parallel to the direction of motion.

Kapandji (1985) stated that the tension of the PCL in extension leads to an outward rotation of the tibia. This statement, which was not further elaborated, caused the translator of Kapandji's book into the German language, J. Koebke (1985), to add a footnote stating that 'according to common perception' the automatic rotation is due to the unwinding of the taut cruciates.

Shaw & Murray (1974) determined the rotation pattern of the intact knee and stated that a severed PCL led to no alteration; a severed ACL, however, led to an additional 'anterior skid' of the tibia relative to the femur, irrespective of whether or not the PCL was intact. These results agree with our own findings: in spite of severed cruciates the curvature of the medial femoral condyle is able to maintain the principle of automatic rotation.

The dog and pig knees showed weak automatic rotation. As, in these species, the slight curvature of the medial femoral condyle lacks articular contact with the intercondylar eminence, it appears probable that this mechanism represents the last link in the motion caused. This probably correlates with human upright posture. The oblique PCL guiding bundle appears to be the decisive factor of the mechanism in question. This would then activate the ACL mechanism: the automatic outward rotation would otherwise lead to a relaxation of the ACL and hence cause instability.

Automatic rotation and the amuscular stance

Two opinions dominate the literature: one maintains that this is the essence of the automatic rotation, i.e. locking the knee in extension and hence securing the amuscular stance, first stated by Meyer (1853). According to the second opinion, automatic rotation is defined as 'a small movement of no particular significance' (Haines, cited by Barnett, 1953). Apparently, Haines stands alone in his opinion; practically all other authors follow the first opinion. Barnett (1953) restricted this teleologic thinking pattern as he stated that 'the alleged cause of the femoral rotation may be in fact merely its result'.

The mechanism of the amuscular stance has been explained as follows. Barnett (1953) stated that the automatic rotation is reversed ('unlocking') faster at the beginning of flexion than it appears in the final extension phase. Benninghoff (1985) explained this principle more radically: the knee cannot be flexed prior to undoing the automatic rotation. This implies that a pure inward rotation of the tibia would have to precede a pure flexion. Automatic rotation, however, cannot be undone faster than it appears.

Another argument against the 'locking theory' is that dogs and pigs display automatic rotation although only the quadriceps compensates the knee flexion tendency of ground-reaction forces. On the other hand, equine and bovine species (as well as giraffes; Künzel & Forstenpointner, 1990) compensate for the flexion tendency by the patellar mechanism. The human amuscular stance functions when the ground-force vector lies in front of the knee axis. The extension tendency is then compensated for by the knee ligaments. The term 'locking' can, however, be related to the rotatory stability in extension, as the collateral ligaments prevent outward, and automatic rotation prevents inward, tibial rotation respectively.

The reversal of automatic rotation

Reversal of the automatic rotation is neither due to the medial femoral condyle (Gerrish, 1899) nor to the popliteus muscle alone (Barnett, 1953; Williams & Warwick, 1980). Both mechanisms together have never been proposed as being or observed to be responsible for reversal of automatic rotation. The mechanism of the popliteus muscle lies in the fact that the distance between its attachments increases during flexion (Fuss, 1989*b*) and that its muscle tone alone suffices to counteract an elongation at the commencement of flexion, hence supporting the reversal of automatic rotation.

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