BIOMECHANICS OF THE POSTERIOR CRUCIATE LIGAMENT AND DESIGN OF A SYNTHETIC REPLACEMENT

by

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ABSTRACT

Review of the literature showed that there was little information and less agreement on the function of the posterior cruciate ligament (PCL) and the best method of treating its rupture. A series of cadaveric studies were designed to establish the structure, function and mechanical properties of the natural ligament. An anatomical study showed that it was possible to divide the ligament by blunt dissection into anterolateral (aPC) and posteromedial (pPC) bundles based on reciprocal tension patterns near extension. Tensile testing showed that the aPC had significantly superior mechanical and material properties (to calculate material properties a new method of soft tissue cross–sectional area measurement was developed). Selective cutting of the two bundles was used to calculate their percentage contribution to resisting posterior drawer over the range of flexion. Miniature pressure transducers were implanted into the two bundles to measure the load in each bundle in response to a posterior drawer force. It was shown that the function of the aPC was to resist posterior drawer from partial flexion to full flexion and that the function of the pPC was to resist posterior drawer in full flexion. It was also shown that structures other than the PCL resisted the majority of a posterior drawer force at low flexion angles. It was concluded that reconstruction of the aPC would give posterior stability below full flexion. In vitro tests of a synthetic aPC implant in a PLC deficient knee showed that normal posterior stability could be restored below 120° flexion. A clinical trial was started. Understanding of the PCL has been improved and clinical recommendations made for restoring posterior stability: 'Secondary' structures are very important near extension, reconstructing the aPC is sufficient for most activities, for stability in deep flexion the pPC and the aPC reconstructed.

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1. INTRODUCTION

The aim of this project was to advance the understanding of the biomechanics of the Posterior Cruciate Ligament (PCL) with a view to improving the treatment of PCL rupture. A series of studies were designed to establish the structure, function and mechanical properties of the natural posterior cruciate ligament. The results of these studies were used to design a synthetic implant and associated surgical tools.

This introductory chapter continues with a review of the literature that is relevant to the understanding of the PCL, PCL injury and its subsequent treatment. In the course of the review the author has attempted to reconcile the contradictions of previous studies and to demonstrate the need for further investigations. A brief description of the anatomical terms and frame of reference used in this thesis can be found in appendix A.1.

1.1 ANATOMY OF THE PCL

1.1.1 Fascicular Anatomy

In the cruciate ligaments collagen fibres are grouped together to form subfascicles which are covered by a thin layer of connective tissue, the endotenon (Figure 1.1). Thicker connective tissue, the epitenon, binds the subfascicles together to form the fascicles that are visible to the naked eye (section 3.1). These fascicles are enclosed by the paratenon and the synovium (Burks, 1990).

The posterior cruciate ligament, or PCL, originates from the medial side of the femoral intercondylar notch and inserts into the central posterior area of the proximal tibia. This statement is uncontroversial, however, studies of PCL anatomy have shown considerable variation in details.

Studies from the early part of the century considered the PCL as a single functional unit and did not discuss the pattern of fibres within the ligament (Smith, 1918; Hey Groves, 1920). Later studies attempted to provide a more detailed description: In 1938 both Horwitz and Palmer described a thick anterior and a thin posterior portion of the

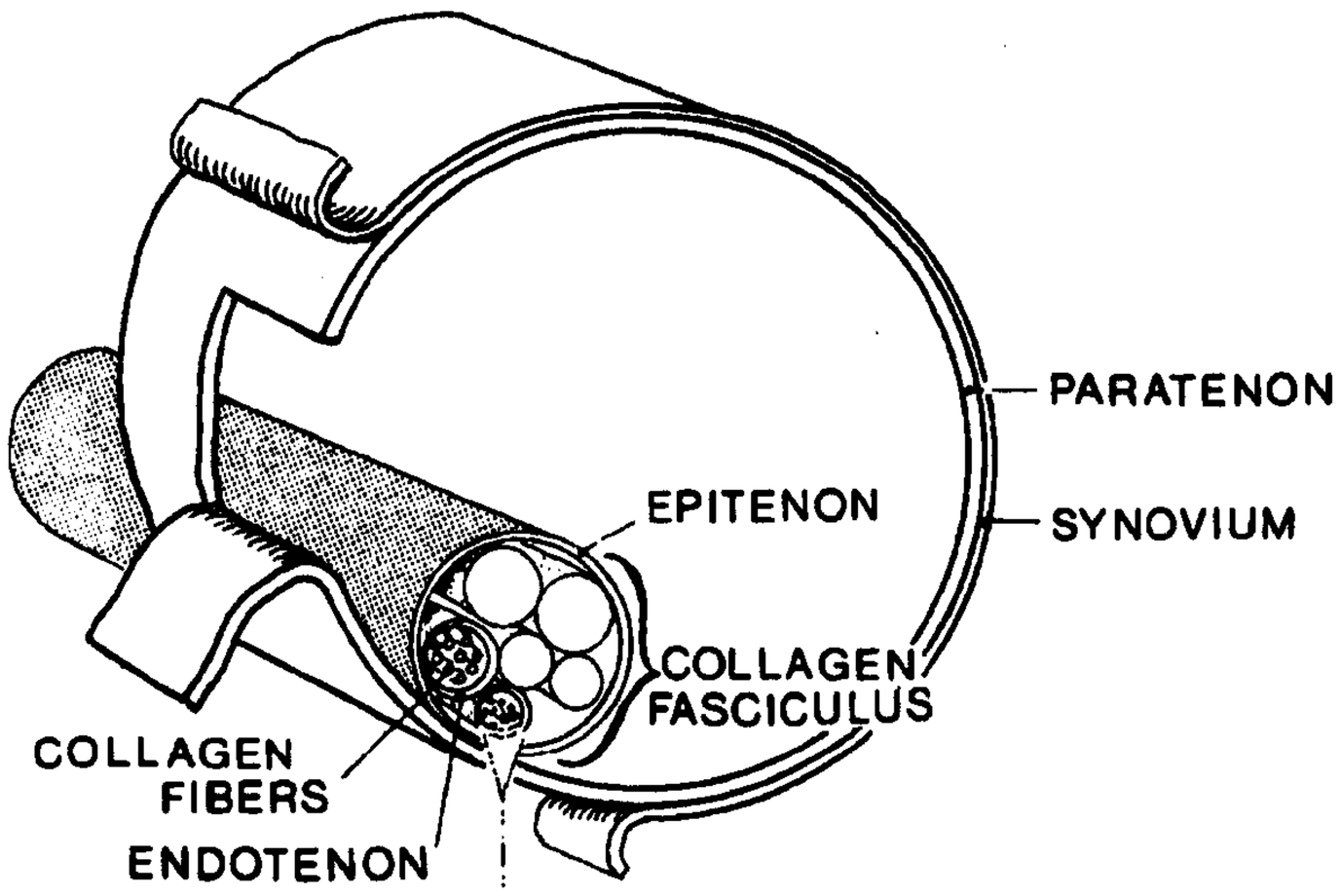


Figure 1.1 Diagrammatic representation of a cruciate ligament. From Burks (1990).

PCL. Palmer called these anterior and posterior portions the 'flexion' and 'hyperextension' sections respectively. This division of the PCL has since been reasserted, most notably by the study of Girgis et al (1975) who described two inseparable fibre bundles; a thick anterior bundle and a smaller, oblique, posterior bundle (There was a small error in Girgis et al (1975), when the ACL and PCL femoral insertions were drawn on condylar profiles they were mixed up, showing the PCL on the lateral condyle and the ACL on the medial condyle. This is mentioned because of the frequency with which these images have been reproduced). The two bundle structure has been supported by many studies (Kennedy and Grainger, 1967; Cross and Powell, 1984; Sepulchre and Blaimont, 1986; Van Dommelen and Fowler, 1989; Burks, 1990; Kannus et al, 1991; Whipple and Ellis, 1991) and is the conventional wisdom accepted by Gray's Anatomy, in which Girgis et al (1975) are quoted. These bundles are known as the 'anterolateral' and the 'posteromedial' or 'oblique' and are referred to in this thesis as the aPC and pPC respectively.

Although the PCL structure described by Girgis et al (1975) is widely accepted there has been some variation on the two bundle theme. Hughston et al (1980) claimed that the aPC originates from the medial side of the femoral notch and inserts into the lateral side of the tibial fovea and that the pPC is shorter and thicker than the aPC and runs without lateral or medial deviation from the medial side of the femoral notch to the medial side of the fovea of the tibia. Claes and Dürselen

(1990) observed that there are three bundles: smaller anterior and posterior bundles either side of a thicker 'middle' bundle. Kapandji (1987) described 'anteromedial' and 'posterolateral' bundles.

Some studies have dismissed the 'bundle' hypothesis and have described the PCL as a continuum of straight fibres which fan out on the femur (Brantigan and Voshell, 1941; Abbott et al, 1944; Fuss, 1989; O'Brien et al, 1992; Friederich 1992). Appel and Gradinger (1989) have claimed to show that the PCL, and also the ACL, are made up not of fascicles containing parallel fibres but rather of branching fibre bundles which mesh to form a continuous structure.

The controversy in the literature suggests that further investigation is warranted.

1.1.2 Vascular Anatomy

The PCL, along with the ACL is predominantly supplied by the middle genicular artery, a branch of which enters the joint through the posterior capsule (Gray's Anatomy). Both cruciates have greater vascularity near their insertions than in their middle portions (Amiel et al, 1990). The PCL has been shown to be surrounded by a greater density of vessels than the ACL (Amiel et al, 1990) and there is a current clinical debate as to whether this gives the PCL a greater capacity to heal. Although the cruciates are extra–synovial it has been demonstrated that they can take up nutrients from the synovial fluid which surrounds them by a process of diffusion (Amiel et al, 1990). The take up of nutrients from the synovial fluid has implications for the healing of cruciate reconstructions which are placed intra–synovially.

1.1.3 Neural Anatomy

The cruciate ligaments are predominately innervated by the posterior articular nerve which branches from the tibial nerve to form the popliteal plexus which penetrates the posterior capsule (Barrack and Skinner, 1990). Pain receptors have been found in the inter–fascicular spaces occupied by blood vessels (Burks, 1990). Mechanoreceptors have been identified by various studies on the surface of the cruciates and near their insertion sites but there exists little consensus as to their type, location, number or function (Barrack and Skinner, 1990).

1.2 FUNCTION OF THE PCL

The PCL is generally regarded as having a primarily mechanical rôle, to help give passive stability to the knee joint and control joint motion by opposing inertial or muscular forces. It has also been suggested that the cruciates give the active, muscular stabilisers of the knee neural feedback on joint position and loading so that the active system can be used to prevent injury. Some workers have suggested that this is the primary rôle of the cruciates. The studies in this thesis are aimed at establishing the mechanical function of the PCL.

1.2.1 Qualitative Observations

Almost all previous studies agree that the PCL is the primary restraint to posterior drawer, that is posterior translation of the tibia with respect to the femur. However more detailed descriptions of the function of the PCL have been variable, sometimes leading to divergent conclusions.

Many authors have made qualitative statements about PCL function, based on in vitro observation of the intact ligament. These statements have described the pattern of fibre tension within the PCL during knee flexion–extension, the response of the PCL to posterior drawer and the role of the PCL in controlling internal-external tibial rotation.

At the beginning of the century anatomists maintained that the PCL was only tightened in the flexed position of the joint (Pringle, 1907). This view was restated by Jones (1914) and Hey Groves (1920) but Smith (1918) reported that the PCL was tight in full flexion and in the extended knee but that it was slack in semiflexion. Later studies offered more sophisticated descriptions. Brantigan and Voshell (1941) and Abbott (1944) reported that as the knee moves from extension to flexion a zone of tension moves from the posterior fibres of the PCL to the anterior fibres. Of the authors who support the concept of two functional, if not anatomical, bundles all bar Horwitz agree: that the aPC is slack in the extended knee and tightens beyond 30° of flexion and that the pPC is slack in flexion, tightens in extension and becomes very taut in hyperextension (Palmer, 1938; Girgis et al, 1975; Hughston et al, 1980; Van Dommelen and Fowler, 1989; Sepulchre and Blaimont,

1986; Burks, 1990; Kannus et al, 1991; Whipple and Ellis, 1991). Horwitz's study differed in that he claimed that the pPC is only taut in combined extension and internal tibial rotation (Horwitz, 1938).

The above observations can be reconciled as follows: In extension the anterior fibres are slack and the posterior fibres are tight but since the posterior fibres make up a smaller fraction of the PCL they have been overlooked by some investigators. As flexion proceeds the posterior fibres slacken and the anterior fibres begin to tighten, giving rise to an overall reduction in tension as noted by Smith (1918). Then as flexion proceeds the anterior fibres become increasingly tight; so the majority of the PCL is tight in flexion, as stated by all the above investigators. The slackness in extension of the anterior fibres of the PCL has been demonstrated in vivo by Magnetic Resonance Imaging (MRI): sagittal plane images have shown a well defined arcuate structure (Bassett et al, 1990; Grover et al, 1990). This arcuate structure in extension has also been demonstrated in vitro (section 3.1.3.2).

As regards the function of the PCL in posterior drawer, there is general agreement that the PCL resists posterior drawer but differing opinions as to its relative importance and its relationship with flexion angle. Hey Groves (1917) stated that the cruciate ligaments are the only structures that resist A–P drawer since no other ligaments in the knee are oblique in the sagittal plane. However, most early investigators reported that the PCL resists posterior drawer in the flexed knee and that in extension the collateral ligaments take over (Hazen, 1910; Jones and Smith, 1914; Smith, 1918; Hey Groves, 1920). Palmer (1938) pointed out that this was an oversimplification as parts of the PCL are tight in extension. This view was echoed by Brantigan and Voshel (1941) and Abbott et al (1944) who reported that the PCL resists posterior drawer at all flexion angles. Dissent came from Horwitz (1938), who claimed that the collateral ligaments are more important at all flexion angles, and Helfet (1948), who considered the cruciate ligaments to be rotational guides during flexion/extension rather than checks against A–P drawer. More recently O'Brien et al (1992) have concluded that the PCL is biomechanically most efficient (at resisting posterior drawer) in extension as they reported that the PCL is 'most horizontal' (most nearly parallel to the direction of posterior drawer) then. This description of the angle of the PCL with respect to the tibia was supported by Kapandji

(1987) but other investigators have described the PCL as more 'vertical' (perpendicular to the direction of a posterior drawer force) in extension and more 'horizontal' in flexion (McMaster, 1975; Van Dommelen and Fowler, 1989).

These recent, contradictory, descriptions probably result from the fact that different investigators have concentrated on different parts of the PCL's broad femoral insertion.

All the above investigators have assigned some significance to the PCL in the control of tibial internal–external rotation. It has often been stated that the cruciate ligaments are relaxed by external tibial rotation and that they resist internal tibial rotation by winding around each other (Hazen, 1910; Brantigan and Voshell, 1941; Last, 1948; Kennedy and Grainger, 1967; Kapandji, 1987). Horwitz (1938) demonstrated this by cutting all structures bar the cruciate ligaments and rotating the tibia: he found that external tibial rotation was unrestricted, to 180°, and that internal tibial rotation was limited, to 15° at 90° flexion, by the 'abutment of the anterior against the posterior cruciate'. This 'cruciate wind up' mechanism implies that the axis of tibial rotation lies between the cruciate ligaments, as it must have done in Horwitz's experiment. However, Palmer (1938) stated that the axis of tibial rotation, in the intact knee, is in the centre of the medial condyle and that previous descriptions of cruciate action in rotation must, therefore, be wrong. The alternative description offered by Palmer was that internal tibial rotation, about a medial axis, relaxes the PCL and tightens the ACL whereas external tibial rotation tightens the PCL and relaxes the ACL: therefore the PCL resists external rotation. Palmer (1938) went on to point out that if the knee is damaged, particularly by rupture of the medial collateral ligament, then the axis of tibial rotation may be displaced to a central or lateral position: in the former case the cruciates will resist internal rotation only and in the latter their usual actions will be reversed.

1.2.2 Proprioception

There has been considerable debate as to the proprioceptive rôle of the cruciates which is largely beyond the scope of this thesis. It has been thought for some time that the cruciates have a proprioceptive function (Barrack and Skinner, 1990). This view has correlated with apparent

loss of proprioceptive function after knee ligament injury and improved function after proprioceptive training secondary to injury. However, this view is not universally accepted. It can be argued that the cruciates cannot be sensitive to joint position as they are isometric, and therefore not strained by passive flexion–extenison, although in vivo there are few situations where one or other of the cruciates are not strained to some degree. It is also possible that even though the cruciates may be sensitive to strain they could not be sensitive to joint position because different combinations of knee rotations and translations could lead to the same strain pattern.

Some workers have stated that muscle and skin are the primary proprioceptive organs (Clark et al, 1979). This contention is supported by subjective reports from athletes. It is a common experience that 'tubigrip' style joint 'supports' give a feeling of security to those that wear them. The structure itself does not have sufficient stiffness to significantly influence joint stability but does serve to amplify the forces experienced by the skin. Some rugby players have refined the procedure and found that binding adhesive tape around the joint line of the knee gives the greatest benefit. The position of the tape means that the skin over the patellar tendon and the hamstrings is compressed when the respective muscle group is activated. However, this hypothesis is, as yet, unproven.

1.2.3 Mechanical and Material Properties

The published data for the mechanical and material properties of the PCL, which shows considerable variation, is summarised in Table 4.1. The variation in results can be explained, in part, by examining the methods used by different investigators. For the entire PCL mechanical properties have been published by Trent et al (1976), Kennedy et al (1976), Marinozzi et al (1983) and Prietto et al (1988). Butler et al (1986) have published results for the material properties of the PCL based on individual fascicles of three donors. Individual fascicles were tested because it was thought that this would result in more consistent results for ligament material properties: initial length is easier to measure for a small specimen and even loading of the fibres is easier to achieve. It was anticipated that smaller specimens would yield superior material properties. Trent et al (1976) tested bone block-ligament-bone block

preparations. They reported sequential failure with 'steps' in the load/extension curve, also the low strain rate led to premature failure by bony avulsion. (The effect of strain rate on failure mode has been demonstrated by Noyes et al (1974)). Kennedy et al (1976) tested ligaments that were excised from their bony attachments and held in mechanical clamps, which can be expected to cause premature failure. Marinozzi et al (1983) tested bone block-ligament-bone block specimens. They did not report avulsion failure but may have subjected ligaments to uneven loading leading to sequential failure: the example graphs of load against deflection, which were taken from tests of the collateral ligaments, clearly showed sequential failure but Marinozzi et al did not comment on this. Prietto et al (1988) tested tibia-PCL-femur preparations and reported a higher maximum load than Trent et al (1976) and Kennedy et al (1976), but this could have been due to the lower age of donor (Hollis et al, 1988) rather than better loading conditions.

Several previous studies of ligament material properties have identified submaximal peaks and referred to them as the 'yield point' (Kennedy et al, 1976; Beauchamp et al, 1979; Cabaud, 1983; Marinozzi et al, 1983). In these cases I think that 'yield point' is a misnomer, and that it actually refers to the onset of ligament fascicle ruptures. Noyes et al (1974) and Noyes and Grood (1976) noted a submaximal peak, labelled as 'linear load'. In the study of Noyes et al. (1974) the text defines 'linear load' as the load at the first failure causing a drop in load of more than 2kgf and goes on to say that this value is an approximation for the onset of failure. Subsequent studies seem to have taken this approximation to be the definition of the 'yield point'. The actual yield point, when the gradient of the load/deformation graph decreases until failure, can only be accurately determined if sequential failure does not occur. I believe that data published previously that included 'yield points' was actually representative of tearing–type sequential failures, which will have caused maximum loads to be underestimated.

The studies of Trent et al (1976), Kennedy et al (1976), Marinozzi et al (1983) and Prietto et al (1988) all suggested material properties for the PCL that appear to be significantly inferior to those published by Butler et al (1986). An explanation for this difference is that the PCL is generally considered to be made up of two bundles (section 1.2.1). Since

the aPC and the pPC are load bearing at different angles of flexion (section 1.2.2.) and have different orientations it is impossible to load the fibres of the whole PCL in parallel. Thus any flexion angle chosen for testing of the whole ligament must compromise one or both of the bundles (leading to peeling failure), and result in uneven fascicle loading (leading to sequential failure). These two effects result in underestimation of total PCL strength, maximum stress and modulus.

1.2.4 Selective Cutting Studies

1.2.4.1 Load Controlled Selective Cutting

Most selective cutting studies have attempted to deduce ligament function from the increase in laxity associated with their removal under a controlled load (Palmer, 1938; Brantigan and Voshell, 1941; Wang and Walker, 1974; Girgis et al 1975; Hsieh and Walker, 1976; Trickey, 1980; Fukubayashi et al, 1982; Gollehon et al, 1985; Nielsen et al, 1985; Nielsen and Helmig, 1986; Gollehon et al, 1987; Rong and Wang, 1987; Grood et al, 1988; Ogata et al, 1988). The experimental method was similar in all of these load controlled studies and is summarised below.

The intact knee was loaded in some or all of the six degrees of freedom (section 1.1.) either in isolation or in combination. The displacement, linear or rotational, of the intact joint was recorded for the chosen load. Then the increase in displacement, under the same load, was measured as ligamentous structures were selectively cut.

Comparison of the results of these studies is sometimes awkward because different investigators used different loads and considered different ranges of flexion angle. Also, different clamping methods were used which meant that different degrees of freedom were constrained.

The light shed by these studies on the role of the PCL in A–P drawer will be considered first. All investigators reported that isolated section of the PCL resulted in increased posterior translation and that this was minimal in extension and increased with flexion (Palmer, 1938; Brantigan and Voshell, 1941; Girgis et al, 1975; Hsieh and Walker, 1976; Trickey, 1980; Fukubayashi et al, 1982; Gollehon et al, 1985; Rong and Wang, 1987; Nielsen et al, 1985; Nielsen and Helmig, 1986;

Gollehon et al, 1987; Grood et al, 1988; Ogata et al, 1988). Some investigators reported that a posterior drawer force produced coupled external tibial rotation (Fukubayashi et al, 1982; Gollehon et al, 1987; Grood et al, 1988). This coupled rotation was shown to be eliminated (Fukubayashi et al, 1982; Gollehon et al, 1987) or much reduced (Grood et al, 1988) by section of the PCL. Fukubayashi et al (1982) reported that when this coupled rotation was restrained, by preventing tibial rotation, posterior laxity was reduced by 30%. Hsieh and Walker (1976) investigated the effect of a compressive load during A–P drawer: they showed that, in an intact knee at 30° flexion, a compressive load equal to body weight reduced posterior laxity by 80% and that a load equal to twice body weight reduced posterior laxity by 88%.

With regard to hyperextension there has been some disagreement between past studies. Brantigan and Voshell (1941) reported that section of the PCL resulted in hyperextension (and hyperflexion) whereas others reported that PCL insufficiency had no influence on hyperextension (Palmer, 1938; Girgis et al, 1975; Trickey, 1980; Nielsen et al, 1985). Grood, Stowers and Noyes (1988) reported that isolated section of the PCL caused a 'small but significant ($p<0.05$) increase in hyperextension'. The increase noted by Grood et al (1988) is not incompatible with the conclusions of Palmer (1938), Girgis et al (1975), Trickey (1980) and Nielsen et al (1985) because it would not have registered on the previous studies' less sensitive apparatus: Girgis et al (1975) quoted results to the nearest 5°.

There has also been some conflict of opinion as to the role of the PCL in controlling forced internal–external tibial rotation. Some investigators noted that after isolated section of the PCL there was increased, though inconsistent, tibial rotation in flexion (Brantigan and Voshell, 1941; Wang and Walker, 1974; Girgis et al, 1975): the data of Girgis et al (1975) show that the average increases in internal and external rotation were 3° ± 2.7°(s.d.) and 7.5° ± 4°(s.d.) respectively. Girgis et al (1975) and Wang and Walker (1974) also noted that there was no increase in tibial rotation in extension. More recent studies have shown that isolated section of the PCL results in no increase in tibial rotation over the range 0°–90° of flexion (Gollehon et al, 1985; Gollehon et al, 1987) or, in the case of the study of Grood et al (1988), that the only effect,

over the range of 0°–90° of flexion, is a small (2.6° ± 1.3°, about 9%) increase in internal rotation at 90° of flexion.

The effect of the PCL on varus–valgus rotation has only been investigated in two of the laxity studies. Gollehon et al (1987) reported that isolated section of the PCL resulted in no significant change in adduction or abduction rotation. Grood et al (1988) reported that there was a 'very small' (<1°, about 10%) increase in adduction and abduction rotation at all flexion angles (0°–90°)

Although load controlled selective cutting studies can yield useful results the information they give is limited: the technique will reveal the most important restraint to the abnormal motion under investigation but it cannot be used to determine the relative contributions of ligaments to stabilising the intact knee. When a ligament is cut the displacement of the joint increases until other ligamentous structures are strained sufficiently to resist the same load: in the intact knee these other structures may, or may not, be loaded. In order to discover the relative contributions of ligamentous structures to knee stability another selective cutting technique was developed: it was based not on the increase in displacement under a controlled load but on the reduction in load under a controlled displacement, (Butler et al, 1980).

1.2.4.2 Displacement Controlled Selective Cutting

When a knee specimen is subjected to a particular displacement various ligamentous structures may be strained and therefore resist the displacement. If one of these resisting structures is cut, and the knee is subjected to the same displacement, then there will be a reduction in resistance. However, all the other structures will be strained exactly as before and therefore each will offer the same resistance as they did in the intact knee. So, the proportional drop in resistance after cutting a ligament is equal to the proportion of resistance offered by that ligament in the intact knee. As long as the geometry of the joint is preserved further structures can be cut and their resistance contributions measured.

Butler et al (1980) measured the contribution of the PCL to resisting posterior drawer at 30° and 90° of flexion for 1mm to 5mm displacements. They tested 14 specimens and reported that, at 5mm

posterior displacement, the PCL resisted 94%(at 90°) to 96%(at 30°) of posterior draw force. Their data showed that the percentage resistances for smaller displacements were lower but that the differences, between 30° and 90° and between 1mm and 5mm, were not statistically significant ($p>0.05$). Piziali et al *(*1980) measured the contribution of the PCL to resisting posterior drawer force in extension in 2 specimens. They reported that in one knee the PCL resisted 93% of posterior drawer force and that in the other the PCL resisted 72% of drawer force. In the latter case 16% of the resistance to posterior drawer came from the lateral collateral ligament. These two studies confirmed that the 'primary restraint', a phrase coined by Butler et al (1980), to posterior drawer is the PCL. Both studies used clamping systems that completely constrained the specimens to movement in the A–P direction. This restraint meant that the geometry of the intact joint was always preserved after cutting, which is required for the method to work. However, straight A–P displacement in response to an A–P load is abnormal: in the unconstrained knee there is coupled tibial rotation (Fukubayashi et al, 1982; Gollehon et al, 1987; Grood et al, 1988) and proximal–distal motion of the tibia (Hsieh and Walker, 1976). Drawing conclusions about the in–vivo state from single degree of freedom tests to is not straightforward.

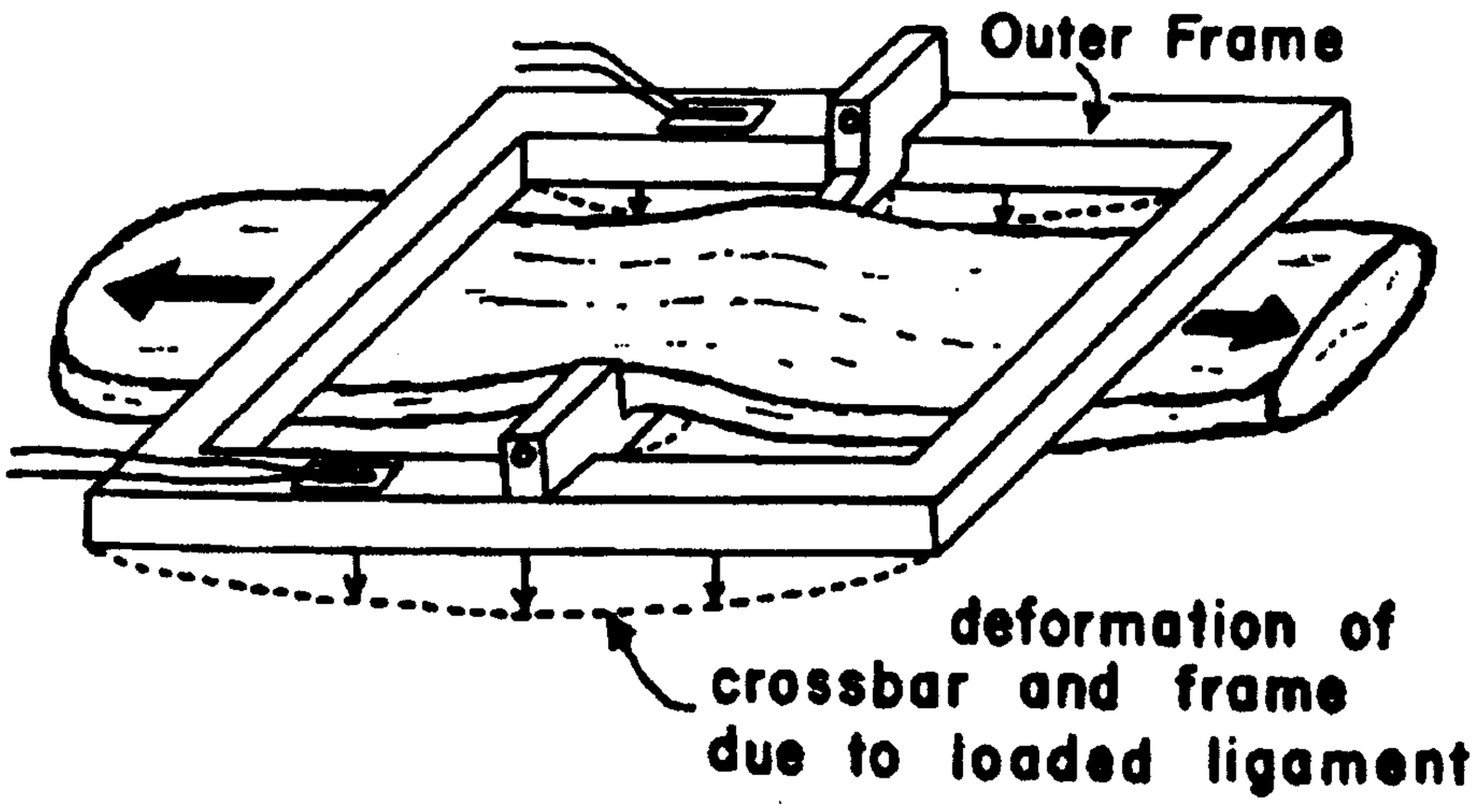


Figure 1.2 Buckle transducer (Lew and Lewis, 1982)

1.2.5 Ligament Loading Studies

Ligament loads have been inferred using buckle transducers (Lewis and Fraser, 1979; Jasty et al, 1982; Lew and Lewis, 1982; Ahmed et al, 1987). These devices consisted of a metal buckle which was placed around the ligament in such a way that tension in the ligament caused the buckle to deform (Figure 1.2). This deformation was measured by strain gauges on the buckle and recorded during various knee motions. The buckle transducer–ligament system was then calibrated by removing individual ligaments, with bone blocks, and recording the buckle transducer's response to known loads. Ligament loads have also been measured directly by attaching a miniature load cell to the femoral insertion of the PCL via a bone tunnel (Wascher et al, 1991). Indirect measurements have been made by recording strain in the ligament substance using Hall Effect strain transducers (Ogata et al, 1988) and by recording bone strain next to ligament insertions (France et al, 1983).

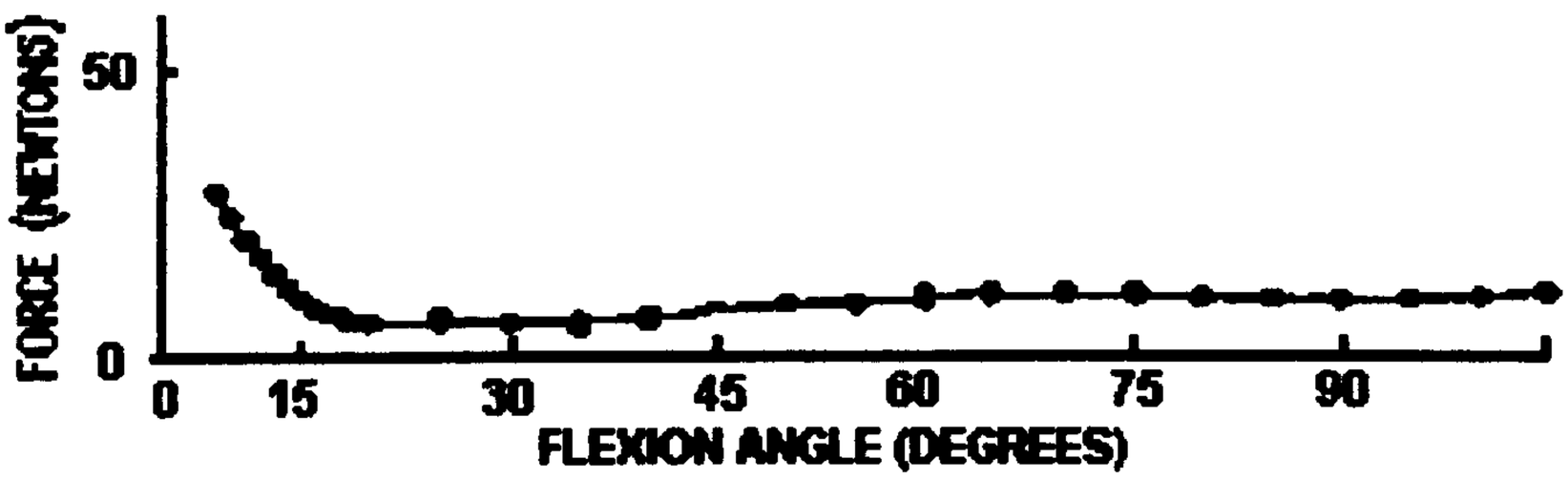


Figure 1.3 PCL load in response to passive flexion. Data from. Wascher et al (1991), n = 12.

The results of those studies which reported loads for passive flexion–extension are in conflict. Some studies measured the load in the PCL as a whole and some considered the anterior and posterior fibres separately. Of the former, Lewis and Fraser (1979), who reported results for one specimen, stated that the PCL bears no load in hyperextension. However, Wascher et al (1991), who tested 12 knees, reported that PCL

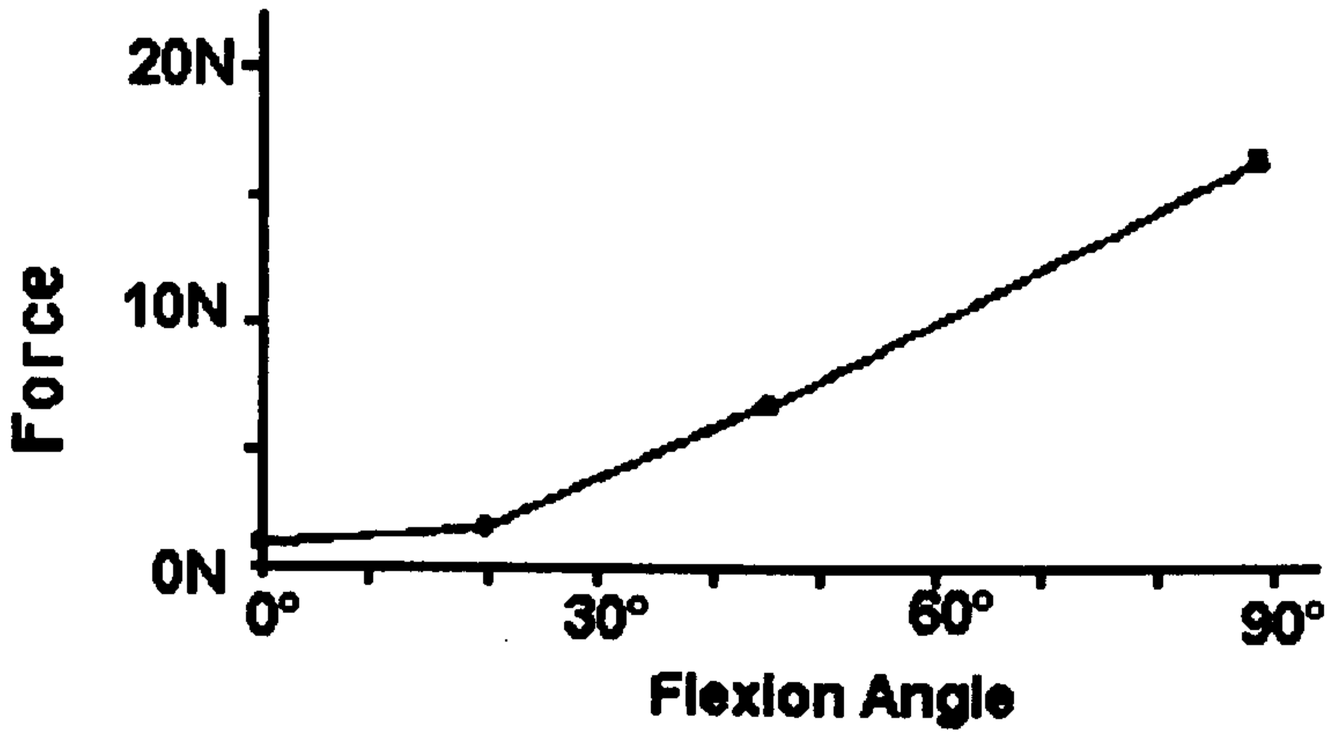


Figure 1.4 PCL load in response to passive flexion. Data from Lew and Lewis (1982), n = 3.

load increased from extension to hyperextension (Figure 1.3). Lew and Lewis (1982) measured the load in the posterior fibres of the PCL in 3 specimens: they reported that the load increased from 0° to 90° of flexion (Figure 1.4). However, France et al (1983), who tested 3 specimens, showed that the load in the posterior fibres was minimal between 10° and 120° of flexion and that it increased in extension and

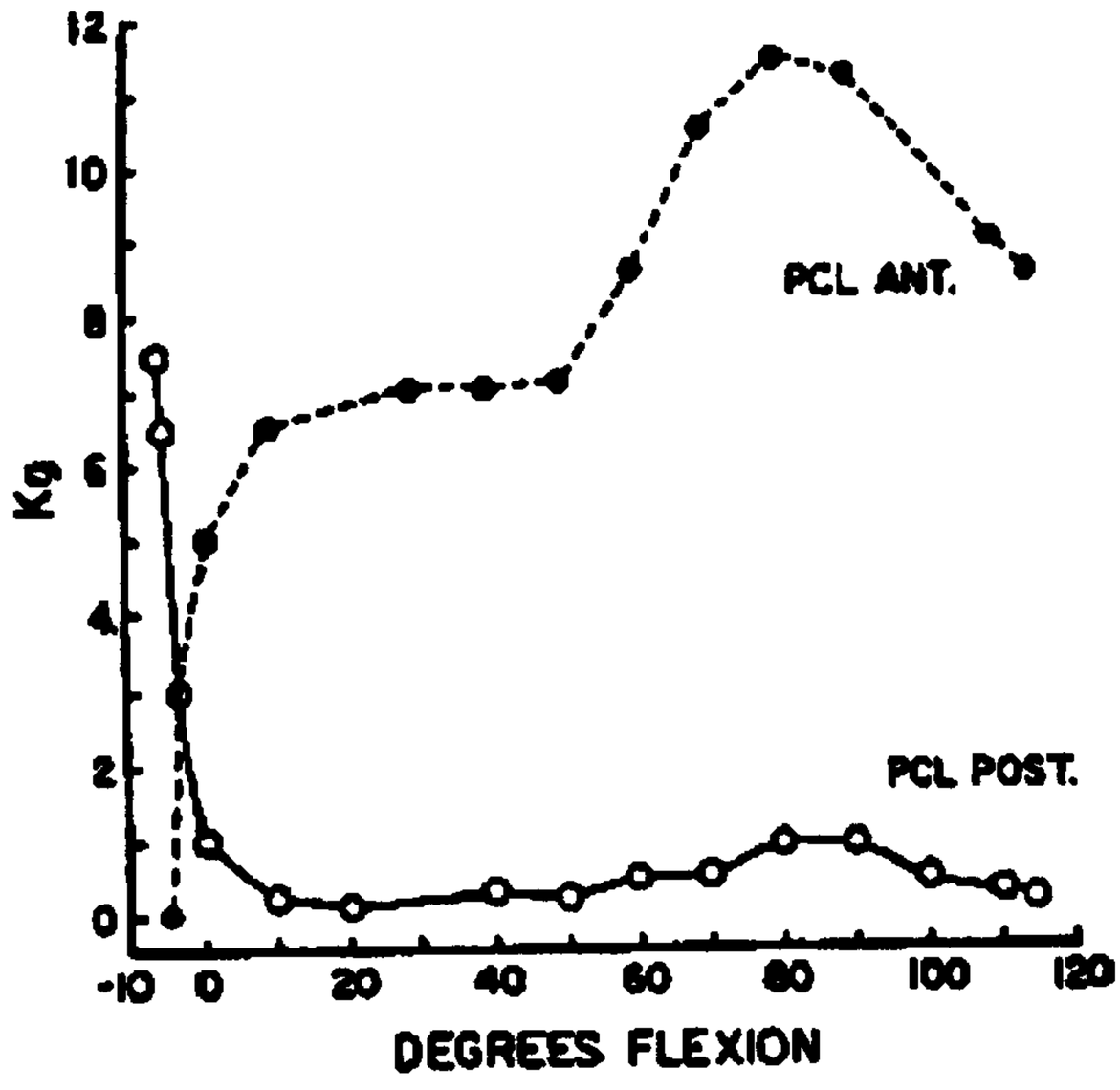


Figure 1.5 aPC and pPC load in response to passive flexion. Data from France et al (1983), n = 1

dramatically increased in hyperextension. France et al (1983) also reported that the anterior fibres were relaxed by hyperextension and maximally taut at 80° to 90° of flexion (Figure 1.5). Ogata et al (1988) implanted Hall Effect strain transducers into the 'central' portion of the PCL in 3 specimens; they showed that the strain in the PCL was minimal at extension and maximal (2.8%) at 90° of flexion (Figure 1.6).

Those studies which measured PCL loads in response to posterior drawer are in general agreement: that the PCL is loaded by posterior drawer at all flexion angles but less so near extension. These studies do,

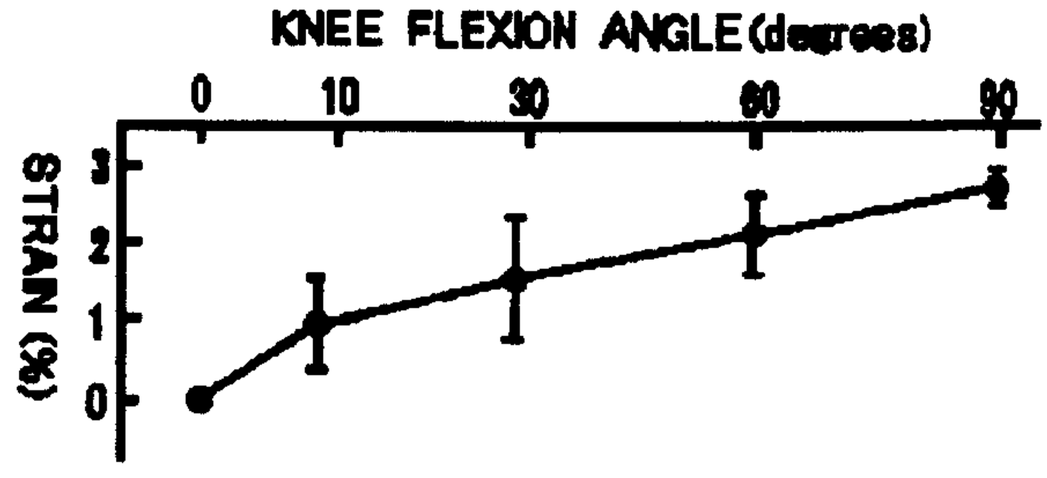


Figure 1.6 Strain in the 'central' portion of the PCL in response to passive flexion. Data from Ogata et al (1988), n=3.

however, differ quantitatively. Lewis and Fraser (1979) reported that in a clinical posterior drawer test at 90° of flexion the PCL was the only ligament loaded but that in the same test in extension the collateral ligaments were also loaded. Jasty et al (1982) applied a clinical load of about 45N and published a 'typical' result which showed that: in extension the PCL was not loaded, at 20° of flexion PCL load was about equal to the applied drawer force, at 45° of flexion the PCL load was about twice the applied force and that at 90° of flexion PCL load was about three times the drawer force (Figure 1.7). The study of Wascher et al (1991) showed that between about 45° and 90° of flexion the load on the PCL was about equal to the posterior drawer force and that below 45° of flexion PCL load decreased until extension, when the PCL load was one fifth of the drawer force (Figure 1.8).

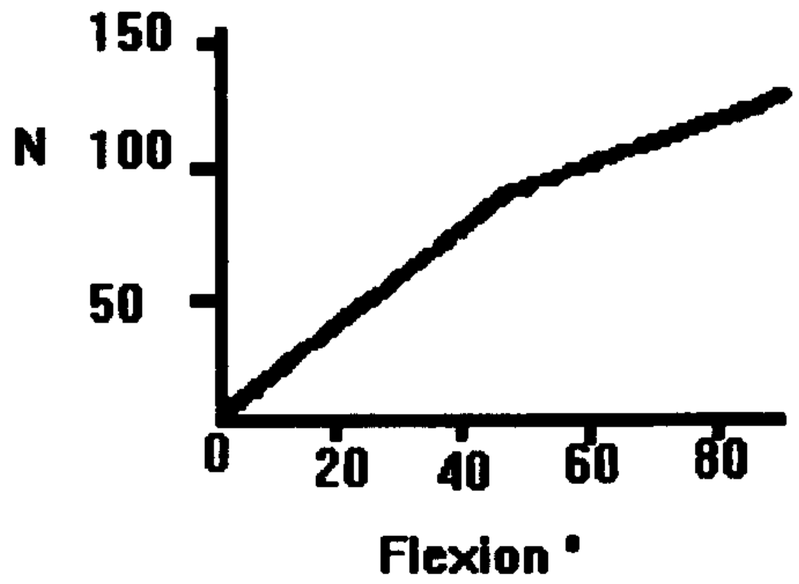


Figure 1.7 PCL load in response to a 45N posterior drawer force. Data from Jasty et al (1982). n = 1.

With regard to tibial rotation, Wascher et al (1991) showed that the PCL resisted internal tibial rotation after 45° of flexion but that it was relaxed by external tibial rotation in the range of flexion considered (0°–90°). Ahmed et al (1987) measured the load on the posterior fibres of the PCL during tibial rotation at 40° and 90° of flexion in 30 specimens. They showed that at 40° of flexion the posterior fibres were not loaded by tibial rotation but that at 90° of flexion the fibres were loaded, to a variable degree, in both internal and external rotation, more so in external rotation.

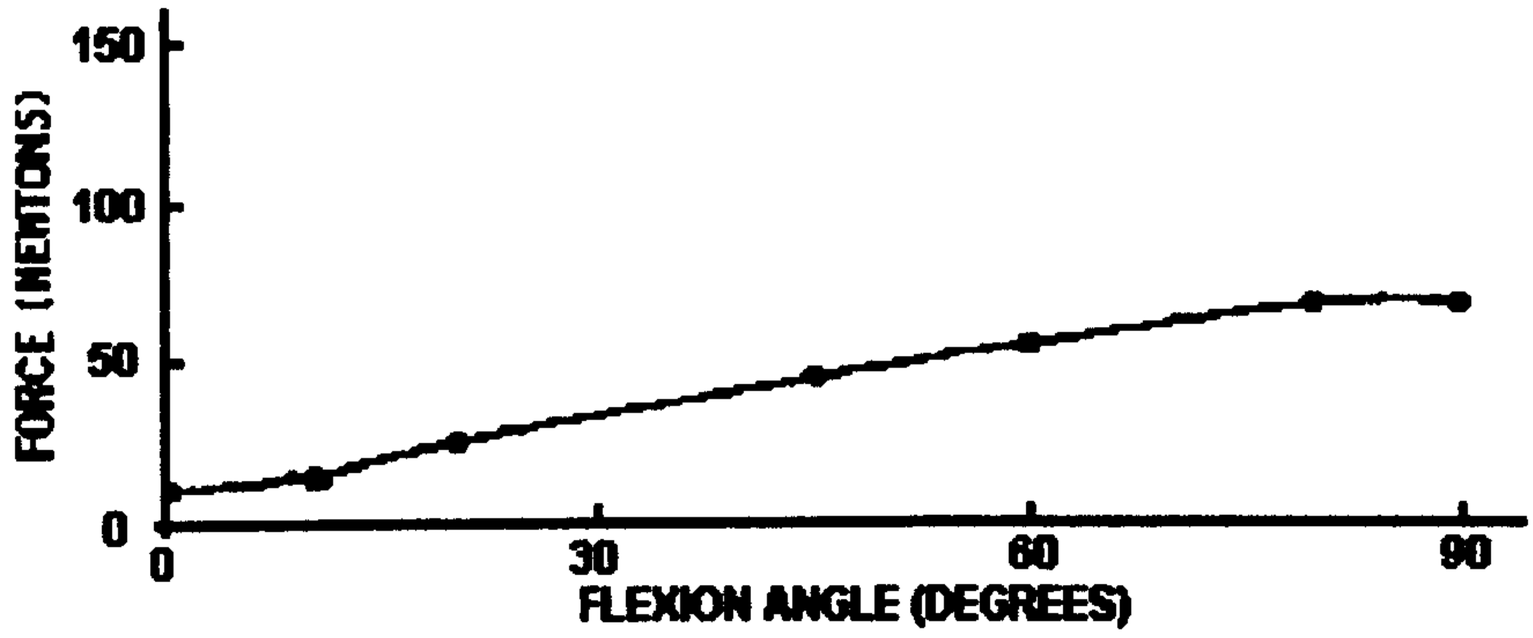


Figure 1.8 PCL load in response to a 50N drawer force. Data from Wascher et al (1991). n = 12

1.2.6 Fibre Length Change Studies

The earliest published length change data was in the study of Horwitz (1938), (Figure 1.9). Horwitz split the femur in a sagittal plane so that the PCL could be directly measured during flexion–extension. Horwitz

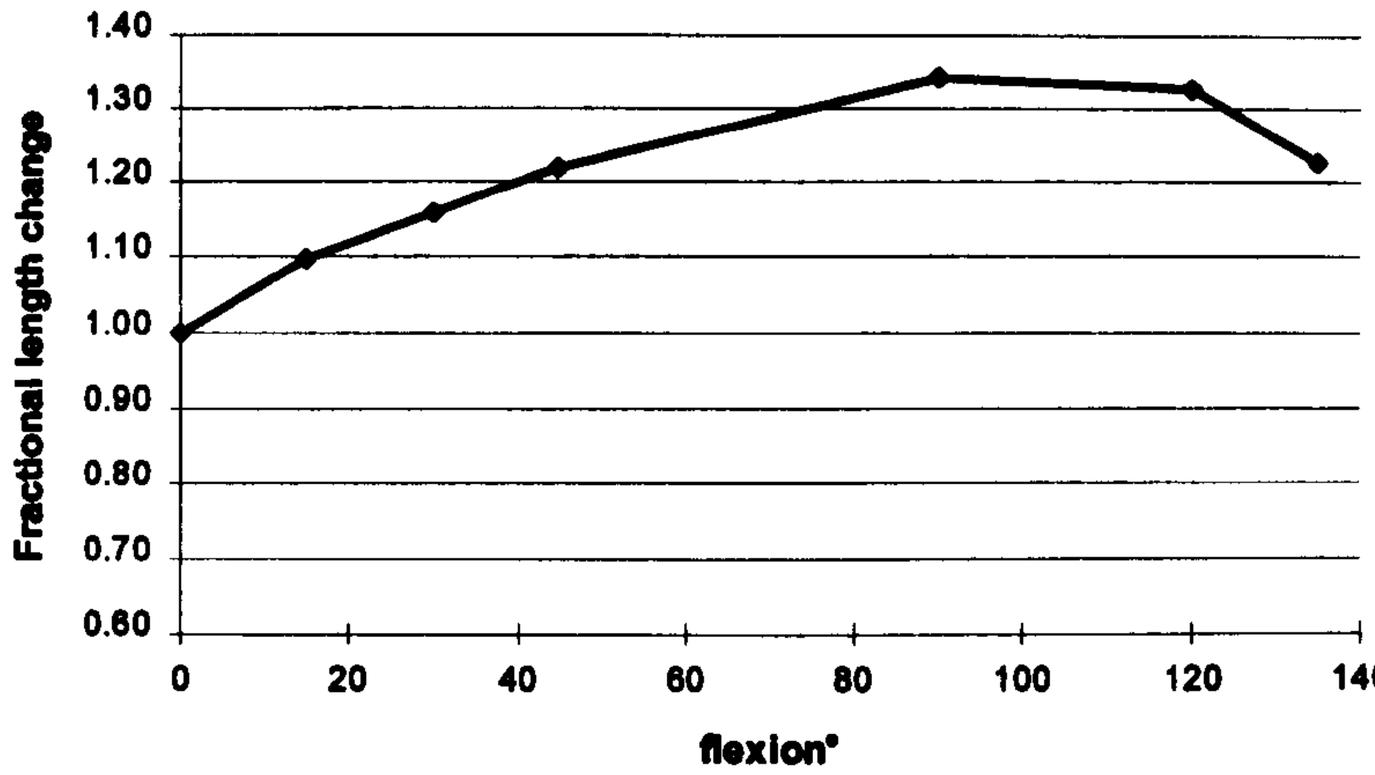


Figure 1.9 Fractional length change of the PCL with flexion. Data from Horwitz (1938), n = 2

(1938) reported that the PCL lengthened in flexion, however, he did not state which part of the PCL was measured. A similar method was used by Satku (1984) who removed the lateral femoral condyle in order to measure the aPC and the pPC with vernier callipers in 3 specimens. It was claimed that normal joint motion was preserved by clamping the condyle back onto the knee when it was moved to a new flexion angle. The data of Satku shows that that the aPC lengthens in mid flexion and that the pPC lengthens at the extremes of flexion (Figure 1.10).

Fibre length patterns of knee ligaments have also been measured using implanted conducting liquid strain gauges (Edwards et al, 1970; Kurosawa et al, 1987; Kurosawa et al, 1991), by recording the three dimensional motion of the joint and calculating the separation of selected bony insertions (Wang et al, 1973; Trent et al, 1976; Selvik, 1979; Hefzy and Grood, 1985; Hefzy et al, 1986; Rovick et al, 1987; Sidles et al, 1988; Fuss, 1989) and by measuring the excursion of an implanted wire with a linear variable displacement transducer (Covey et al, 1992). These studies are discussed below.

Edwards et al (1970) implanted a mercury filled rubber tube strain gauge into the ligaments of 13 specimens. They stated that the device was implanted into the 'central section' of the PCL. Their data showed that the PCL was most elongated in mid flexion (Figure 1.11). Kurosawa et al (1987; 1991) used multiple electrolyte filled strain gauges implanted in different fibre regions of the ligaments in 3 knees. They reported that the posterior fibres were at maximum length in extension and that the anterior fibres were at maximum length at about 50° of flexion (Figure 1.12).

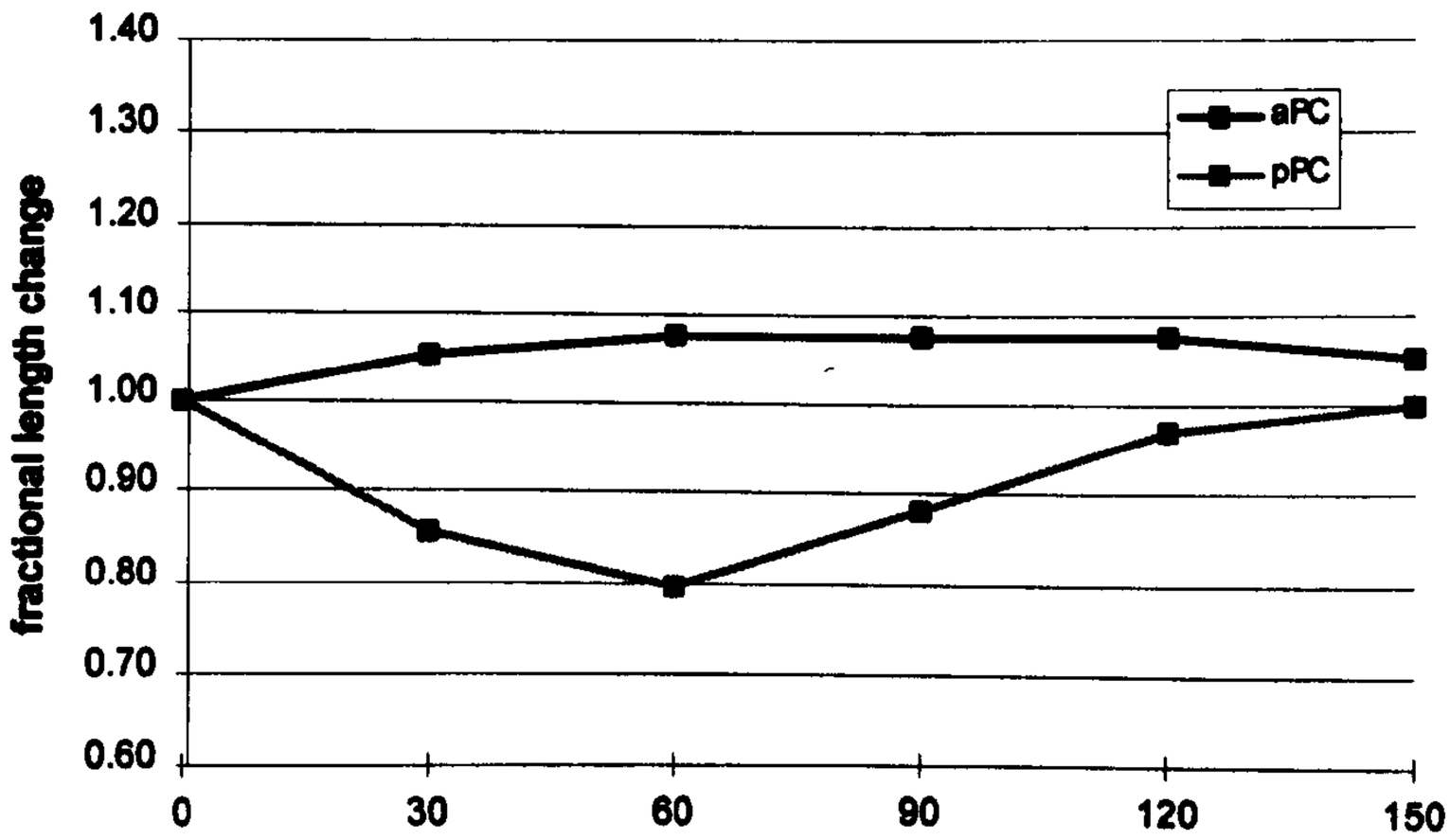


Figure 1.10 Fractional length change of the aPC and pPC with flexion. Data from Satku (1984), n=3

Wang et al (1973) inserted 2mm diameter rods into the femur and tibia with the tip of each rod at a ligament bundle insertion, which was verified by palpation. By measuring the co-ordinates of two extra-articular points on each rod they were able to calculate the position of the intra-articular tip of each rod and hence the separation of the ligament bundle insertions. They tested 12 specimens and their data showed that, in passive flexion-extension, the central fibres of the PCL were at maximum length in extension and minimum length at 30° of flexion (Figure 1.13). For 5 of the knees tested by Wang et al (1973) they also calculated the length changes of the anterior and posterior fibres and reported that the anterior fibres were at maximum length at 60° of flexion and that the posterior fibres were at maximum length at 120° of flexion (Figure 1.14)

Trent et al (1976) recorded the motion in passive flexion–extension of 3 knee joints photographically using extra–articular marker rods. They calculated fibre length changes in flexion–extension over the range 0° to 90° and, in another 7 knees, the centre of tibial rotation. They reported that the lengths of the posterior fibres of the PCL were 'virtually constant' and that the middle and anterior fibres both lengthened in flexion (Figure 1.16). Trent et al also reported that the function of the PCL in controlling tibial rotation was variable and depended on the relative position of the axis of tibial rotation: when the axis of rotation was central the PCL was lengthened by internal rotation and slackened by external rotation, when the axis was in the medial compartment PCL function in tibial rotation was reversed. This study provided quantitative backup to Palmer's (1938) statement on the function of the cruciates in tibial rotation (section 1.2.1).

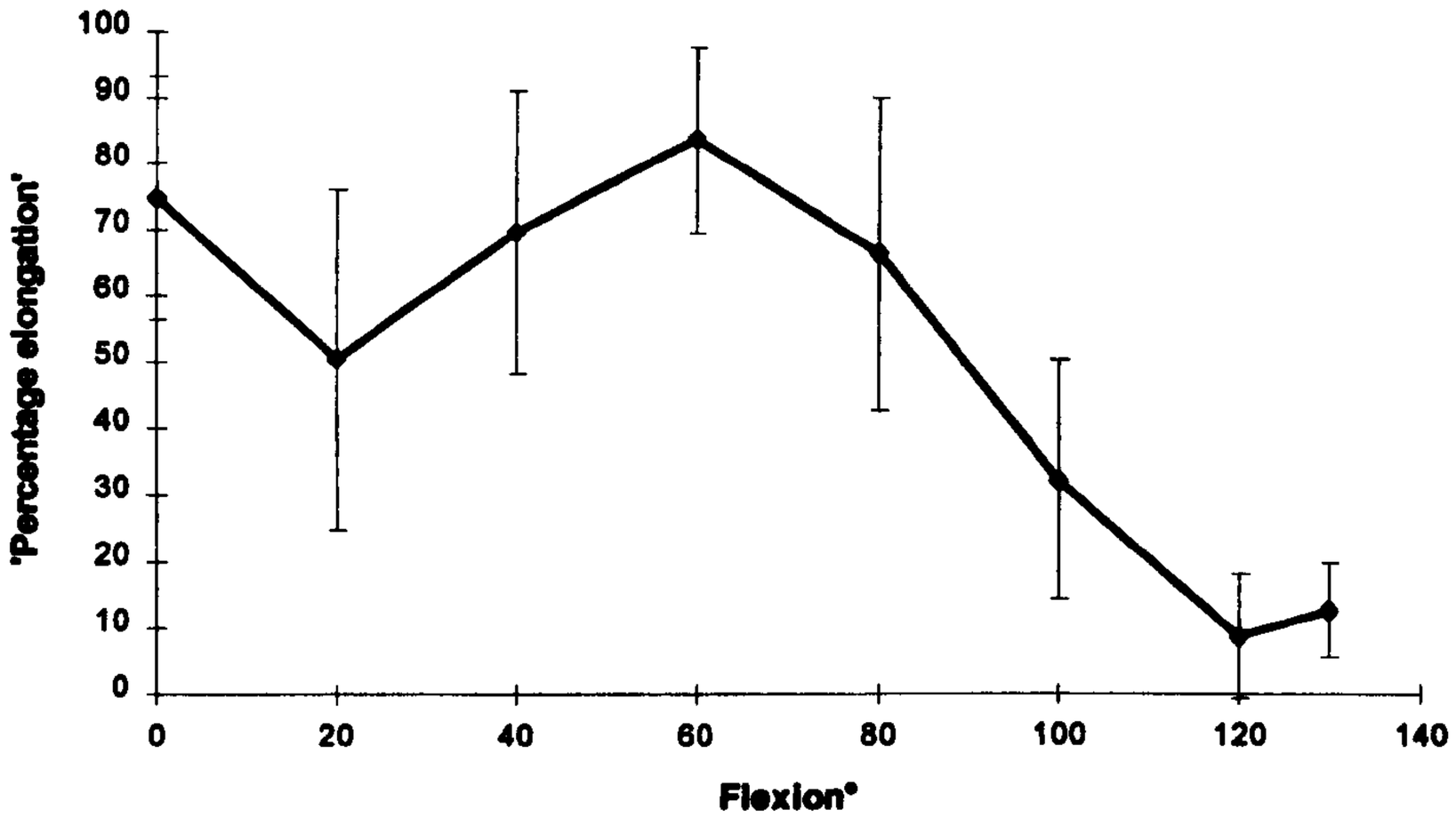


Figure 1.11 Length change of the PCL with flexion as a percentage of maximum length change. Data from Edwards, Lafferty and Lange(1970), n=13, ± s.d.

Rovick et al (1987) measured the 3-D motion of knee specimens in an instrumented rig as they were put through dynamic flexion–extension by a quadriceps force. His results showed the anterior fibres of the PCL gradually lengthening from extension to full flexion. That result is in line with the results of Kurosawa et al (1987) for the same loading conditions, however, they cannot usefully be compared with the passive flexion studies. Isolated quadriceps contraction is no more

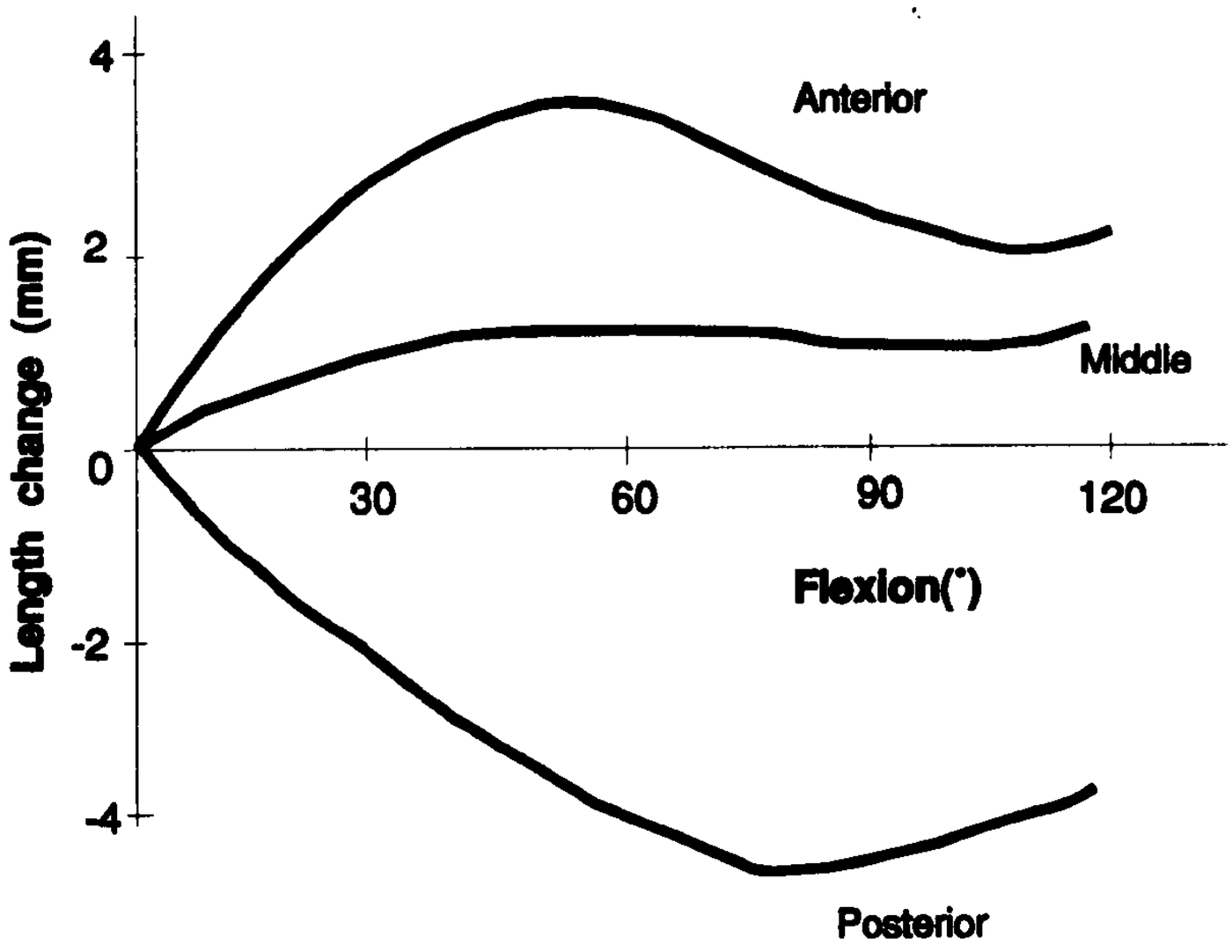


Figure 1.12 Length change with flexion for the anterior, middle and posterior fibres of the PCL. Data from Kurosawa et al (1991), n = 3.

physiologically 'normal' than passive flexion–extension: in vivo there are usually antagonistic muscle forces to balance knee loads.

Selvik (1979) reported a stereophotogrammetric method using tantalum balls as radio–opaque markers. He only tested two specimens but made the important observation that in unconstrained passive flexion–extension the geometry of the joint could be altered by small forces, especially the rotational position of the tibia. He therefore recommended

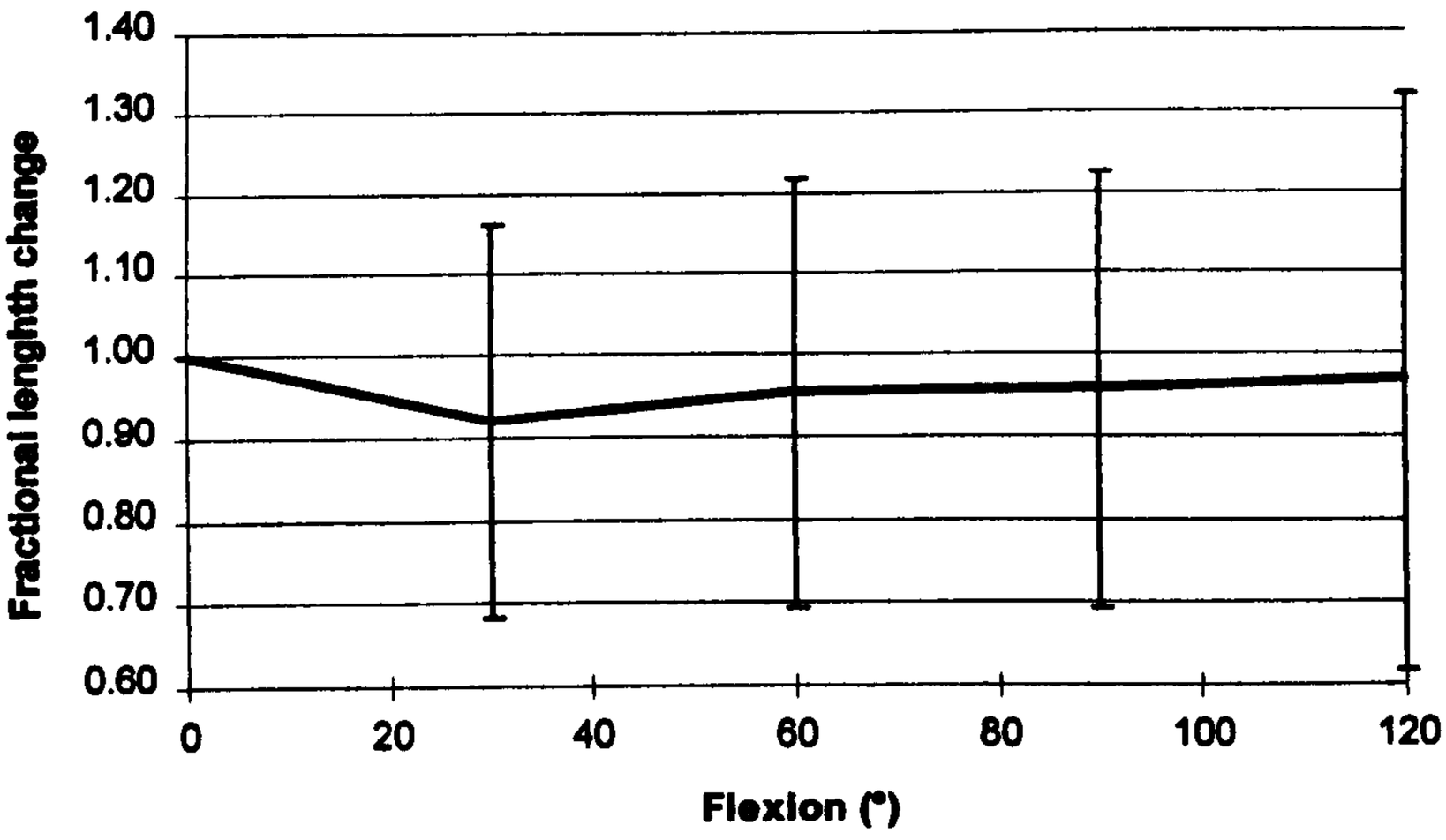


Figure 1.13 Fractional length change of the PCL with flexion. Data from Wang et al (1973), n = 12.

that in future studies the load on the joint, in all degrees of freedom, should be carefully controlled.

In the studies of Hefzy et al (1986) and Hefzy and Grood (1985) knee motion was recorded by a six degrees of freedom instrumented spatial linkage (ISL). The ISL recorded the relative positions of the tibia and femur, it was later used as a 3–D digitiser, after the knee had been dissected, to record the relative positions of selected points within the bony insertions of the PCL. Knee specimens were tested in the range 0°

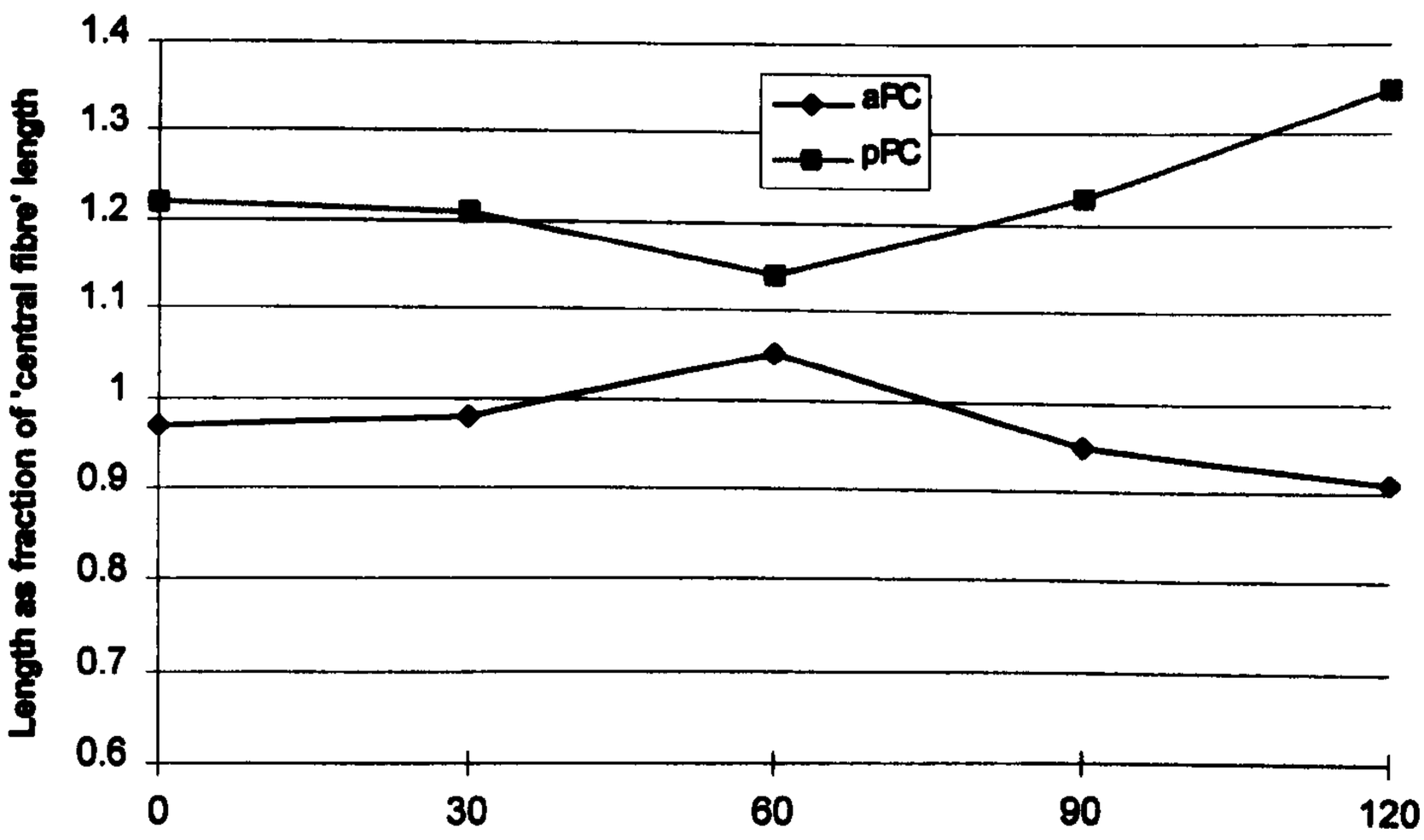


Figure 1.14 Length of the aPC and the pPC as a fraction of PCL central fibre length . Data from Wang et al (1973), n = 5.

to 90° (Hefzy et al, 1986) and 0° to 110° (Hefzy and Grood, 1985) while subject to a 100N posterior drawer force. This posterior drawer force was used to ensure loading of the PCL and would also have masked the effect of any small forces unintentionally introduced during the flexion–extension cycle. In both studies anatomical fibres were not considered: length changes were calculated from a central tibial location to various femoral locations. It was claimed that the tibial insertion position of a fibre did not significantly affect its length change pattern.

Five knees were tested by Hefzy et al (1986); they reported that a small group of fibres in the posterosuperior corner of the femoral origin of the PCL shortened from 0° to 90° of flexion and that the bulk of the ligament lengthened over the same region (Figure 1.15). Hefzy et al (1986) also considered the effect of tibial rotation: they found that the length change due to rotation was small compared to the effect of flexion–extension. They also reported that the effect of tibial rotation on the PCL depended on the axis of the rotation, which varied from

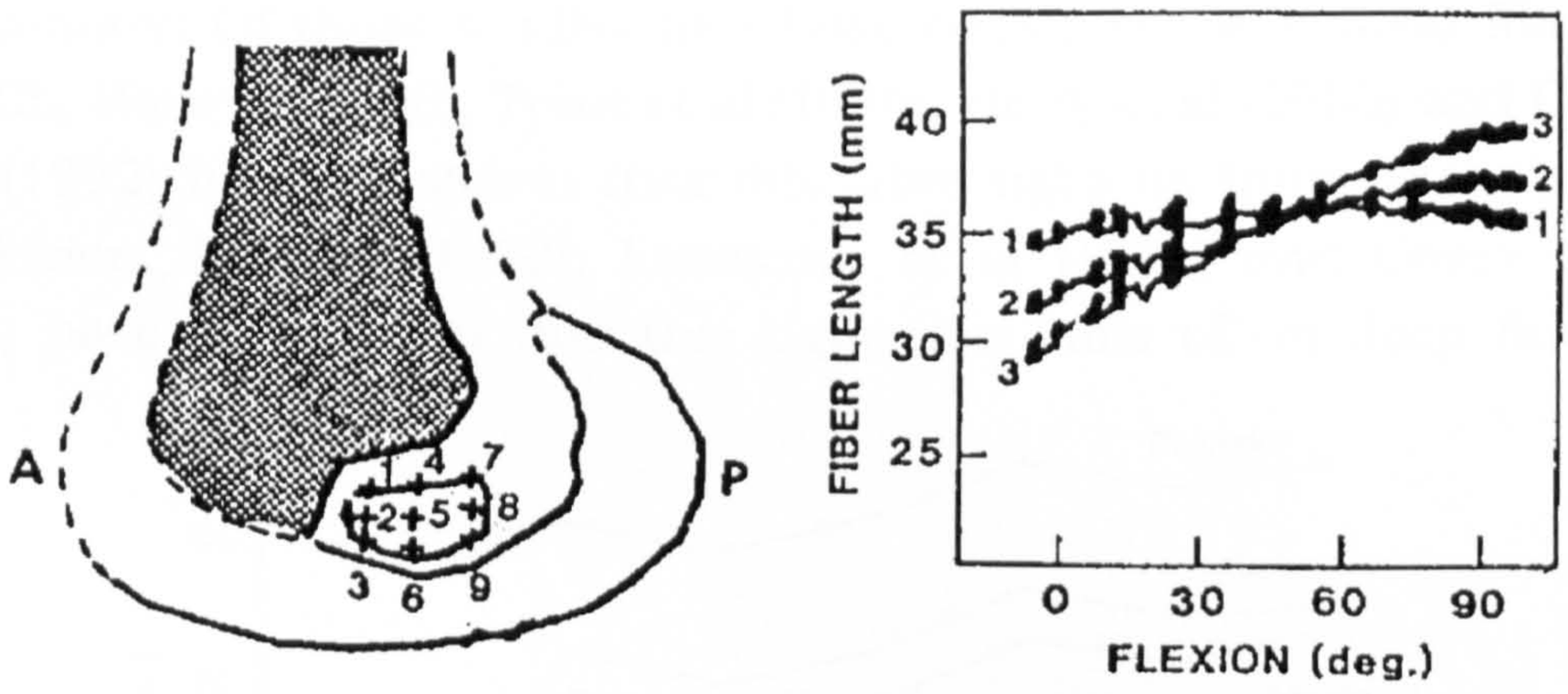


Figure 1.15. Length change with flexion for a range of PCL femoral locations. Data from Hefzy et al (1986), a 'typical' specimen.

specimen to specimen, as reported by Trent et al (1976). Only one knee was tested in the study of Hefzy and Grood (1985); they found that the posterior fibres shortened from extension to about 40° of flexion and then lengthened with further flexion and that the anterior and inferior fibres lengthened steadily from extension to flexion. They found smaller length changes in the central and superior fibres.

Covey et al (1992) implanted wires into four areas of the PCL in six knees. The wires were linked to a linear variable displacement transducer to record length changes directly. The wires were spring loaded to 10–15N. Covey et al (1992) found that the anterior and central fibres of the PCL lengthened considerably with flexion and that the posterior longitudinal fibres shortened and then lengthened with flexion (Figure 1.17). The posterior oblique fibres were nearly isometric.

In summary: Of those studies that have considered a 'central fibre' of the PCL, Horwitz (1938), Trent et al (1976), Hefzy et al (1986) and Covey et al (1992) have all shown that this fibre tightens from extension to mid-flexion. Horwitz (1938), Kurosawa et al (1991) and Covey et al (1992) have also shown that this tightening tails off in deep flexion.

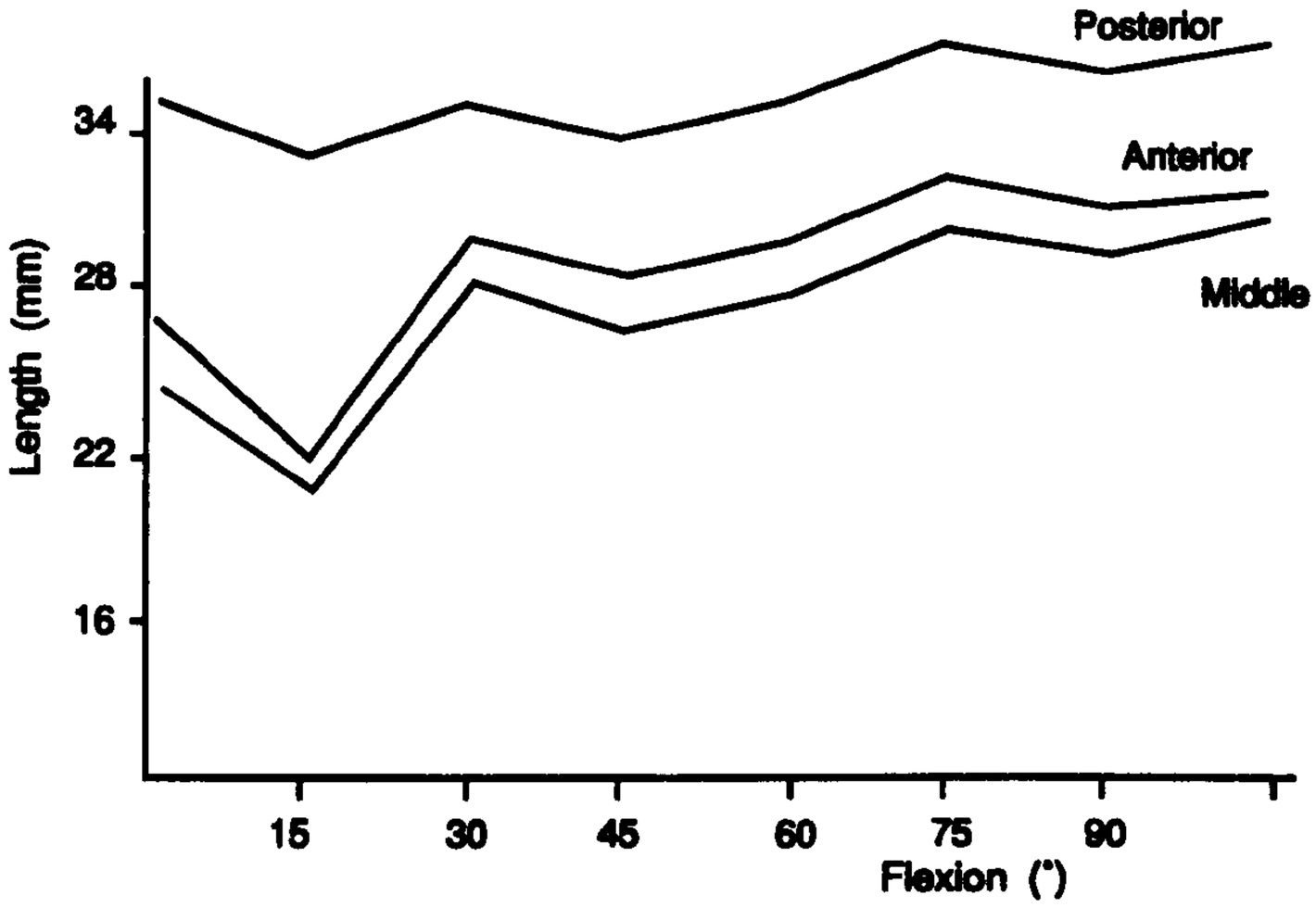


Figure 1.16 Length change with flexion of anterior, middle and posterior fibres of the PCL. Data from Trent, Walker and Wolf (Trent et al, 1976), n = 3

However, in the study of Wang et al (1973) the central fibre showed the opposite behaviour. This discrepancy was probably due to different methods of defining the 'central' fibre of the PCL.

With regard to the anterior fibres of the PCL, all the above studies have shown that these fibres tighten from extension to mid-flexion (Wang et al, 1973; Trent et al, 1976; Satku et al, 1984; Kurosawa et al, 1991; Covey et al, 1992). The studies of Kurosawa et al (1991), Satku (1984) and Wang et al (1973) have also shown that the anterior fibres slacken from mid-flexion to deep flexion.

With regard to the posterior fibres of the PCL, Trent et al (1976) and Covey et al (1992) reported that they were of near constant length whereas Kurosawa et al (1991), Satku et al (1984) and Wang et al (1973) showed that they tightened in extension. Satku et al (1984) and Wang et al (1973) also showed that the posterior fibres tightened in deep flexion

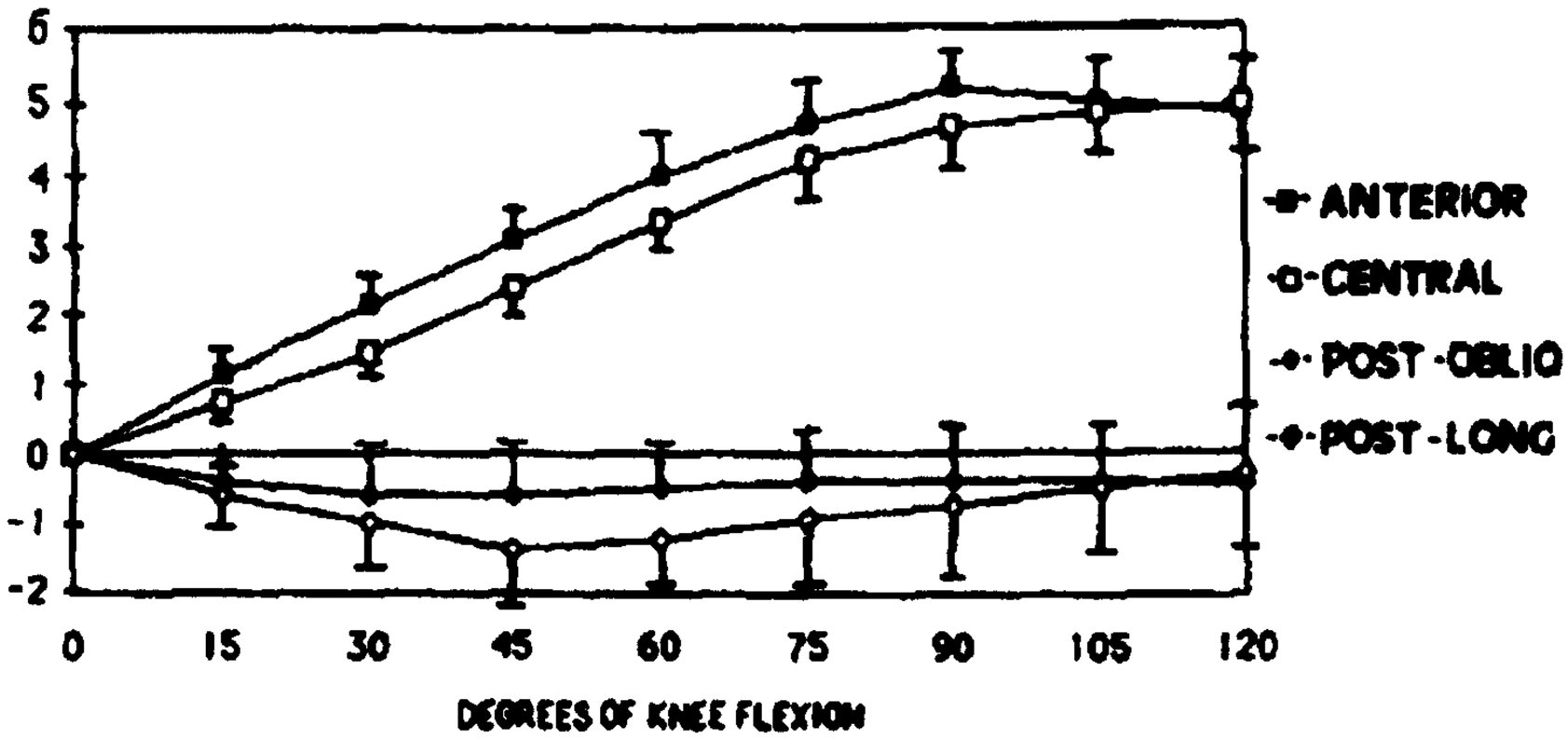


Figure 1.17 Graph of length change in millimetres with flexion for four areas of the PCL. Data from Covey et al (1992), n = 6.

All the above length change studies measured the distance between bony insertions and therefore suffered from the same problem: if a fibre was slack in some part of flexion-extension then the 'functional' range of the fibre could not be determined if its zero strain length was not known. If a fibre was buckled below its rest length then it cannot have been acting to control joint motion or resist any load. Since it is impractical to measure the zero lengths of fibres in the intact knee some investigators have attempted to deduce length changes by mathematical modelling

1.2.7 Mathematical Models

There have been many mathematical models of the knee (Hefzy and Grood, 1988) but only a few have given detailed consideration to the ligaments. These ligament based models have been used to estimate 'unmeasurable' parameters of physical experiments, to describe the kinematics of the knee, and to study a greater variety of conditions than could easily be achieved in a physical experiment.

1.2.7.1 The Rest Length Problem

As mentioned in section 1.2.6, ligament 'length change' studies have measured fibre insertion site separation. To deduce the actual length change of a real fibre from this data requires knowledge of the fibre's zero strain or rest length: it is possible for a fibre to buckle as its bony insertions become closer together than its rest length. This problem is also of paramount importance to mathematical models, since changes to fibre rest length or pretension are reported to make large differences in A-P stability results (Wismans et al, 1980).

Arms and Butler (1989) found 'optimised' zero strain fibre lengths for a simple model based on insertion site location data from a single knee. The co-ordinates of the tibial and femoral insertion sites of fifteen fibre bundles within the ACL and thirteen bundles within the PCL were recorded, in the neutral position, at 30° intervals from 0° to 90° of flexion. It was assumed that those cruciate fascicles that had the greatest insertion site separation at 90° would be simultaneously ruptured by 8mm of pure anterior or posterior displacement. It was further assumed that the bundles would all fail at a strain of 15%. This allowed the calculation of zero strain lengths for those fibres which were longest at 90°. Failure of these fibres under pure A-P motion at the lower flexion angles was then used to define the rupture displacement for those bundles which were longest at each particular flexion angle.

Once the zero strain lengths were calculated Arms and Butler (1989) used the model to predict the A-P load/displacement characteristic of the knee. To estimate the forces resulting from the strains derived from the model they assumed that each bundle had a cross–sectional area of $4mm^2$. They further assumed that each bundle had a stress/strain characteristic defined by a third order polynomial fit to experimental data for ACL fascicles from a previous study. They reported that the resulting load/displacement curves were within the range of those reported in the literature. This was a small claim as it has long been realised that there is a considerable range of 'normal' joint laxity (Hazen, 1910). Unfortunately they did not report the load/displacement curves for the knee used to generate the model, which would have provided more direct validation.

Arms and Butler (1989) stated that one source of error may have been that different regions of ligaments may have different material properties. One curious feature of their model was that the ACL was 15% larger, and therefore 15% stronger, than the PCL. All previous mechanical properties studies have shown the PCL to be much stronger than the ACL (Trent et al, 1976; Kennedy et al, 1976; Marinozzi et al, 1983).

Blankevoort et al (1991) also resolved the rest length problem by using assumed maximal fibre strains. The reference point they used was the maximum insertion site separation during passive flexion-extension. They then calculated fibre rest lengths by assuming that at the point of maximum insertion site separation a fibre was under a strain of 5% or 10%. Knees were installed in a six degrees of freedom loading jig. The position of the tibia and femur were recorded using Roentgen stereophotogrammetry based on tantalum marker pellets. Position data for four specimens were collected for passive flexion from 0° to 90°, 30N A-P drawer loads and 3Nm internal–external tibial torques.

To allow for the uncertainty in assumed maximal strain they reported results as 'recruitment probabilities'. A bundle at the assumed maximal strain, which was at the greatest insertion site separation, was assigned a recruitment probability of 1. A bundle at, or below, the 'zero strain' length derived from the assumed maximal strain was assigned a recruitment probability of 0. Intermediate pseudo–strains were assigned intermediate values.

For passive flexion, over the range 0°-90°, Blankevoort et al (1991) concluded that the aPC was increasingly recruited from 60° to 90° of flexion and that the pPC was recruited in extension. Neither bundle was recruited between 10° and 45° of flexion. For 3Nm internal–external tibial torques they concluded that the aPC was recruited in external rotation, at higher flexion angles, and that the pPC was recruited in internal rotation at 90°.

For a 30N posterior drawer they reported that at 90° the aPC had a high recruitment probability and the pPC a low recruitment probability. At 20° of flexion neither bundle was ever recruited. Blankevoort et al (1991) suggested that this finding, which was described as surprising,

was because some intermediate fibres of the PCL, not considered in their study, were functional in that region. They did not report considering that the posterior drawer force may have been resisted by some fibres other than the intermediate PCL which were also not represented in their model. This possibility is certainly suggested by previous studies that have shown that the arcuate complex resists posterior drawer below 30° of flexion (Gollehon et al, 1985; Grood et al, 1988).

Mommersteeg et al (1994) recorded the kinematics of knee specimens under various loading conditions using Roentgen stereophotogrammetry. They then isolated the cruciates and collaterals in bone–ligament–bone preparations. A number of fibre bundles were identified and digitised. The mechanical properties of these bundles were determined by an optimisation model based on repeated multiple direction tensile tests. The model ligament data was then combined with the previously recorded kinematic data which allowed bundle lengths to be computed. The mechanical properties data was then used to calculate bundle loads and their components in relevant directions. Mommersteeg et al (1994) investigated the loading of the cruciates and collaterals for combinations of 150N compression with internal–external and adduction–abduction rotation and A-P drawer. With regard to posterior drawer they found that the load in the PCL was greatest at extension and least at 30° of flexion. It was also found that the load in the PCL shifted from the posterior to anterior bundles with flexion. The horizontal component of the PCL force was considerably less than the applied drawer force for all flexion angles: this meant that other structures or articular contact were resisting most of the posterior force. The contribution of the PCL to resisting posterior drawer was about 45%, 8%, 23% and 20% for flexion angles of 0°, 30°, 60° and 90° respectively. These load shares appear to contradict the conventional wisdom that the PCL resists the majority of posterior drawer force. However, Hsieh and Walker (1976) found that compressive loading drastically reduced posterior laxity at 30° of flexion. The compressive loads used by Hsieh and Walker (1976) were higher than the 25% of body weight used by Mommersteeg et al (1994) but the biggest drop in posterior laxity occurred on the application of the first 50% of body weight.

1.2.7.2 Four–bar linkage Models

The four–bar linkage concept is often used as an aid to the understanding of the cruciate ligaments (Kapandji, 1987) and it has been used to model the kinematics of the knee since 1917 (Strasser, 1917). In recent times the four–bar linkage has been used in the models of O'Connor et al (1990), Fuss (1989) and Zavatsky and O'Connor (1993). Figure 1.18 shows the physiological basis for the four–bar linkage according to O'Connor et al (1990).

By assuming that the knee is a planar mechanism controlled by isometric ligament fibres the four–bar linkage can easily model the angles between the cruciates and the femur and tibia. The instant centre of rotation of the knee is simply calculated by virtue of the fact that it must lie on the intersection of the two cruciate links.

In order to explore the effect of changing the parameters of the four–bar linkage the equations given in the thesis of Zavatsky (1993) were used to generate a Microsoft Excel spreadsheet. The spreadsheet was set up to produce plots of the linkage with both the femoral and tibial links fixed and the instant centres of rotation overlaid. The angles between the PCL and the tibia and femur were also calculated over the range of flexion. The plots of the four–bar linkages were imported into a standard drawing package (Corel Draw) to give the correct aspect ratio and scale. The available published data was input to generate Figure 1.19 and Figure 1.20.

One of the fundamental assumptions of the four–bar linkage model is that there exist points on the femur and tibia which are equidistant over the range of flexion; these points give the locations of the 'neutral' fibres in the cruciate ligaments. The variability of the length change studies reviewed in section 1.2.6 shows that choosing an isometric point is difficult.

In the model of O'Connor et al (1990) the average centres of the anatomical cruciate insertions of four cadaveric knees were used as the

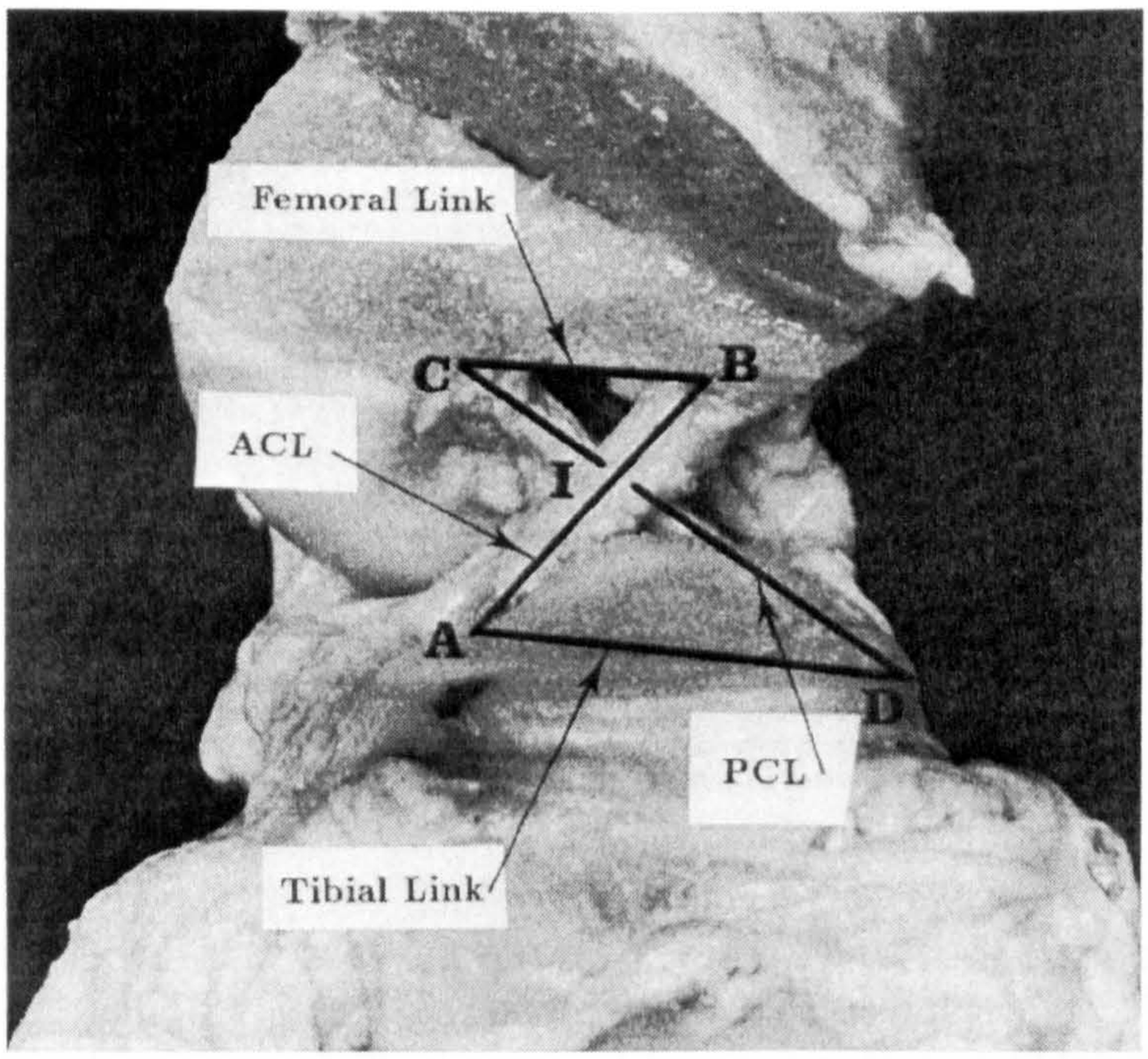


Figure 1.18 The physiological basis for the four–bar linkage according to O'Connor et al (1990).

'neutral' insertion sites. Although isometry in general and of the neutral fibres specifically was discussed they did not provide any evidence that the centres of the anatomic insertions behaved isometrically. Comparison of Figure 1.18 and Figure 1.19 shows that the links are of quite different proportions although the links in Figure 1.18 appear to have been constructed in accordance with the reported procedure used to generate the links in Figure 1.19. This discrepancy confirms the difficulty of choosing general 'neutral fibres'.

Zavatsky (1993) criticised the central fibre equals neutral fibre assumption of O'Connor et al (1990) on the grounds that some fibres were always anterior to the flexion axis. This meant that 'such fibres would offer resistance to flexion which is not observed in the human joint'. This criticism is not in itself sufficient as it is still possible to have

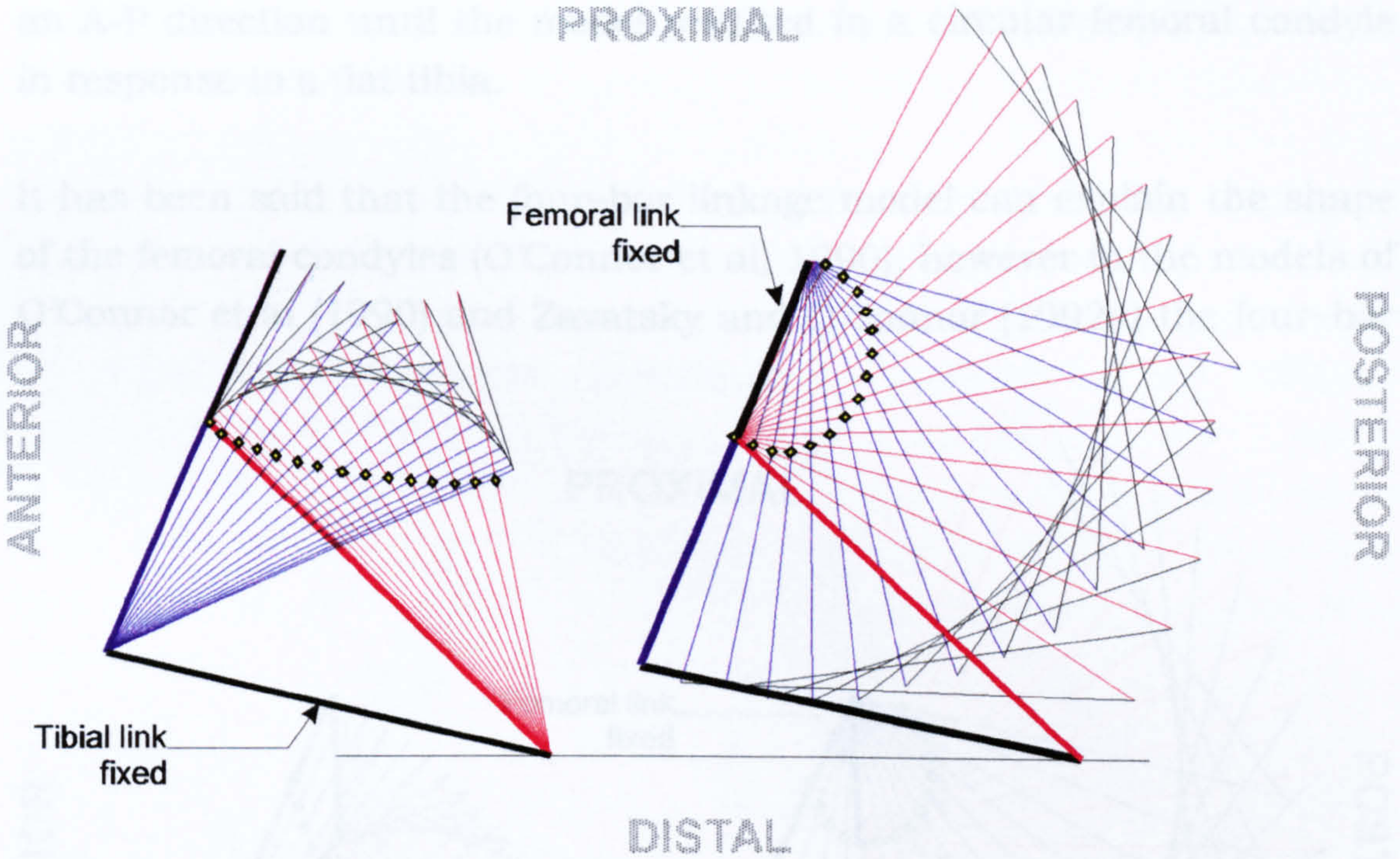


Figure 1.19 The four–bar linkage model of O'Connor et al (1990) drawn at 10° intervals between 0° and 140° of flexion. The extension position of the linkage is shown by the bold lines. The ACL is drawn in blue, the PCL in red and the femoral and tibial links are drawn in black. The instant centres of rotation are plotted as black dots

negligible resistance to flexion if some fibres tighten and some slacken with flexion (flexion–extension would still require some energy, due to hysteresis and friction). Also, natural ligament fibres have a 'toe' region of low stiffness which will offer very little resistance to a flexion moment if small (1%/0.3mm) fibre extensions are involved, especially considering the very small moment arms of the anisometric cruciate fibres about the flexion axis.

Zavatsky and O'Connor (1992a) chose a neutral ACL fibre based on several studies which showed that the most isometric fibre of the ACL was its most anterior in extension. This neutral fibre fitted in with Zavatsky's requirement that other ACL fibres should not be stretched with flexion. A PCL neutral fibre was based on the posterior oblique band which was found to be most nearly isometric in the study of Covey et al (1992). The femoral insertion of the neutral fibre was adjusted in an A-P direction until the model resulted in a circular femoral condyle in response to a flat tibia.

It has been said that the four–bar linkage model can explain the shape of the femoral condyles (O'Connor et al, 1990), however in the models of O'Connor et al (1990) and Zavatsky and O'Connor (1992a) the four–bar

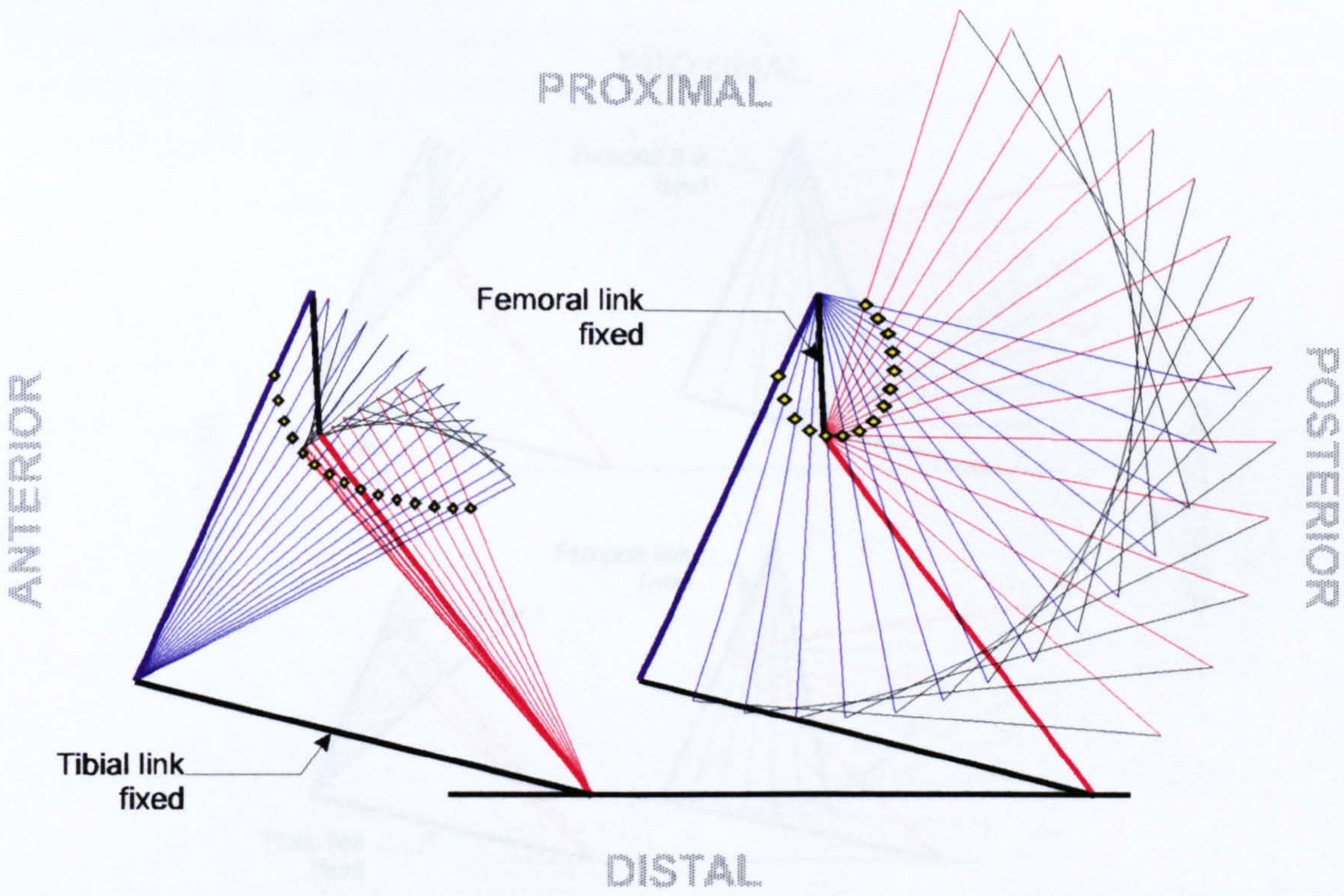


Figure 1.20 Four–bar linkage, drawn at 10° intervals, from the data of Zavatsky and O'Connor (1992a). The extension position of the linkage is shown by the bold lines. The ACL is drawn in blue, the PCL in red and the femoral and tibial links are drawn in black. The instant centres of rotation are plotted as black dots.

linkages were deliberately adjusted until they produced the desired circular femoral condyle in response to a flat tibia. While the geometries of the femoral and tibial condyles are not incompatible with a four–bar linkage, for any linkage there exists no unique solution for matching surfaces (O'Connor et al, 1990). It has been popular to suggest that the

geometry of the cruciates defines the geometry of the condyles, that nature started with the four bar link and then developed the knee. It is of course equally appropriate to suggest that the geometry of the condyles and the menisci define the insertions of the cruciates. Given the capacity of bone and soft tissue to remodel it is hard to define any 'fixed' property of the human body. The explanation for the *particular* shape of the condyles and for their asymmetry must lie in a more subtle analysis. I suspect that the condyles are optimised for muscle actions and ease of locomotion.

As stated earlier, the four–bar linkage can easily model the change in angle between the cruciates, femur and tibia with flexion. The models of O'Connor et al (1990) and Zavatsky and O'Connor (1992a), both based on apparently reasonable assumptions, produce rather different

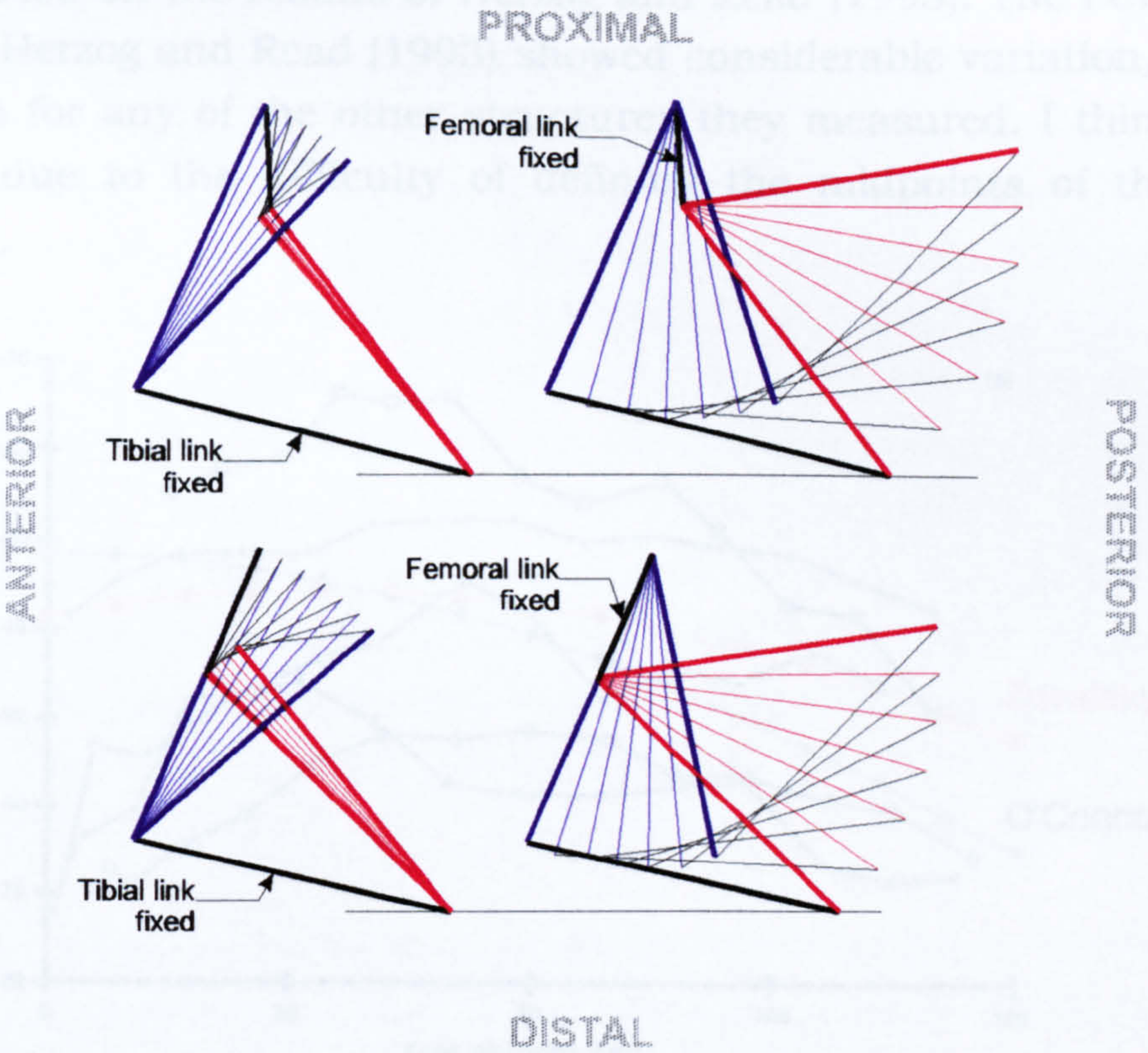


Figure 1.21 The four–bar linkages of Zavatsky and O'Connor (1992a), top, and O'Connor et al (1990), bottom. Drawn at 10° intervals from extension to 60° of flexion, this is the range of flexion experienced during normal gait (Lafortune et al, 1992). The 0° and 60° positions of the ACL and PCL are shown by bold lines.

answers (Figure 1.19 and Figure 1.20). For flexion between 0° and 140°, the model of O'Connor et al (1990) predicts angular changes between the tibia and the PCL and the femur and the PCL of 34° and 106°

respectively, whereas the model of Zavatsky and O'Connor (1992a) predicts changes of 17° and 126°. The 'extra' 3° (17+126≠140) in the model of Zavatsky and O'Connor (1992a) comes from the initial counter rotation of the PCL due to the uncrossed starting configuration. The sagittal plane lines of action of the major knee ligaments during passive flexion have been measured experimentally by Herzog and Read (1993) using a 3–D mechanical digitiser. Obviously the choice of measured fibre affected the results. For the PCL Herzog and Read (1993) chose to measure the line of action of a pseudo–fibre joining the 'midpoints of the insertion area on tibia and femur'. This pseudo–fibre definition was identical to the definition used by O'Connor et al (1990) to define the neutral fibre of the PCL in their four–bar linkage model. Figure 1.22 shows the theoretical line of action of the PCL from the models of O'Connor et al (1990) and Zavatsky and O'Connor (1992a) superimposed on the results of Herzog and Read (1993). The PCL angle results of Herzog and Read (1993) showed considerable variation, much more than for any of the other structures they measured. I think that this was due to the difficulty of defining the midpoints of the PCL insertions.

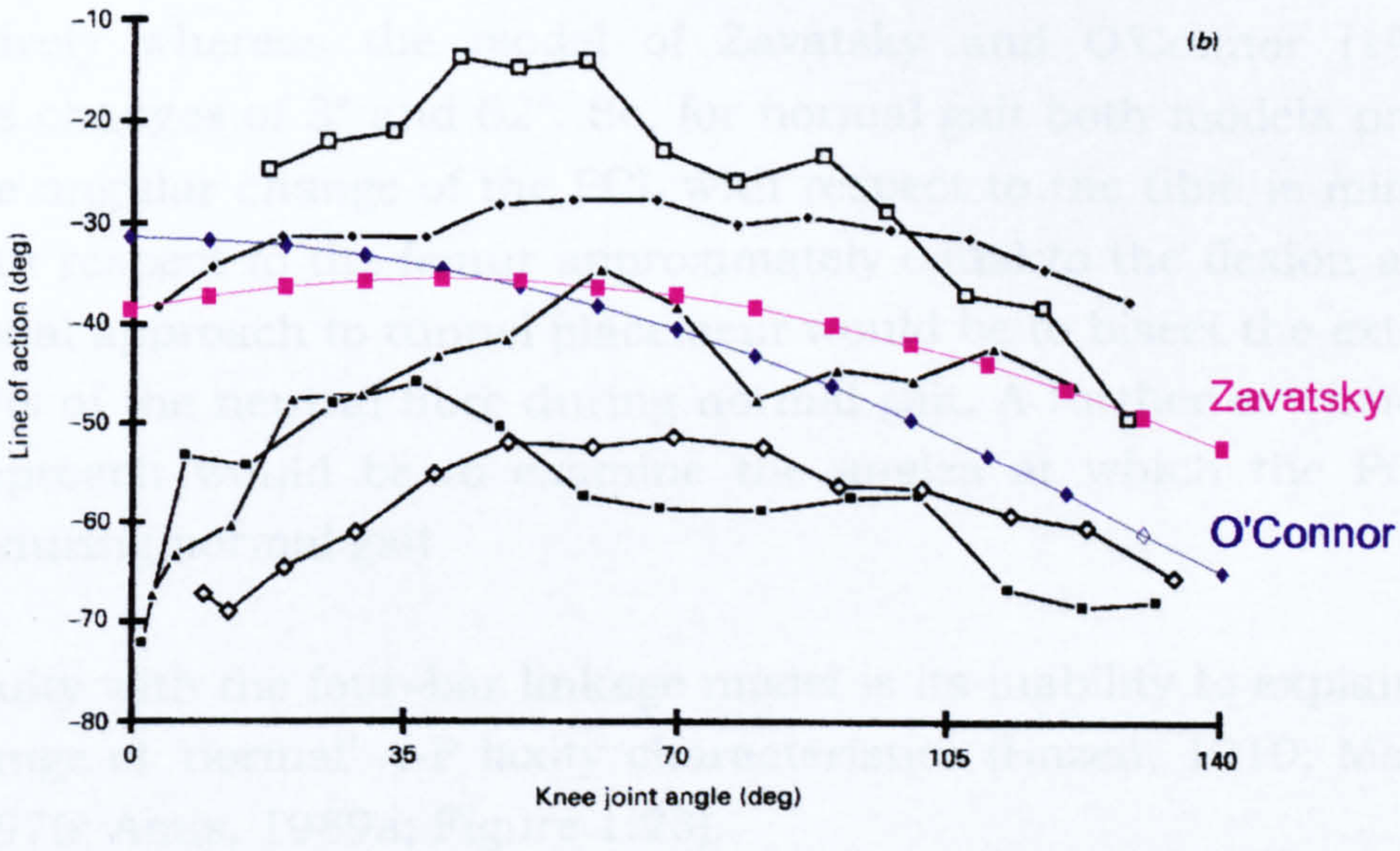


Figure 1.22 Graph of the change in angle between the PCL and the tibia with flexion, data from Herzog and Read (1993). The coloured lines represent the results of the models of Zavatsky and O'Connor (1992a) and O'Connor et al (1990) scaled to match the axis of the experimental data.

The angle between the PCL and the femur and tibia is pertinent to the choice of bone tunnel direction in reconstructive surgery and to the

orientation of specimens for mechanical testing. O'Connor et al (1990) suggested that the bending of implants would be minimised by choosing a tunnel angle that bisected the extreme positions of the neutral fibre. This could be described as the 'median' angle approach. Close examination of Figure 1.19 and Figure 1.20 will reveal that, assuming all flexion angles occur equally, the median angle is not coincident with the mean angle. This is because equal steps in flexion angle did not result in equal steps in the angle between the femur and PCL or the tibia and PCL. The rate of change of the angle between the PCL and the femur was greatest at extension and decreased with flexion. The rate of change of the angle between the PCL and the tibia was minimal near extension and increased with flexion. Thus a 'mean' angle approach would be to bias the tibial tunnel towards the extension position and the femoral tunnel towards the flexion position. I suggest that the best approach may be 'modal': the most frequent loading of a ligament implant will occur during normal gait. During normal gait the knee flexes between 0° and 60° (Lafortune et al, 1992). Figure 1.21 shows the four–bar linkages of O'Connor et al (1990) and Zavatsky and O'Connor (1992a) constructed for 0° to 60° of flexion: the model of O'Connor et al (1990) predicts angular changes of 7° and 53° with the tibia and femur respectively whereas the model of Zavatsky and O'Connor (1992a) predicts changes of 3° and 62°. So, for normal gait both models predict that the angular change of the PCL with respect to the tibia is minimal and with respect to the femur approximately equal to the flexion angle. The modal approach to tunnel placement would be to bisect the extreme positions of the neutral fibre during normal gait. A further extension of this approach would be to examine the angles at which the PCL is loaded during normal gait.

A difficulty with the four–bar linkage model is its inability to explain the wide range of 'normal' A-P laxity characteristics (Hazen, 1910; Markolf et al, 1976; Amis, 1989a; Figure 1.23).

In the model of Zavatsky and O'Connor (1992a) it was assumed that the ligaments were at zero strain at some reference position, given by the maximum insertion site separation, and that passive flexion–extension away from this position caused fibre slackening. This assumption led to a model in which no fibres were loaded in A-P neutral and in which the 'neutral' fibres were just taut (at zero strain). This result is compatible

with the laxity characteristic shown by specimen (b) in Figure 1.23: The cruciates are just unloaded in A-P neutral and progressively recruited by anterior or posterior displacement.

Fuss (1989) also assumed that fibres were just taut when their insertion site separation was maximal. It should be noted that his justification of this assumption was rather unconventional: 'Owing to their extremely low elasticity module, the collagenous fibres have a practically invariable length; therefore they are taut when the distance between origin and insertion is equal to this given length. This is the greatest possible distance between origin and insertion since a further increase would mean rupture of the fibres.'

Other workers have made radically different assumptions about fibre strain in A-P neutral. In the study of Blankevoort et al (1991) it was

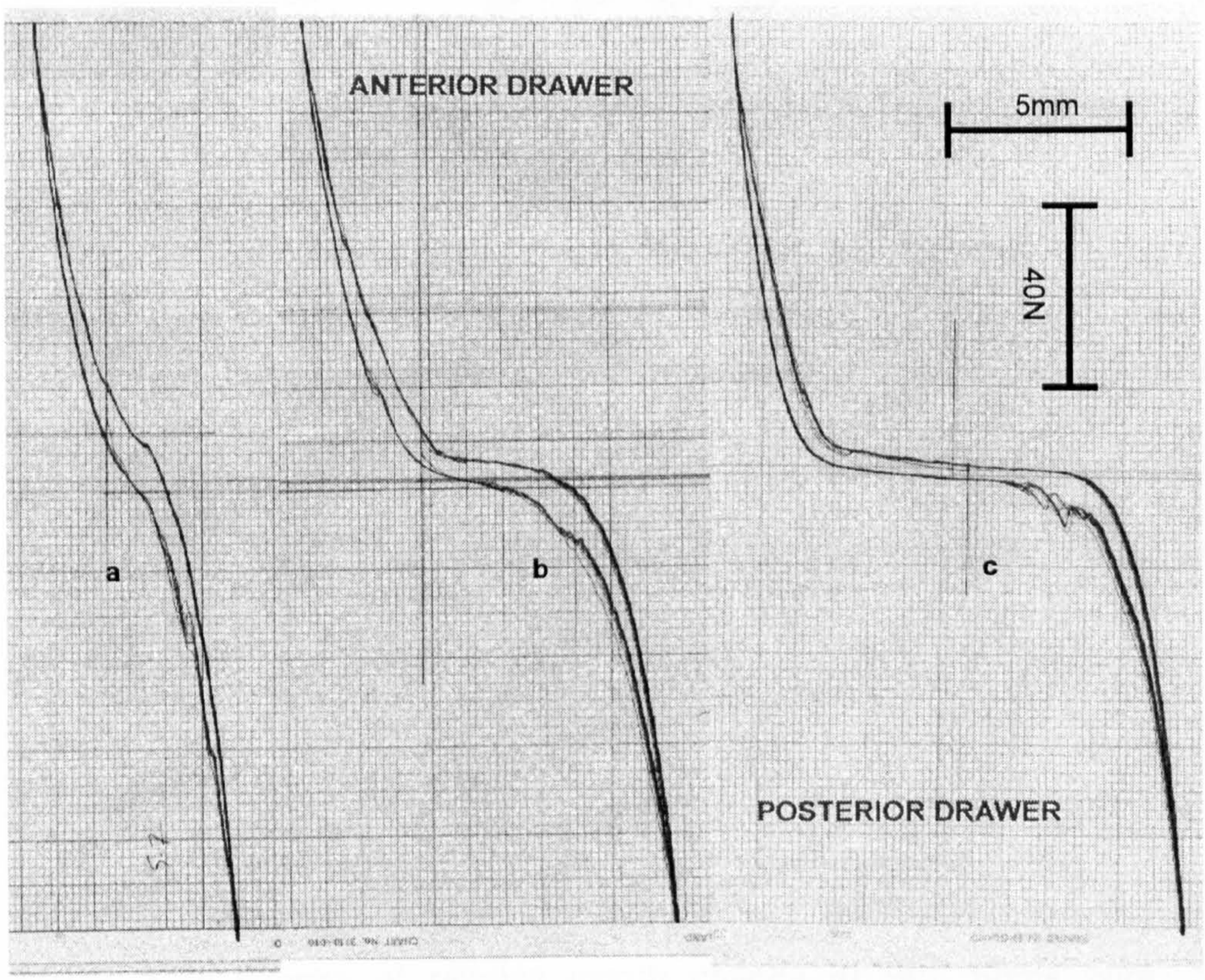


Figure 1.23 A-P drawer characteristics at 90° of flexion for three 'normal' knees (output from Instron chart recorder). Note that the ultimate stiffnesses of the knees, when the cruciates are loaded, are very similar. In a 'tight' knee (a) the cruciates are antagonistically loaded near A-P neutral, shown by the sharp change in gradient. In an 'intermediate' knee (b) the cruciates are just unloaded at A-P neutral, shown by a region of near zero stiffness. In a 'loose' knee (c) there is a region of near zero stiffness of several millimetres where no structures, cruciates or 'secondary' restraints, are loaded by A-P drawer.

assumed that passive flexion–extension generated maximum strains of 5% or 10% in each fibre. This assumption led to a model in which there was some probability that the cruciates were antagonistically loaded in A-P neutral at 90° of flexion. This result is compatible with the laxity characteristic shown by specimen (a) in Figure 1.23. At lower flexion angles there were regions were both cruciates were slack. Crowninshield (1976) also assumed a 5% maximum strain for passive flexion–extension (1.2.7.3)

Four–bar linkage models have been used to investigate length changes in the non-neutral fibres. Extending the four–bar linkage model to

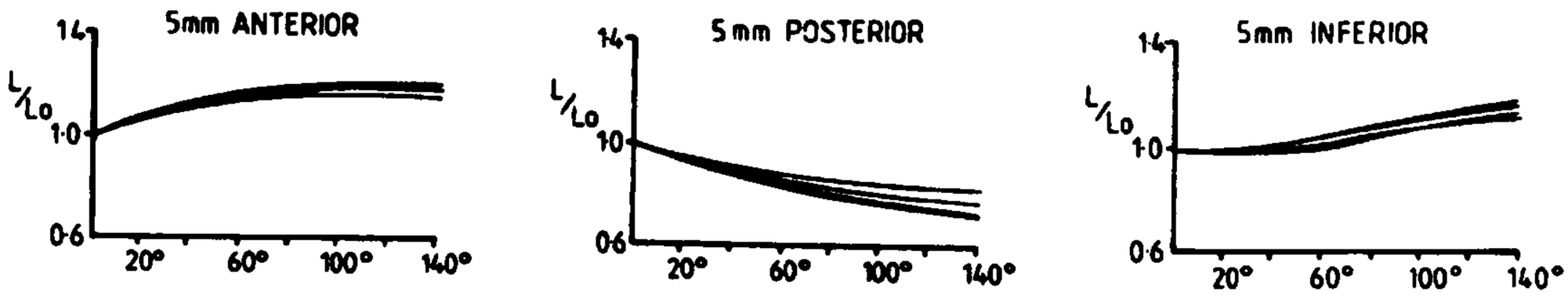


Figure 1.24 Relative length change between attachment sites 5mm away from the femoral insertion of the PCL neutral fibre. From the model of O'Connor et al (1990)

include non–neutral fibres has required some strategy to define the insertions of non–neutral fibres, or fibre mapping. Obviously it had to be assumed that length changes in the non–neutral fibres did not affect

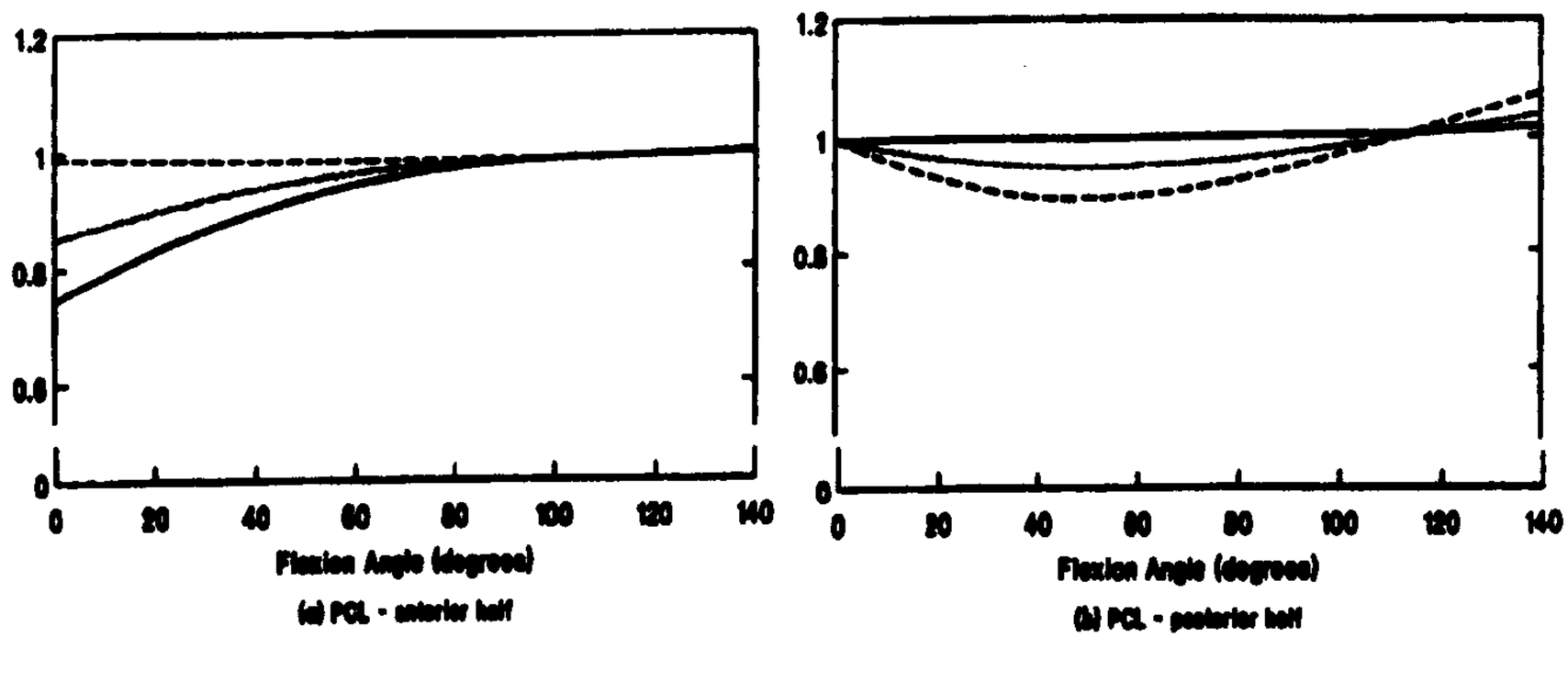


Figure 1.25 Graphs of relative length change of PCL fibres with passive flexion, from the model of Zavatsky and O'Connor (1992a). The solid and dashed lines represent the most anterior are the most posterior fibres respectively. Note that the most posterior and intermediate fibres of the posterior half of the PCL lengthen beyond 115° of flexion.

the kinematics of the joint. O'Connor et al (1990) bypassed this problem by suggesting that there may not be any physical neutral fibres, rather

the complex pattern of fibres found in the joint *combine* to behave as if there *were* an isometric neutral fibre. One can imagine different patterns of fibres which could combine to form this 'isometric illusion' in passive flexion, however different patterns could respond very differently to, say, A-P drawer. O'Connor et al (1990) suggested that clinical observations of the cruciates were best explained by a model in which the non–neutral fibres of each cruciate crossed in the sagittal plane. This was based on the finding that, for passive flexion between 0° and 140°, uncrossed ACL fibres resulted in strains of more than 20% whereas crossed fibres were more isometric: this was because the crossed fibres passed closer to the flexion axis. Zavatsky's design process was in part controlled by the need to have all fibres at no more than zero strain throughout the range of flexion. Zavatsky and O'Connor (1992a) chose to use uncrossed fibre mapping, based on published in–vitro investigations.

According to the model of O'Connor et al (1990) a PCL fibre 5mm anterior to the neutral fibre femoral insertion lengthened with flexion (Figure 1.24). In the model of Zavatsky and O'Connor (1992a) the anterior fibres (which were also distal to the neutral fibre) showed a similar pattern of lengthening with flexion (Figure 1.25) although in this model the fibre was considered to be at rest length in full flexion: this was necessary to maintain the condition that fibres should not be stretched by passive flexion.. For the posterior fibres of the PCL the model of O'Connor et al (1990) shows consistent fibre slackening with flexion (Figure 1.24) whereas the model of Zavatsky and O'Connor (1992a) shows fibres slackening and then retightening (Figure 1.25). The most posterior PCL fibres in the model of Zavatsky and O'Connor (1992a) lengthened about 8% beyond their rest length in deep flexion, which appears to have been in contravention of the model's rule that there should be no resistance to passive flexion.

Bradley et al (1988) published graphs for '...the length–change outcomes which result from moving the attachments of the anterior *(and posterior)* cruciate ligament...' However, these length changes were based on the assumption that the motion of the knee was *still controlled* by the *isometric* 'neutral' fibres chosen to represent the normal cruciate structure. Clearly, if the attachment of a ligament which controls knee motion is moved, the motion of the knee is likely to be influenced: the

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kinematics of a knee with an anisometric cruciate graft will not be governed by a 'neutral' fibre that is no longer present.

The model of Zavatsky was also used to investigate fibre recruitment in response to A-P drawer (Zavatsky and O'Connor, 1992b). Ligaments were assigned widths and material properties based on published experimental results. The widths of the ligaments were then adjusted to match the resulting force–displacement curves with published experimental data. Because different fibre regions were at different degrees of laxity over the range of flexion (Figure 1.25) there was progressive recruitment of fibres with increasing A-P displacement: initially only the neutral fibre was loaded followed by the adjacent fibres which were least lax. This mechanism would have been slightly altered at higher flexion angles where the posterior fibres of the PCL were strained in the 'neutral' position. Because the fibres in the array were at different initial laxities the strain pattern and resultant force were dependant on flexion angle and A-P displacement.

Four–bar linkage models are somewhat limited in that they cannot be used to model knee function in abnormal states, such as cruciate insufficiency, or model the effect of non–isometric ligament reconstructions.

1.2.7.3 Constrained Anatomical Models

In the model of Edwards et al (1970) the kinematics of the knee were fixed, and based on previously reported data. Ligament insertions were measured for a 'typical' knee in extension and single line elements were used to represent the PCL, ACL, LCL and MCL. The MCL element was divided in two to allow for wrapping around the tibial plateau. Joint motion and insertion site location were defined using an orthogonal co-ordinate system fixed to the centre of the tibia. Passive flexion was assumed to result in three components of femoral motion: rotation about the medial–lateral axis, translation along the longitudinal axis of the tibia and rotation about a parallel axis centred on the medial tibial condyle. Over the flexion range 0° to 130° the medial–lateral axis of rotation was assumed to move 10mm proximally and to rotate 30° about the proximal–distal axis at the centre of the medial tibial condyle. These coupled motions were assumed to occur in linear proportion to flexion.

Edwards et al (1970) used the model to calculate ligament length change with flexion. In reporting their results they translated length changes into 'percent elongation' by which an element's minimum length was defined as 0% and maximum length as 100%. Because of this it is difficult to compare their results with other studies which have reported length changes as absolute values or as some proportion of ligament length. They did not report considering the rest length problem.

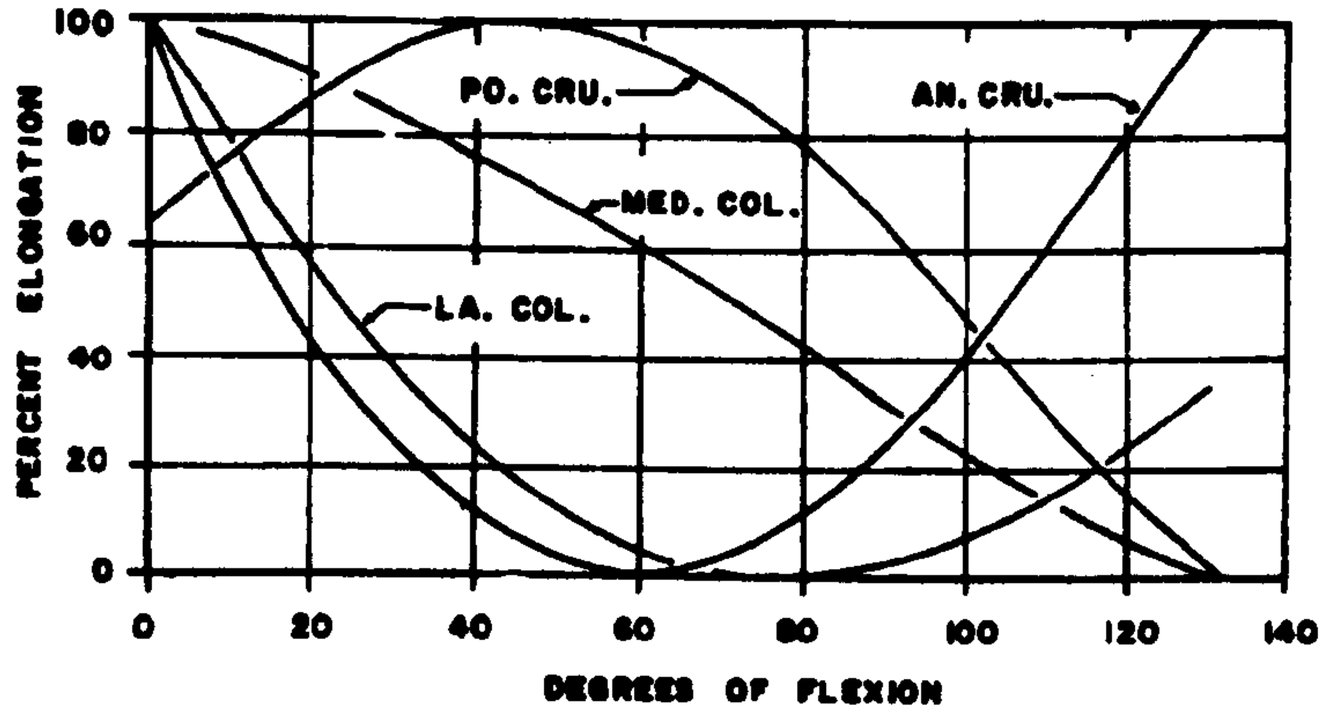


Figure 1.26 Ligament length changes in response to passive flexion from the model of Edwards et al (1970). 0% elongation = minimum length, 100% elongation = maximum length.

Crowninshield et al (1976) used a similar principle to Edwards et al (1970), anatomically based insertion co-ordinates moving according to a predetermined kinematic pattern, and developed the concept further. The ligaments of the knee were represented by thirteen line elements which included posterior capsule structures and posterior and anterior

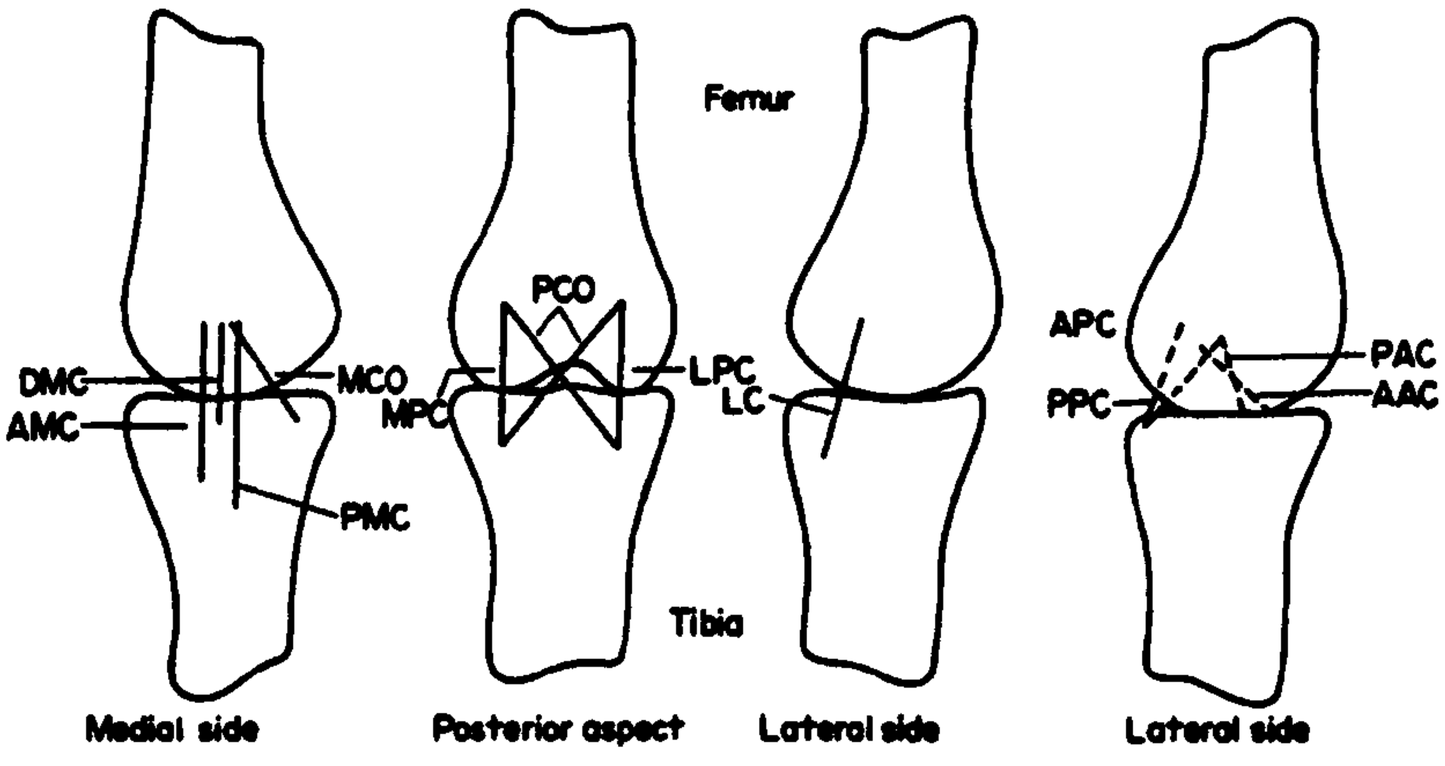


Figure 1.27 Line elements used in the model of Crowninshield et al (1976)

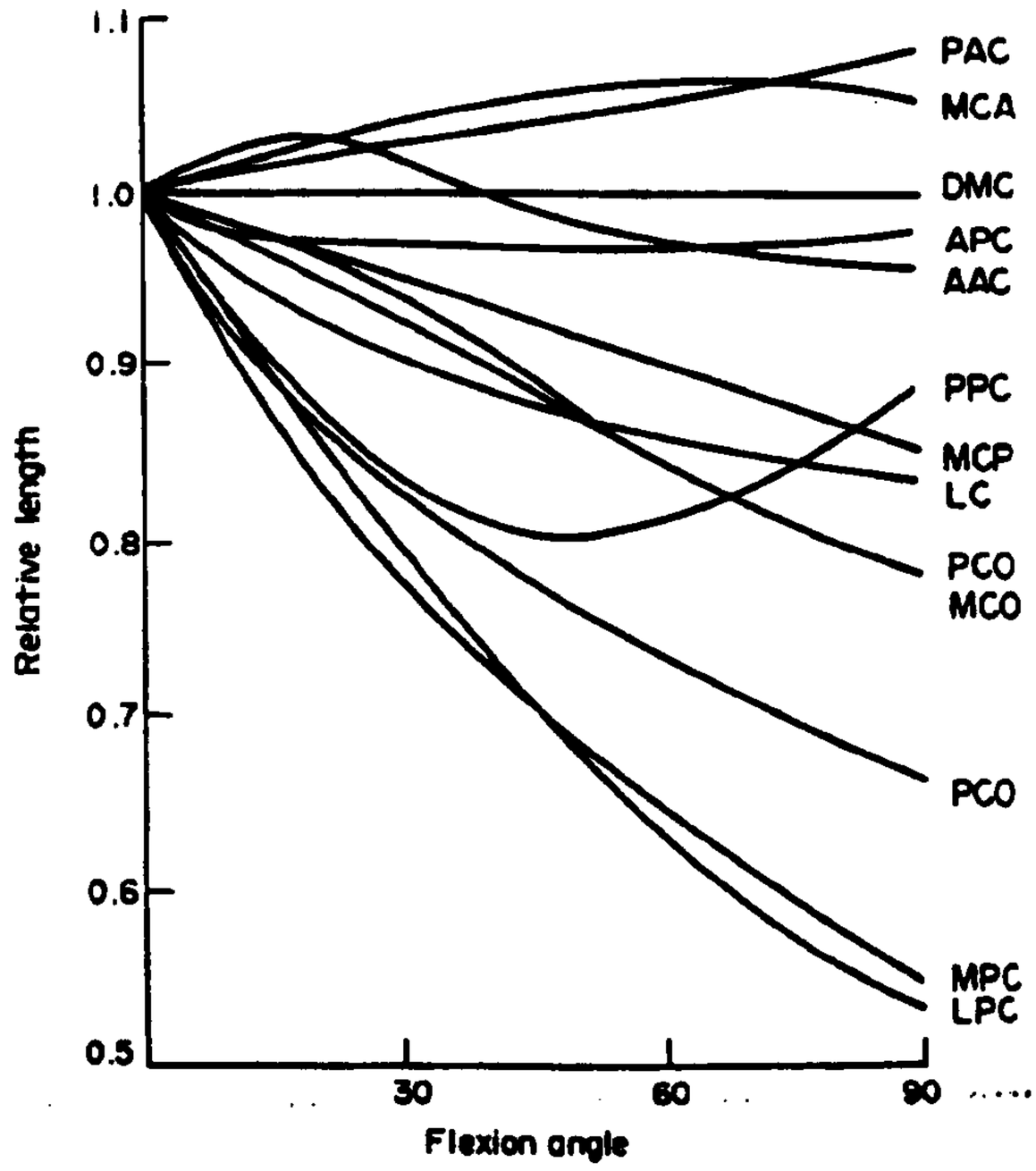


Figure 1.28 Relative length changes of line elements for the model of Crowninshield et al (1976). The line elements are shown in Figure 1.27.

portions of the cruciates (Figure 1.27). The kinematics of passive flexion were changed or refined. Rather than having the flexion axis move 10mm distally with flexion Crowninshield et al (1976) chose to move it 9mm posteriorly with flexion, again assuming the displacement was linearly proportional to flexion. The rotation of the flexion axis about the proximal–distal axis was assumed to be less than that used by Edwards et al (1970), 15° rather than 30°, and it was assumed that it occurred towards the start of flexion. This assumption better fitted the commonly observed pattern of terminal rotation of the tibia. The required non-linear pattern of rotation was implemented by a function of the fifth root of the flexion angle. These kinematic formulae allowed Crowninshield et al (1976) to calculate fibre length changes with flexion (Figure 1.28).

Figure 1.27 and Figure 1.28 show that the length changes calculated for the PCL by Edwards et al (1970) and for the aPC and pPC by Crowninshield et al (1976) are quite incompatible.

Crowninshield et al (1976) also used their model to calculate joint stiffness in response to various motions. To this end they had to assign mechanical properties and rest lengths to the line elements of the model. Ligament material properties were based on mechanical tests of

MCL specimens. MCL stiffness was found to be a linear function of strain up to 5% so the mechanical properties of each structure, which were assumed to be of the same material, depended on their cross–sectional area and the linear stiffness function. The cross–sectional areas were measured in cadaveric specimens using an unspecified technique. To solve the fibre rest length problem they assumed that passive flexion generated maximal strains of 5% in all line elements.

Crowninshield et al (1976) reported result in terms of normalised stiffness as a function of flexion angle in response to internal–external rotation, adduction–abduction rotation and A-P drawer. Results were also reported for PCL, ACL and MCL deficient knees. They did not report how they calculated stiffness so it is difficult to compare their results with other studies. Their own validation study showed reasonable agreement for the rotations but poor agreement for the A-P drawer tests. The model applied completely constrained A-P drawer but it was not clear what constraints were used in the validation study.

1.2.7.4 Unconstrained Anatomical Models

Some workers have sought to create models which describe the whole knee, including ligamentous and geometric constraints. In these models the kinematic behaviour of the joint is not restricted by the nature of the model, as in the four–bar linkage or constrained anatomical models, but is defined by the input data. With such models it becomes possible to model 'real' knees: data can be input from 'tight' and 'loose' knees to give distinctly different characteristics, as observed in 'normal' knees. There are, of course, many assumptions made in generating these models which have various consequences: clearly, a model which does not include the menisci cannot answer any questions about the effects of meniscectomy, a model that treats ligaments as single line elements cannot simulate a partial tear or reveal the interfascicular variation in load share.

Wismans et al (1980) fitted third or fourth order polynomial surfaces to the articular surfaces of a real knee. These surfaces, which were assumed to be rigid, were used to provide geometric constraints for the model. The soft tissues joining the tibia and femur were modelled as seven line elements with quadratic load elongation characteristics. The ACL, PCL and LCL were modelled as single elements and the MCL was divided into anterior and posterior portions. The menisci were not

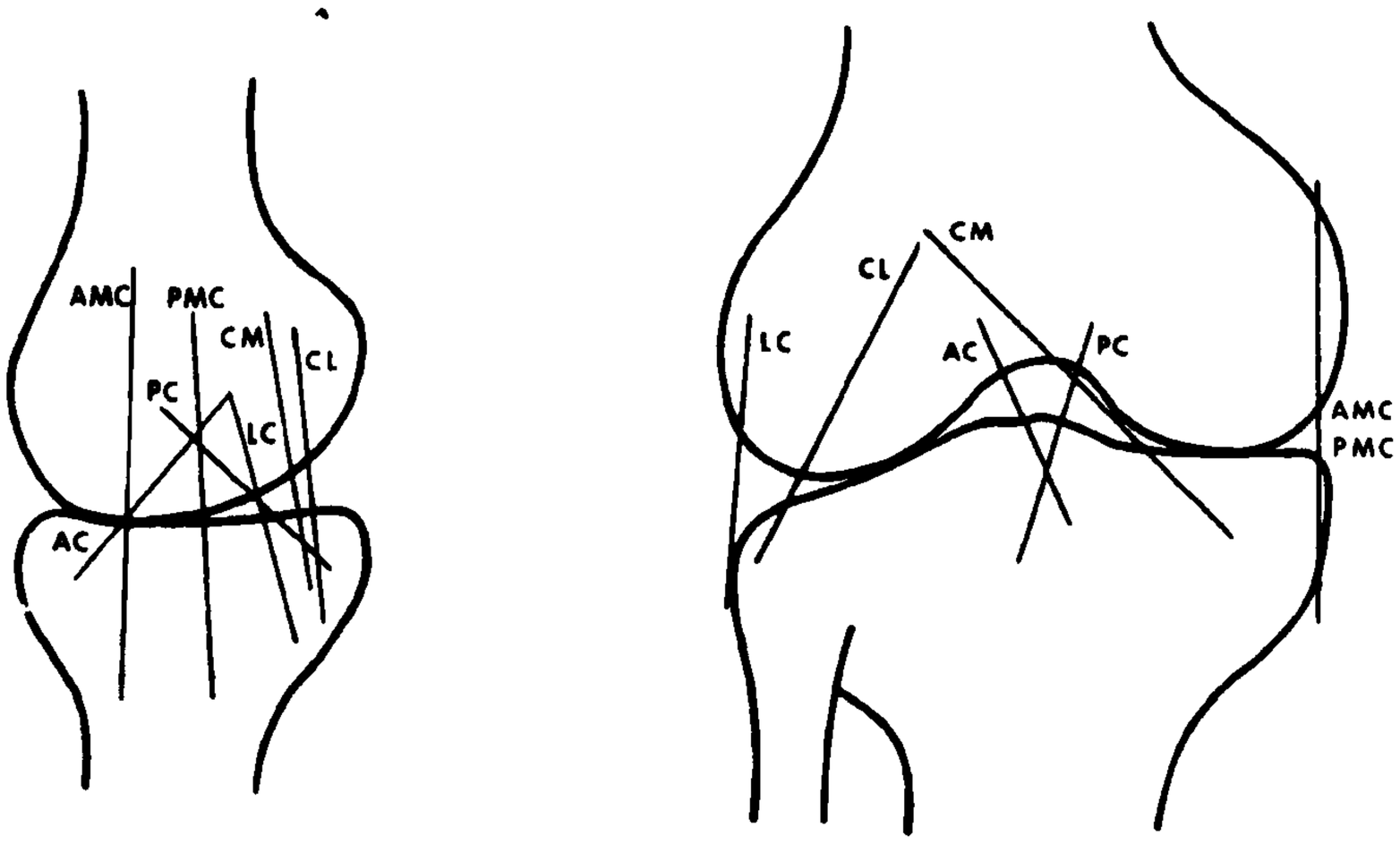


Figure 1.29 Medial–Lateral and Anterior–Posterior views of the line elements used to represent ligamentous structures in the model of Wismans et al (1980).

included in the model but the posterior capsule was modelled by two line elements, one lateral and one medial (Figure 1.29).

Results were presented for the strain in line elements in response to passive flexion (Figure 1.30) and the A-P laxity envelope for ±10N (Figure 1.31). Figure 1.30 shows that the cruciate line elements chosen by Wismans et al (1980) were far from isometric. Examination of the results of PCL length change studies for unloaded flexion–extension discussed in section 1.2.6 reveals no obvious correlation. However, the results of Hefzy et al (1986) for flexion–extension subject to 100N posterior drawer are not entirely incompatible: point #5 (Figure 1.15), at the centre of the femoral attachment, shows minimal length change between extension and 30° of flexion followed by lengthening at a decreasing rate. Wismans et al (1980) reported that the initial strains, at extension, of the line elements were chosen on the basis of qualitative descriptions in the literature. The initial strain on the PCL element was defined as -1%, presumably due to the common observation that the bulk of the PCL is slack in extension (section 1.2.1).

Their results for A-P laxity showed an order of magnitude difference between the laxity at 30° of flexion and that at 0°, 60° and 90°. This is understandable from Figure 1.30 which shows that the PCL was slack below 40° of flexion. The posterior laxity which resulted decreased towards extension when the posterior capsular elements were strained.

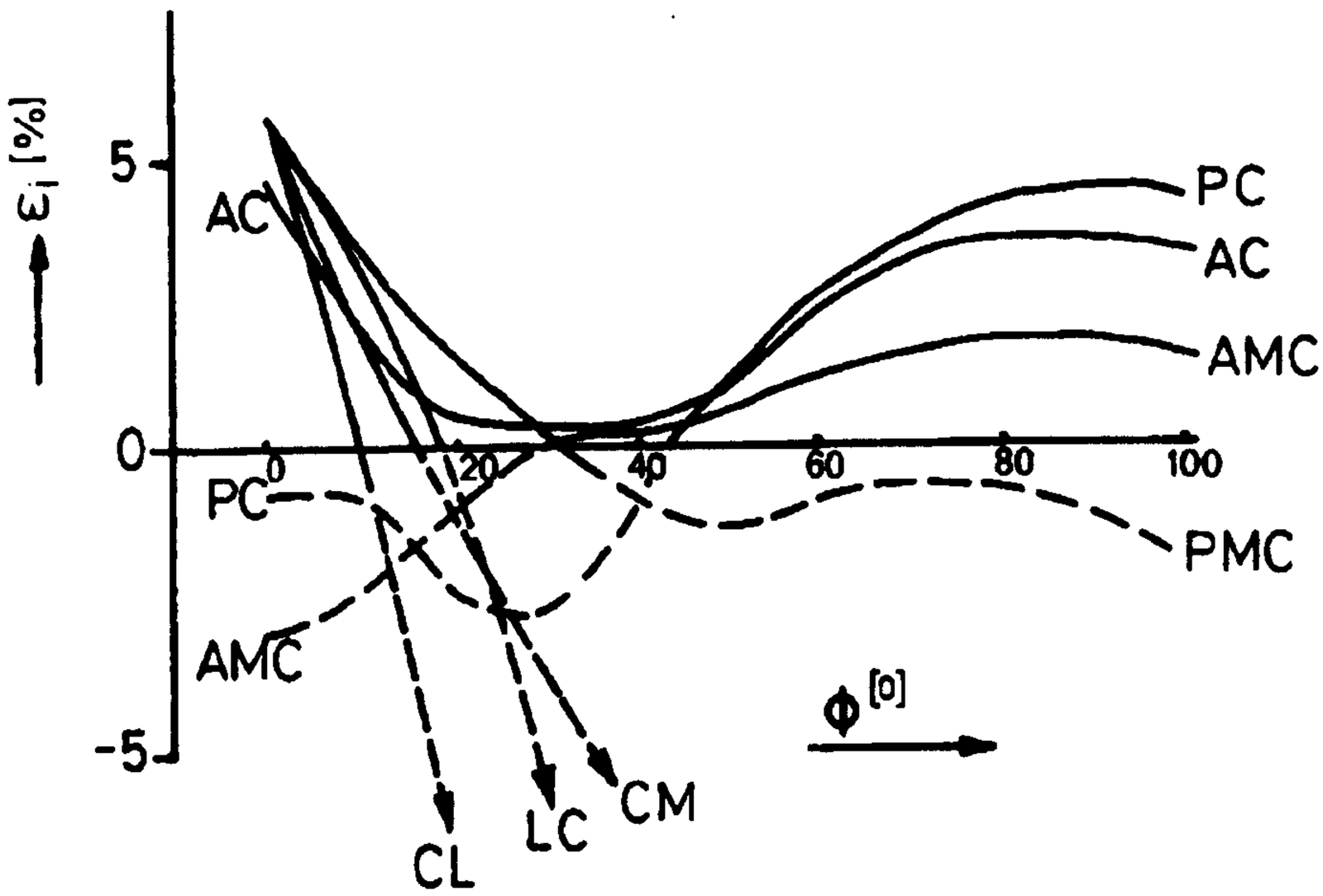


Figure 1.30 Graph of strain in response to passive flexion computed for the ligament line elements of the model of Wismans et al (1980). The line elements are shown in Figure 1.29

When Wismans et al (1980) simulated a PCL–deficient knee they found that there was an increase of 24mm in A-P laxity at 90° of flexion due to an A-P force of ±10N. The increase in laxity lessened at lower flexion angles to the point where there was no increase in A-P laxity at extension. There was also no increase in A-P laxity at extension when either the MCL or the LCL was cut. It can, therefore, be concluded that the posterior capsule elements resisted all of the posterior drawer force in extension. This result, with important consequences for the analysis of posterior stability, seems to have been overlooked by workers who subsequently developed this model without any capsular fibres (Blankevoort et al, 1991).

Andriacchi et al (1983) used finite element methods to model the knee. Twenty one spring elements, of various linear stiffnesses, were used to represent the ligamentous structures. The PCL, ACL and LCL were represented by three springs, the MCL by four springs and the joint capsule by eight. The compliance of the articular cartilage was modelled using ten hydrostatic surface elements. This model also included the menisci, which were capable of resisting shear, bending and compression. Data for the shape of the condyles and the stiffness of the ligaments was based on experimental results from the literature.

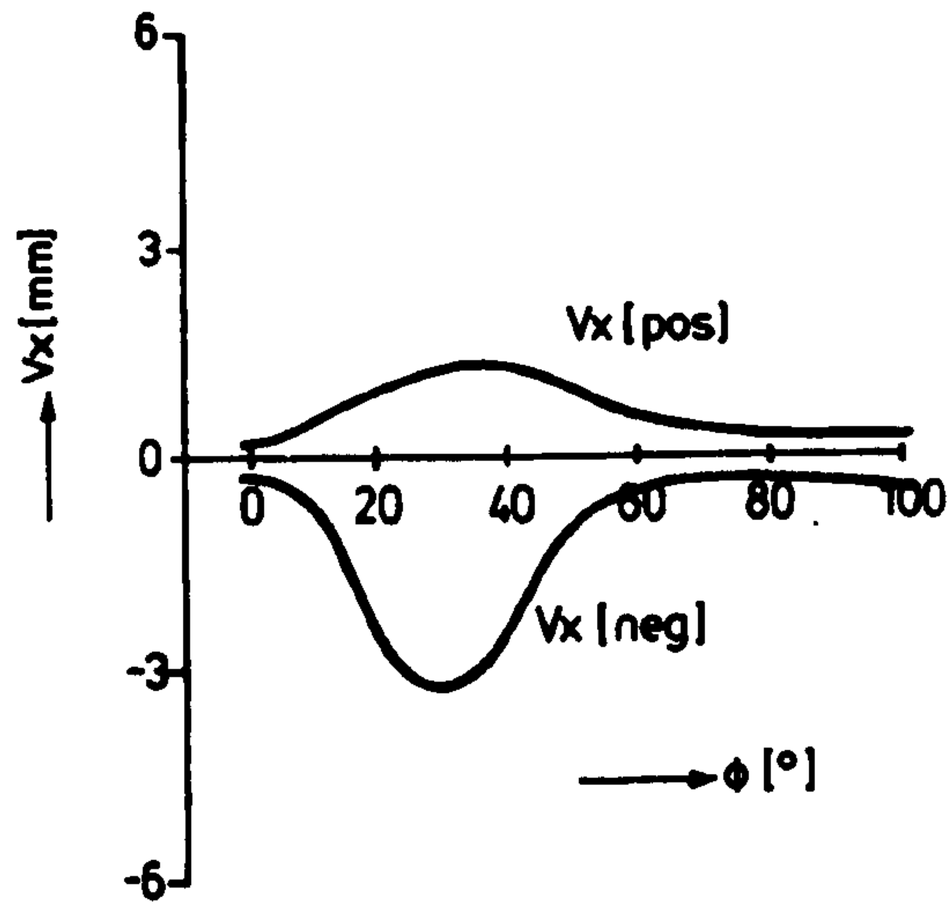


Figure 1.31 Graph of A-P displacement in response to a ±10N A-P drawer over the range of flexion, from the model of Wismans et al (1980).

The results presented by Andriacchi et al (1983) concentrated on varus (sic) rotation. Unfortunately the model was not used to investigate A-P stability beyond calculating A-P drawer stiffness in extension. The calculated stiffness, 63N/mm, was well matched with the average mid–range, or neutral stiffness of 65N/mm found experimentally by Markolf et al (1976). It should be noted that the average value reported by Markolf et al (1976) based on 35 knees had a standard deviation of 76N/mm or 117%.

It was suggested by Andriacchi et al (1983) that non–linear load/elongation characteristics could be included in future versions of their model, although they acknowledged that there would be problems with solution convergence. At the time of writing this does not appear to have been done.

Andriacchi et al (1983) made the important observation that constraining coupled motions and constraining the axis of rotation both had a significant effect on the model's results. They therefore advised caution in the design and interpretation of experiments.

The model of Andriacchi et al (1983) was later used to investigate the fractional change in the PCL force generated by posterior drawer when the insertions of the PCL were displaced (Bach et al, 1992). Unfortunately they did not report absolute values for PCL load, so the model's predictions cannot be compared with experimental results. It

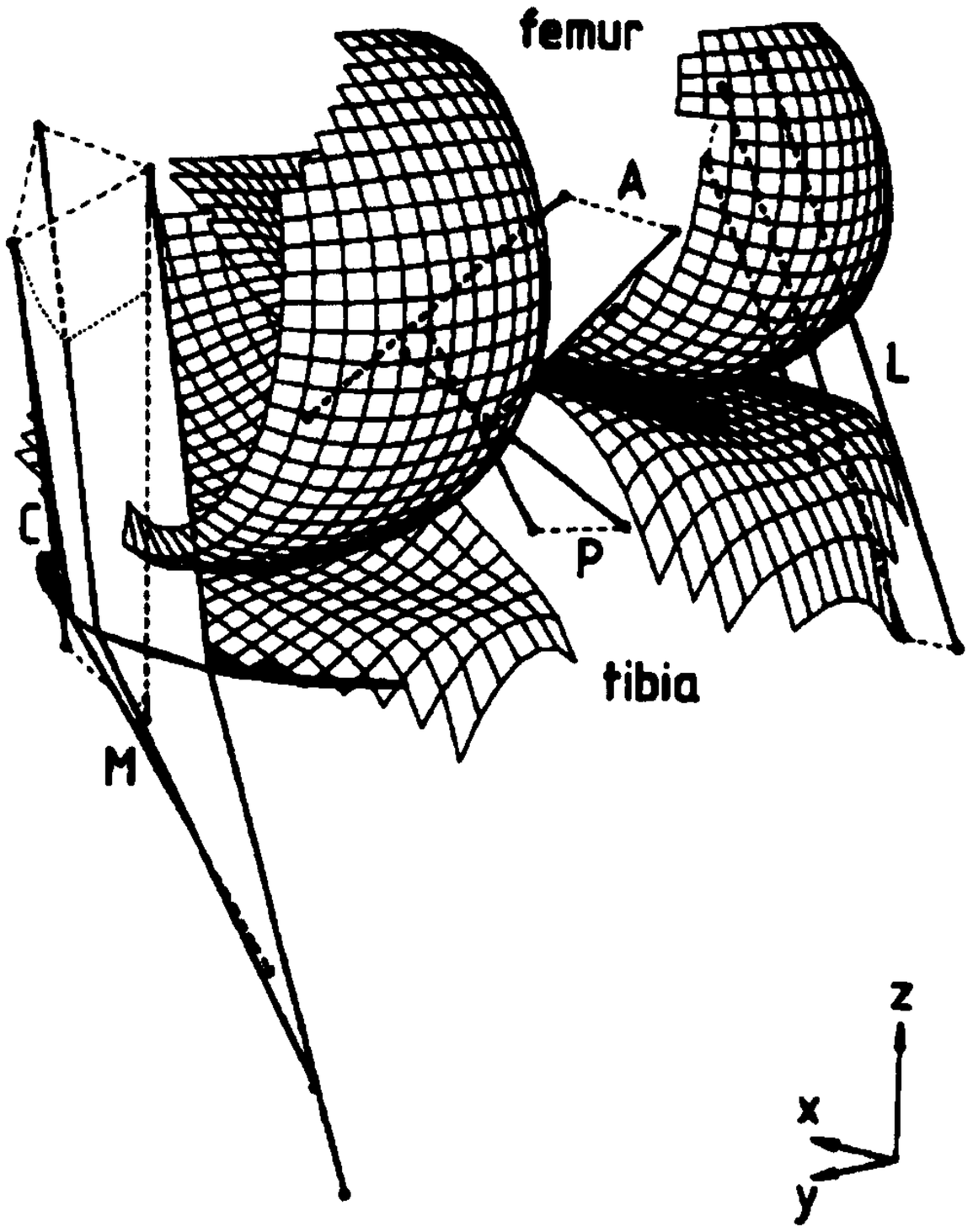


Figure 1.32 Line elements used in the model of Blankevoort et al (1991). **P**- PCL, **A**- ACL, **M**- MCL, **L**- LCL.

was found that the femoral insertion was more sensitive than the tibial. The femoral insertion was most sensitive to antero–posterior displacements and the tibial insertion was most sensitive to medial–lateral displacements. For the flexion range 0° to 90° they found the greatest fractional change in load at extension. The relative location of the three line elements that were used to represent the PCL were not reported.

Blankevoort and co-workers have developed a model using the approach of Wismans et al (1980). Rather than rely on experimental results from the literature Blankevoort formed an integrated strategy of experimental studies, model development and validation. This led to the formation of a specimen specific model which could be directly validated.

The first model produced by Blankevoort et al (1991) differed from that of Wismans et al (1980) in that more line elements were used to represent the cruciates and collaterals and the posterior capsule was not included. Other developments were deformable articular contact

surfaces and ligament wrapping about bone. The results of this model that relate to the PCL are discussed in section 1.2.7.1.

1.2.7.5 Summary

With the notable exception of Blankevoort and co-workers, current knee models have been poorly validated and have not been extensively used to realistically address clinical issues.

Clearly, any model that uses predetermined kinematics is unsuitable for *investigating* knee kinematics. It may, however, be useful for explaining joint motion.

The most exciting prospect for mathematical models is in combination with advances in minimally–invasive imaging. In the future there will be models that can accept CT/MRI images as input and predict the results of surgical intervention and subsequent bone remodelling. This could greatly enhance the efficiency and success of orthopaedic procedures.

And finally, it is worth noting that mathematical modelling has led to some bizarre conclusions. Langrana and Bronfeld (1985) measured the relative positions of ligament insertions in a cadaveric knee and then solved the force equilibrium equations for a posterior tibial displacement based on clinical data. They reported, with exquisite precision, that for an intact knee at 90° of flexion, the majority of the posterior force was resisted by the ACL (33.3%), followed by the PCL (33.2%), the LCL (31.0%) and the MCL(2.5%). On the basis of what was clearly an erroneous result, in the light of the many studies showing that the ACL slackens in posterior drawer (Butler et al, 1980), they concluded that the posterior drawer test at 90° of flexion was an insensitive indicator of PCL deficiency.

1.2.8 Appropriateness of investigative methods

Results of all investigations must be considered carefully in order to come to safe conclusions. It is not always possible to be confident that the results of changing an experimental model\mathematical model parameter will be reflected in vivo. Characteristics that are not active in 'normal' conditions, and so 'safely' ignored in the process of

simplification, may be highly active in the 'abnormal' conditions which are of the greatest interest. To model abnormal and not directly validated conditions a model must be complete.

'Complete' is used here to describe a whole system. A mathematical model would be defined as complete if it modelled every element of a system or complete sub-system. The in-vivo system is, by definition, complete. It is useful to consider passive joint structures as a complete subset of this complete system: the effects of any changes to this complete sub-system, measured by an in–vitro experiment or deduced from a complete mathematical model, would, largely, be reproduced in the in-vivo sub-system. This is not a perfect division as there is some cross–over between active and passive structures as some ligamentous structures, like the MCL, are influenced by muscular activity.

A *complete* mathematical model of the passive sub-system of the knee would require the modelling of the articular geometry, the properties of the articular cartilage, the properties of the menisci and the properties of the interconnecting ligamentous and capsular structures that join the femur, tibia, fibula, (fabella) and menisci. To be truly *complete* would require modelling at the molecular level. Clearly, there would be diminishing returns in terms of macroscopic accuracy as microscopic completeness is approached. There are different, lesser, levels of completeness that can be aimed for: modelling individual fibres and cells or modelling groups of fibres/areas of cartilage/bone with homogenised properties.

The simplifications inherent in current modelling have not led to the creation of a *complete* sub-system. The existence of numerous in-vivo parameters of the *complete* sub-system that are outside the various pseudo-sub-systems chosen by different workers has meant that the *predictions* of these models have been uncertain.

For experimental work near completeness is much easier to achieve: the passive sub-set can be complete if all of the fibres that make up the passive connections between the elements of the joint are intact. Of course it is necessary to ensure that the transition from in-vivo to in-vitro conditions produces the minimum change in the system. The

difficulties inherent in experimental work have more to with control of forces/motions and measurement than structural inaccuracies.

Having a complete model of a system, mathematical or physical, it is then necessary to devise clinically appropriate tests. In any trauma repair, reconstructing normal anatomy would, of course, give a perfect result, normal function. However, since this is not a viable option at the present time a far less sophisticated intervention must be designed. Since an unsophisticated intervention cannot hope to fulfil all normal functions it must be targeted at a smaller range of function, hence the requirement for clinically appropriate tests. It must be determined what are the most important functions of a system element so that an imperfect solution can be targeted to improve some crucial aspect of a malfunctioning element. If a crucial function can be identified and an intervention, albeit imperfect, can be designed to fulfil this function without excessive side effects then there may be a net benefit to a patient.

As an example consider anterior-posterior stability. Clinical experience has shown that A-P instability, as measured by the A-P drawer test, correlates to some degree with disability and degenerative changes, so much experimentation has been done to establish the causes of A-P stability, instability and its correction. Clinically, the A-P drawer test is done with the foot fixed, so the tibia rotates about the ankle. For a small angular displacement this rotation gives an approximate A-P motion of the tibia at the joint line. The femur is free to rotate at the hip so compression-distraction of the knee joint is unconstrained (although it results in a small change in flexion angle at the knee). Medial-lateral motion of the tibia relative to the femur is unconstrained. Internal-external rotation of the tibia is allowed subject to the rotational stiffness of the ankle. So the clinical test approximates to unconstrained A-P drawer at a fixed flexion angle. Some of the studies referred–to in section 1.2.4 used pure A-P translation, all other rotations and translations were constrained. Although it does have the attraction of simplicity and precision, pure A-P displacement is not something that can generally occur in-vivo and is not guaranteed to be an accurate or useful test for benefit to a patient. For example, if compression-distraction were to be constrained then very large forces could be generated by the interaction of the tibia and the femur. The magnitude

of these forces would depend on the amount of compression/distraction introduced when the position of the joint was fixed and on local variations in the slope of the condyles with respect to the direction of translation. These induced forces could mask the effects of a procedure. The constraints on the other degrees of freedom, which would restrict coupled motion, would also change the force balance in a joint, which could lead to results that do not aid the understanding of in–vivo conditions. Consider the investigation of reconstructive procedures. If a completely dislocated joint were to be repaired with an MCL graft then an experiment using pure A-P displacement would find that A-P stability could be restored with an appropriate graft tension. An experiment which allowed unconstrained motion, at a certain flexion angle, would obviously give a negative result: the tibia would externally rotate and posteriorly translate. Unconstrained A-P motion, while not necessarily being a common event, will not introduce 'impossible' forces or lead to improbable conclusions.

1.3 INJURY MECHANISMS

One of the earliest discussions of injury mechanisms is due to Pringle (1907) who was the first investigator to draw attention to the two possible failure modes of the cruciate ligaments: ligaments can fail by rupture of the ligament substance or by avulsion fracture of the bony insertion. Pringle (1907) quoted the work of Pagenstecher who was able to produce isolated PCL failure in cadavers by means of a blow to the anterior surface of the tibia in the flexed knee, which normally resulted in failure by tibial avulsion. This mechanism for isolated PCL rupture has since been widely reported (Palmer, 1938; Abbott et al, 1944; McMaster, 1975; Meyers, 1975; Torisu, 1977; Johner et al, 1979; Torisu, 1979; Trickey, 1980; Dandy and Pusey, 1982; Fowler and Messieh, 1987; Kannus et al, 1991; Pournaras and Symeonides, 1991; Whipple and Ellis, 1991). Isolated PCL rupture has also been observed after hyperflexion (Fowler and Messieh, 1987) and after hyperflexion with internal rotation (Stanish et al, 1986). Combined ligament injuries, including rupture of the PCL, have been observed after hyperextension (Drucker and Wynne, 1975; Trickey, 1980; Dandy and Pusey, 1982; Itokazu et al, 1990; Kannus et al, 1991). Palmer (1938) carried out in vitro hyperextension tests and reported that the posterior fibres of the PCL started to fray at about 30° of hyperextension but that the primary

injury was ACL rupture. Kennedy and Grainger (1967) also carried out such tests and reported that the PCL failed by tibial avulsion, after the posterior capsule had torn, beyond 30° of hyperextension. Subsequent investigators have reported that PCL failure in hyperextension is secondary to ACL rupture (Drucker and Wynne, 1975; Dandy and Pusey, 1982; Kannus et al, 1991). This contention is supported by selective cutting and length change studies (section 1.2.4and 1.2.6). Another combined ligament injury is PCL deficiency with rupture of the posteromedial or posterolateral structures. This injury results from an off centre blow to the tibia as in the cases reported by Baker et al (1984), where a blow to the anteromedial aspect of the tibia had caused both the PCL and posterolateral structures to rupture.

1.4 DIAGNOSIS OF PCL DEFICIENCY

Given that the primary function of the PCL is to resist posterior displacement of the tibia on the femur (section 1.2) the most obvious diagnostic sign of PCL deficiency would seem to be abnormal posterior displacement. This was the method of diagnosis used by Battle (1900) in the first published case of cruciate ligament repair. Battle (1900) noted that, under anaesthesia, there was free anterior–posterior movement in the flexed knee and concluded that the cruciate ligaments were ruptured. Hazen (1910) pointed out that there was considerable variation of anterior–posterior laxity in 'normal' knees and that therefore the uninjured joint should be used as a reference in deciding what constitutes abnormal motion. Abnormal posterior tibial motion has been demonstrated by active posterior drawer, where a posterior force is applied to the tibia by a clinician (Hey Groves, 1920; Hughston, 1969; Moore and Larson, 1980), and also by what has been variously called 'spontaneous drawer backwards', the 'Gravity Sign' and 'Posterior Sag' (Palmer, 1938; Trickey, 1980; Insall and Hood, 1982; Satku et al, 1984; Kannus et al, 1991), where the weight of the tibia causes posterior subluxation in the flexed knee. Various devices have been produced which claim to give consistent quantitative data from clinical drawer tests. However, independent studies have shown that there is significant inter–device and inter–examiner variation (Daniel, 1991; Anderson et al, 1992).

Several surgeons have observed that the posterior drawer sign can be obscured by muscle spasm and by haemarthrosis (Palmer, 1938; Abbott

et al, 1944; Trickey, 1968). Palmer (1938) noted that in some cases a posterior drawer sign could be evoked immediately post–trauma but that on re–examination hours later posterior drawer was negative because of a protective 'reflex increase in tonicity' of the muscles that actively stabilise of the knee. Therefore Palmer (1938), and others (Abbott et al,1944; Trickey, 1968; Trickey, 1980) have recommended examination under anaesthesia, when the active stabilisers will be relaxed. Since swelling of the joint can blur shifts in bone position it has been common practice to look for the posterior drawer sign by comparing sagittal plane X–ray images before and after the application of posterior force (Hey Groves, 1920; Jacobsen, 1977; Satku et al, 1984). X–ray images have also been used to directly diagnose avulsion fractures (Jones and Smith, 1914; Trickey, 1980) and, by injection of a contrast medium, ligament rupture (Palmer, 1938; Pavlov and Freiberger, 1978). More sophisticated, three dimensional, X–ray images have been produced by computerised tomography (Pavlov et al, 1979; Passariello et al, 1986)

In recent years arthroscopic examination, which can allow direct visualisation of a PCL injury, has become widespread (Gillquist et al, 1979; DeHaven, 1980; Lysholm and Gillquist, 1981; Passler et al, 1989; Schreiber, 1991). Although high accuracy has been reported for this technique it involves anaesthesia and the opening of the knee joint, with the associated risks. Magnetic Resonance Imaging (MRI), which is non–invasive, has been used successfully in the diagnosis of PCL injury (Turner et al, 1985; Gallimore, Jr. and Harms, 1986; Reicher et al, 1987; Polly, Jr et al, 1988; Bassett et al, 1990; Grover et al, 1990).

Trickey (1968) suggested that PCL failure had often been missed at the time of diagnostic tests, especially in cases where there was no tibial avulsion, or misdiagnosed as ACL rupture. This view has since been supported by others (Torisu, 1979; Dandy and Pusey, 1982). Some surgeons have stressed the importance of deducing the injury mechanism as an aid to diagnosis, especially recognition of an abrasion or contusion haematoma on the anterior aspect of the proximal tibia as an indication of PCL rupture (Palmer, 1938; Trickey, 1980; Kannus et al, 1991). It has also been observed that in cases of chronic PCL laxity the presenting symptom can be patellar pain (Barton et al, 1984).

1.5 TREATMENT OPTIONS

There is general agreement in the literature that cases of avulsion fracture of the PCL should be treated by open reduction (Lee, 1937; Butel et al, 1978; Trickey, 1980; Bianchi, 1983; Barton et al, 1984; Loos et al, 1981; Satku et al, 1984; Chiu et al, 1994b). Fixation of the avulsed fracture has been achieved by wire suture (Brennan, 1960), staple (Torisu, 1977; Torisu, 1979; Yerys, 1991), crossed K-wires (Galle, 1979) and bone screw (Abbott et al, 1944; Trickey, 1968; Burks and Schaffer, 1990). There is no such agreement on the best management of a PCL rupture. The various approaches to this problem are discussed below.

1.5.1 Conservative Management

Many surgeons are of the opinion that a ruptured PCL does not require surgical treatment. There have been many reports of a satisfactory outcome from conservative management of PCL rupture (Dandy and Pusey, 1982; Satku et al, 1984; Cross and Powell, 1984; Parolie and Bergfeld, 1986; Fowler and Messieh, 1987). Some athletes even maintain pre–injury levels of performance, for example Cain and Schwab (1981) reported the case of an American Football player who was competing successfully with chronic PCL deficiency. They observed that early quadriceps contraction in the gait cycle gave dynamic stability.

From an evolutionary perspective it seems highly unlikely that such a strong and consistent structure in the body should be redundant and yet it can appear to be so. There are two explanations which may resolve this paradox. Selective cutting studies have shown that section of the PCL causes a considerable increase in laxity in flexion but that this increase is minimal in extension (section 1.2.4). Therefore, a PCL deficient patient may well not experience instability during normal gait because the knee is then only loaded near extension. If such a patient has a low activity level they may well never experience functional impairment. At the other end of the scale are the elite sports players. Such individuals clearly have an unusual aptitude for physical balance, timing and training and can learn to cope with an unstable joint.

It has been observed that there is a period of functional adaptation to PCL deficiency, lasting 6-18 months, during which intensive physiotherapy can be used to gain dynamic stability (Dejour and Walch, 1987; Kannus and Jarvinen, 1990). The affected joint may be immobilised for a short time because of pain but quadriceps training is started as soon as possible: quadriceps strength is the key to successful conservative management (Kannus et al, 1991). Not only must the quadriceps be strengthened but the patient must learn, with a variety of balancing exercises, to use the muscle group to stabilise the knee. The degree to which this is successful is to a large extent dependent on the patients commitment to training (Kannus and Jarvinen, 1990). It could be argued that all patients can be satisfied with conservative treatment as a patient's desired activity level will correlate strongly with their commitment to training.

Some longer term studies have shown that, even if dynamic stability is achieved, there are degenerative changes in the knee: quadriceps atrophy, chondromalacia of the patella and of the medial femoral condyle (Dejour et al, 1988; Torg et al, 1989; Wirth, 1989; Geissler and Whipple, 1993). These authors have suggested that degenerative changes may have been missed by studies that did not follow up for more than five years. However, proponents of conservative techniques have pointed out that there have been no reports of large numbers of ligament deficient patients requiring total knee arthroplasty (TKA). This lack of correlation with TKA may be explained by three possibilities:

- PCL injury does not lead to serious degenerative changes?
- Ligamentous integrity has not been rigorously assessed at the time of TKA so the correlation has been missed?
- Degenerative changes cause patients to reduce their activity level and allow the joint to recover?

Assuming that the reported degenerative changes with chronic PCL deficiency are due to simple mechanical overload of the articular cartilage then the third possibility seems quite plausible. A patient who has experienced articular cartilage damage is likely to be inspired by pain to stop whatever activities were causing the mechanical overload and thereby to halt the degenerative process. Whether or not this is acceptable depends on the individual patient's expectations and the healing capacity of the damaged cartilage.

Because of the suspected poor long-term prognosis for chronic PCL laxity many surgeons have chosen to reconstruct the ligament, especially in younger patients with high expectations of future activity (Kennedy and Grainger, 1967; Trickey, 1968; Hughston et al, 1980; Moore and Larson, 1980; Fleming, Jr et al, 1981; Loos et al, 1981; Hughston and Degenhardt, 1982; Bianchi, 1983; Clancy, Jr et al, 1983; Wirth and Jager, 1984; Wirth, 1989; Chiu et al, 1994a).

Finally, it should also be noted that a frequently given reason to manage PCL ruptures conservatively has been that the results of operative procedures have been variable (Fowler and Messieh, 1987; Kannus et al, 1991). While this negative decision making may be in the best interest of patients in the short term it must be realised that the existence of bad surgical procedures should not deter one from developing good surgical procedures.

1.5.2 Surgical Intervention

Surgical intervention after PCL rupture can be divided into two broad approaches, active and passive reconstructions. Active reconstructions have aimed to restore dynamic posterior stability by altering the structure of the knee: muscular insertions have been moved or re-routed to give improved, active, control of posterior tibial motion. These reconstructions have used either the popliteus (Barfod, 1971; McCormick et al, 1976) or the medial head of the gastrocnemius (Moore and Larson, 1980; Hughston and Degenhardt, 1982; Insall and Hood, 1982; Kennedy and Galpin, 1982; Tesmoingt and Dossa, 1982; Tibone et al, 1988; Roth et al, 1988). The results of these treatments are highly dependent on the patient's ability to train these re-routed muscles, as with the quadriceps training during conservative treatment.

The more common aim of surgical intervention has been to repair or reconstruct the natural PCL to give 'passive' joint stability. As stated earlier, the results of PCL reconstructions have been very inconsistent (Fowler and Messieh, 1987; Kannus et al, 1991). The key variables of passive reconstructive procedures, graft material, graft placement and graft fixation/tension are discussed separately below.

1.5.2.1 Graft Materials

Early attempts at PCL repair relied on silk sutures to repair the ruptured ligament (Battle, 1900) but no long term success was reported. Since that time various surgeons have advocated reconstructing the PCL using tissue harvested from the injured or contralateral limb. These autogenous reconstructions have used the fascia lata (Cubbins et al, 1939), the meniscus (Lindström, 1959; Bruckner and Gutschmidt, 1980; Hughston et al, 1980), the semitendinosus or gracilis tendons (Hey Groves, 1920; Gallie and LeMesurier, 1927; Pickett and Altizer, 1971; Ficat et al, 1975; Southmayd and Rubin, 1980; Trickey, 1980; Lipscomb et al, 1981; Fleming, Jr et al, 1981) and one third bone–patellar tendon–bone(B–pt–B) (Campbell, 1939; Clancy, Jr et al, 1983; Holzach et al, 1985; Eriksson et al, 1986; Verdonk et al, 1986).

All the above grafts have appealed to clinicians on the grounds that they comprise autogenous tissue which can be 'spared'. It would seem reasonable to assume that if anything is to be implanted into the body then autogenous tissue will generate the most benign immunological reaction. The body's reaction to a PCL graft is of crucial importance: for long term success the implanted tissue must revascularise and be capable of remodelling to maintain a strong, ligament–like, collagenous structure. However, tissue reaction is not the only factor governing success: if the graft is not mechanically competent at implantation then it will break, or stretch to the point of non–funtion, before remodelling is an issue.

Noyes et al (1984) published mechanical and material properties for common autografts: They showed that for young donors (26yrs±6yrs) the failure loads of the gracilis, semitendinosus and central third bone–patellar tendon–bone (B–pt–B) were 838N±30N, 1216N±50N and 2900N±260N respectively. The failure load of the PCL in young donors has been shown to be 1627N±491N (Prietto et al, 1988). Only the patellar tendon autograft matches this at harvest.

It was stated earlier that the graft must be capable of revascularisation and remodelling to be successful, but the remodelling process creates its own problems. The 2.9kN failure load for a B–pt–B graft may appear to offer a generous safety factor but the strength of transplanted tissue decreases rapidly postoperatively as the graft is invaded by cells which

digest and remodel the tissue. Then, after several months the strength of the graft increases. In a study of B–pt–B ACL reconstructions in primates it was shown that at 7 weeks post implantation the failure load of the graft had reduced to 16% of the ACL control strength (Butler et al, 1989). The same study showed that after one year the graft strength had increased to 39% of the ACL control strength. If this relationship were to hold true for humans then the failure load of a B–pt–B autograft at one year would be 675N.

The use of autogenous material has the advantage that there is no foreign body reaction. However, the removal of healthy, functional parts of the limb is biomechanically questionable. Also, harvesting of graft tissue can lead to complications. For B–pt–B autografts there are reports of patellar fracture after bone block removal, patellar tendon avulsion, patellofemoral pain and patellar tendon contracture (Manaster et al, 1988; Christen and Jakob, 1992; Strover, 1992)

Because of the problems of autogenous reconstructions allografts of B–pt–B and Achilles tendon have been used (Arnauw et al, 1991). However, allografts carry the risks of host reaction and disease transmission. To protect against disease transmission, especially HIV, allografts are gamma irradiated. Unfortunately, the irradiating process has been shown to significantly weaken B–pt–B grafts (Feder et al, 1994). It has also been shown, in a goat model, that fresh frozen B–pt–B allografts did not perform as well as B–pt–B autografts (Jackson et al, 1991). Bovine xenografts were briefly experimented with but wear debris caused a chronic inflammatory response and they did not promote tissue ingrowth or remodelling and so were abandoned (Claes et al, 1987; Allen et al, 1987; Berry et al, 1988).

1.5.2.2 Graft Placement

In cruciate ligament surgery there has been much emphasis on the importance of isometric graft placement. This fashion for isometry has been persistent in spite of the fact that the majority of the PCL is distinctly anisometric (section 1.2.6). The purpose of using an isometric reconstruction has been to ensure that the degree of tautness of the graft is constant over the range of flexion so that it is not overloaded or excessively slack as the knee moves. Previous studies have shown that it is possible to achieve a near–constant tautness over the range of

flexion using a femoral drill hole in a number of positions (Sidles et al, 1988; Grood et al, 1989). However, different 'isometric' reconstructions will have resulted in different graft orientations over the range of flexion. If the angle of a graft in the sagittal plane is different then its effect on posterior drawer will be different. Thus, a series of reconstructions which were all 'isometric' and tensioned to the same degree may have led to different results in terms of A-P stability.

There has been a growing movement to perform more anatomical reconstructions (Covey et al, 1992). Some autogenous reconstructions have even attempted to reproduce the frequently described 'double bundled' structure of the PCL (Mott, 1983; Wirth and Jager, 1984).

1.5.2.3 Graft Fixation

All soft tissue grafts are difficult to secure. Comparing the strength of grafts and of anchors shows that tendon based reconstructions will always fail at the anchorages (Kurosaka et al, 1987; Butler, 1987). This means that a return to normal joint function is not possible until the graft has become fixed by fibrous/bone ingrowth. It is possible that some poor results for PCL reconstructions were due to loosening of the grafts by fixation failure. This problem, combined with a desire for early mobilization has led the development of more carefully planned rehabilitation programs.

1.6 SYNTHETIC LIGAMENTS

Synthetic implants appear to offer many benefits over autogenous grafts.

- There is no donor site morbidity.
- The implant can be manufactured with a specific strength.
- Graft strength will not be reduced by tissue remodelling.
- Fixation can be achieved by mechanical interlocking to give immediate, secure fixation which allows rapid rehabilitation.
- The length of the implant can be adjusted for surgical convenience.
- Operating time can be reduced.

These potential advantages led to a considerable interest in synthetic implants in the late 1970's and the following decade. Although many

ligaments have been developed none are commonly used at present. The brief surgical fashion in favour of synthetic implants is currently subject to a backlash generated by a history of poor long term results (James et al, 1979; Scharling, 1981; Claes et al, 1987; Bray et al, 1988; Turner and Thomas, 1990; Bartolozzi et al, 1990; Arnauw et al, 1991; Jenny et al, 1991; Lopez Vazquez et al, 1991; Andersen et al, 1991; Klein and Jensen, 1992; Paulos et al, 1992; Richmond et al, 1992; Barrett et al, 1993; Wilk and Richmond, 1993). Although there have been many reports of poor outcome from synthetic implants it is also true that autogenous reconstructions have produced very variable results without being dismissed altogether. With a small degree of cynicism one can observe that in the case of a poor autogenous result the technique of the surgeon is open to criticism whereas a poor synthetic result is more easily attributed to other factors. Curiously, one of the most successful, and ongoing, series of PCL reconstructions has been performed using a synthetic implant (Laboureau and Dericks, 1992)

Undoubtedly, the present antipathy towards synthetic graft material is based on the poor results of many commercially available implants. However, before dismissing synthetic materials out of hand the *reasons* for the failure of those implants should be addressed. So, given the apparent attractions of synthetic materials, what went wrong?

In the following discussion of the development of synthetic implants most of the studies cited are concerned with ACL reconstruction, but the lessons to be learned are equally applicable to the PCL.

1.6.1 Mechanical Design of Implants

Unfortunately early synthetic implant development had no input from engineers and the material and mechanical properties of natural ligaments and of the prosthesis were not considered (Amis, 1989b). This has led to very serious mismatches between natural ligaments and implants. In the study of Beuchamp et al (1979) an implant with a yield point of 160N was used. A solid ACL implant made of ultrahigh–molecular–weight polyethylene that was commercially produced in the 1970's was found to have a yield strength of 420N (Grood and Noyes, 1976). The fact that the PCL and ACL had been shown to have far higher strengths (Kennedy et al, 1976; Noyes and Grood, 1976)

Table 1.1 Rupture loads and anchorage methods for some 1980's synthetic ligaments.

IMPLANT	RUPTURE LOAD (kN)	ANCHORAGES
Apex™	5.2[1]	Screws and grommets
L.A.R.S. Laboureau	5.0[1]	Interference screws
Gore–Tex™	4.8[2]	Screws
Surgicraft® ABC	3.0[1]	Toggle and bollard
Leeds–Keio	2.0[1]	Bone plugs
Stryker Dacron	1.7[1]	Staples
Corin APS™	Not Published	Screws and staples

1– (Manufacturer's data)
2– (Bolton and Bruchman, 1985)

demonstrates that failure of these implants should have been predicted at the design stage. Because the implant was solid there was no possibility of soft tissue ingrowth, so the implant was expected to remain load bearing for many decades. But a solid, rather than fibrous, structure was more liable to fatigue failure. It is difficult to understand the thought processes that led to these devices being implanted.

As a result of increasing use of engineering analysis (Grood and Noyes, 1976; Chen and Black, 1980) implant mechanical properties were generally improved during the 1980's (Table 1.1). The Stryker Dacron implant was designed to match the ACL's widely reported rupture strength of 1.7kN but many of the poor results referred to above involved this implant. Later tensile testing studies have shown that the strength of the natural ACL had been underestimated: according to Hollis et al (1988) the ACL had a failure load of 3kN in young adults. This finding cast doubt on the mechanical sufficiency of the Stryker Dacron, Surgicraft® ABC and Leeds–Keio implants for ACL reconstruction. Studies that have measured the failure load of the ACL and the PCL have consistently shown the PCL to be stronger (Trent et al, 1976; Kennedy, Hawkins, Willis et al, 1976; Marinozzi et al, 1983). In the light of this fact it would seem wise to assume that an implant intended for PCL reconstruction should have a failure load in excess of 3kN.

So, one answer to the question 'what went wrong?' is that most implants have simply been too weak: the very weak implants will have

failed by direct yielding and the 'matched' implants may have failed by fatigue. An understrength implant is not a natural consequence of using a synthetic material, just a design failure. It appears that much of the antipathy towards synthetic materials is misplaced: a more useful lesson might be that clinicians should consult biomechanical engineers when trying their hand at engineering design.

1.6.2 Biocompatibility and Tissue Ingrowth

Biocompatibility is of great importance in implant design: good mechanical function is irrelevant if the joint cannot be moved due to pain from synovial irritation and effusion. Tissue ingrowth is desirable for three reasons, firstly to cover the implanted fibres so that wear debris are not released into the joint, secondly to anchor the implant to bone and thirdly to reinforce and possibly take over the mechanical function of the implant as it inevitably degrades.

Carbon fibre implants were used in the 1970's and initially seemed to encourage the growth of a neo-ligament (Jenkins et al, 1977; Jenkins, 1978; Leyshon et al, 1984). Further studies have shown that the new

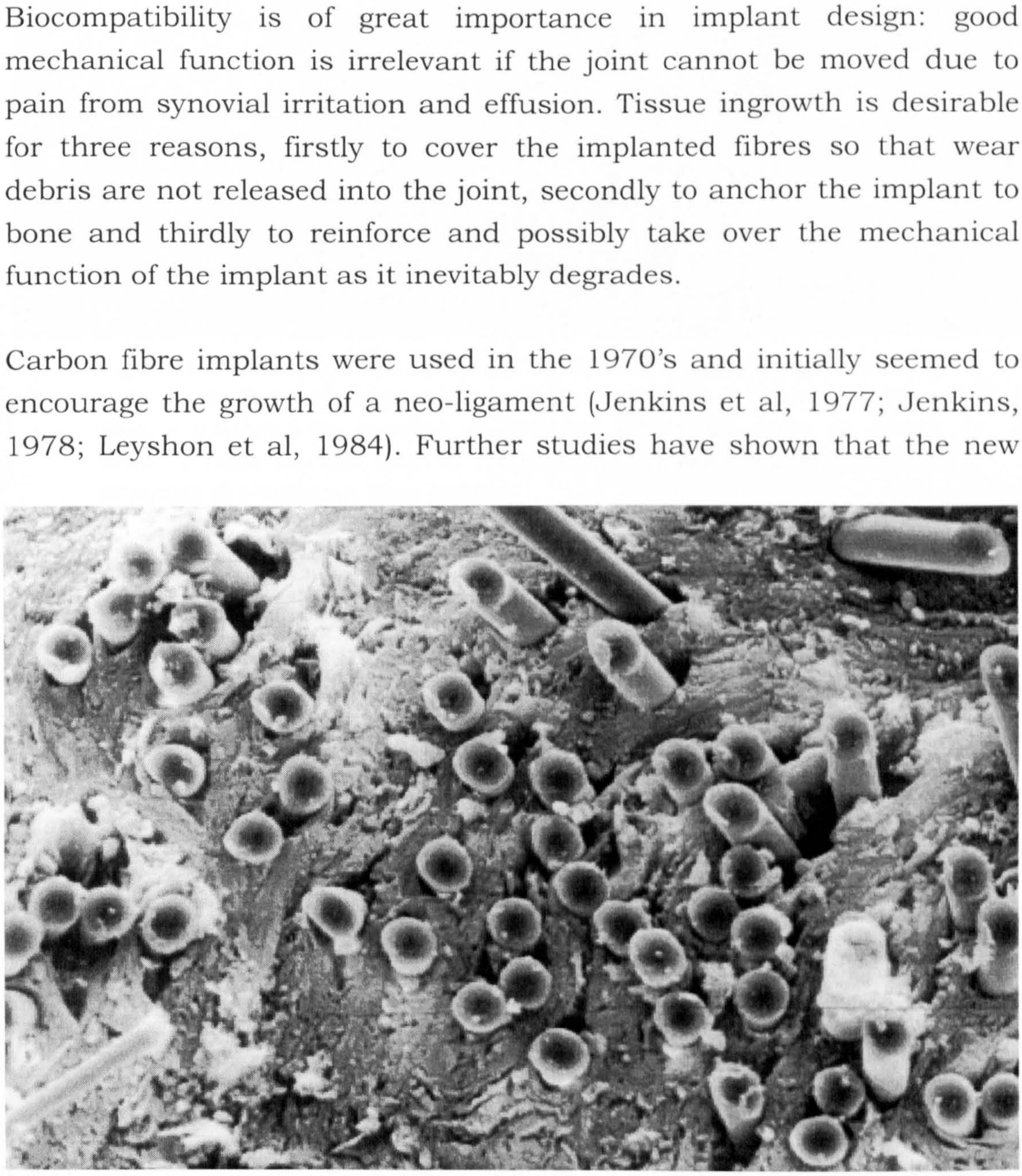


Figure 1.33 Electron micrograph of a small biopsy taken 6 months after implantation of an Apex™ ACL replacement. The parallel fibred implant has been well invaded by soft tissue.

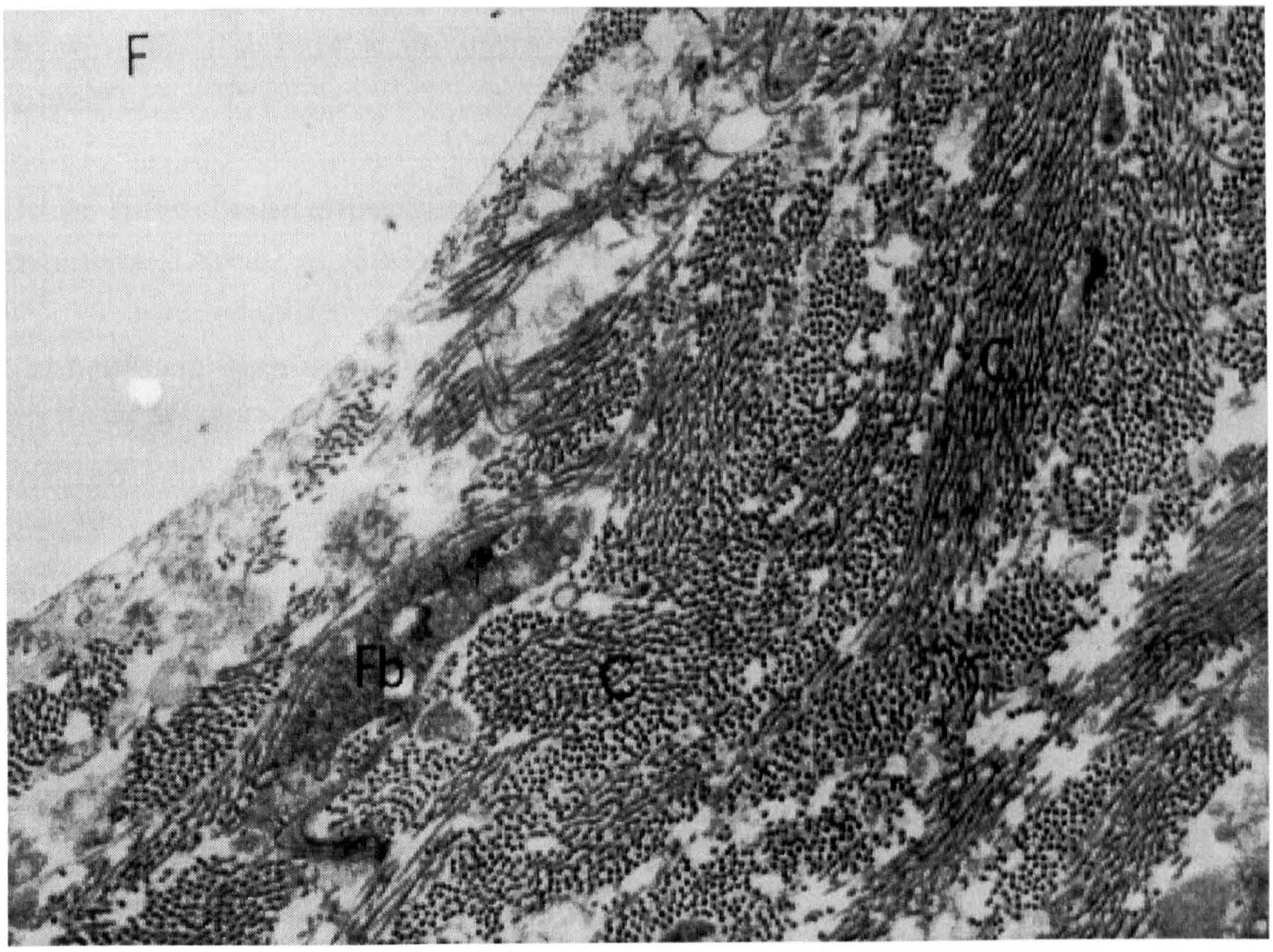


Figure 1.34 Electron micrograph (x20,500) showing an Apex implant fibre (F) at six months post implantation in a human knee. The fibre was surrounded by bundles of collagen fibres (C) some of which were in close contact with it. Fibroblasts (Fb) were found dispersed amongst the collagen fibre bundles.

tissue growth was restricted to the outside of the implant and that there was no penetration of cells into the body of the ligament (Amis et al, 1988). It has also been shown that collagen fibres that are laid down in association with higher modulus materials like carbon fibre lack the crimped structure of normal soft tissue collagen fibres whereas lower modulus fibres of polyester and nylon were shown to induce normal, crimped collagen (Goodship, 1985).

There were also biocompatibility problems with carbon fibre implants. Carbon fibres are relatively brittle and small fragments were found to break off and migrate through the joint causing an undesirable tissue reaction. This effect has been reduced by wrapping the implant in autogenous material (Strover and Firer, 1985).

Polyester has proved to be more amenable to tissue ingrowth (Amis et al, 1988; Amis et al, 1992; Harries and Amis, 1995). It has also been shown to be biocompatible with none of the adverse reactions seen with

carbon fibre debris(Amis et al, 1988), so most current implants use polyester.

There have been experiments with synthetic collagen fibres for use as implants (Law et al, 1989; Dunn et al, 1992) but no clinical trials as yet.

Although it has always been a goal of synthetic implant design to encourage tissue ingrowth, nearly all implants have had a plaited or knotted structure. Any braided or knotted structure generates considerable inter-fibre pressures when loaded, which is not conducive to cell invasion. A complex fibre structure can be used to reduce the stiffness of an implant (Claes et al, 1983) but at the expense of tissue ingrowth.

With regard to tissue ingrowth 'what went wrong' was that the use of unsuitable materials and unsuitable implant constructions resulted in many cases where tissue ingrowth did not occur. This has resulted in a general acceptance amongst surgeons that tissue ingrowth, of the correct type, is not possible with a synthetic implant. However, it has been well established that given a suitable material and construction ingrowth *does* occur. The parallel fibred Apex™ implant has been shown to be well suited to soft tissue invasion (Amis et al, 1992; Harries and Amis, 1995). The fine polyester fibres were evenly separated and invaded by fibroblasts which laid down collagen fibres parallel to, and in contact with, the implant fibres (Figure 1.33 and Figure 1.34). This parallel fibre structure was also recently introduced in the intra-articular portion of the L.A.R.S. Laboureau PCL implant.

The results of modern, well designed, synthetic implants suggest that it would be reasonable to conduct a prospective randomised trial comparing synthetic with autogenous reconstructions.

1.6.3 Implant Fixation

Early mobilisation after reconstructive surgery is desirable to prevent muscle atrophy and joint stiffening. To enable immediate mobilisation an implant fixation must be strong enough to resist the loads encountered in everyday activity. The study of Morrison (1969) estimated that for various walking activities the maximum loads on the

ACL and PCL were 0.44kN (on descending a ramp) and 1.2kN (on ascending stairs) respectively.

One of the major advantages of a synthetic ligament implant is the possibility of immediate, secure fixation. Natural soft tissue is very difficult to anchor (Butler, 1987) whereas a synthetic structure can be designed with strength of fixation in mind. Several different anchorage strategies have been devised. Many implants have been designed with end loops (Gore–Tex™ , Surgicraft® ABC, Apex™). If an implant has a loop then fixation can be achieved by mechanical interlocking rather than friction. Other implants have been anchored by staples (Stryker Dacron), bone plugs in a hollow implant (Leeds–Keio) screw and staple (Corin APS™) and interference screws (Laboreau L.A.R.S)

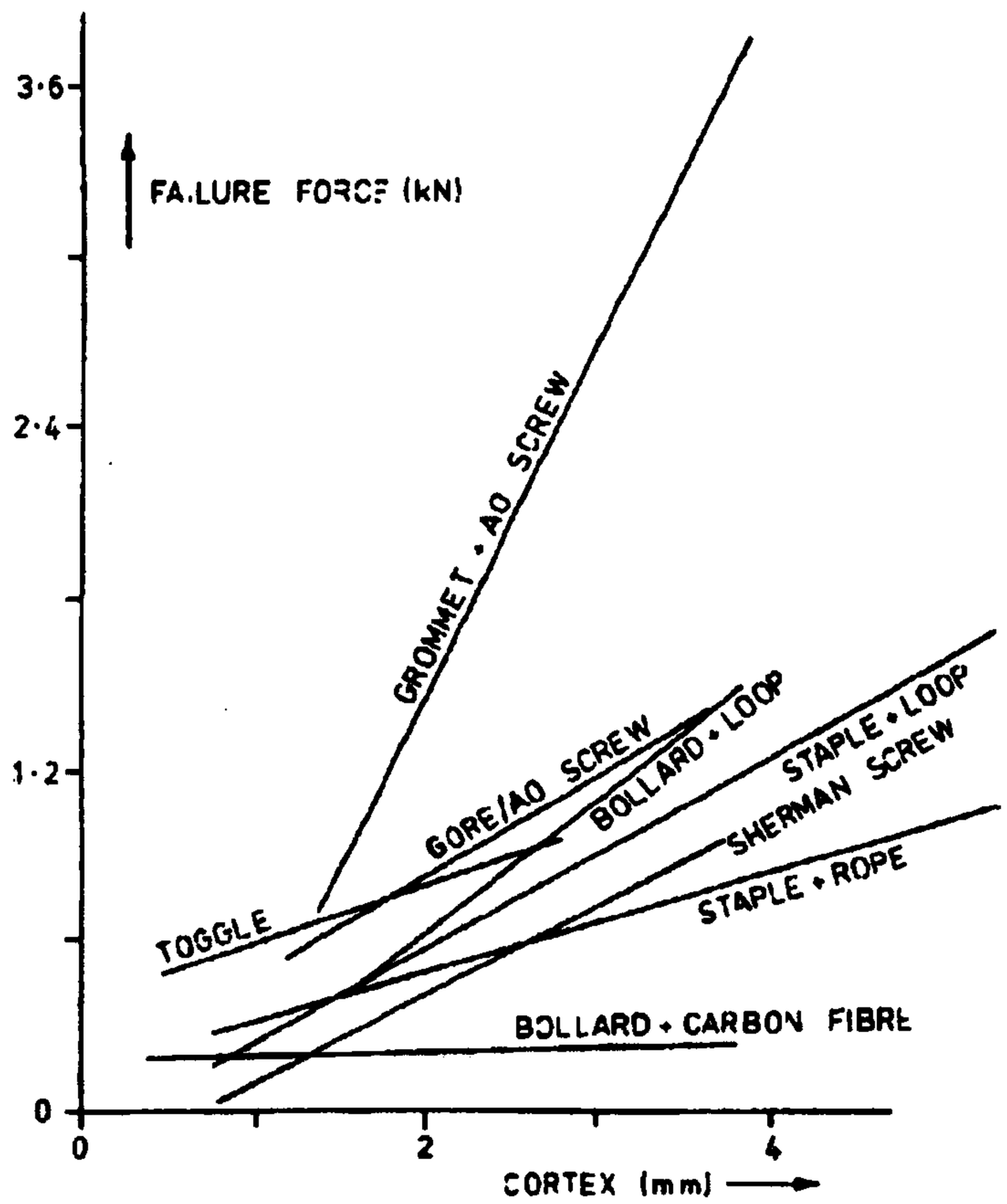


Figure 1.35 Variation of fixation strength of artificial ligament anchors with cortical thickness. Data from Amis (1988)

The study of Amis (1988) showed that most implant fixation techniques would be adequate for 'normal' ACL loads and that the Gore/AO screw, bollard and loop and staple and loop might be adequate for 'normal' PCL loads if placed in sufficiently thick cortical bone (Figure 1.35).

However, only the Apex™ AO screw and grommet would give any margin of safety for 'normal' PCL loads.

Most fixation devices are much weaker than the implants that they fix (Figure 1.35, Table 1.1) and therefore rely on tissue ingrowth to achieve full function. This ingrowth requirement has meant that these synthetic implants have been subject to the same problems of loosening as autogenous reconstructions. So, with regard to implant fixation 'what went wrong' was that many implant designers failed to realise the opportunity for immediate mobilisation by sound fixation. Again, this was not a fundamental flaw in synthetic materials but a design failure that could, and should, have been resolved before clinical use.

1.7 SUMMARY

In comparison to the ACL there has been little work done on the PCL. Those studies that have been published show disagreement about the structure, function and mechanical properties of the ligament. Therefore, further studies of the PCL are required to understand its normal properties and function. The lessons learned from such studies will be of use in improving the treatment of PCL insufficiency.

There is no doubt that in the PCL deficient knee dynamic stability can be achieved by vigorous physiotherapy and gait training. However, the long term consequences of posterior laxity may be disabling. So, surgical reconstruction of some kind is needed. A synthetic PCL implant offers the prospect of a relatively simple operation and a rapid return to activity. Although confidence in synthetic ligaments is currently at a low ebb I believe that the development of a well designed PCL implant is worthwhile. Even at the present time there is a small demand for synthetic implants for salvage cases where no autogenous material is available.

2. EXPERIMENTAL TECHNIQUES

2.1 SPECIMEN ACQUISITION AND STORAGE

Knee specimens were obtained at post-mortem from donors within two days of death. Storage of knees at room temperature for up to four days has been shown not to significantly affect intra–articular ligament properties (Viidik et al, 1965). The knee joints were extracted complete with surrounding soft tissue and about 100mm of the extra–capsular femur and tibia. Specimens were stored until needed in sealed polyethylene bags, to prevent dehydration, at -20°C. This, too, has been shown not to affect ligament properties significantly (Tkaczuk, 1968). When required, the specimens were thawed, still in bags, in warm water.

2.2 BONE POTTING

In order to provide secure anchorage points for mechanical testing bones were set in stainless steel pots. First, the region of bone to be potted was stripped of soft tissue. Then the periosteum was scraped off with the back of a scalpel. The bone was then scored with a rasp and degreased with alcohol. It was then held vertically and coaxially in a cylindrical stainless steel mounting pot by three grub screws. A rigid mounting was achieved by filling the pot with polymethylmethacrylate* (pmma). The powder and liquid were stored at 4°C to increase the pot life of the mixture. The pmma was mixed by hand and had a curing time of 15 minutes. While the pmma was curing the knee specimen was covered by a wet paper towel to prevent dehydration.

2.3 DATA ACQUISITION

Digital data acquisition was facilitated by a 486DX33 PC based system. A National Instruments 'Lab-PC' general purpose data acquisition card was fitted to the PC and controlled by software written by the author

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* 'Simplex Rapid, clear', Austenal Dental Products, Nobel-Pharma, Harrow, Middlesex, England.

using the National Instruments 'Lab Windows' package. The Lab-PC card had eight analogue inputs which were converted by a 12–bit Analogue–Digital Converter. There were also three 8–bit digital Input/Output ports. The analogue inputs were used to monitor a potentiometer used to measure flexion angle (section 5.2) and the output of a strain gauge amplifier. The amplifier was used for a LVDT (section 5.2) and miniature pressure transducers (section 6.2.2). The digital I/O ports were used to capture cross–head extension data from an Instron 1122 materials testing machine (section 6.2.2).

2.4 CROSS-SECTIONAL AREA MEASUREMENT[1]

2.4.1 Introduction

To calculate the mechanical and material properties of soft tissue, the physical dimensions of the sample must be known. The accuracy of calculations for stress, elastic modulus and energy density for a sample are dependant on the accuracy of the measured cross-sectional area. Currently there are two commonly cited methods of cross-sectional area measurement:

(i) Constant pressure area micrometer (Ellis, 1969). This device squashes the tissue into a channel of known section with a constant force. The cross-sectional area of the sample is then read from a suitably calibrated LVDT or dial gauge. To prevent overestimation, caused by gaps between the tissue and the channel, sufficient force must be applied to deform the sample so that it conforms to the shape of the channel. This loading results in underestimation which depends on the pressure applied and the time allowed for the tissue to deform. Standard values of 0.12MPa applied pressure and 2 minutes deformation time have been used to allow results to be compared (Danto *et al*, 1989; Butler et al, 1986)

(ii) Laser micrometer (Lee and Woo, 1988). This device rotates the sample through 180° in a wide laser beam. The width and position of the shadow are recorded at 3° intervals. From this data the cross-

[1] The material in this section has been accepted for publication as a technical note in *J.Biomec.*, Co-author A.A. Amis.

sectional area of the sample is calculated. This method assumes the tissue to be convex; concavities in the cross-section lead to overestimates, which cannot be standardised.

To determine the mechanical properties of the two bundles of the PCL the author developed a novel replica moulding technique of cross-sectional area measurement (section 4.2.2.2). It was hypothesised that this technique, which does not squash the ligament and allows for concavities, would be more accurate than the methods outlined above. This study aims to establish the accuracy of the technique, to quantify the errors involved and to compare the results with those of an area micrometer and a laser micrometer.

2.4.2 Method

2.4.2.1 Replica Technique

Moulds were made of five diameters of silver steel rod in the range 1.6mm to 8.0mm diameter. The steel rods were each cut into five 30 mm lengths each of which was placed coaxially in a cardboard tube of 35 mm diameter. Liquid silicone rubber* was then mixed with curing agent and poured into the tubes. When the silicone rubber had set the cardboard tubes were removed and the steel rods pushed out. The resulting moulds were then used to cast a set of polymethylmethacrylate (pmma) replicas (n=25). In order to check that mould thickness would not affect the dimensions of the replica mouldings, a further set (n=5) replicas were made using cardboard tubes of 20mm diameter. When a bone-ligament-bone preparation is being measured then the mould is cut on one side, parallel to the fibres, and then peeled off. In order to quantify the error produced by cutting the moulds a further set of moulds and replicas were produced (n=20): the moulds were cut and peeled off the rods and then carefully closed and held together with adhesive tape.

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* 'Silcoset 105', with rapid curing agent, from; Ambersil, Bridgewater, Somerset, England.

The pmma was mixed by hand with 6ml of monomer liquid to 10g of polymer powder. This was the highest proportion of polymer to monomer that remained sufficiently fluid to fill the moulds. A high proportion of polymer is desirable because the shrinkage of pmma is primarily caused by the polymerisation of the monomer (Chan and Ahmed, 1991). The pmma and silicone rubber were stored at 4°C to increase pot life. This ensured that the rubber conformed to the specimen, and the pmma to the mould, before starting to set, which occurred after 1 minute in both cases. The tests were also repeated with pmma at 20°C to establish if starting temperature changes were a source of variation in shrinkage (n=25).

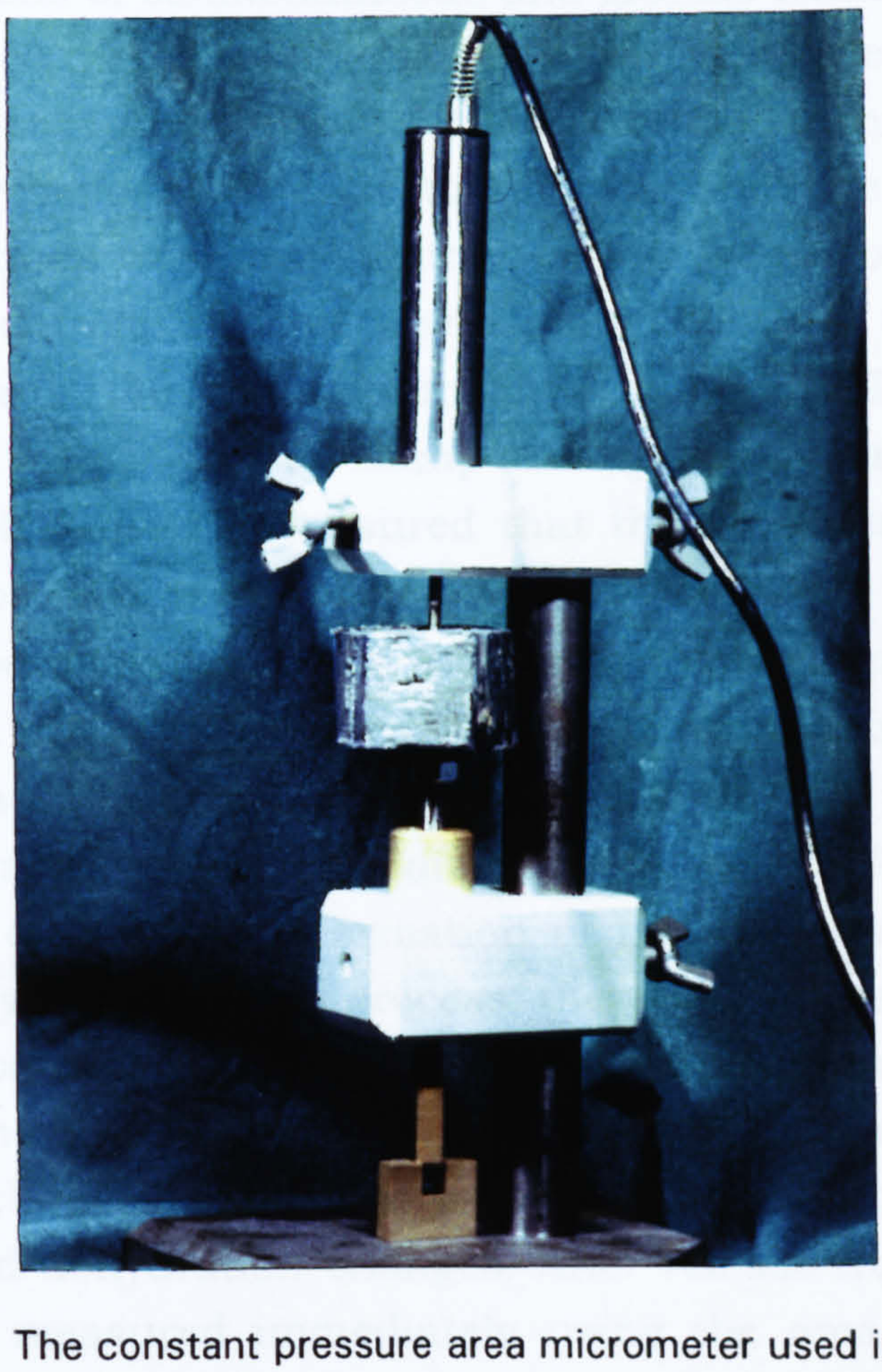


Figure 2.1 The constant pressure area micrometer used in this study.

The replicas were sectioned and stained with black ink and then photographed with a steel rule in the plane of the section. The black and white negatives were then placed in a standard enlarger and the cross-sections were traced, along with a 10mm reference, onto graph paper using a 0.25mm pen. The images were magnified by a factor of between

10 and 20. The cross-sectional areas were then determined by square counting.

2.4.2.2 Comparison With Area Micrometer

An area micrometer was built which used the same channel section and applied pressure as that used by Butler et al (1986) (Figure 2.1).Displacement was measured by an LVDT which was monitored using the data acquisition system described in section 2.3. A program was written to calculate and display the cross-sectional area of a specimen every second.

Twelve specimens of semitendinosus and gracilis tendon were measured using the replica method. The tendons were from knees which had been obtained at post-mortem within 48 hours of death and stored at -20°C in sealed polythene bags prior to thawing and use. For the replica method the tendon was held at either end by tissue forceps. One clamp was suspended from a retort stand and the other, which hung free, supported a steel tube (30mm diameter, 40mm long). The steel tube was fixed coaxially with the tissue clamp and ligament using modelling clay: this gave a 2N load which ensured that the specimen was straight. A cardboard tube was then formed around the steel tube to coaxially enclose the specimen and allow the silicone rubber mould to be made, as before (fig). Dehydration was prevented by covering the apparatus with a wet towel. The towel was not in contact with the specimen but served to maintain 100% humidity. When the moulds were removed there was no discernible deformation of the tendons that might have been caused by the moulding process: there was no visible discontinuity between that portion of the tendon that had been covered by the silicone rubber and that portion which had not. The silicone rubber did not adhere to or chemically react with the tendon and its presence would have prevented dehydration changes. After release from the mould the tendons were measured immediately using the area micrometer. The effect of duration of pressure application on the area micrometer measurements was investigated in 5 specimens for 10 minutes. Dehydration was prevented by covering the apparatus with a wet towel, as before.

2.4.2.3 Comparison With Laser Micrometer

Since the replicas of known sections had shown no artifactual section irregularities, the validity of the assumption of convexity of the cross-

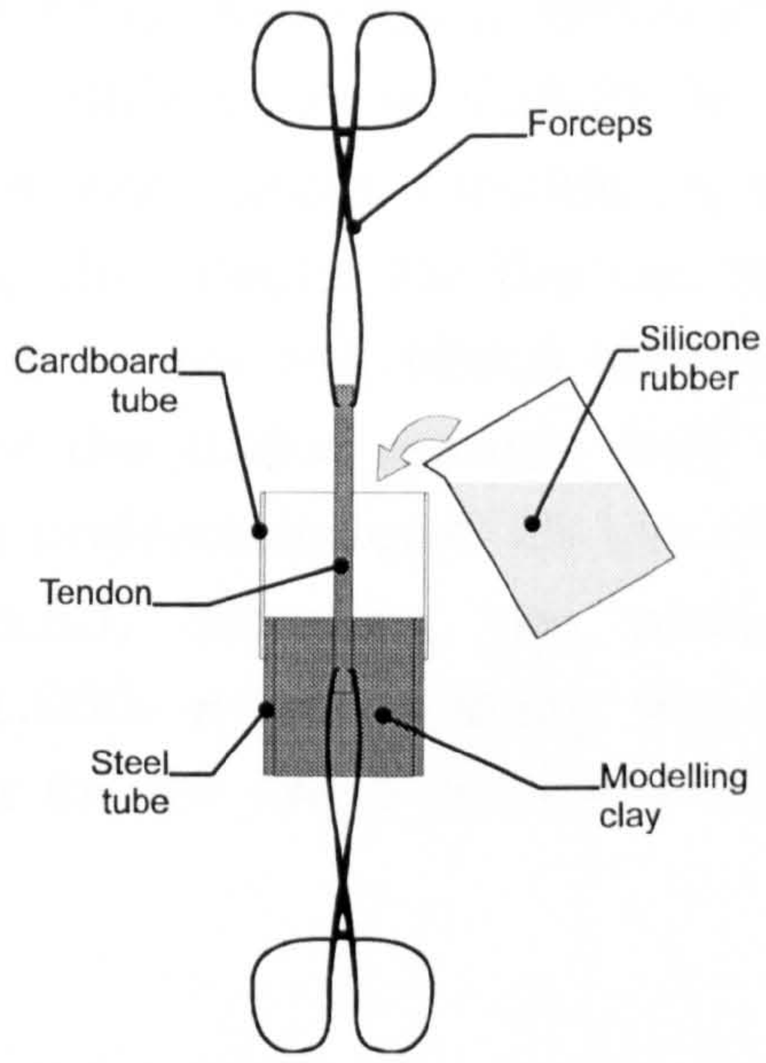


Figure 2.2 Schematic cross-section showing the replica method as applied to tendon specimens in the present study

sections of ligaments and tendons for the laser micrometer was tested by analysing the cross-sections produced by the replica method for the above tendon specimens (n=12) and also cross-sections from a previous study of the anterolateral and posteromedial bundles of the PCL (n=18). Tangential lines were drawn around the perimeter of the graph paper tracings enclosing any concavities and giving a theoretical 'best performance' limit for the laser micrometer, which would apply if the rotation increment tended to zero between measurements. The total area of the concavities was then determined and the resulting overestimate calculated.

2.4.3 Results

2.4.3.1 Replica Technique

There was a systematic underestimation of cross-sectional area by the replica method due to shrinkage, which could be allowed for and so did not affect the overall accuracy. The thickness of silicone rubber mould did not significantly affect the measured cross-sectional area (p=1).

Multiple Student's t tests, using Boniferroni's correction (5 groups, therefore significance level=0.01), showed that the mould/replica shrinkage was not significantly affected by the diameter of the replica section ($p=0.02$) except for the 1.6mm diameter rod ($p<0.001$). Paired comparisons of replicas made in the same moulds at with pmma mixed at 4°C and at 20°C showed no significant difference ($p=0.26$). There was a small but significant difference ($p=0.023$) in the underestimation calculated from the cut and uncut moulds. A correction factor was established by grouping the results for the cut moulds, excluding the results for the 1.6mm diameter rod which were significantly different. The underestimation for the uncut moulds was 7.5% ± 0.3% (s.e.m.), 1.2% (s.d.). The average underestimation for the cut moulds was 6.2% ± 0.4% (s.e.m.), 1.7% (s.d.), therefore, the correction factor for the systematic error was 1.066. After applying the correction factor, the remaining random error for the cross-sectional areas using the replica method was ±1.8%.

2.4.3.2 Comparison With Area Micrometer

The effects of time on the area micrometer measurement can be seen in Figure 2.3. Figure 2.4 shows a semitendinosus specimen after removal from the area micrometer after 2 minutes.

The tests directly comparing the areas calculated by the area micrometer and by the replica method, with 1.066 correction for shrinkage of the replicas, showed that the mean difference between them was 16.2% ± 6.9% (s.d.) with a range of 6.7%–28.9%. The area micrometer gave the lower estimate, as expected.

2.4.3.3 Comparison With Laser Micrometer

Analysis of the cross-sections of the tendon specimens which would have been derived by the laser micrometer showed that the average overestimation was 2.3% ± 1.5% (s.d.) with a range of 0.9%–4.9%; this was not significantly different from the errors in the replica method found in this study ($p=0.27$, 2-tail t-test, assuming unequal variances). Analysis of the PCL bundles showed that the average overestimation for the apparently 'round' anterolateral bundles was 4.7% ± 2.5%(s.d.) with a range of 1.6%–7.6%; this error was significantly greater than that found for the replica method ($p=0.013$, 2-tail t-test assuming unequal

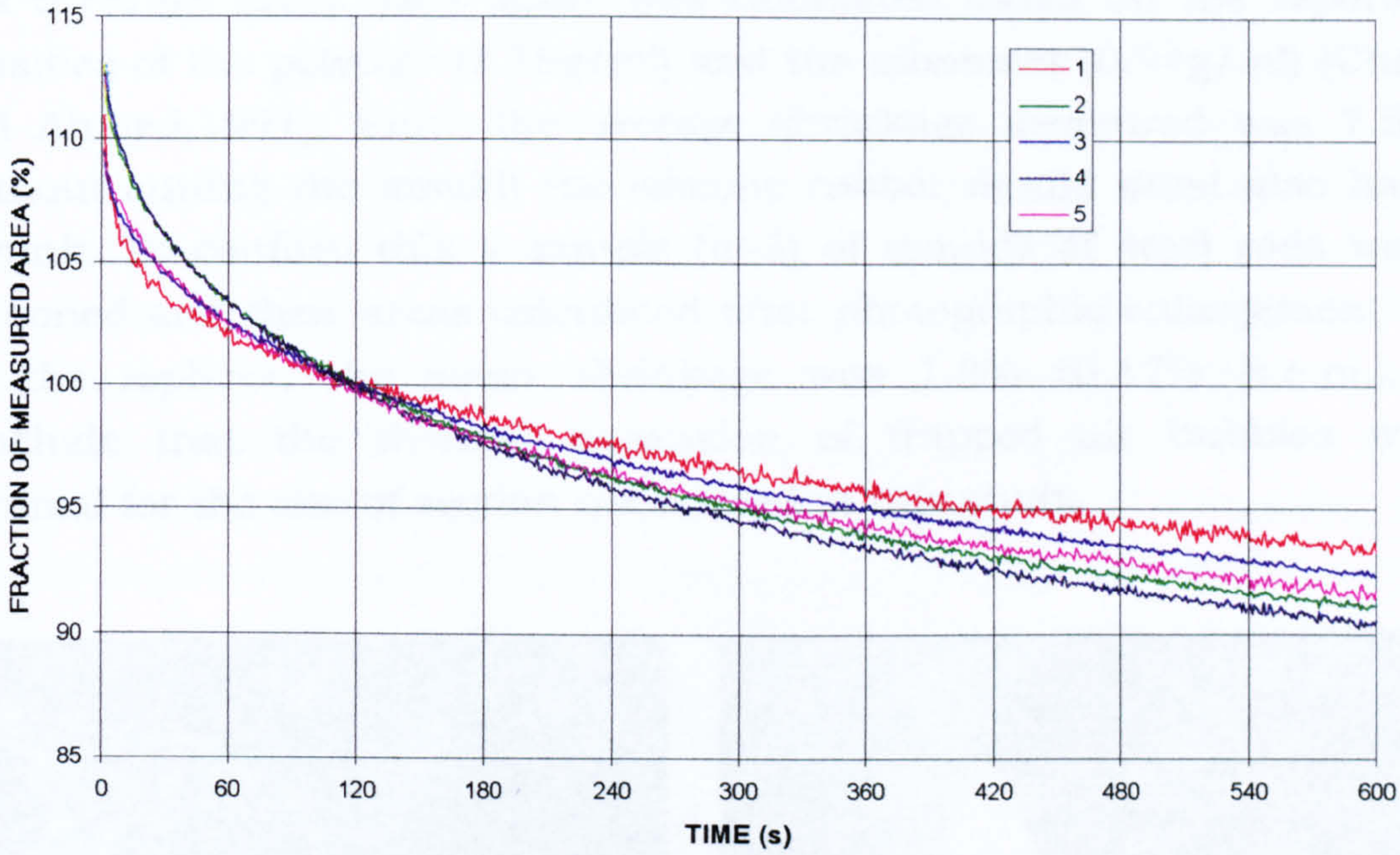


Figure 2.3 Graph of measured area against time from the constant pressure area micrometer. The measured area data for each of the five tendon specimens has been scaled to give 100% at 2 minutes

variances). The posteromedial bundles had obvious concavities and the average overestimation was 19.1% ± 6.4%(s.d.) with a range of 10.9%–29%, this error was also significantly greater than that found for the replica method (p=0.0003, 2-tail t' test assuming unequal variances). Typical bundles are shown in Figure 2.5, along with a section of an entire PCL.

2.4.4 Discussion

2.4.4.1 Replica Method

The replica method recorded the shape and cross-sectional area of specimens, including concavities. The same moulding materials have been used previously to pick up the fine surface detail of articular cartilage (Sayles et al, 1979). Shrinkage of the pmma during polymerisation led to undersized replicas, but most of the error was systematic and could be corrected for using a factor based on the measurement of replicas of known sections. The theoretical maximum shrinkage for the pmma mixture used, which would occur if there was

no thermal expansion of trapped air bubbles (Chan and Ahmed, 1991), was 5.7% (in area). This figure was calculated based on the reported densities of the polymer (1.18g/ml) and the monomer (0.94g/ml) (Chan and Ahmed,1991). Since the average shrinkage measured was 7.5% (without cutting the mould) the silicone rubber mould must also have shrunk. To confirm this a sample (n=3) of moulds of steel rods were sectioned and their areas calculated after photographic enlargement as for the replicas; the mean shrinkage was 1.8% ±0.17% (s.e.m.). I conclude that the thermal expansion of trapped air bubbles was minimal for the size of section considered in this study.



Figure 2.4 Semitendinosus specimen before (left) and after (right) 2 minutes deformation in the area micrometer.

I think that the 14% shrinkage for the 1.6 mm diameter replica was due to the pmma not flowing well through such a small hole and a greater proportion of monomer entering the mould. Therefore I do not recommend the pmma replica method for sections under 3mm diameter. It is possible that another casting compound may give better results for small sections and/or shrink less in curing. The shrinkage of the moulds (1.8% in area) meant that the soft tissue specimens must have been slightly compressed during the curing process. Although soft tissue specimens appeared to be unaffected by the moulding process it

is possible that the silicone rubber caused some chemical change in the specimen; this has yet to be investigated.

An obvious route to the reduction of systematic error would be to dispense with the pmma replica and directly measure the silicone rubber mould. However, the advantages of using the pmma replica are: *(i)* It is much easier to visualise the shape of the tissue being studied and come to a considered decision about the orientation of the plane in which to section the replica. *(ii)* Any number of replicas can be made from a mould and sectioned in a variety of ways but sectioning the mould itself means that some information is permanently lost.

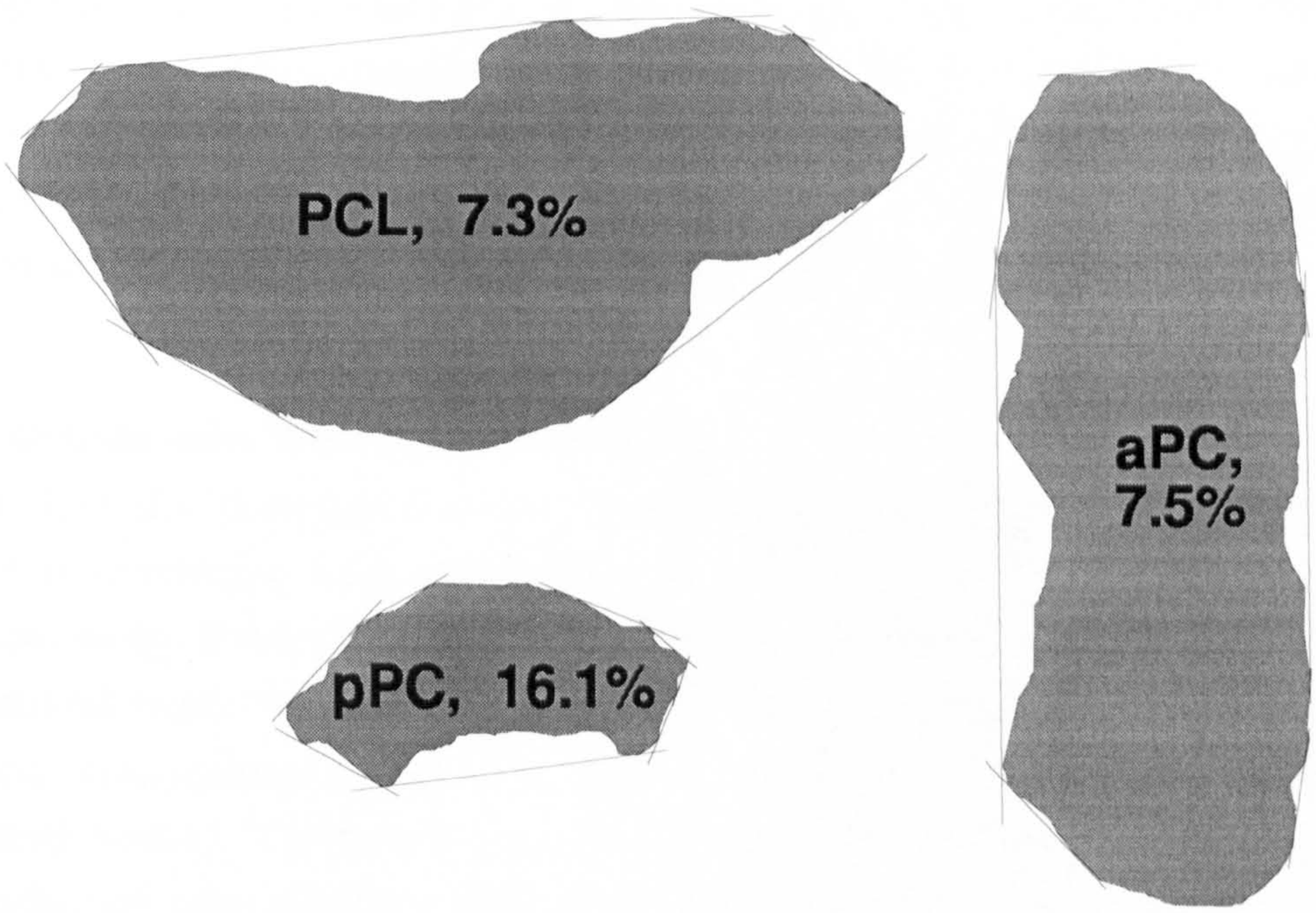


Figure 2.5 Typical cross-sections, showing the aPC, pPC and PCL. Tangential lines are shown which mark the concavities. The percentage figures give the error that would result if the sections were considered to be convex or flat.

The overall random error was ±1.8% (s.d.); this error has several possible components which are discussed below. The error involved in square counting was quantified by measuring the areas of five identical circles, drawn with a compass, of a similar size to the traced areas. The standard deviation of the square counts was 0.17%. The pmma liquid and powder were measured to within 0.05ml and 0.02g respectively; these figures gave the probable variability of shrinkage of the pmma as ±0.03% (of cross-sectional area). Cutting the moulds increased the

random error by 0.5%. It was concluded that the remaining 1.1% of the overall random error was due to errors which occurred when the section outlines and scales were traced onto the graph paper.

I consider the accuracy of the replica method to be superior to that of both the area micrometer and the laser micrometer, particularly when measuring irregular specimens such as cruciate ligament bundles. However, as described above it is labour intensive. If a digitiser were used to measure the traced areas it would save some time but the accuracy of the result would be limited by the accuracy with which the outline of the section could be followed by the stylus and the resolution of the digitiser. The photography and square counting could both be by-passed using a video dimension analyser with a suitable macro lens. I have done some experiments with a V.D.A and have found that great care and consistency is needed in calibration, lighting and setting threshold levels if the accuracy is to match that achieved 'by hand'.

2.4.4.2 Comparison With Area Micrometer.

The fact that the measured cross-sectional area decreases with time in the area micrometer appears to be due to the flow of liquid/ground substance away from the compressed tissue: when a tendon specimen was removed from the area micrometer after 17 minutes the part that had been compressed had the yellow appearance and stiffness of dehydrated tissue. Therefore the area micrometer is not measuring the cross-sectional area of the entire, composite, tissue with the result that material properties cannot be accurately calculated: specimens with different percentages of ground substance and different fibre densities will result in different underestimates of cross-sectional area. This hypothesis is supported by the variation in the measured area/time characteristics for different specimens (Figure 2.1). A 'functional cross–sectional area', that of the load bearing collagen fibres, may be hypothesised but any relationship to either the undeformed or deformed (by the area micrometer) cross–sectional area is unknown at present.

2.4.4.3 Comparison With Laser Micrometer.

Unlike the area micrometer and the replica method, the laser micrometer is affected by specimen geometry: Lee and Woo (1988) reported that measuring at 1° intervals the laser micrometer gave the

cross-sectional areas of a circle, a hexagon and a triangle to within +0.1%, +0.3% and +2.1% respectively. They also reported that at 3° measuring intervals the laser micrometer was accurate to within +5% for a triangle, and this angular increment has been used for subsequent ligament studies. The laser micrometer is also affected by concavities (Lee and Woo, 1988; Woo et al, 1990); this study has shown that the uncontrollable error that results from concavities is variable and may be much larger than the random error. So, while the laser micrometer is potentially the most precise of the three methods, since its precision increases with decreasing angular measurement increments (Lee and Woo, 1988), it is also potentially the least accurate because of unpredictable concavities.

Measurements of cross-sectional area are usually made in order to assess the material properties of a specimen, which involves tensile testing. The laser micrometer system requires all-round visibility which in the case of the cruciate ligaments entails the removal of bone adjacent to the ligament attachments (Harner et-al, 1995). Great care must be taken to avoid compromising the ligament insertions.

2.4.4.4 Other Studies

This study suggests that the average difference between cross-sectional areas of ligaments measured by the laser micrometer and by the area micrometer is 21%, which is the sum of the average overestimate and underestimate by the two micrometers for the aPC. Previously published data from other studies, of rabbit and pig ligaments, show differences between the two micrometers of 19% ± 3% (s.e.m.), range 4%-36% (Woo et al, 1990) and of 17% ± 2% (s.e.m.), range 8%-25% (Lee and Woo, 1988). These figures are consistent with the differences found in the present study and demonstrate the variability of area/laser micrometers.

2.4.4.5 Conclusion

The replica method is particularly useful when studying cruciate ligaments. Their wide multi-fascicle structure is deformed significantly by the area micrometer, and I am not convinced that the microstructure of fibrils and ground substance would return to normal even on re-hydrating. This would therefore compromise subsequent mechanical

tests. The laser micrometer is not easily applied to cruciate ligaments because of the need for all-round illumination. This entails removal of parts of the bones, which could prejudice insertion strength and does not account for the large concavities in the ligament cross-sections.

3. ANATOMICAL STUDY

3.1 FASCICULAR STRUCTURE

3.1.1 Introduction

A search of the literature showed that the fascicular structure of the PCL was not agreed upon (section 1.2.1). Opinion was divided as to the presence and number of discreet functional bundles. Therefore, a qualitative investigation of PCL fascicular anatomy was conducted.

3.1.2 Method

Initially five knees were dissected; anatomical observations were also made of five knees used in the study described in section 4. All soft tissue was removed bar the PCL, MCL and the menisci. The lateral femoral condyle was then removed to allow the whole of the PCL to be visualised. The synovial covering of the PCL was carefully removed by sharp dissection to reveal the fascicular structure. The meniscofemoral ligaments present were noted and then excised. Blunt dissection was then used to determine the course of the fascicles. Sutures of various colours were lightly wrapped around groups of adjacent fascicles as a visual aid. During the dissection process the fascicles were observed as the knee was flexed and extended.

3.1.3 Results and Discussion

3.1.3.1 Meniscofemoral Ligaments

The anterior meniscofemoral ligament, or ligament of Humphry, was found in all ten knees but varied in size (Figure 3.1 and Figure 3.4). The posterior meniscofemoral ligament, or ligament of Wrisburg, was only present in four knees (Figure 3.2). The meniscofemoral ligaments helped to control the motion of the lateral meniscus. There was no direct connection between the tibia and femur due to the meniscofemoral ligaments.

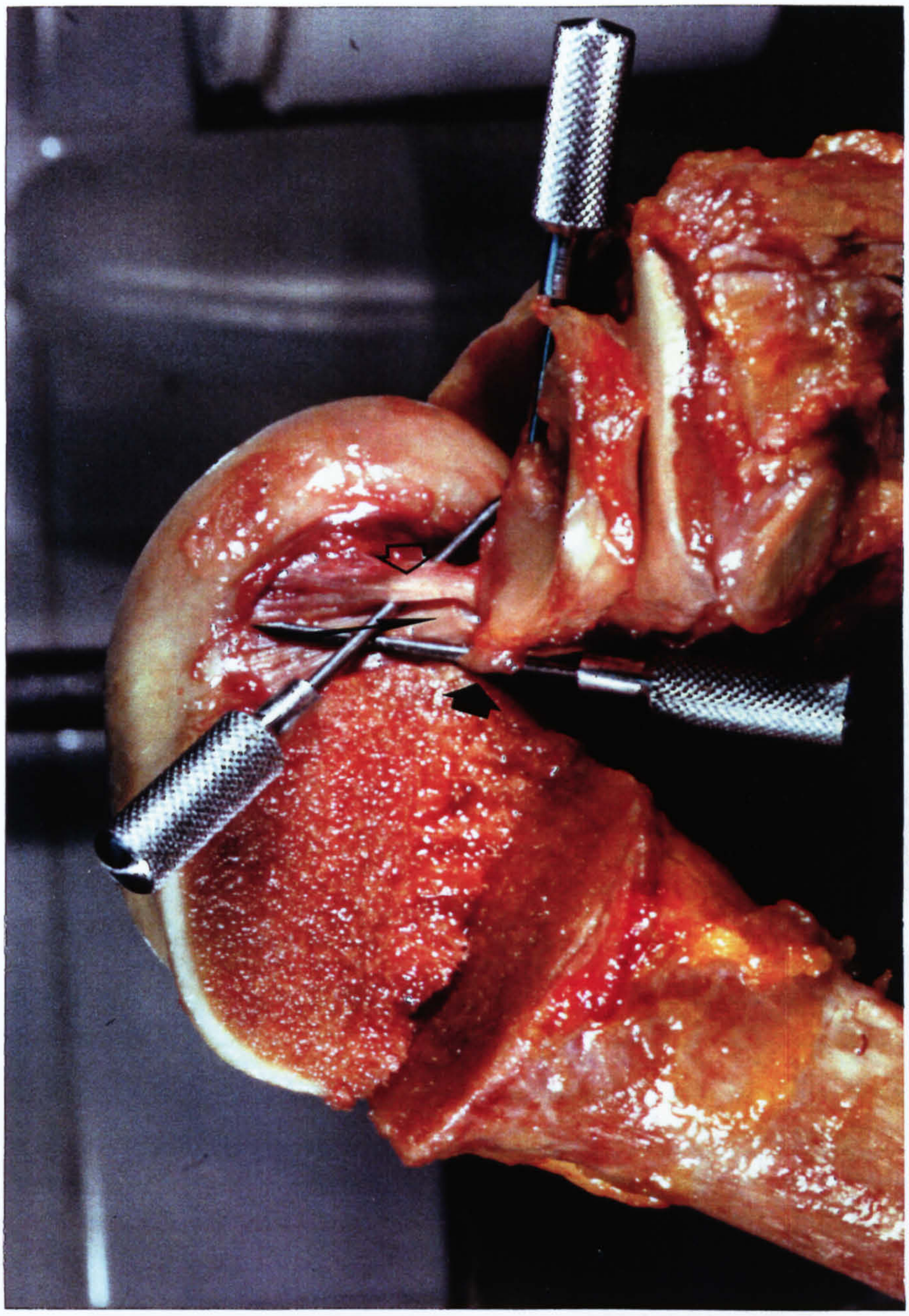


Figure 3.1 Flexed knee viewed from the lateral side with the lateral condyle removed to display the femoral attachments of the posterior cruciate ligament. Pins have been placed between the PCL (⟶) and the anterior (⌂) and posterior (⬟) meniscofemoral ligaments.

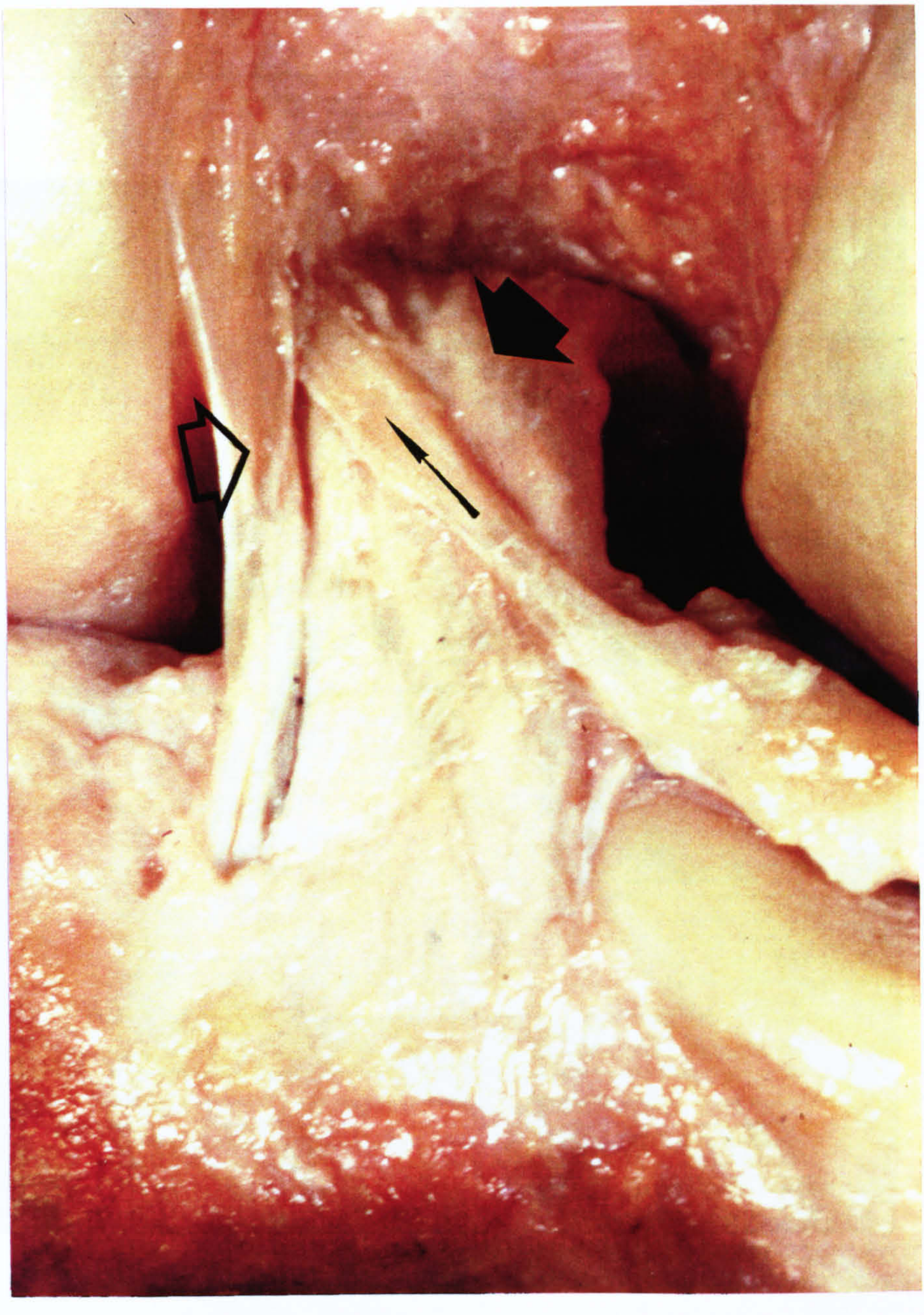


Figure 3.2 Posterior aspect of the extended knee showing the femoral insertion of the posterior meniscofemoral ligament (⟶) between the anterior (◀) and posterior (◁) fibres of the PCL.



Figure 3.3 Lateral aspect of the tibia with the lateral tibial condyle removed to show a sagittal section of the PCL insertion. The bulk of the fibres insert into the flat of the intercondylar fovea (⬟). Some of the posterior fibres insert much more distally (⌓).

3.1.3.2 Structure of the PCL

It was observed that the bulk of the PCL originated from the roof of the femoral notch (Figure 3.2 and Figure 3.4). In the literature most diagrams of the PCL femoral origin show only a sagittal plane projection which is quite misleading. In the tibia the majority of the PCL fibres inserted into the 'flat' of the fovea between the tibial condyles. Some of the posterior fibres ran over the back of the tibia and inserted up to 15mm distally to the fovea (Figure 3.3).

The fibres that originated from the roof of the notch were consistently present and aligned in an approximately sagittal plane. The posterior fibres that originated from the medial femoral condyle showed some variation in structure. Two main posterior fibre groups were observed. One group was aligned approximately to the sagittal plane and inserted medially into the tibia, while the other was aligned obliquely and inserted laterally into the tibia (Figure 3.6). The proportion of these posterior bundles varied: Figure 3.5 shows a knee with a large oblique bundle and no sagittal bundle.

3.1.3.3 Qualitative Observations of PCL Function

While flexing and extending the dissected knees it was discovered that the fascicles of the PCL could be separated, by blunt dissection, into two groups: those fascicles which tightened in extension and those fascicles which tightened in mid-flexion. Separation was achieved by slowly introducing a MacDonald blunt dissector from the lateral side and feeling the tension generated by flexion-extension. When the dissector had been steered through to the medial side of the PCL its progress was halted by the thick synovial covering. The medial synovial covering was then punctured with a scalpel, after which the blunt dissector could be used to complete the separation.

The finding that there were reciprocal tensions in two fibre groups of the PCL led to the conclusion that it was useful to consider the PCL as a two-bundled structure comprising the anterolateral (aPC) and posteromedial (pPC) bundles. The aPC was made up of the fibres that inserted into the roof of the femoral notch. The pPC was made up of the fibres which inserted into the medial femoral condyle; these included the oblique and sagittal posterior fibres referred to in section 3.1.3.2.

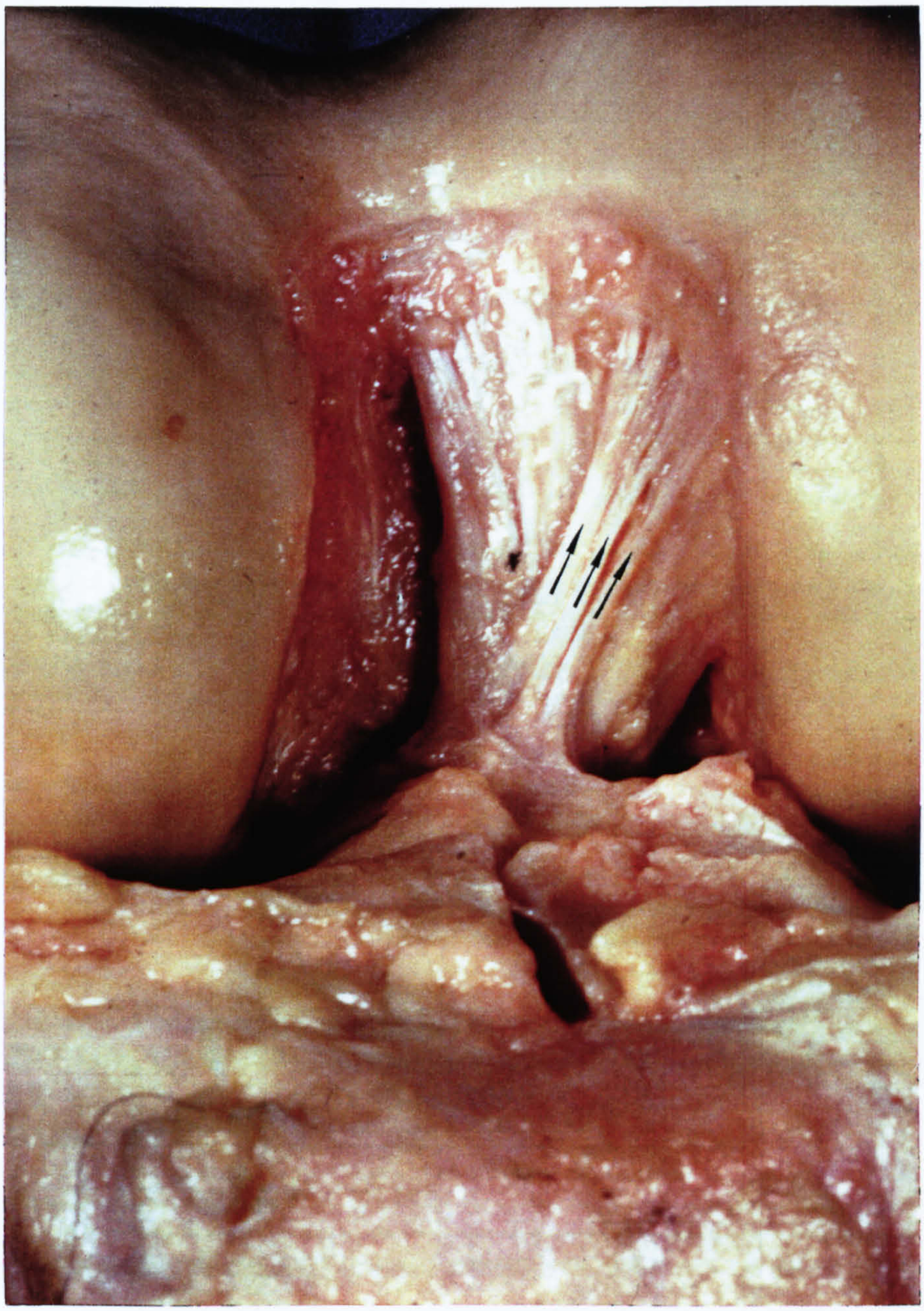


Figure 3.4 Anterior aspect of the flexed knee. The three oblique fascicles (⟶) made up the smallest observed anterior meniscofemoral ligament.

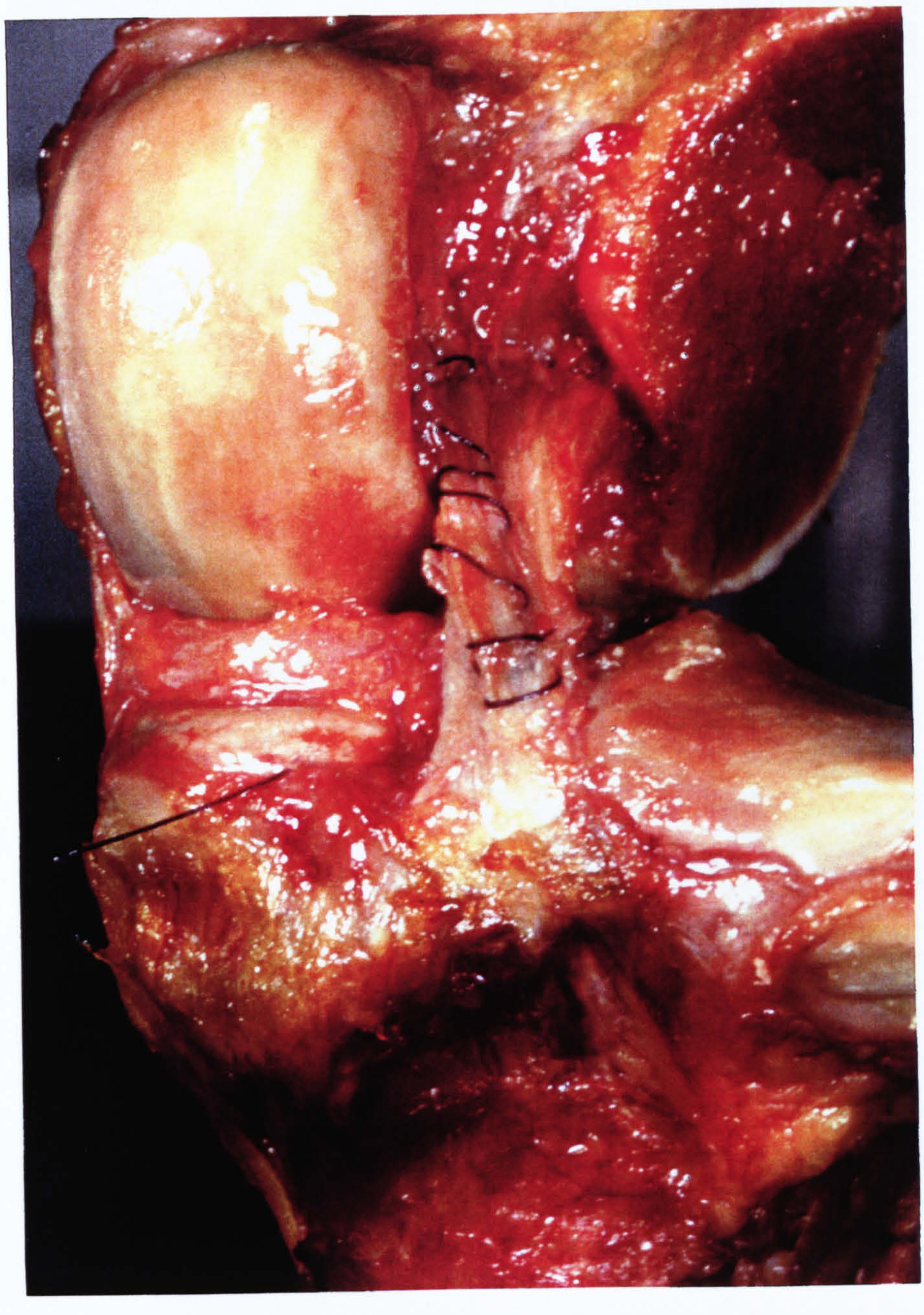


Figure 3.5 Posterior aspect of the extended knee with the lateral femoral condyle removed. The oblique posterior fibre bundle has been picked out with a suture.

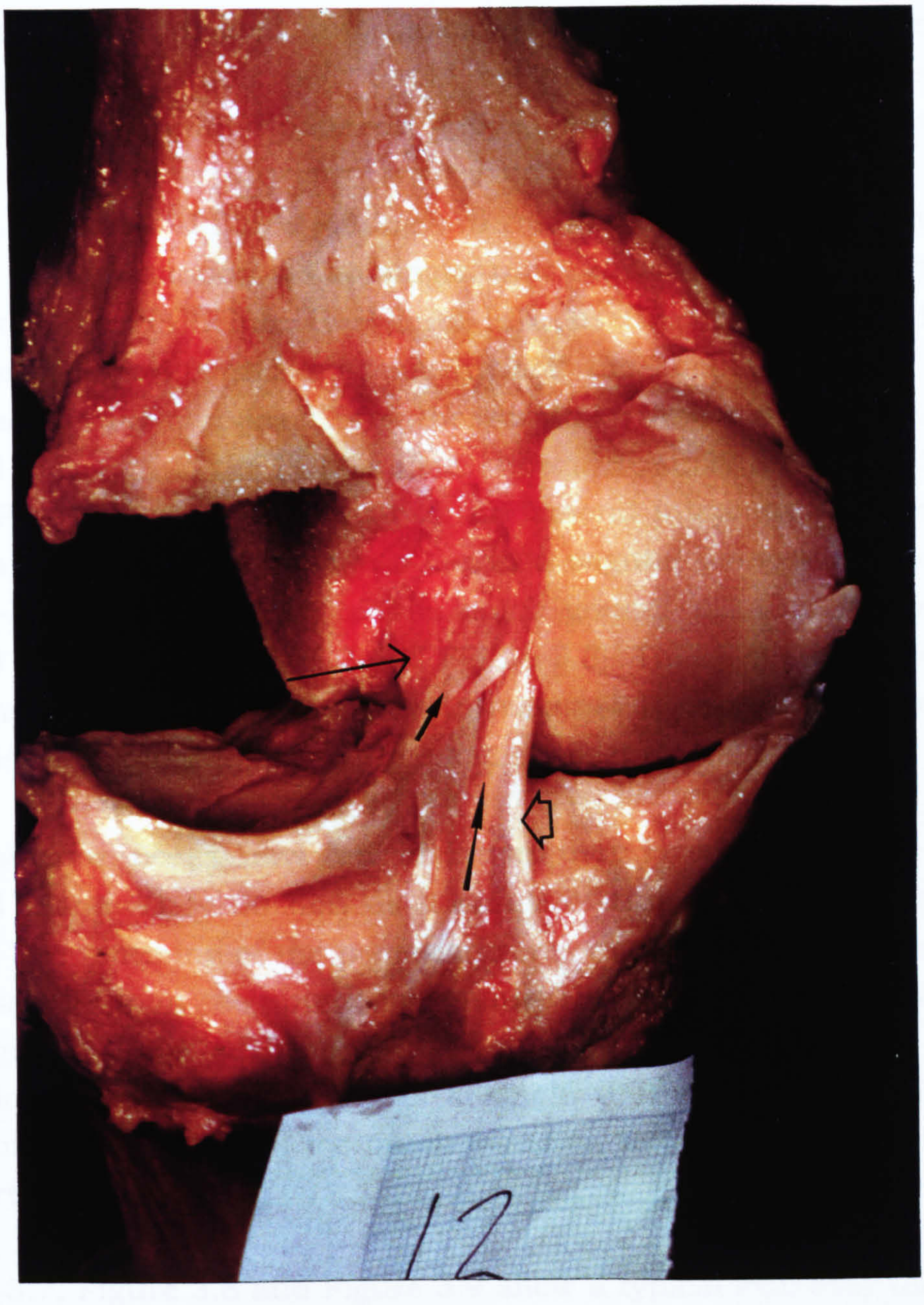


Figure 3.6 Posterior aspect of the extended knee with the lateral femoral condyle removed. Note the sagittal (⌓) and oblique (⟶) posterior fibres of the PCL. The anterior fibres of the PCL (→) are partly obscured by the posterior meniscofemoral ligament (➡).

The PCL could, in theory, have been divided into any number of fibre bundles by grouping fibres within certain envelopes of insertion site separation in response to flexion: the smaller the envelope of insertion site separation the greater the number of bundles. The ligament could also have been divided into fascicle–based bundles. However, the division into aPC and pPC could be performed with an ease and consistency which other patterns of division did not seem likely to offer.

Clearly, an array of fibres joining two rotating and translating surfaces cannot really function as a single unit. However, I think that the division of the PCL into two units is a useful simplification for the purpose of experimental analysis. In fact, the simplification is not as gross as it might appear from the sagittal plane view in which the femoral insertion of the PCL is often shown. Because the femoral origin of the PCL is largely in the roof of the femoral intercondylar notch the fibres of the aPC overlap each other in a sagittal plane projection: the medial–lateral width of the aPC is much greater than its thickness in sagittal projection (Figure 3.2 and Figure 3.8). These 'overlapping' fibres will function very much as a unit in response to sagittal plane motion, which is the motion that has the greatest effect on the cruciates.

The posterior fibres have an insertion that is more parallel to the sagittal plane. They do not, as a result of this, overlap to any great extent. The grouping of the posterior fibres into a single functional unit was less safe than the anterior grouping: there was a greater spread in the sagitally projected femoral insertions of the fibres. In spite of this limitation, the pPC was kept as a single unit because the posterior fibres of the PCL showed too much variation to be reproducibly divided any further.

Figure 3.7, Figure 3.8 and Figure 3.9 show a typical PCL over the range of flexion. In extension the aPC was conspicuously slack but as flexion progressed the aPC became taut. In full flexion the aPC came into contact with the roof of the notch which made it hard to judge its tension. The pPC was tight in extension but slackened with flexion (Figure 3.10). As flexion progressed the pPC passed from 'behind' the aPC (Figure 3.7), to 'in front' of it (Figure 3.9).

In extension it did not seem that the aPC could resist posterior drawer since it was so slack. The aPC appeared able to efficiently resist posterior drawer in mid–flexion as it was taut and well aligned to the direction of the drawer force. As flexion progressed the aPC remained taut but became more vertical and so less well aligned to resist posterior drawer.

The function of the pPC was not clear from this study. It has often been observed that the pPC is taut in extension (section 1.2.2), and this was confirmed by the present investigation. However, because the pPC's alignment was almost perpendicular to the tibial plateau in extension the pPC cannot have resisted much posterior drawer force. As flexion progressed the pPC did not appear to become better suited to resist posterior drawer as it rapidly became slack. In mid–flexion and full flexion it was hard to judge the tension in the pPC because it was obscured by the aPC. If there were any tension in the pPC in these regions then the pPC would have functioned to resist posterior drawer because its alignment improved with increasing flexion.

This study raised questions about the function of the PCL at the extremes of flexion. It did not appear that the PCL could resist posterior drawer in extension and the relative importance of the two bundles in flexion was unclear.

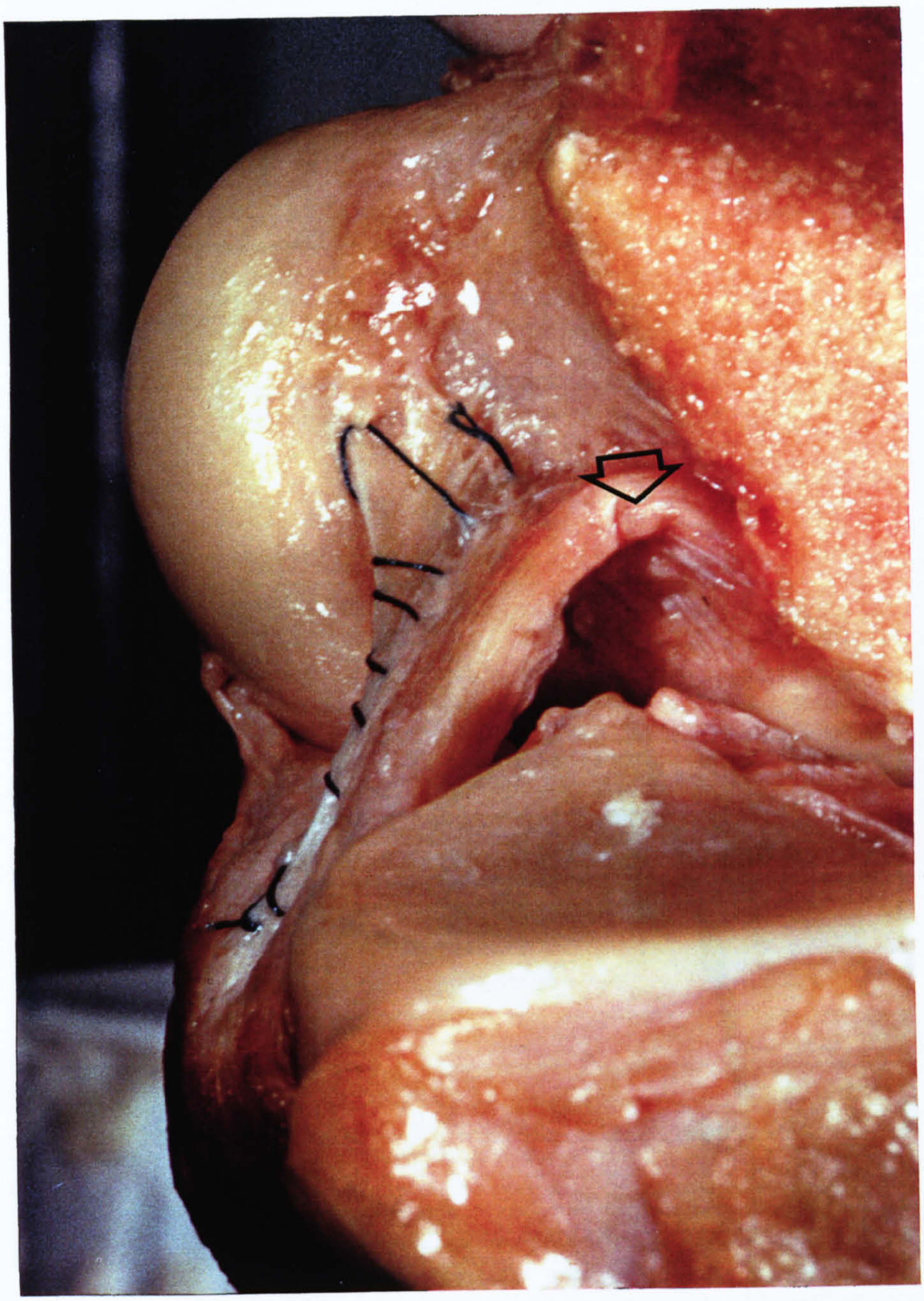


Figure 3.7 lateral aspect of the extended knee with the lateral femoral condyle and lateral meniscus removed. The posterior fibres of the PCL have been wrapped with blue suture. Note that the posterior fibres are taut and that the anterior fibres are slack (⌓).

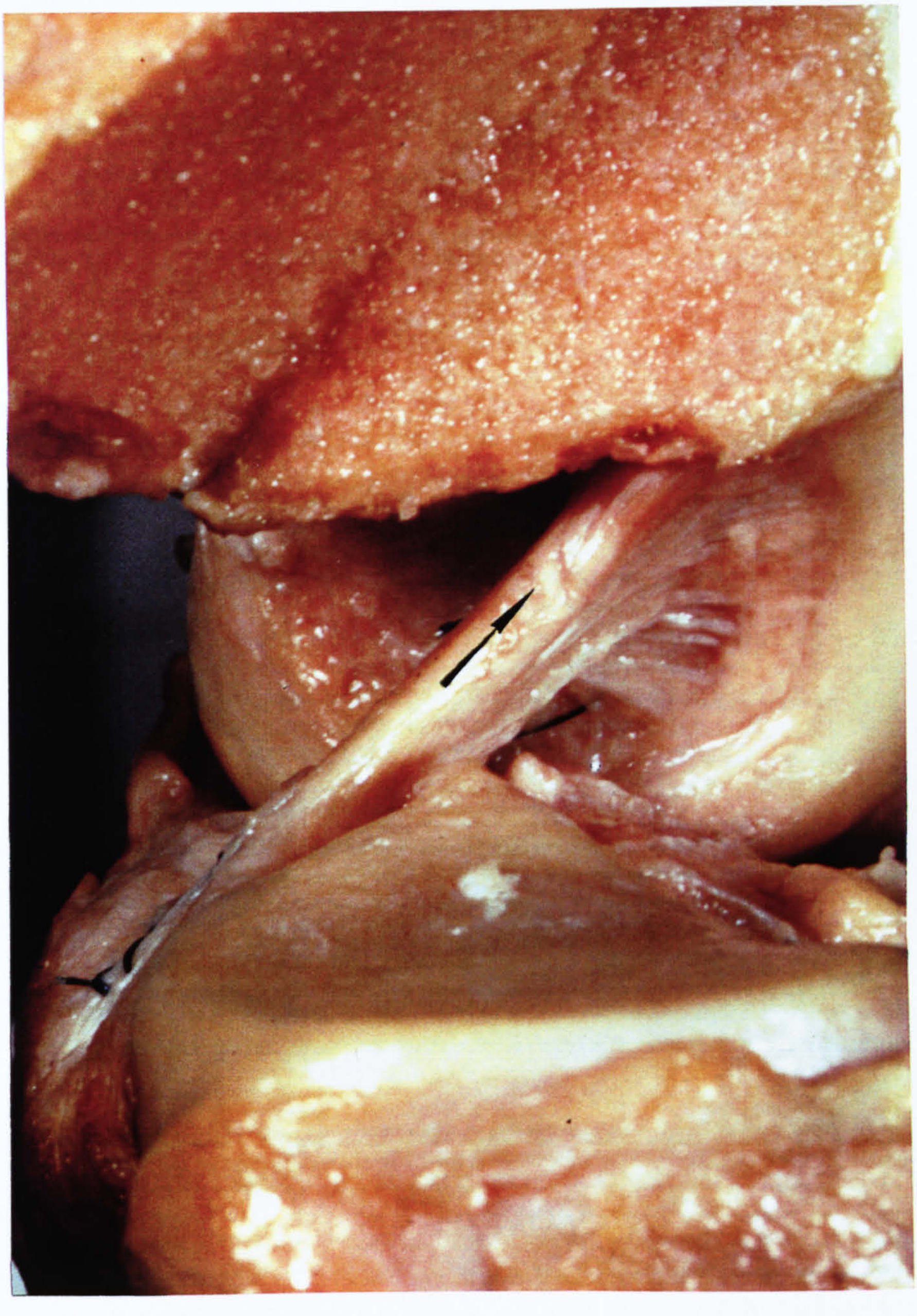


Figure 3.8 Flexed knee viewed from the lateral side with the lateral condyle removed to display the femoral attachments of the posterior cruciate ligament. Note that the anterior fibres of the PCL (⟶) are taut.

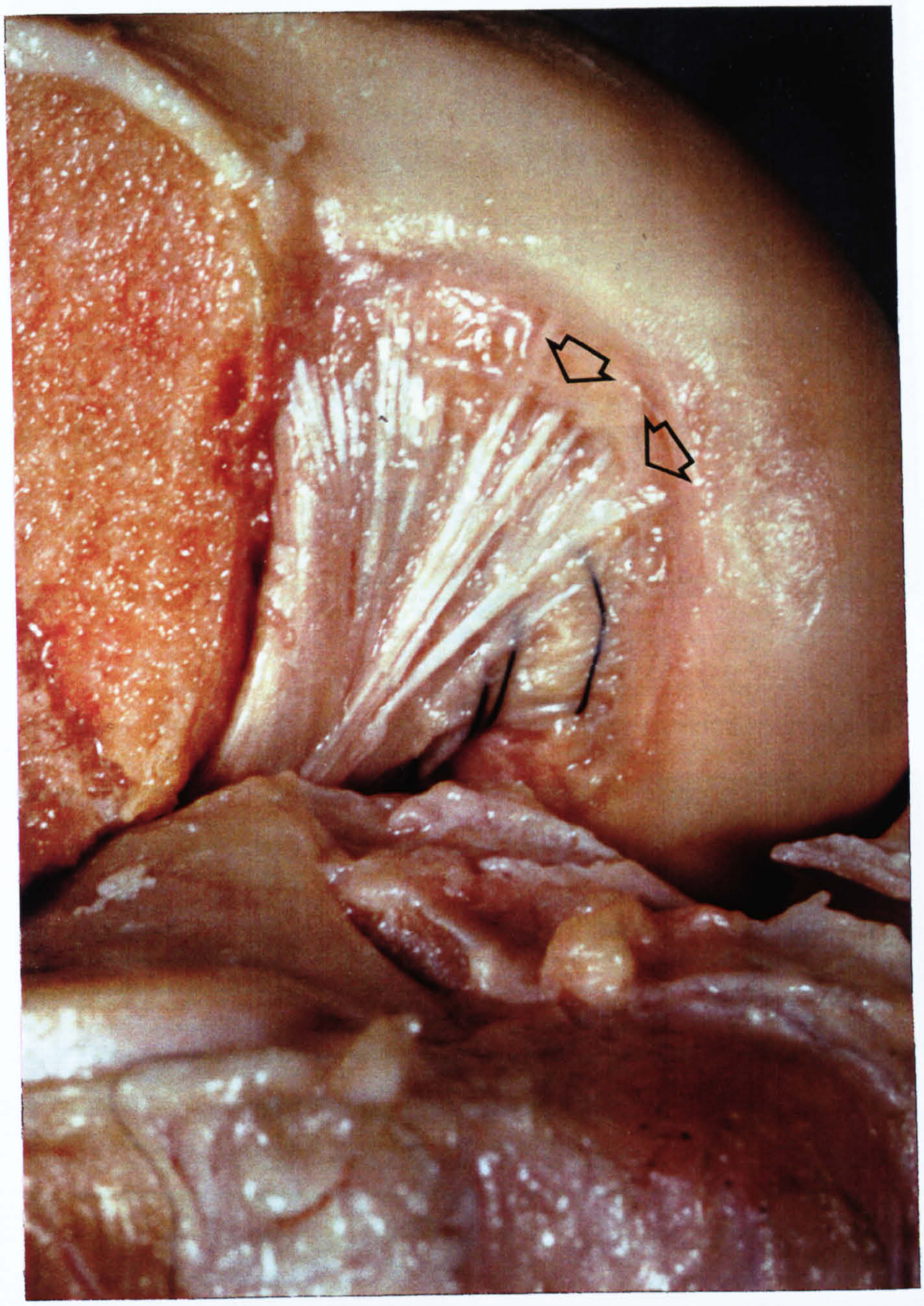
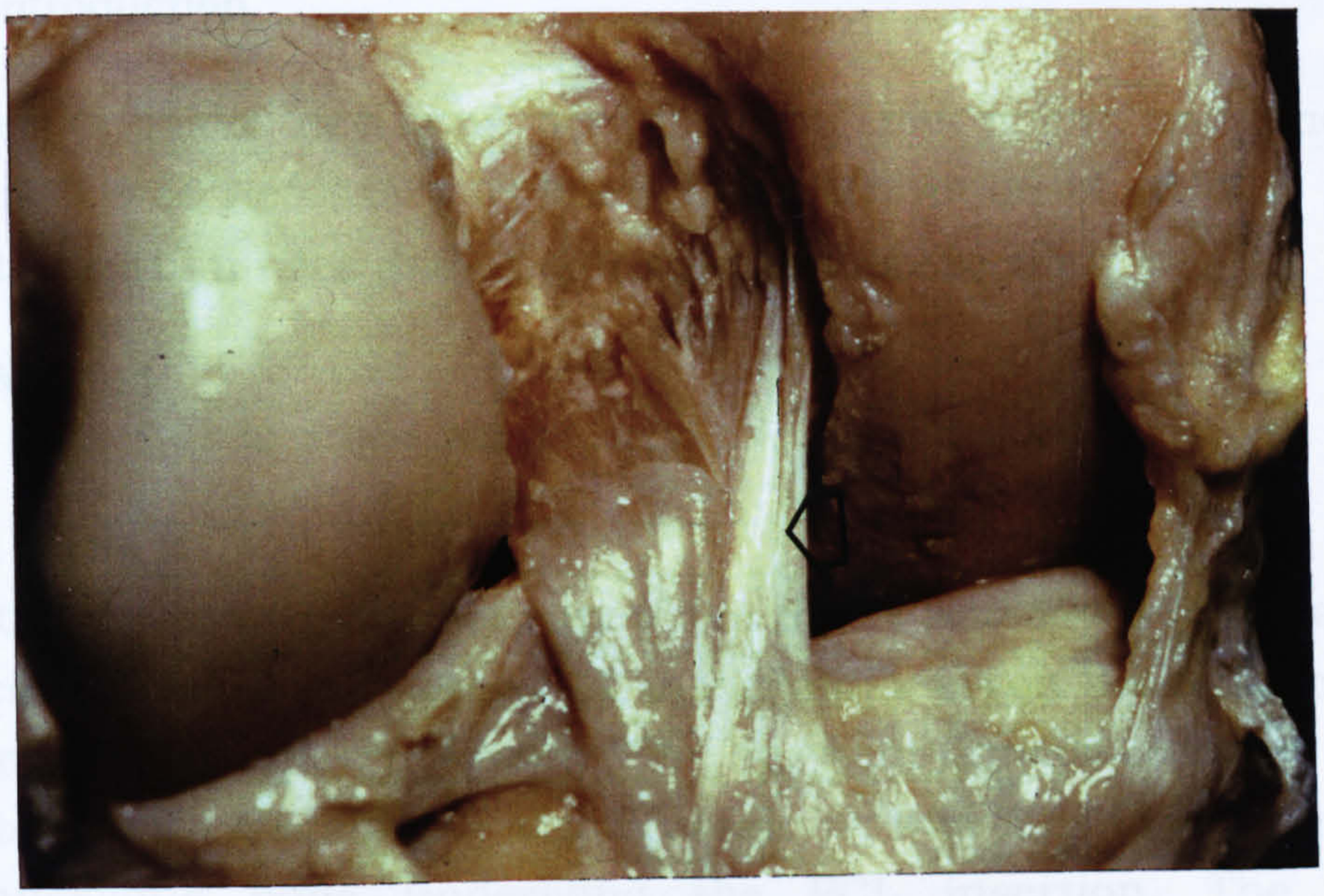


Figure 3.9 Medial side of the intercondylar notch with the lateral femoral condyle and lateral meniscus removed. In full flexion the posterior fibres of the PCL came to the front of the joint and were well aligned to resist posterior drawer. The pPC has been marked with suture, although some of the anterior and distal fibres of the pPC were missed (⌓).



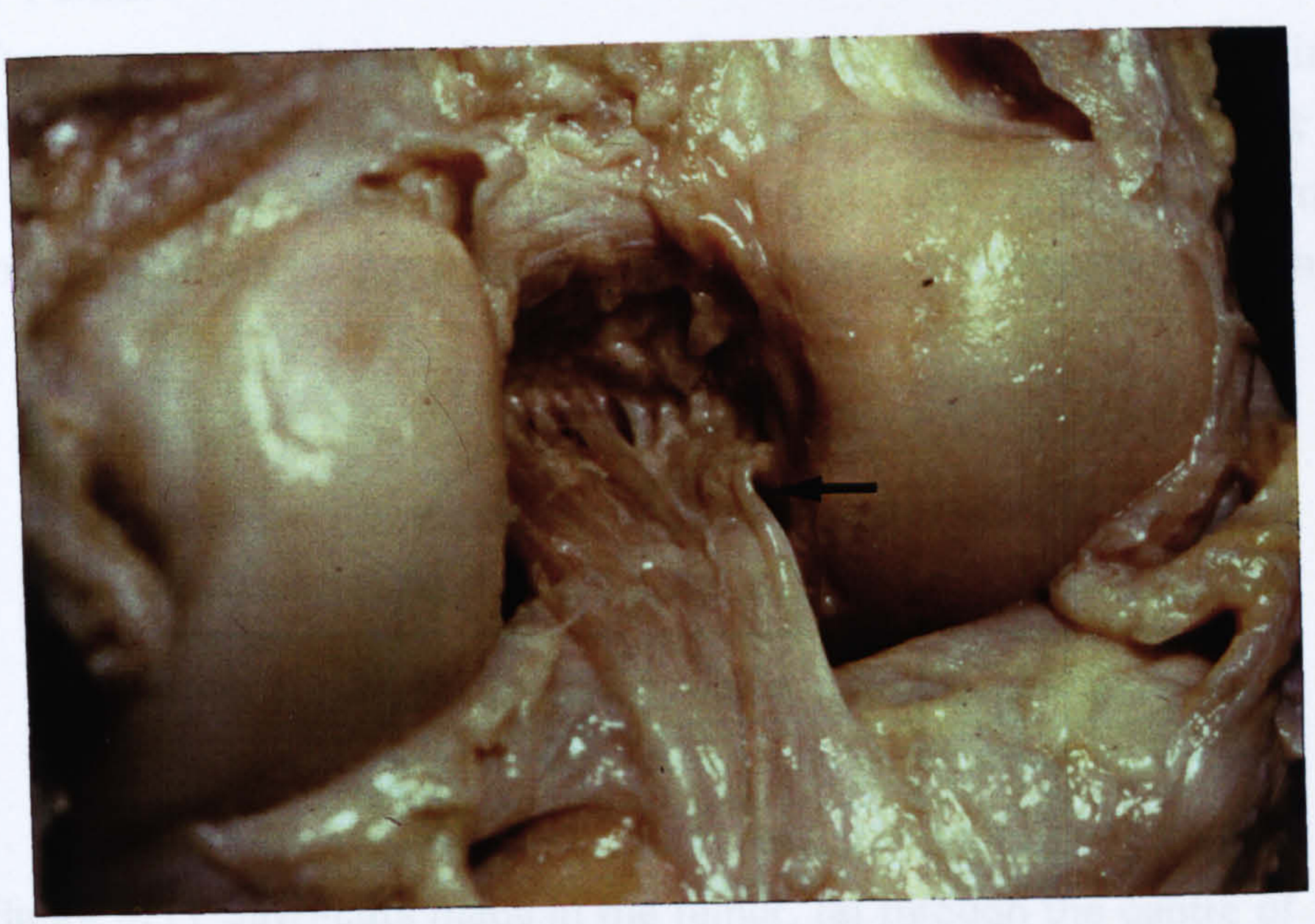


Figure 3.10 Posterior aspect of the knee with the posterior capsule removed. In the extended knee, top, the posterior fibres of the PCL are taut (⌓). In the partially flexed knee, bottom, the posterior fibres of the PCL slacken (⟶).

3.2 CORTICAL THICKNESS OF THE DISTAL FEMUR AND PROXIMAL TIBIA.

3.2.1 Introduction

In order to make decisions about tunnel placement and anchorage methods it was necessary to know the distribution of cortical bone at the PCL insertion sites and at potential anchorage sites.

3.2.2 Method

3.2.2.1 Tibia

Seven tibiae were stripped of superficial tissue and sawn in a plane that ran through the centre of the PCL insertion, between the tibial condyles and through the tibial tuberosity. The ligament fibres were then scraped of the bone with a scalpel. A small osteotome was then used to cut away the cancellous bone underlying the PCL insertion. The cortical thickness was then measured with vernier callipers.

3.2.2.2 Femur

Three femurs were stripped of superficial tissue and then cut in three planes which were perpendicular to the insertion of the PCL. The planes passed through the centres of the aPC and the pPC and an intermediate location (Figure 3.11). As with the tibia ligament fibres were scraped off

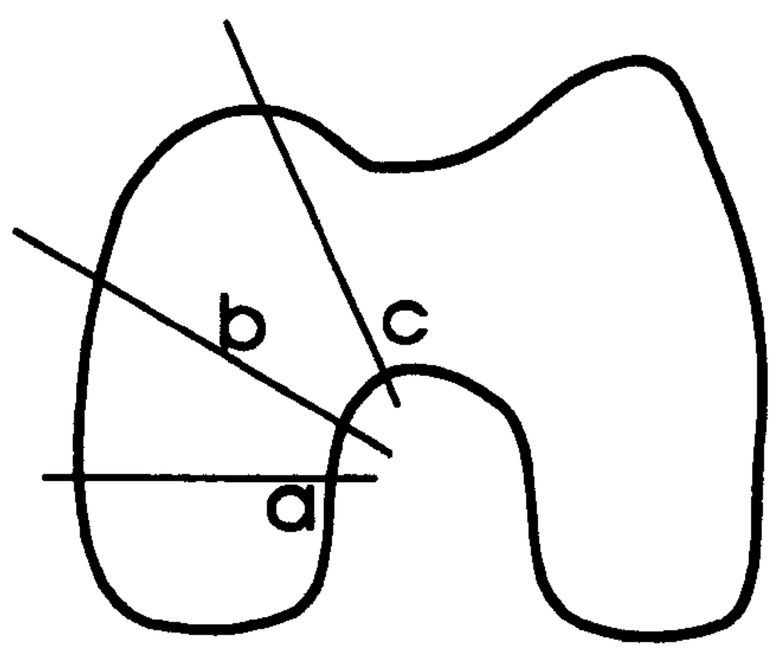


Figure 3.11 Saw–cuts made in the femur. (a) through the centre of the pPC, (c) through the centre of the aPC and (b) intermediate position.

and cancellous bone removed to allow measurement of the cortex. The thickness of the cortex at the PCL insertion was measured at the three saw–cuts. Also, the cortex of the epicondyle was measured at cut (b) in Figure 3.11 because this was a potential anchorage site for the implant.

3.2.3 Results and Discussion

3.2.3.1 Insertion Sites

The cortical thickness of the tibial insertions of the PCL are shown in Table 3.1. It was found that the cortex of the femoral insertion was significantly thicker than that of the tibial insertion. It was also qualitatively observed that the cancellous bone underlying the femoral insertion was much denser than that underlying the tibial insertion. These two factors appear to explain the clinical observation that PCL avulsion fractures almost always occur in the tibia (Barton et al, 1984).

Table 3.1 Cortical thickness, in (mm), of the insertions of the PCL.

		Mean ± s.d.
Tibial		1.3 ± 0.3
Femoral	pPC	1.5 ± 0.3
	intermediate	2.2 ± 0.1
	aPC	2.5 ± 0.3

3.2.3.2 Femoral Anchorage Site

The cortical thickness of the medial epicondyle was 0.5±0.2mm. It was noticed that the cancellous bone was very easily removed from the cortex. This was probably because the trabecula were aligned parallel to the cortex.

4. MECHANICAL PROPERTIES OF THE TWO PCL BUNDLES

4.1 INTRODUCTION[1]

A search of the literature revealed that there has been little data published on the mechanical and material properties of the PCL and that the results show much variation (Table 4.1).

In the study presented in section 3 it was found that the PCL could be consistently divided into two groups of fibres based on their reciprocal tightening and slackening near extension. Although this functional division of the PCL has been suggested before (1.1.1) the mechanical and material properties of the aPC and pPC have not been measured previously. Since these two bundles appear to have different functions (indicated by reciprocal tightening and slackening) it was hypothesised that they may have different mechanical and material properties. Therefore, the purpose of this study was to measure and compare the mechanical and material properties of the two separate PCL bundles and to clarify the mechanical properties of the whole ligament.

4.2 METHOD

Specimens from 10 donors were acquired and stored as described in section 2.1. The mean age of donor was 75 years (53-98).

4.2.1 Specimen Preparation

Each knee specimen was prepared as follows: The extracapsular tibia was stripped of all soft tissue and the fibula was removed. The tibia was then potted, as described in section 2.2.

The specimen was then clamped in a vice via the pot. Soft tissue was excised to expose the PCL, taking care not to damage the ligament itself. The lateral femoral condyle was then sawn off, taking care not to disrupt the ligament origin, to give a better view of the whole PCL. The synovial covering of the ligament was then carefully removed, by sharp

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[1] The material in this section has been published in *J. Biomec.*,v27, pp.13–23, 1994. Co-author A.A. Amis.

Table 4.1 Data for the mechanical and material properties of the posterior cruciate ligament (PCL) previously published and data from the present study for the anterolateral (aPC) bundle of the PCL.

	Trent PCL	Kennedy PCL	Marinozzi PCL	Prietto PCL	Butler fascicles	This study aPC
Donors	6	10	5	4	3	7(*10)
Age	29-55	62(20-75)	55-90	22.5 ± 3	21,30,30	75 ± 14
Length(mm)	-	-	25 ± 4	31.4 ± 2.0	31.0 ± 1.1	35.3 ± 3.4
Area(mm2)	110	-	-	62.8 ± 12.5	-	43.0 ± 11.3
Extension Rate(%/s)	≅2	≅24	≅7	100	100	≅47
Max Load(N)	739 ± 368	1051 ± 237	855 ± 225	1627 ± 491	-	*1620±500
Stiffness(N/mm)	180 ± 58	-	145 ± 66	204 ± 49	-	*347±140
Linear Strain(%)	-	18.8 ± 9.6	17 ± 4	22.1 ± 4.3	-	12.3 ± 5.7
Linear Stress(MPa)	-	-	-	-	-	28.7 ± 17.0
Modulus(MPa)	-	-	-	109 ± 50	345 ± 107	248 ± 119
Max Strain(%)	-	24.2 ± 6.3	20 ± 5	28.5 ± 9.1	15.0 ± 3.8	18.0 ± 5.3
Max Stress(MPa)	-	-	-	26.8 ± 9.1	36.4 ± 12	35.9 ± 15.2
Energy Density At Failure(J/cc)	-	-	-	-	2.53 ± 1.2	3.12 ± 1.4

Mean ± s.d.

dissection, along with any meniscofemoral ligaments to reveal the fascicular structure of the PCL. The anterolateral and posteromedial bundles were then identified and separated using blunt dissection. The bundles were always joined by synovial tissue but could be easily identified because of the differential tensions in flexion/extension (section 3.1.3.3 and Figure 4.1). The femoral origins of the two bundles were then separated using a chisel placed between the bundles, parallel to their fibres. Two femoral bone blocks, incorporating the origins of the bundles, were then cut with a hacksaw (Figure 4.2).

The specimen was then held, inverted, in a retort stand and the femoral bone blocks suspended, in turn, in small mounting pots. The bone blocks were carefully arranged so that the bundle fibres were parallel to the cylindrical axis of the pots and entering the bone in what seemed, based on descriptions of PCL function, to be a normal load bearing direction. For the aPC this was at about 100° to the femoral shaft, the equivalent of about 70° flexion, and for the pPC this was at about 15° to the femoral shaft, the equivalent of about 0° flexion. These angles were based on observation of knee joints with the lateral femoral condyle removed and were accurate to ±10°. The intention of this study was to avoid sequential failure of the ligament fibres, so the final position of the bone blocks was adjusted to give the most even fibre tension. The bone

blocks were then secured with pmma, which also embedded a crossbar that prevented slipping under axial load. The pmma was poured into the pots so that it covered the bone. After a few minutes, when it had partially set but was still soft, the pmma was pushed away from the ligament so that it did not restrict the fibres or present any sharp edges.

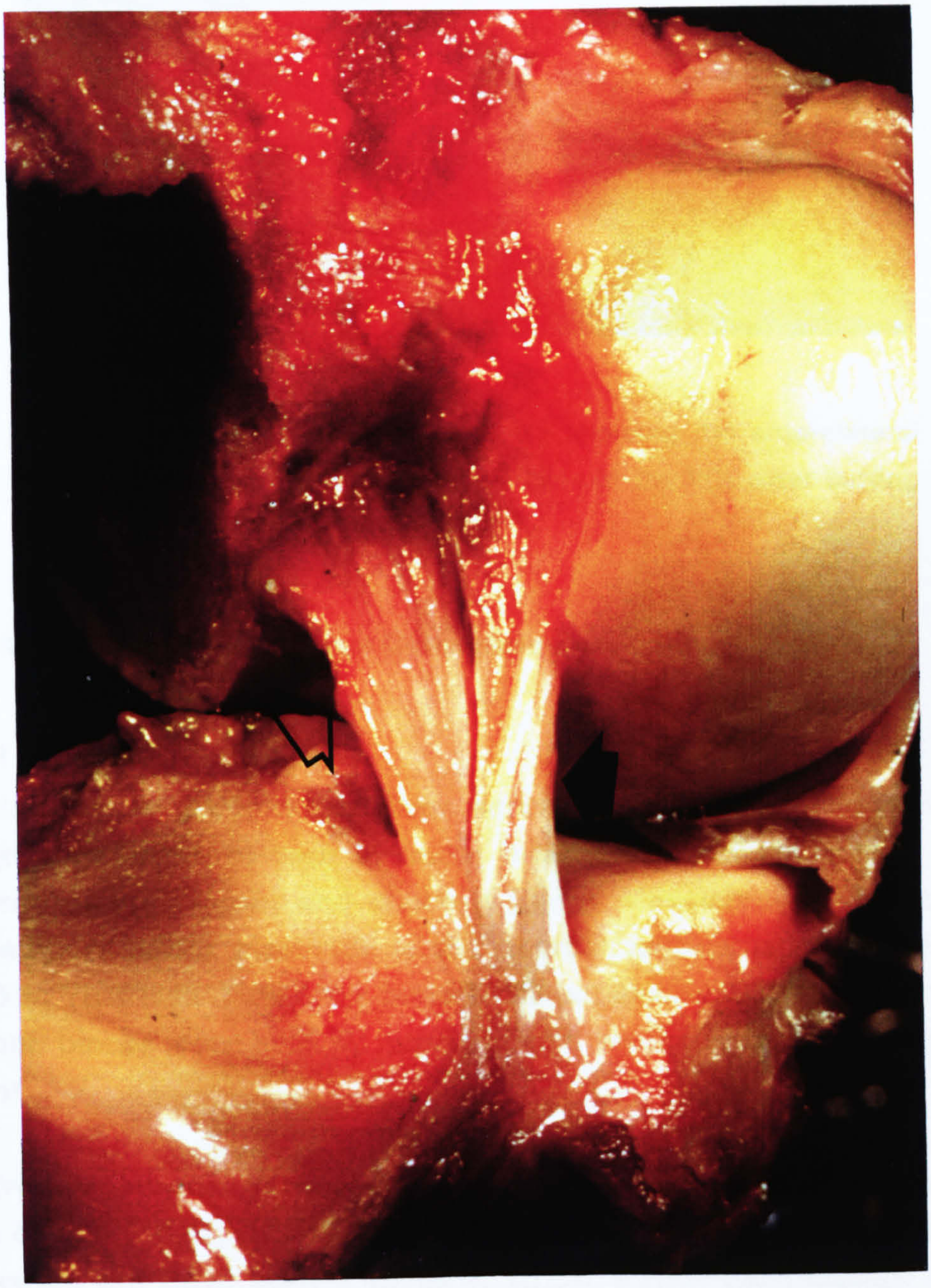


Figure 4.1. Posterior and lateral aspect of the extended knee with the lateral femoral condyle and lateral meniscus removed. Using blunt dissection the PCL has been separated into anterolateral (⇦) and posteromedial (⇦) bundles.

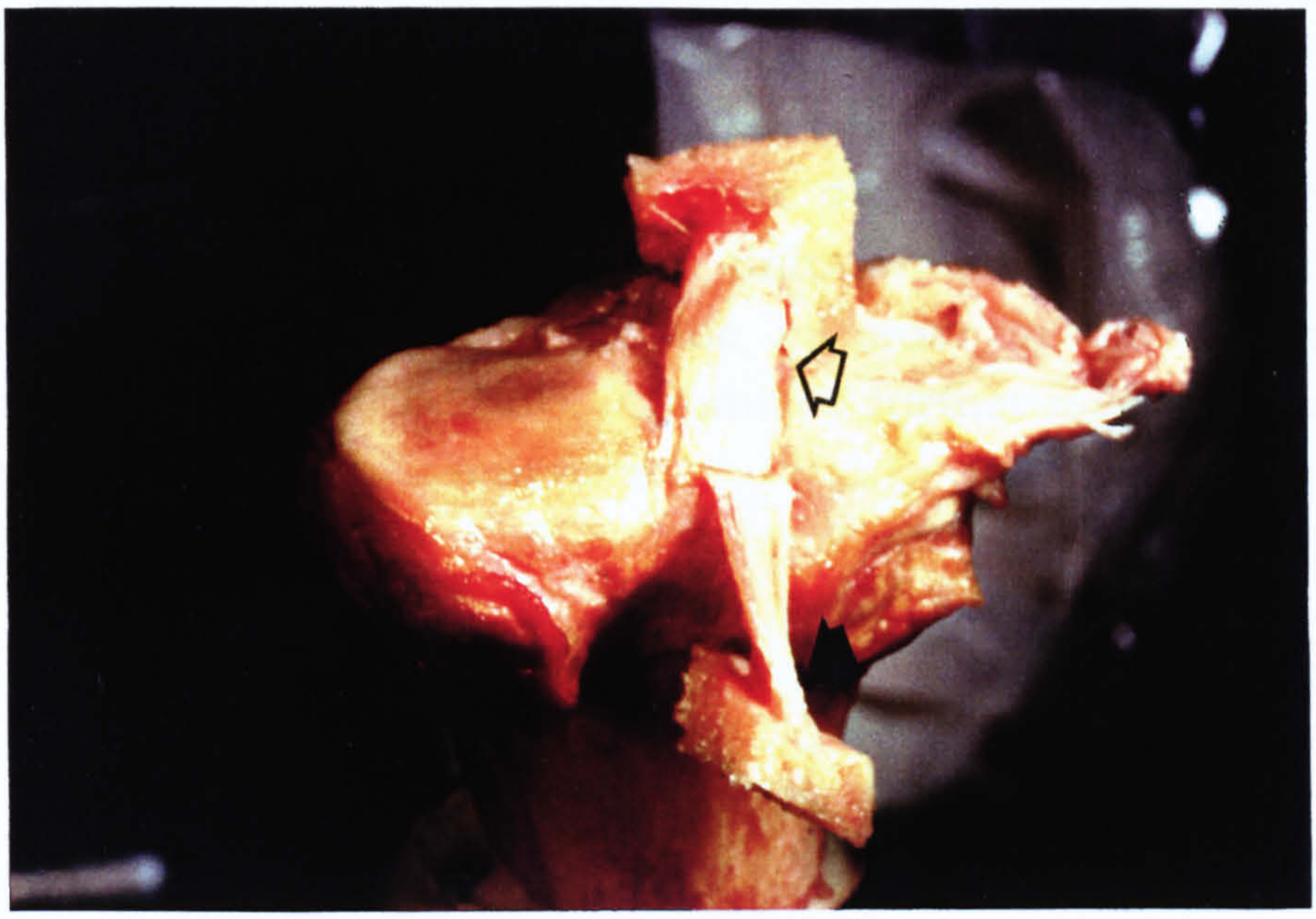


Figure 4.2 Posterior aspect of the tibia showing the anterolateral (⌂) and posteromedial (⬟) bundles of the PCL after cutting femoral bone blocks.

4.2.2 Measurement Of Physical Dimensions

4.2.2.1 Length

The dimensions of bundles from seven knees were measured. The average fibre length of each ligament bundle was determined by taking the mean of the longest and shortest fibres. Measurements were made with callipers and a steel rule to the nearest millimetre, which equates to ±1.5%. This was done after bone block separation and before potting. The bone blocks were held by hand so that the ligament fibres were just straight.

The average spread in fibre lengths was ±1.5mm, which equates to ±4.5% of rest length. Obviously, the spread of fibre lengths meant that there could not have been a uniform strain within each bundle in–vivo or in–vitro no matter how the bundles were loaded. In spite of this limitation the two bundle division was maintained: the consistent and clear difference in the gross behaviour of the bundles suggested that this level of precision would be sufficient to demonstrate some gross

differences in function, mechanical and material properties that would advance the understanding of the PCL. Greater precision in terms of applied strain would have been possible by dividing the PCL into smaller units but this would have sacrificed more fibres at the edges of bone blocks and so compromised the accurate determination of mechanical properties. The division into smaller units would also have required additional measurements to be made in order to define consistent groups of fibres: this would have introduced further inaccuracies which may have made the results less clear.

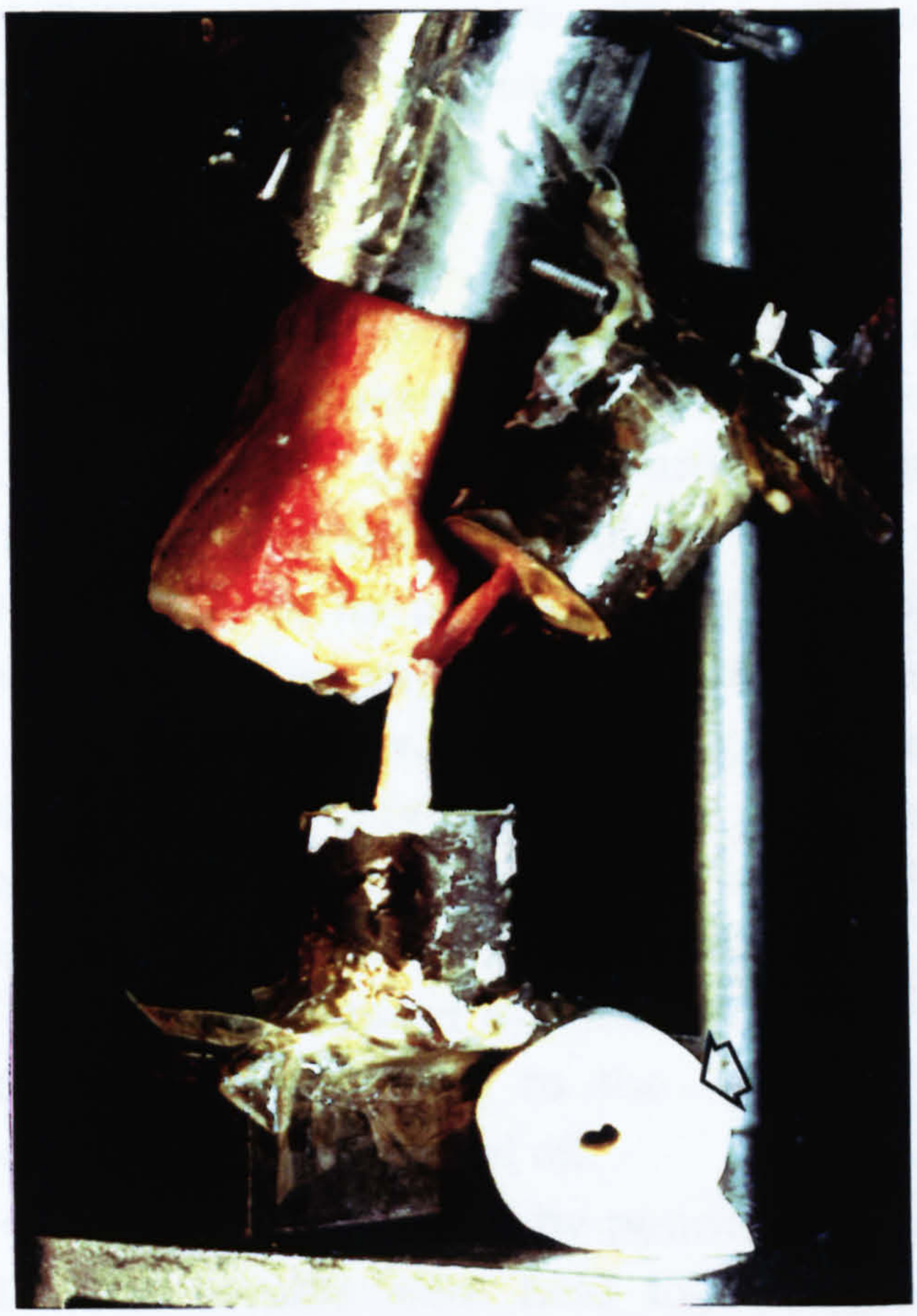


Figure 4.3 Tibia inverted with the anterolateral bundle hanging below. The silicone rubber mould (⌂) has just been removed.

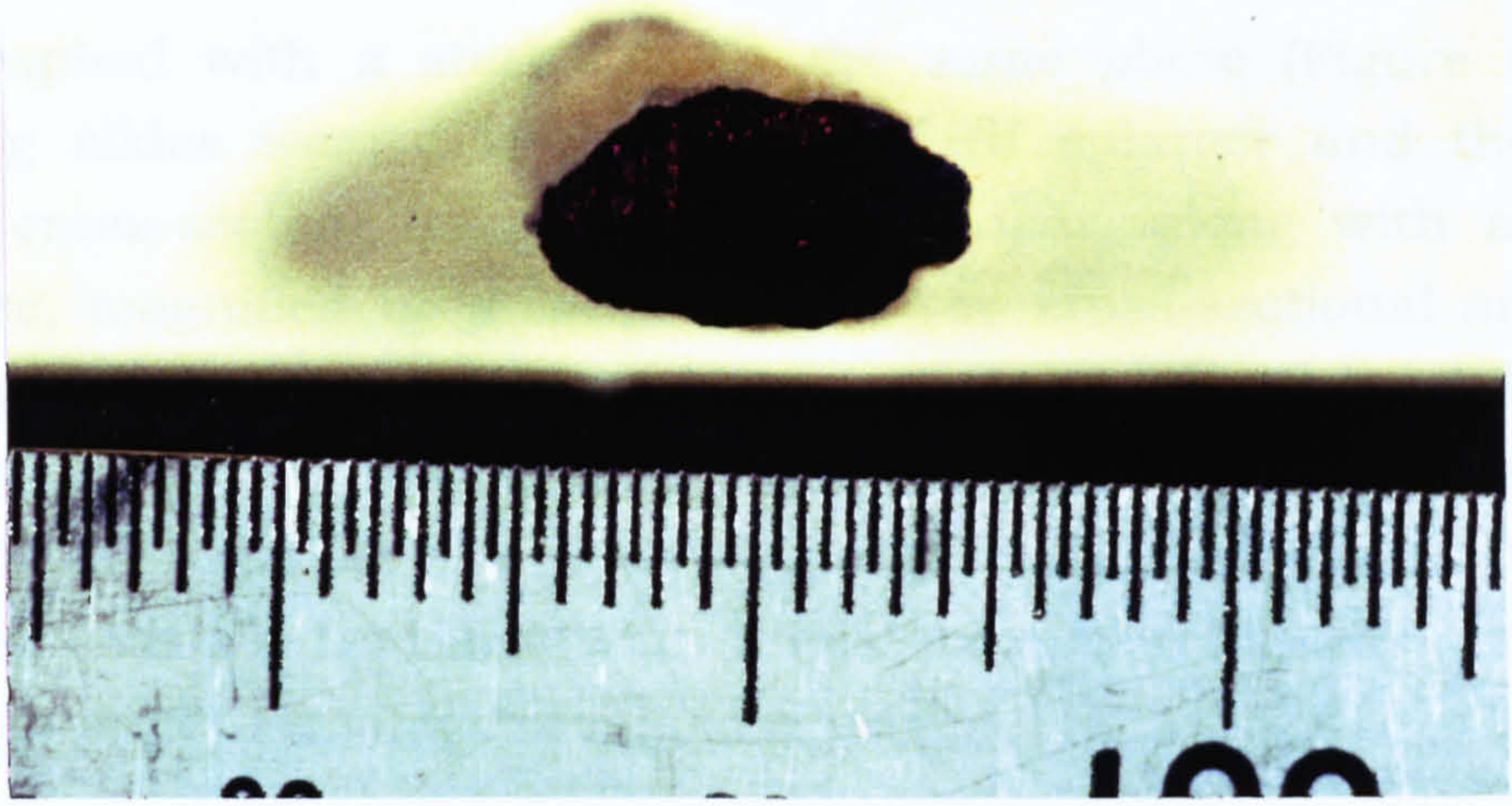


Figure 4.4 Typical section of a replica of the anterolateral bundle, with millimetre scale. Note the concavities.

4.2.2.2 Cross-sectional Area

The cross-sectional areas of the bundles were measured as follows. Moulds of the bundles were taken after the bone blocks had been set in mounting pots. A strip of thin cardboard was taped around the mounting pot to form a tube (38mm in diameter) enclosing the ligament bundle. The bone block, in its pot, was allowed to hang free with the ligament fibres parallel. This gave a 2.3N load, which was sufficient to straighten the ligament fascicles. Quick setting silicone rubber was then poured around the ligament (Figure 4.3). The rubber used was 'Silcoset 105'* with rapid curing agent. It was mixed by hand to give a setting time of 15 minutes. When the rubber had set the cardboard was removed. The mould was removed by making a single radial cut with a scalpel, carefully avoiding damage to the ligament bundle. This cut allowed the mould to be easily peeled off.

Ligament bundle replicas were made by pouring pmma into the silicone rubber moulds. The moulds were held together with adhesive tape. When the replicas had set they were removed by cutting the tape.

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* Ambersil, Bridgewater, Somerset, England.

The pmma replicas were scrubbed with disinfectant and detergent and then soaked in 'Hibiscrub' bactericidal cleansing solution. Sections 5 mm long were cut from the middle and the two faces filed perpendicular to the bundle axis. These faces were then stained with black ink and photographed with a steel rule in the same plane (Figure 4.4). The resulting slides were placed in a standard enlarger and the bundle replica cross-section traced onto graph paper, along with a 10 mm reference, magnified by a factor of ten. The cross-sectional areas were then determined by counting the enclosed squares and scaling according to the 10 mm reference. The smaller of the two areas for each replica was taken as the cross-sectional area of the bundle: careful dissection meant that ligament fibres were not removed, but some synovium might remain, causing over estimation.

The accuracy of the cross-sectional area measurement technique is evaluated in Section 2.4. The cross–sectional areas of the bundles were measured with the same fibre/bone block orientation that was to be used for the mechanical testing. It has been suggested that the cross–sectional area of the cruciates may change with flexion angle (Zavatsky and O'Connor, 1992a). If the cruciates were made up of a range of discrete fibres of zero thickness then the area 'occupied' by the ligament would, obviously, reduce in cross–sectional area when flexion caused the fibres to become more nearly parallel to the planes of their insertion. The 'cross–sectional area occupied' would be maximal when the fibres were most perpendicular to their planes of insertion and minimal when the fibres were most parallel to their planes of insertion. In actual cruciate ligaments the collagen fibres have some thickness and exhibit some branching, there also exists interfibre ground substance. For the cross–sectional area of a real ligament to change in the same way as a zero thickness fibre model would require the flow of ground substance into and out of the cruciates and the compression of collagen fibres. This may, or may not occur, the role of ground substance is not yet well understood. The aim of this study was to characterise the composite material properties of the PCL bundles during normal function. Therefore, the whole cross–sectional area was considered rather than just the area, or number, of collagen fibres and the cross–sectional areas were measured for the flexion angles where the bundles appeared to be tightest and were assumed to be functional. These angles, corresponding

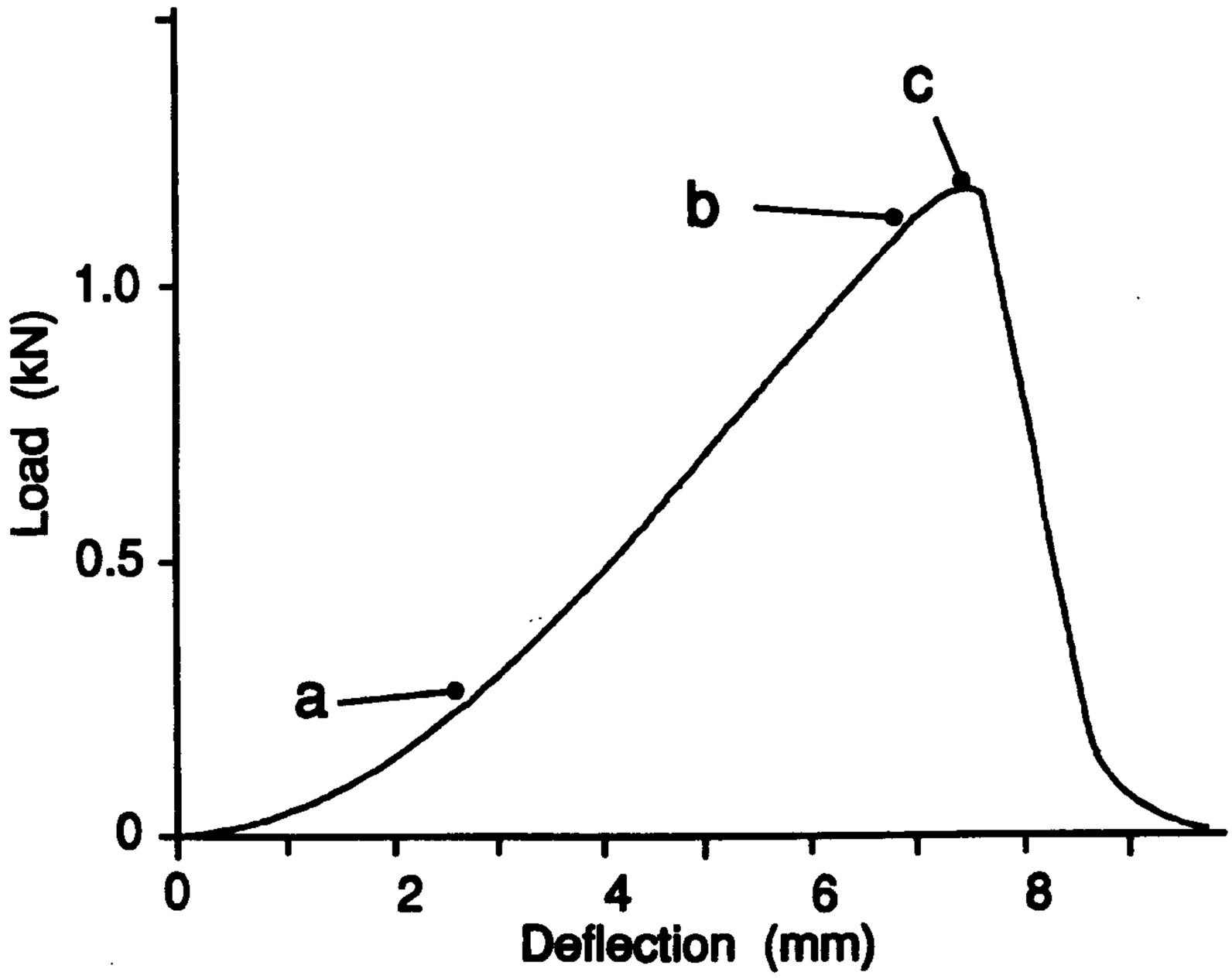


Figure 4.5 Typical load/deflection graph for the aPC: (a) beginning of linear region, (b) yield point, (c) max load.

to flexion angles of 0° for the pPC and 70° for the aPC were also used when the bundles were set up in the Instron for mechanical testing.

4.2.3 Tensile Testing

The pPC and aPC bundles were distracted to failure in an Instron 1122 materials testing machine with a 5kN load cell. The tibia and femoral bone block (in their pots) were orientated so that the load acted along the axis of the ligament bundle. The bundles were tested in neutral rotation, the tibia was angled so that the pPC was tested at an angle of about 75° to the tibial plateau, the equivalent of 0° flexion and the aPC was tested at an angle of about 60° to the tibial plateau at 70° flexion: these were the same flexion angle equivalents used when potting the femoral bone blocks (4.2.1). Again, small adjustments were made to ensure even fibre loading. The smaller pPC was ruptured first; this did not appear to compromise the tibial attachment of the aPC. A crosshead speed of 1000mm/min was used to give a high strain rate (approximately 50%/sec). This ensured that ligament failure occurred rather than bony avulsion (Noyes et al, 1974). The load/crosshead-displacement data was captured on a Nicolet digital oscilloscope and

then output to an X-Y plotter. The mechanical and material properties were then calculated from these graphs and the physical dimensions of the bundles.

4.3 RESULTS

A typical graph of load/deflection is shown in Figure 4.5. The properties of the two bundles of the PCL measured in this study are shown in Table 4.2 along with p values for paired Student's t-tests. The aPC had four

Table 4.2 Mechanical and material properties of the two bundles of the PCL. 'p' values are for the significance level of differences between the anterolateral (aPC) and posteromedial (pPC) bundles in a paired t test

	pPC	aPC	p (Paired t)
LENGTH(mm)	33.8 ± 3.2	35.3 ± 3.4	*>0.1*
AREA(mm2)	10.0 ± 1.3	43.0 ± 11.3	<0.001
MAX LOAD(N)	258 ± 83	1620 ± 500	<0.001
STIFFNESS(N/mm)	77 ± 32	347 ± 140	<0.001
LINEAR STRAIN(%)	12.9 ± 2.9	12.3 ± 5.7	*>0.1*
LINEAR STRESS(MPa)	18.7 ± 10.3	28.7 ± 17.0	*0.086*
MODULUS(MPa)	145 ± 69	248 ± 119	0.011
MAX STRAIN(%)	19.5 ± 5.4	18.0 ± 5.3	*>0.1*
MAX STRESS(MPa)	24.4 ± 10.0	35.9 ± 15.2	0.027
ENERGY DENSITY AT FAILURE(J/cc)	2.06 ± 0.73	3.12 ± 1.4	0.045

Mean ± s.d.

times the cross-sectional area of the pPC. As would therefore be expected the mechanical properties of the bundles were different: the maximum load and stiffness of the aPC bundle were much higher than those of the pPC. The maximum strain for both bundles was the same but the maximum stress, modulus and energy density were significantly higher for the aPC, therefore the material properties of the two bundles were significantly different ($p<0.05$). The large standard deviation in the stress values was predominantly due to one exceptionally strong knee from a 53 year old donor. This knee had a failure stress double that of the rest of the sample. This may have reflected an unusually high activity level.

4.4 DISCUSSION

4.4.1 Sources Of Error

In this study the strain data was based on the grip to grip displacement of the Instron. This data was subject to systematic errors: the compliance of the clamping system, of the tibia and of the bone-ligament junction.

The stiffness of the clamping system plus tibia was tested by applying a load, via a wire attached to the PCL insertion, in the same manner as for the aPC test. The deflection of the tibia, relative to the Instron base plate, was measured with a dial gauge accurate to 0.01mm. The stiffness of the clamp/tibia system was found to be 3.4kN/mm. This figure was used as a correction factor in calculating the data in Table 4.2. The compliance of the bone-ligament interface was harder to establish. Some workers have suggested that video dimensional analysis (VDA) is the answer (Woo, 1982), However, this technique was rejected by the author as it did not promise significantly better results — VDA is limited to surface strain measurement and since the bundles had a spread of fibre lengths of up to 3mm it was not considered that the surface strain was necessarily representative of the whole bundle. Also, Butler et al (1990) have shown that surface strain varies along a fascicle. VDA is also subject to error due to progressive fibre failure blurring the reference lines. The author considered that the grip to grip displacement gave a more representative estimate of the bone to bone extension of the ligament bundles as a whole. (This method was also more convenient than accurately mounting a clip gauge on the bone attachments within the small pots).

The extension signal from the Instron was accurate to 0.01mm; which gave an average error, for the extensions measured, of ±0.2%. The graphs of load/extension were read to an accuracy of 1%. The error in average rest length measurement was ±1.5%. Thus, the overall error in average strain was ±2.7% of the given value.

Since there was a spread of fibre lengths the strain for fibres at the extremities was within ±4.5% of the average. This spread in fibre lengths must have caused some sequential failure. However, since no 'steps'

were observed in the load/extension graphs the effect must have been small (Figure 4.5).

The accuracy of calculated stress was dependent on the error in measured load and cross-sectional area. The error in the Instron load signal was ±0.5% and the plotted graphs were read to ±1%, therefore the error in measured load was ±1.5%. The error in cross–sectional area measurement was ±1.8% (based on repeated tests on silver steel specimens of known dimensions, Section 2.4.3.1). This is comparable to the accuracy reported for the laser micrometer (Lee and Woo, 1988). However, a laser micrometer could not be used on the pPC because of obvious concavities. Even in the 'rounder' aPC the present study indicates that assuming convexity would result in overestimations of areas of between 3% and 8% (Figure 4.4). By comparison, the area micrometer of Butler et al (1986) deliberately applied a constant pressure of 0.2Mpa, which was needed to force the specimen to conform to a rectangular aperture in the apparatus. The author estimated that this load regime caused the area micrometer to give areas approximately 16% lower than those found using the replica technique in this study (Section 2.4.3.2).

The accurate determination of stress is also dependent, as is the maximum load, on the even loading of ligament fibres. Uneven fibre loading causes progressive failure and therefore underestimation of maximum load and stress. As stated earlier this type of error would have occurred if the whole ligament had been tested. However, since the two bundles were tested separately and care was taken to align the bone blocks, the fibres across each bundle were loaded as near simultaneously as possible, given the spread of fibre lengths, and progressive tearing was limited.

4.4.2 Age Effects

Noyes and Grood (1976) and Hollis et al (1988) have shown that there is a correlation between ACL strength and age. Both studies show that the mean strength of the ACL in the younger population (under 26 years) is greater, by a factor of about 2.5, than the mean strength in the older population (over 50 years). The cause of the above correlation is not known, therefore comparisons with the present study based on age

cannot be definitive. The validity of comparisons is dependant on the probability that the variable/s responsible for the changes found in the studies of Noyes and Grood (1976) and Hollis et al (1988) show the same correlation with age in all the compared samples. Given the above caveat, the data in this study, which is of an older sample (53-98 years), suggests that the mean strength of the PCL in young adults may be 1.6*2.5=4kN. Noyes and Grood (1976) also showed that between the younger and older populations the maximum stress decreased by a factor of 2.8; the modulus decreased by a factor of about 1.7; and the strain energy density decreased by a factor of about 3.3. Noyes and Grood suggest that the factors for maximum stress and strain energy are overestimates because the older ligaments failed prematurely by bony avulsion but that the ageing effects were still significant.

4.4.3 Comparison With Work Previously Published

A summary of previously published data is presented in Table 4.1 (p107), along with data from this study for the aPC. Given the age effects outlined above, this study would be expected to give mechanical and material properties inferior to those using younger donors and equal to those using older donors. Table 4.1 shows this not to be the case, therefore either the age effects are not valid or previous studies have underestimated PCL strength and material properties. Trent et al (1976) reported premature failures by bony avulsion, therefore the figure given for maximum load must be an underestimation. Kennedy et al (1976) stated that there was no correlation between age and ligament strength. However, the very low strength reported (compared to this study) suggests that the failure mechanism was dominated by the effects of clamping the soft tissue directly, which would have masked any correlation with age. Marinozzi et al (1983) and Prietto et al (1988) tested the whole PCL, therefore these studies' figures for maximum load are likely to be underestimates due to uneven fibre loading. The low modulus reported by Prietto et al (1988) is probably also due to overestimation of cross-sectional area which was determined with callipers (they did not report the assumed geometry so the error cannot be quantified). Butler et al (1986) used an area micrometer to measure cross-sectional area, therefore that study's figures for modulus and maximum stress are likely to be an overestimation due to underestimation of cross-sectional area because of ligament deformation in the micrometer.

4.4.4 Clinical Relevance

The results of this study enable a more informed decision to be made about the strength requirements of PCL reconstructions. This study indicates that PCL reconstructions for young active people should be designed to match a natural structure with a strength of 4kN. The results also enable a more accurate definition of the material properties of the anterior and posterior parts of the PCL in mathematical models which rely on published data. The data from this study was used in the mathematical knee model of Zavatsky and O'Connor (1992b).

It has been shown that the two bundles have different mechanical and material properties. The aPC is approximately six times as strong as the pPC. Therefore, I conclude that it is the aPC (which resists posterior drawer in flexion, Section 1.2) which is primarily responsible for the stabilising effect of the PCL. This strength difference and the superior material properties of the aPC suggest the hypothesis that the aPC is loaded either more frequently or to a higher stress than the pPC during normal activity, but data on this is not available.

Since the two PCL bundles have reciprocal actions it is impossible to reproduce the action of the entire PCL with a single strand of material. To reproduce the natural PCL a double bundle, aPC and pPC, reconstruction is required. However, since the pPC has been shown in this study to contribute only one quarter of the bulk and one sixth of the strength of the aPC the surgical complexity of a double bundle reconstruction may not be justified. Further work is required to establish the functions of the two bundles before a reconstruction system can be designed with confidence.

5. FIBRE LENGTH CHANGES

5.1 INTRODUCTION

In the study described in section 4 it was shown that the two bundles of the PCL have different mechanical and material properties. This finding, combined with the qualitative observation that there were reciprocal tensions in the aPC and pPC near extension (section 3.1.3.3), led to the hypothesis that the aPC was loaded more frequently or to a higher stress than the pPC (section 4.4.4). The purpose of this study was to quantify the strain pattern,of the two bundles of the PCL over the range of flexion as an aid to the understanding of PCL function.

Previous studies of the length changes of the two bundles have not been conclusive (section 1.2.6). All previous measurements have been based on the separation of points on the tibia and femur which represented pseudo–fibre insertion sites. These measurements assumed that the ligament pseudo–fibres remained straight and taut. Since the aPC is clearly not straight in extension (Figure 3.7) such length change data must be inaccurate. It has been shown that the linear strain of the aPC was 13±6% (section 4.3) which for the average aPC represents a length change of 4.7mm. Assuming that non–destructive loads were used, any measured length change in excess of 5mm must have included some bone–bone length change when the real aPC fibres were slack. For passive flexion-extension it is unlikely that any real fibre would be close to maximum linear strain so it is probable that measured length changes of more than 2mm or 3mm represented a slack ligament fibre.

It was thought that greater accuracy might be achieved by using a minimally loaded flexible suture, connected to an LVDT, which could follow the path of a real fibre.

5.2 METHOD

This study used a method similar to that of Amis and Dawkins (1991) in their analysis of the ACL. Knee joints were obtained at post-mortem as described in section 2.1. The femur was stripped of muscle and fat

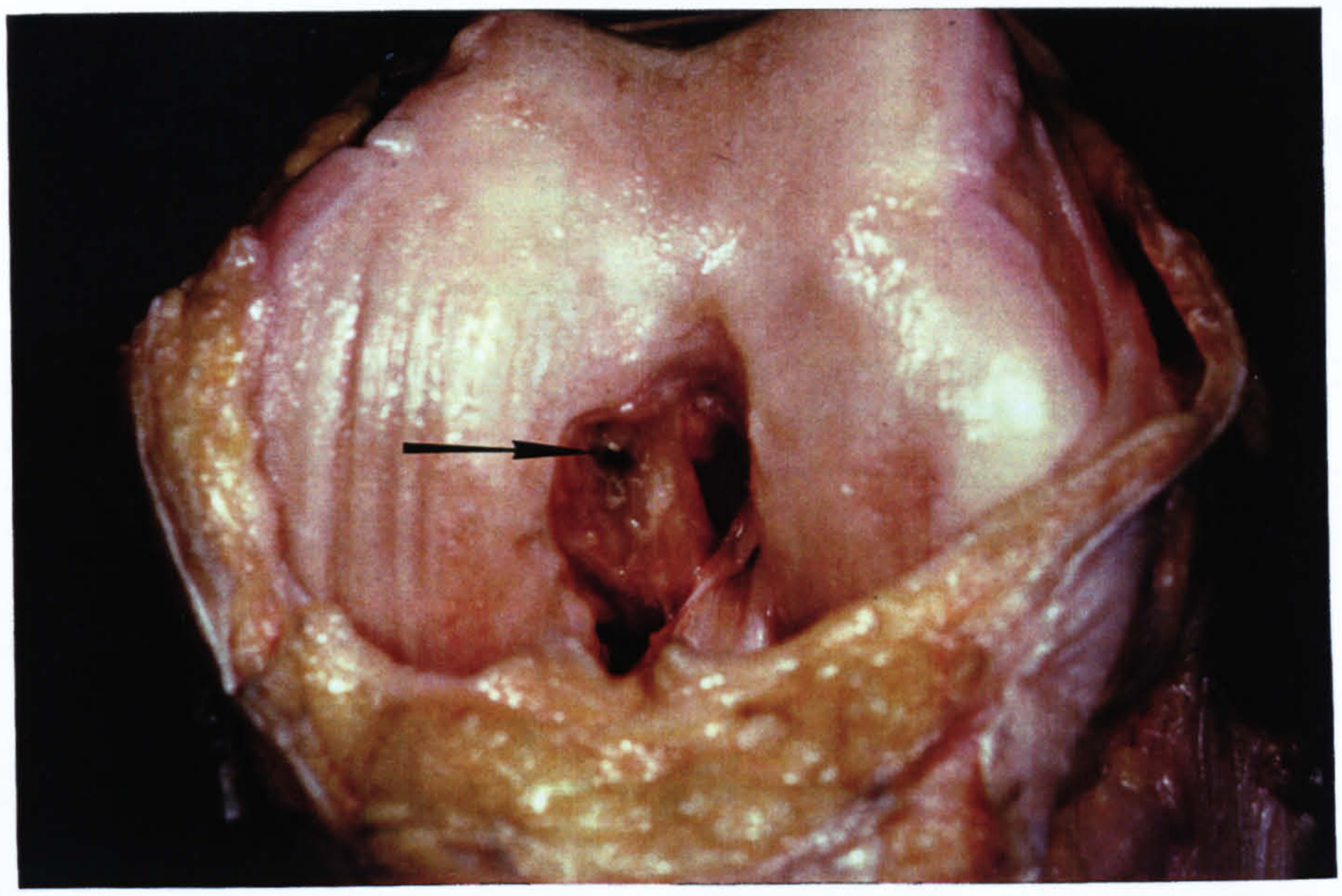


Figure 5.1 Anterior aspect of the flexed knee showing the metal pin (⟶) anchoring a suture implanted in the central region of the aPC.

tissue, taking care to preserve the ligaments and joint capsule. The femur was then held in a vice while the rest of the joint was worked on.

Since the foot and ankle were not present in the specimens used in this study the lateral collateral ligament was compromised: in an intact knee the fibula is distally stabilised by its attachments to the talus, calcaneus and tibia and by the interosseous membrane. In order to stabilise the fibula, and therefore restore the functionality of the lateral collateral ligament, the fibula was fixed to the tibia by two transcortical bone screws. Before stripping the tibia and fibula of tissue the separation of the two bones allowed by the intact interosseous membrane was measured. Non–ligamentous tissue was then removed to allow screws to be placed 10mm and 25mm distal to the edge of the superior tibiofibular joint. These bone screws were adjusted so that the separation of the fibula and tibia was within ±1mm of that allowed by the intact interosseous membrane. After the fibula had been fixed the tibia was potted, as described in section 2.2.

An intra–medullary rod was then hammered into the femur to give a flexion angle reference. Flexion angle was measured by a high quality potentiometer and plumb–line clamped to the intra–medullary rod

(Figure 5.3). The potentiometer had an active range of 270°, so care was required to mount it so that it would not travel out of its active range. It was thought that the plumb–line system would cause some noise by acting as a pendulum but it was used in preference to a goniometer as it did not restrict joint motion.

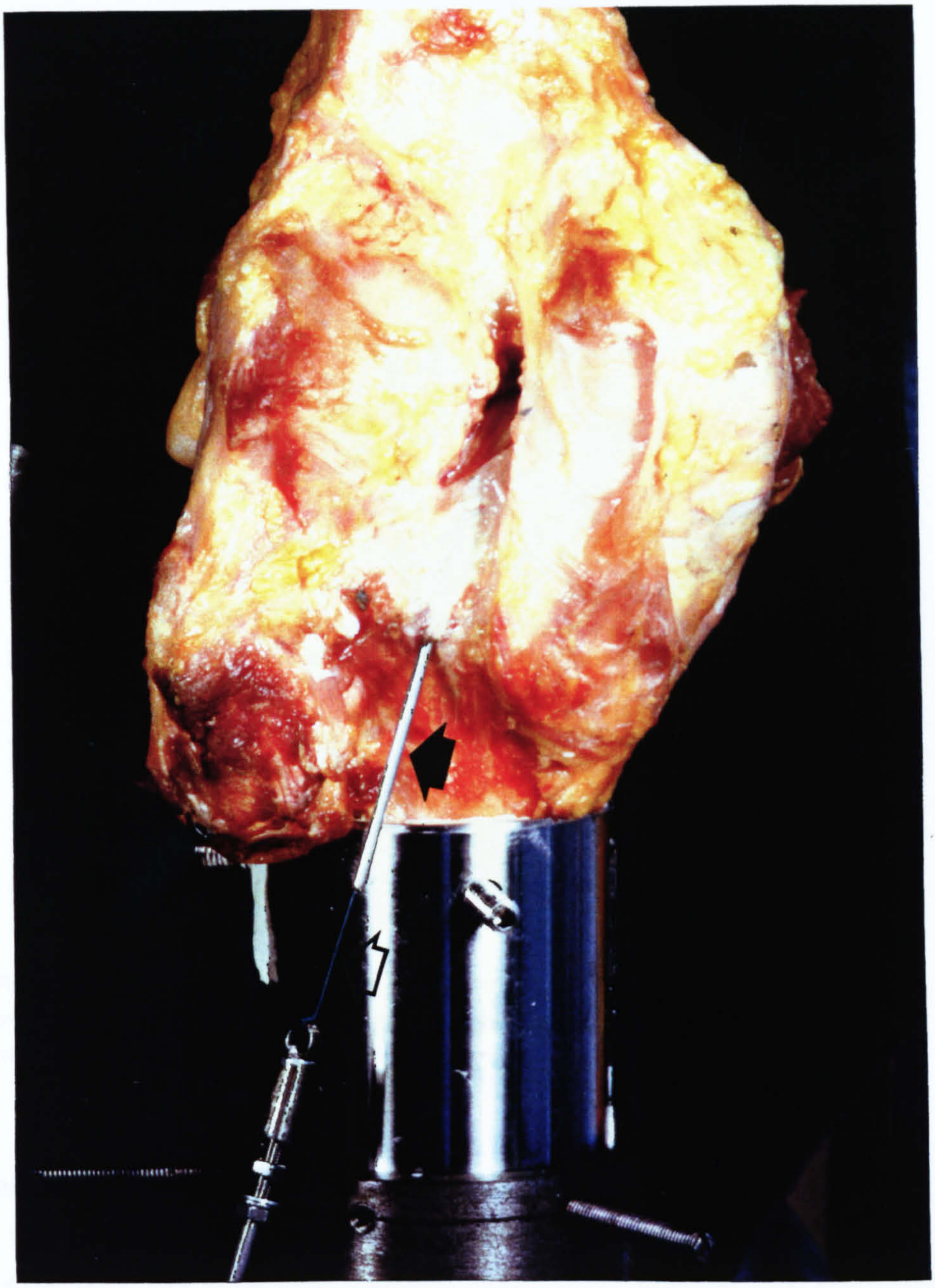


Figure 5.2 Posterior aspect of the extended knee showing the suture (⌓) passing through the tibial tube (⬟) to the LVDT.

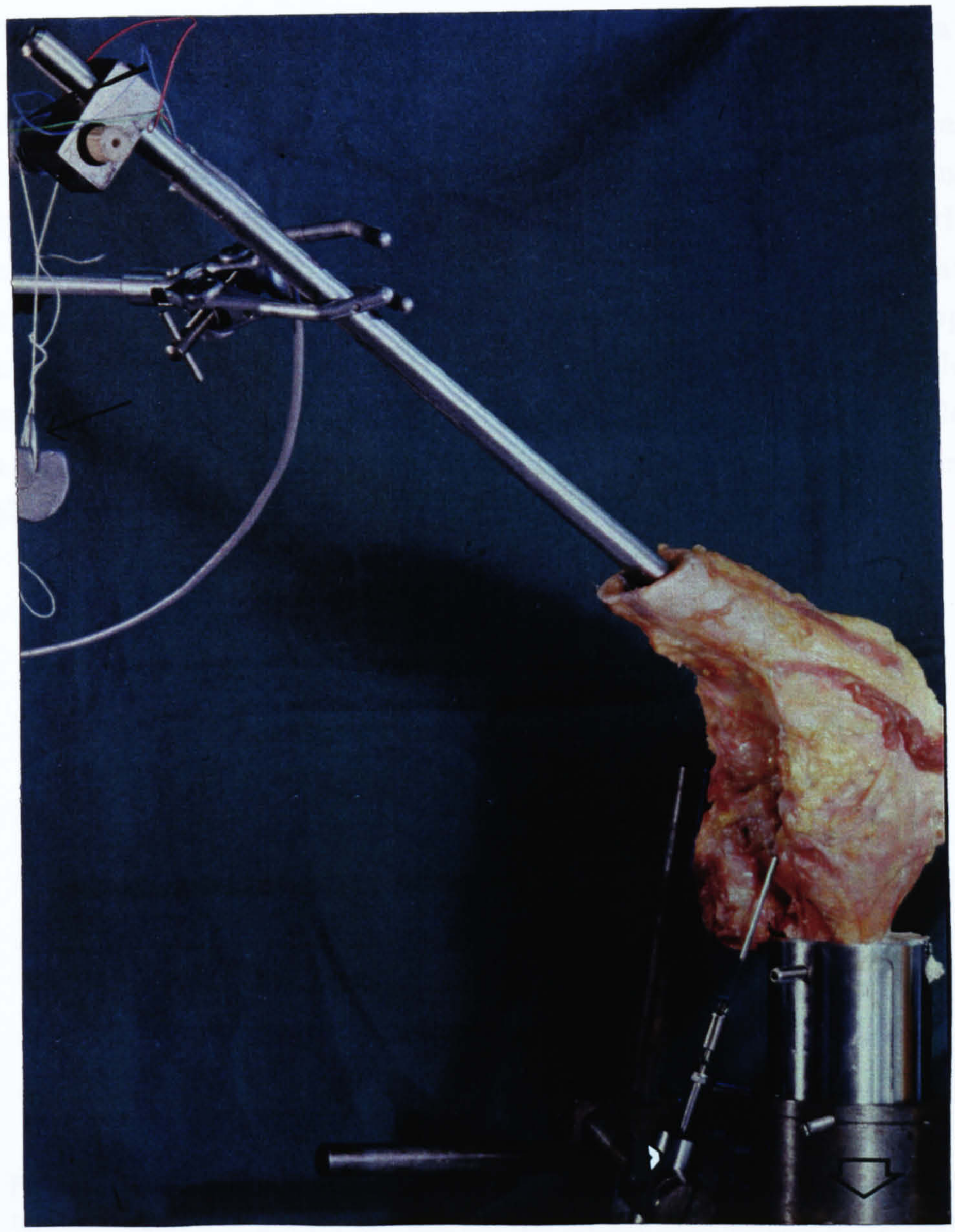


Figure 5.3 Flexion-extension rig. The tibia and LVDT (⌓) were mounted on a vertical axis bearing (⌓). Flexion angle was measured by the potentiometer (⟶) and plumb–line (⟶).

In order to get a clear view of the femoral insertion of the PCL the patella was removed. It was not thought that this would affect PCL fibre length changes because the patella has been shown not to affect A-P drawer significantly (Amis, 1989a). The posterior capsule was opened by a sagittal cut between the femoral condyles. The synovial covering of the PCL was then carefully cut away. Blunt dissection was then used to separate the fascicles of the PCL to allow a suture to be introduced. The blunt dissection was continued up to the ligament insertions. It was not

possible to use staples to fix the suture into the femoral insertion of the PCL because the cortex was too thick (section 3.2.3.1). So, a 2mm diameter hole was drilled from the PCL's femoral insertion out to the medial epicondyle. Then the periosteum was scraped away from around the hole in the epicondyle. A wire loop was then passed through the hole into the knee joint. The fascicles around the hole were held apart with a blunt dissector to allow the wire loop through. The measuring suture was then pulled through the condyle with the wire loop. The suture was then knotted at the epicondyle, pulled tight and held with cyanoacrylate adhesive. A short pin was then forced into the hole from within the joint to prevent the suture from moving from side to side in the hole (Figure 5.1).

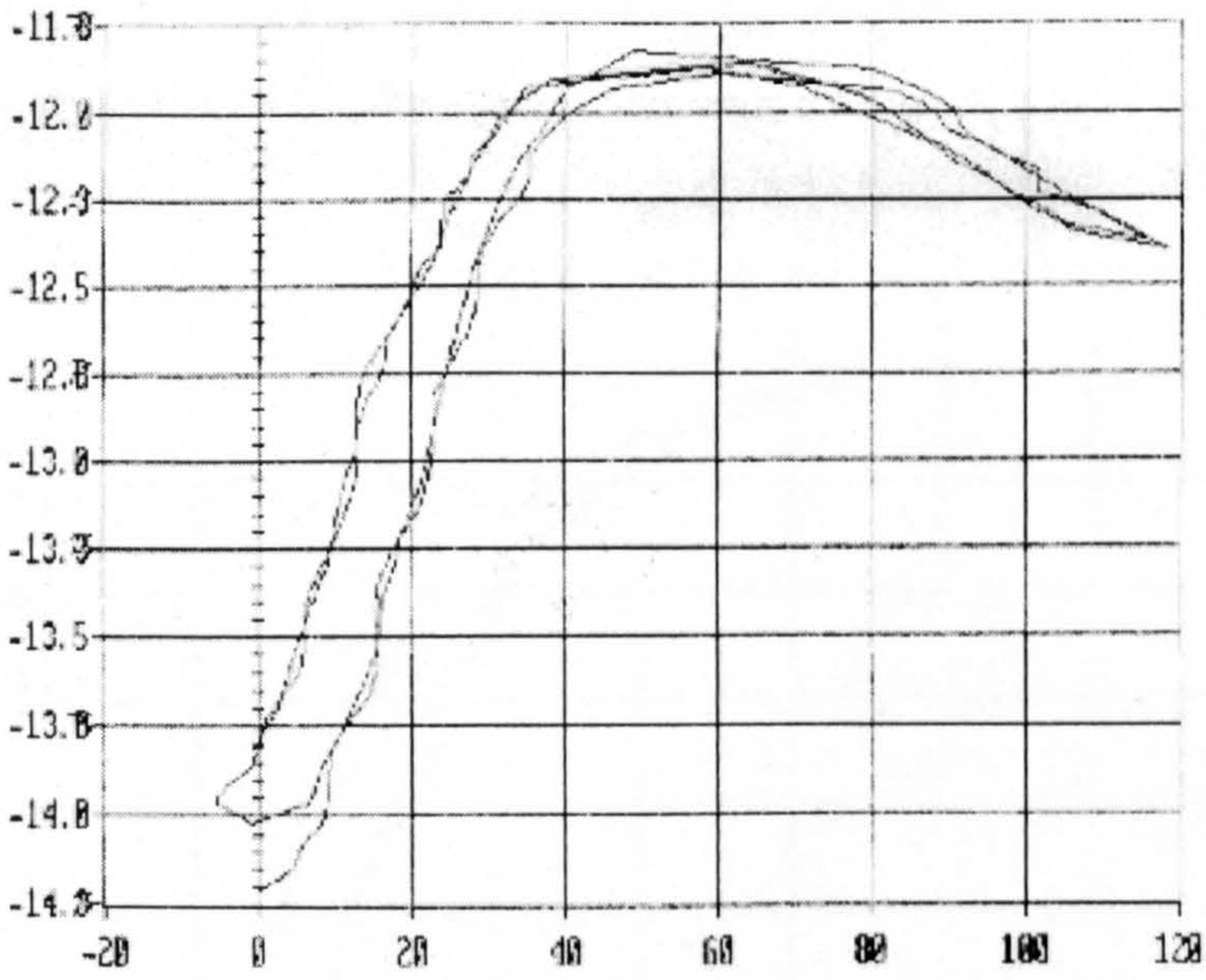


Figure 5.4 Specimen (A). Graph of length (mm, arbitrary zero) against flexion angle (°) for the central fibre of the posterior surface of the aPC during passive flexion-extension.

The desired tibial location for the suture was identified and a drill guide tip introduced. A 2mm diameter hole was then drilled to the chosen tibial location aligned with what was visually estimated to be the mean direction, over the range of flexion, of the fibres adjacent to the suture. A brass tube was then passed through the hole so that its end was flush with the cortex at the tibial insertion of the PCL. The loop of wire was then pushed up through this tube and used to pull the suture through the tibia. For the pPC a drill hole was not required; the suture was laid along the fibre path over the posterior aspect of the tibial intercondylar fovea.

The knee was then mounted in the flexion-extension rig (Figure 5.3). The tibia was clamped vertically to a spindle that was mounted in opposed tapered roller bearings. The assumed axis of internal–external rotation was coincident with the axis of the spindle to ensure that tibial rotation was unrestricted. The tibial clamp was fitted with an adjustable holder for the LVDT(stroke 30mm). This holder allowed the LVDT to be aligned with the brass tube in the tibia so that the suture could move freely.

Then, with the knee in extension, the suture was attached to the LVDT so that the core was at the midpoint of its travel (Figure 5.2). The only load imposed on the suture was the weight of the core which resulted in an estimated tension of 0.1N.

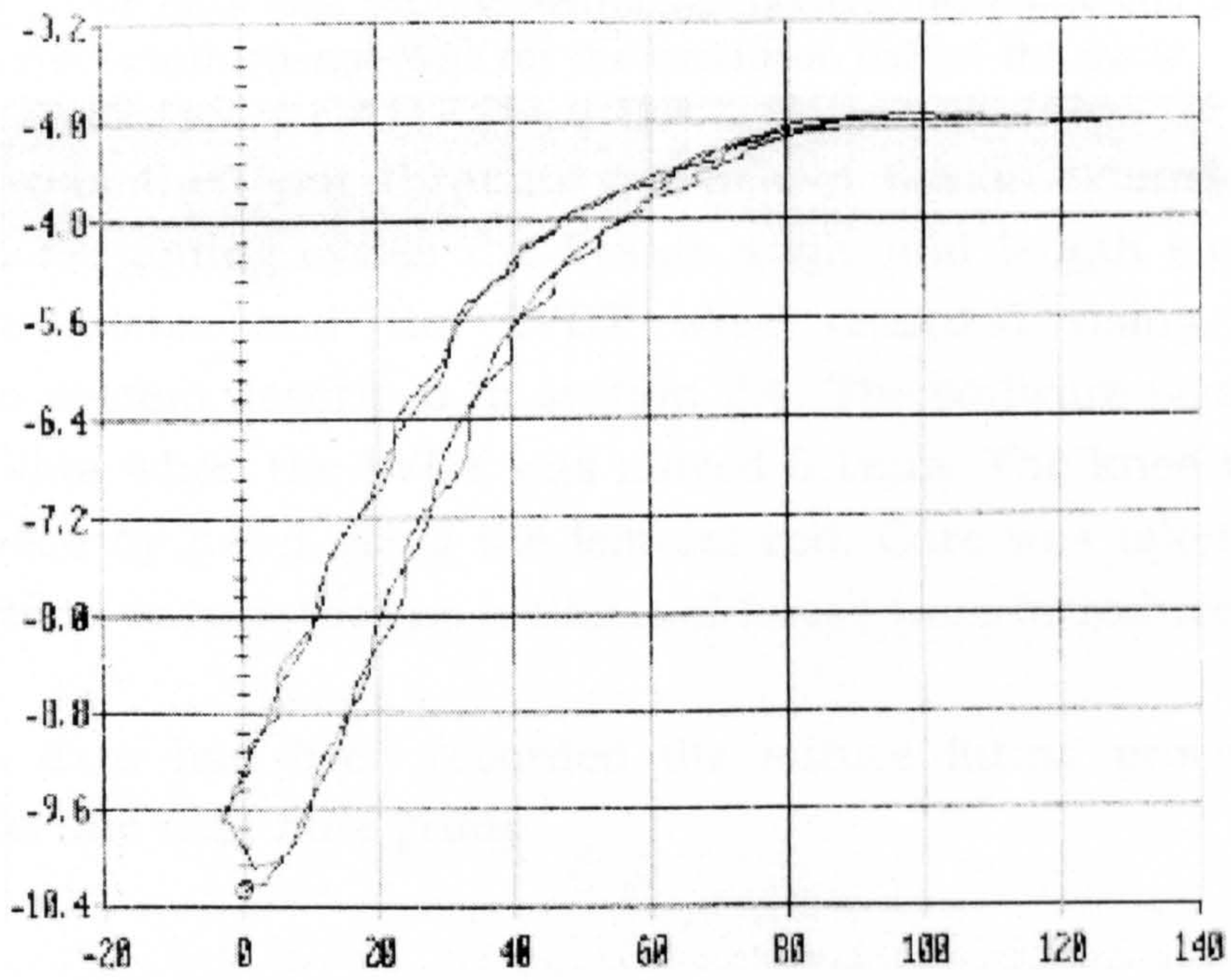


Figure 5.5 Specimen (A). Graph of length (mm, arbitrary zero) against flexion angle (°) for the central fibre of the aPC during passive flexion-extension.

The knee was held in extension and zero readings were taken from the LVDT and the potentiometer. The LVDT and the potentiometer were calibrated before each experiment using a micrometer spindle and a protractor respectively. Extension was defined by aligning the femoral rod to the vertical in a frontal plane, using the plumb–line as a reference.

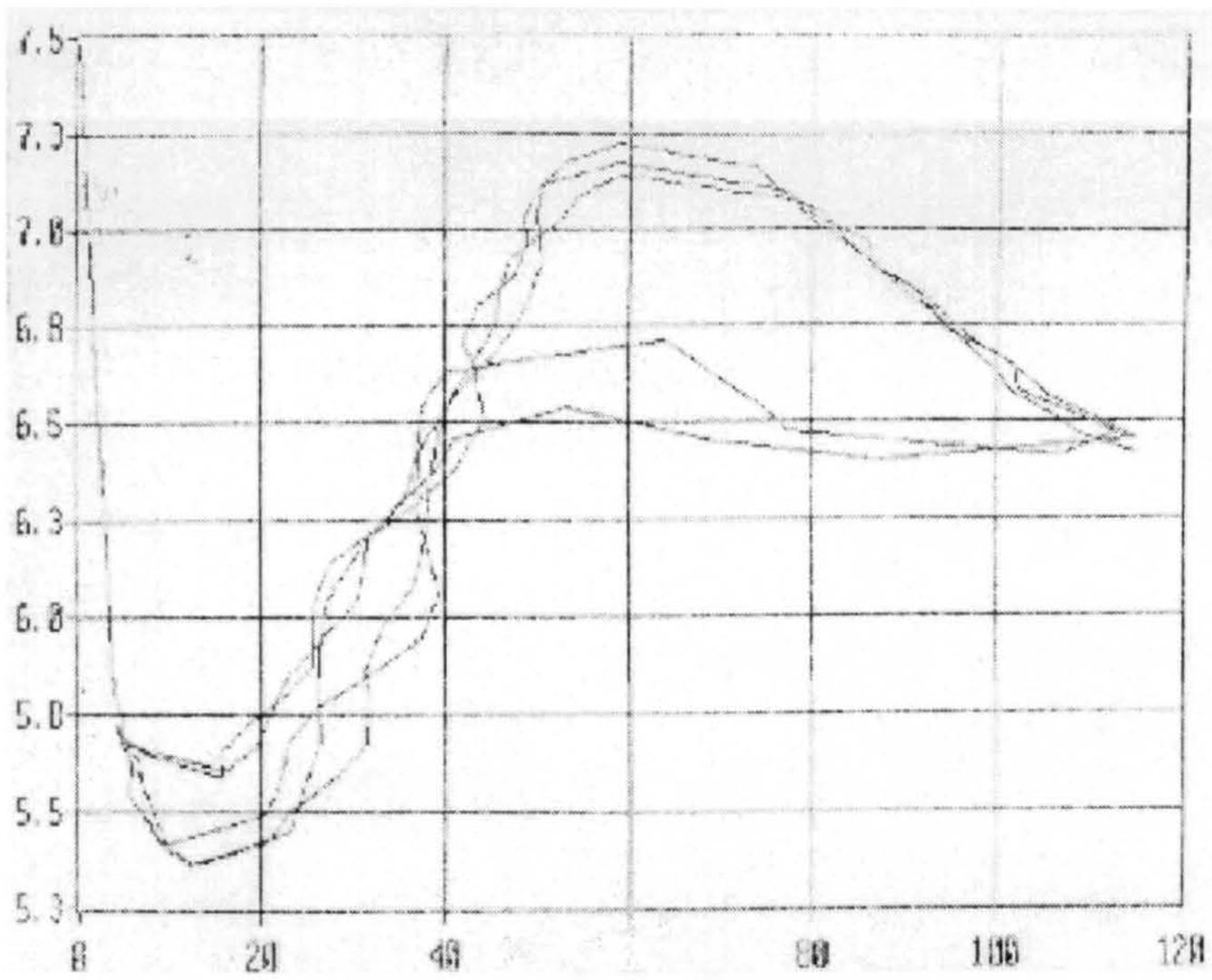


Figure 5.6 Specimen (B), graph of length(mm, arbitrary zero) against flexion angle(°) for the middle fibre of the posterior surface of the aPC. This data was for unconstrained passive flexion-extension. The greater length change was for the extension half of the cycle.

The knee was then put through a series of flexion-extension cycles. After 10 conditioning cycles the flexion angle and length signals from the potentiometer and the LVDT were recorded using the data acquisition system described in section 2.4. The software was designed to record data when the LVDT was moved 0.1mm. The knee was flexed and extended by hand using the femoral rod. Care was taken to use a 'light touch' to ensure that no artifactual forces were introduced.

When the data had been recorded the suture fitting procedure was repeated for the next fibre group.

5.3 RESULTS AND DISCUSSION

The first fibre to be examined was that which ran along the middle of the posterior surface of the aPC. Although there was noise due to the plumb–line swinging, as expected, reproducible length change data was recorded (Figure 5.4). The total length change for this fibre was 2.1mm. In specimen (A) the central fibre of the aPC was also measured. Unlike the posterior central fibre of the aPC, which was of maximum length at 50°–60°, the central fibre of the aPC was at maximum length in full

flexion (Figure 5.5). The total length change for the central fibre of the aPC was 6mm.

In specimen (B) the fibre that ran along the middle of the posterior surface of the aPC was again measured first. The total length change was 1.8mm. There was a large difference in behaviour between flexion and extension (Figure 5.6). It appeared that this was due to laxity in the

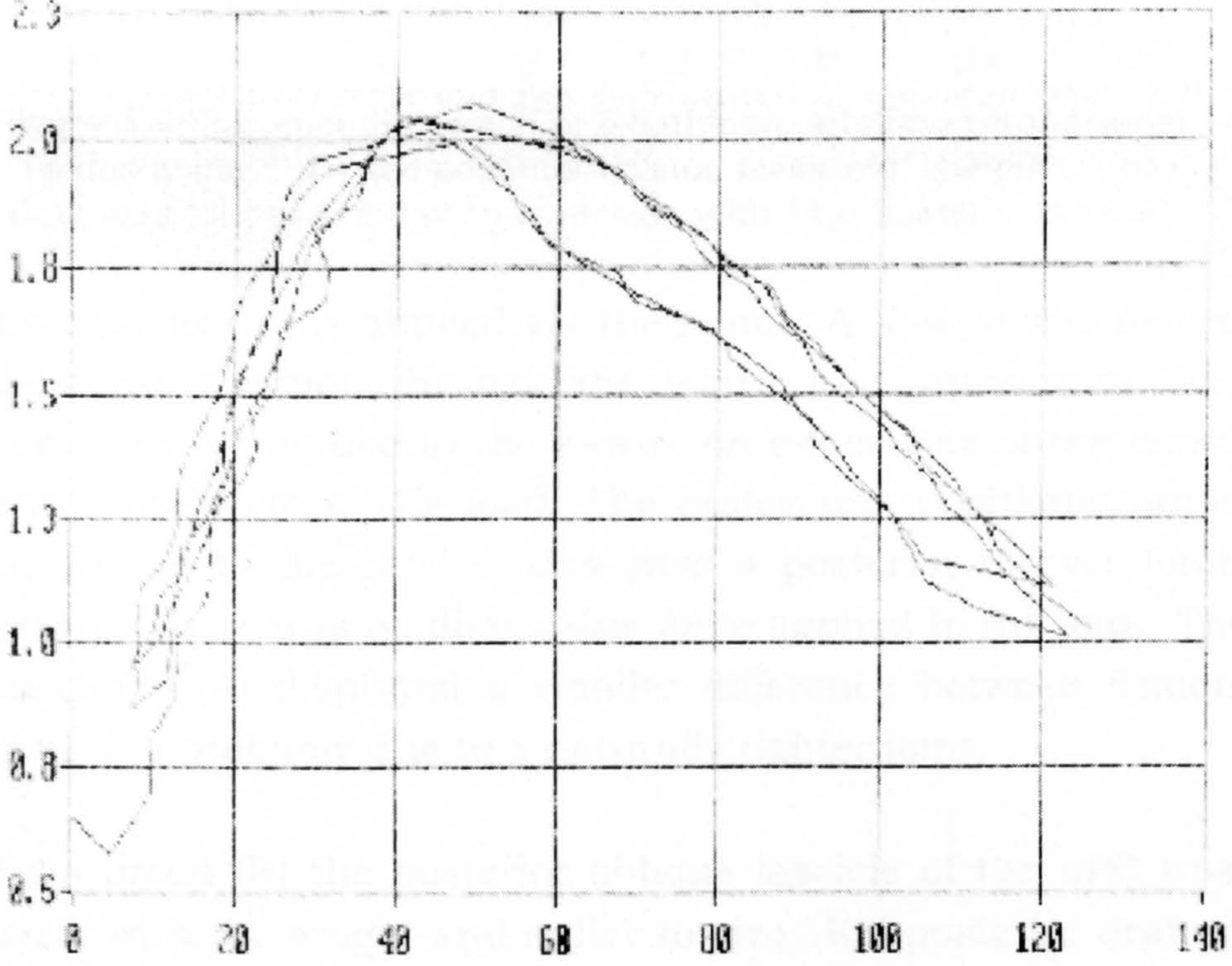


Figure 5.7 Specimen (B). Graph of length(mm, arbitrary zero) against flexion angle(°) for the middle fibre of the posterior surface of the aPC, as in Figure 5.6, with a 1kgf posterior drawer force. Flexion and extension patterns were closer but both were changed with respect to the unloaded condition.

joint which allowed different patterns of sliding and rolling in flexion and extension. During flexion the ACL was taut and forced the femoral condyles to slide. When extension started the PCL was not taut and the femoral condyles were allowed to roll anteriorly until the PCL was taut. The rolling of the femoral condyles effectively caused a small anterior drawer during flexion and a small posterior drawer during extension. If this theory were correct then applying a posterior drawer load should have brought the fibres flexion behaviour into line with its extension behaviour. This was found to be the case although the total length change was reduced to 1.2mm (Figure 5.7).

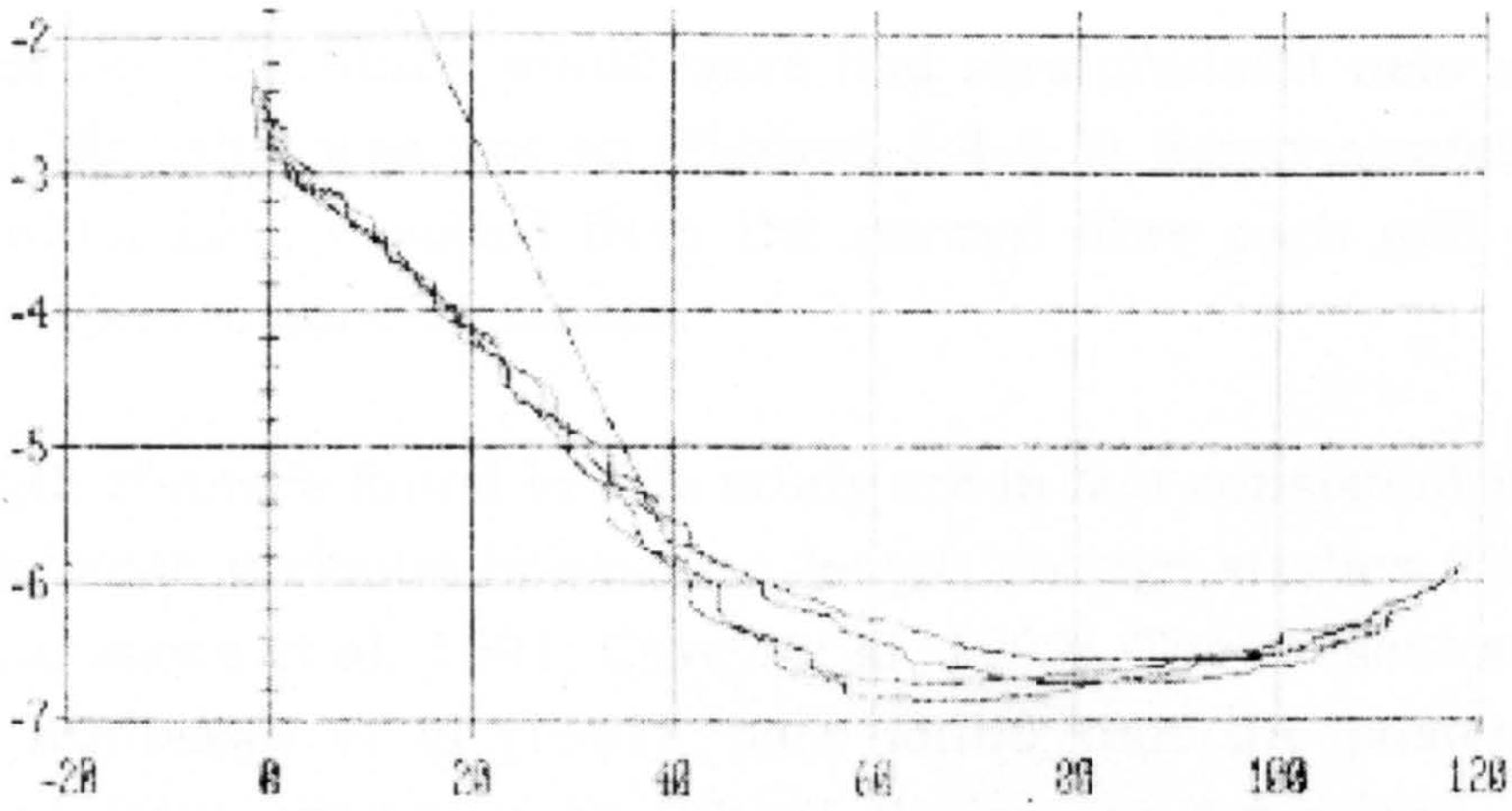


Figure 5.8 Specimen (B), graph of length(mm, arbitrary zero) against flexion angle(°) for the posterior oblique fascicle of the pPC. This data was for passive flexion-extension with 1kgf posterior drawer.

The posterior load was applied via the femur. A K–wire was drilled in a medial–lateral direction through the femur just anterior to the MCL insertion. A cord was tied to the K–wire on either side of the femur and run over a pulley to a 1Kg load. The pulley was positioned anteriorly and just distal to the K-wire, this gave a posterior drawer force and ensured that there was no distracting force applied to the joint. The fact that specimen (A) displayed a smaller difference between flexion and extension was probably due to a naturally tighter joint.

In the specimen (B) the posterior oblique fascicle of the pPC was also measured, with the weight and pulley to give 1Kgf posterior drawer. The total length change was 4mm (Figure 5.8).

5.3.1 Clinical relevance.

The large difference in length change pattern for a small change in experimental conditions invited caution in the interpretation of length change data.

On the basis of qualitative observations of PCL function it was deduced that the length change of the aPC fibres must be zero in the initial stages of flexion. The aPC was observed to be slack in extension (section 3.1.3.3), so real fibres could not have lengthened until the aPC was taut. As was stated in the introduction (section 5.1) it was hoped that the lightly loaded suture would follow the course of the natural fibres. If the suture had followed the natural fibres then the length/flexion angle

graph for the aPC fibres would have had zero gradient near extension. The fact that this was not so (Figures 5.4–5.7) demonstrates that the suture must have deviated from the normal fibre path and tended to measure bone to bone distances.

The length changes found in this study are in fact consistent with those found in some previous bone–bone length change studies (Hefzy et al, 1986; Kurosawa et al, 1991; Covey et al, 1992). The present study, and that of Kurosawa et al (1991), both found that the posterior fibres shortened from extension to 90° of flexion by 4mm and that they lengthened slightly with further flexion (Figure 5.8, Figure 5.9). The shortening of the pPC up to 90° of flexion was also demonstrated in the study of Hefzy et al (1986) (Figure 5.10, fibre #7). As stated in the introduction, length changes over 2–3mm indicate that some buckling of the fibres must have occurred. It was clear that the posterior fibres were tense in extension, but it was not possible to determine their strain at that point. Also, it was not possible to see when the posterior fibres buckled. So, although consistent posterior fibre length changes were found by three studies it is difficult to relate the results to PCL function.

With regard to the aPC, it was hard to judge the position of anterior fibres from the sagittal projection of the PCL femoral insertion published by Hefzy et al (1986) but fibres #1 and #2 in Figure 5.10 appeared to correspond to the posterior (Figure 5.4 and Figure 5.7) and central (Figure 5.5) fibres of the aPC measured in the present study. The length

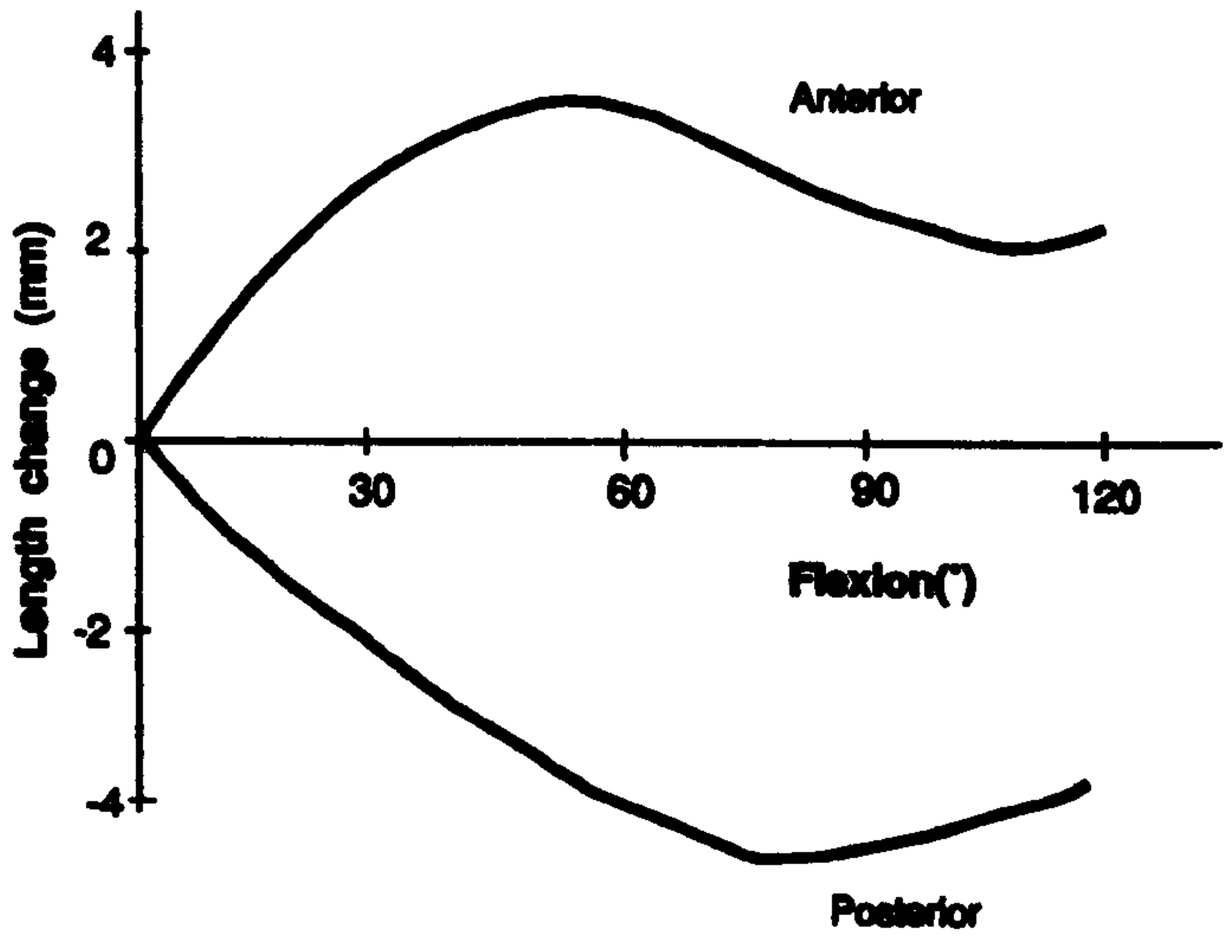


Figure 5.9 Length change with flexion for the 'anterior' and 'posterior' fibres of the PCL. Data from Kurosawa et al (1991).

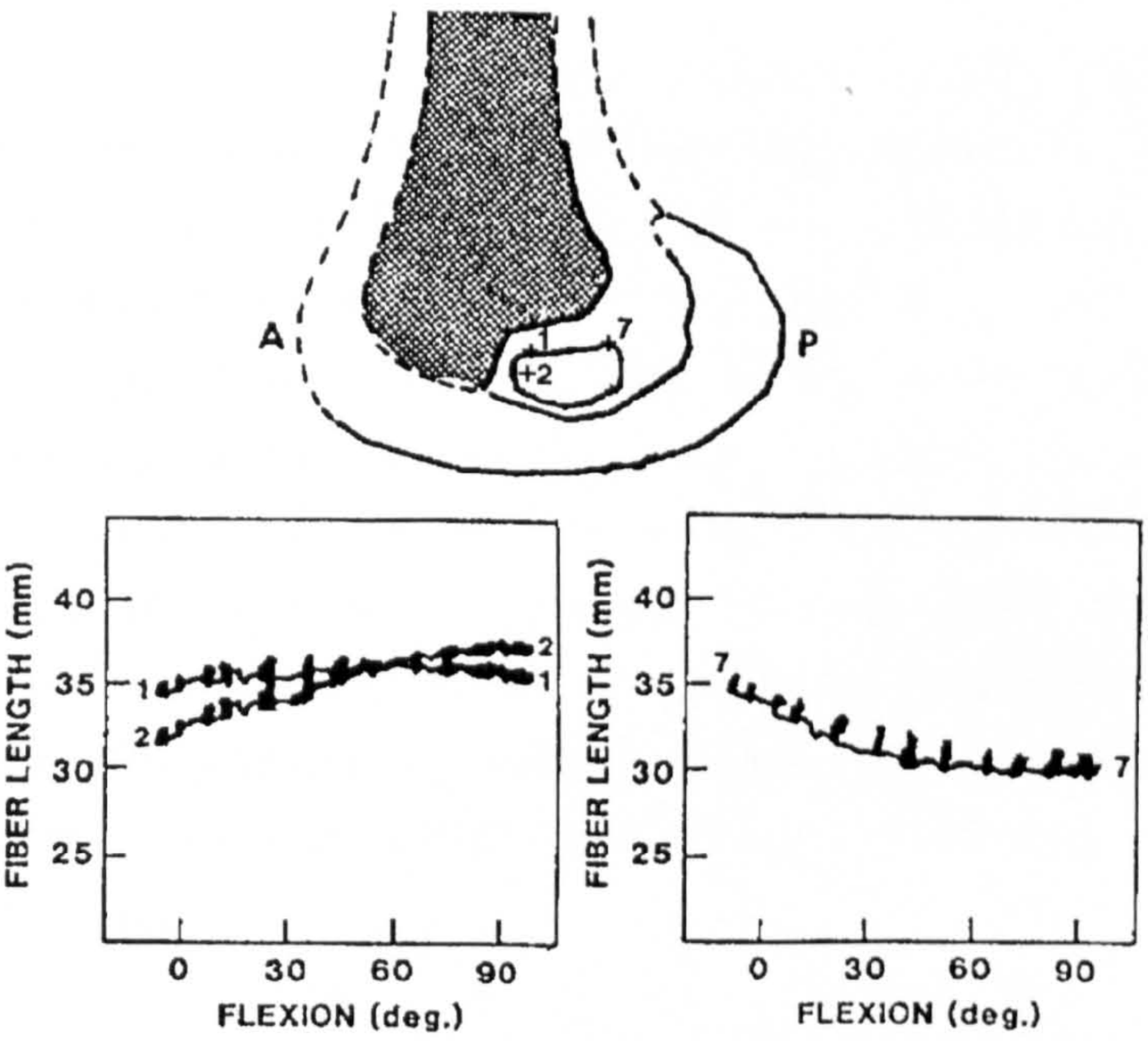


Figure 5.10 Length change with flexion for PCL femoral locations similar to those used in this study. Data from Hefzy, Grood and Lindenfeld (1986). 1– Posterior fibre of the aPC. 2- Central fibre of the aPC. 7- pPC.

change measured for the 'anterior' fibres of the PCL in the study of Kurosawa et al (1991) was of a similar pattern to that of the posterior fibres of the aPC in the present study and that of Hefzy et al (1986) but of a greater magnitude: 3.5mm as opposed to 1.5–2mm found in the present study. According to the maximum linear strain limit discussed earlier these length changes could be related to 'real' strains. However, it was observed that the fibres of the aPC were buckled in extension, so these length changes must be overestimates of real fibre elongation. Again, it was not possible to observe when the fibres became taut. The fact that the fibres did not all tighten simultaneously and the existence of a 'toe' in the load/elongation characteristic of the fibres meant that there was no obvious discontinuity in the length/flexion angle curve that might suggest at which point the fibres became taut.

The central fibre of the aPC was found to lengthen 6mm from extension up to 80° of flexion when its length remained constant (Figure 5.5). This length change, pattern and magnitude, was also demonstrated in the studies of Hefzy et al (1986) (Figure 5.10 fibre #2) and Covey et al (1992) (Figure 5.11).

The results of the present study correlated well with previously published work but did not serve the intended purpose. The purpose of this study was to gain understanding of the function of the PCL by quantifying the strain pattern of the fibres within it over the range of flexion. It was hoped that using a lightly loaded suture, as opposed to the 10–15N load applied in the later study of Covey et al (1992), would have resulted in true fibre elongation data. However, the method used in this study did not appear to produce the real fibre elongation data which was required to calculate strain. Bone–bone length change data, which this study did appear to produce, is hard to convert into real fibre strain. This is because it is extremely difficult to determine the true rest length of a real fibre.

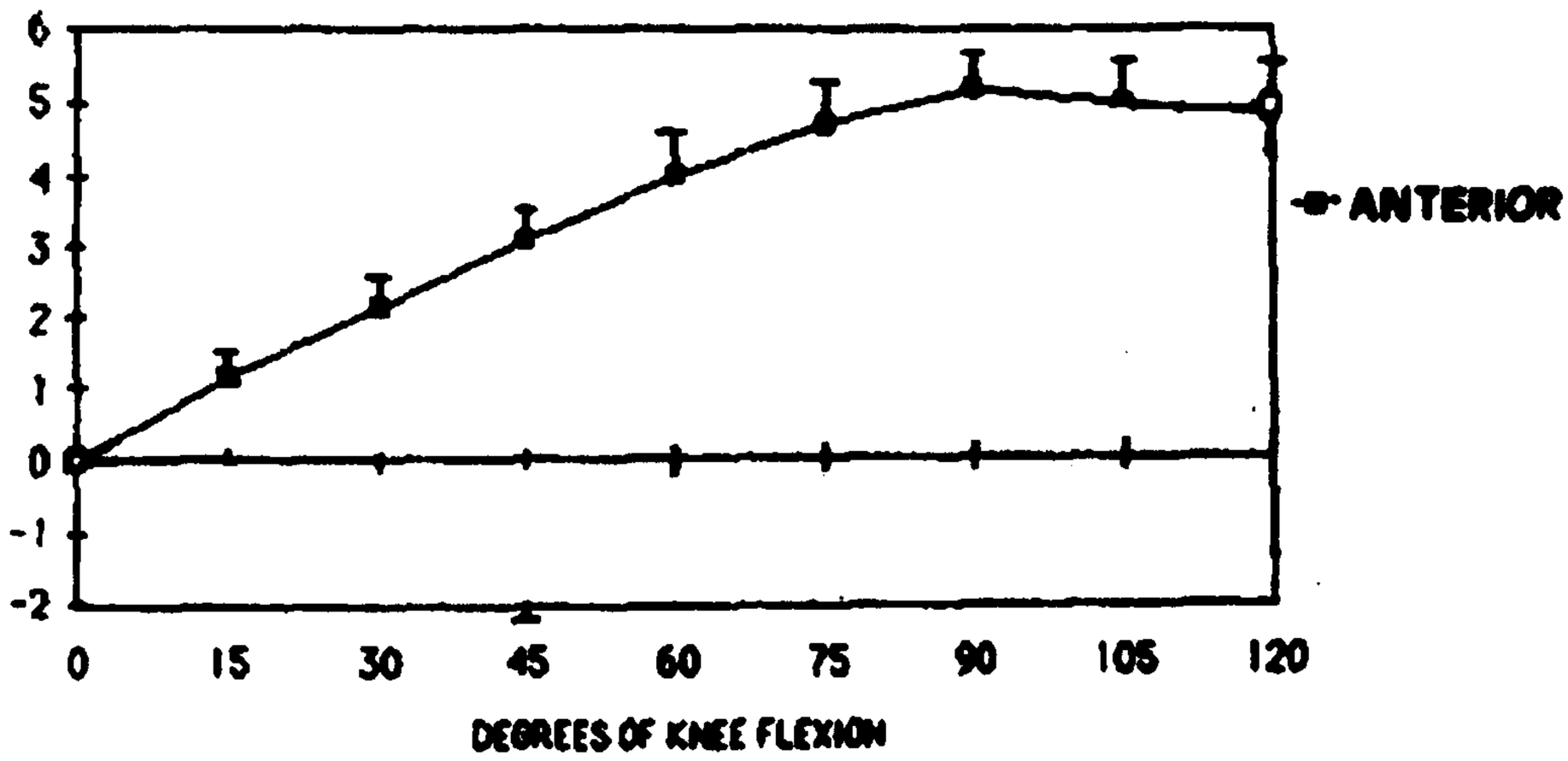


Figure 5.11 Length change (mm) with flexion for an anterior fibre of the PCL, similar to the central aPC fibre measured in the present study. Data from Covey et al (1992).

This study only served to replicate the results of some previous insertion site separation studies. Because new information was not generated and because of the uncertainties in determining fibre rest length I did not feel that it was worth pursuing this study. Therefore, fibre length change measurement was abandoned and a more direct method of establishing PCL function was sought.

6. LOADING OF THE PCL IN POSTERIOR DRAWER

6.1 INTRODUCTION

In the fibre length change study reported in section 5 the behaviour of the aPC and pPC was examined in passive flexion-extension. This study did not provide conclusive evidence of the function of the two bundles. It was thought that it would be more useful to examine the loading of the PCL in posterior drawer.

Many investigators have reported, on the basis of in vitro observation and palpation, that in extension the aPC is slack and that the pPC tightens from extension to hyperextension (Palmer, 1938; Girgis et al, 1975; Hughston et al, 1980; Sepulchre and Blaimont, 1986; Van Dommelen and Fowler, 1989; Burks, 1990; Kannus et al, 1991; Whipple and Ellis, 1991). However, in extension the pPC is aligned perpendicularly to the direction of a posterior drawer force (section 3.1.3.3) and therefore cannot efficiently resist it. Since the PCL has been shown not to be a primary restraint to hyperextension (Girgis et al, 1975; Nielsen et al, 1985; Grood et al, 1988) the function of the pPC is unclear. Also, since the aPC is slack in extension it is not clear what structures resist posterior drawer near extension. The purpose of this study was to clarify the role of the two bundles of the PCL in posterior drawer and to test the hypothesis that the aPC and the pPC have different functions over the range of flexion. It was hoped that direct measurement of in vitro forces would give more definite answers than inferences drawn from bone to bone length change studies (sections 1.2.2.2 and 5.4).

6.2 METHOD

Knee specimens were obtained at post-mortem and frozen until used, as described in section 2.1. The mean age of donor was 79 years (range 56–88) The tibia and femur were stripped of muscle and fat tissue, taking care to preserve the ligaments and joint capsule, and then potted, as described in section 2.2. When the tibia was potted it was aligned so that the axis of the pot, which was designed to be coincident with the axis of the clamp, passed 5mm medially to the medial tibial

spine. This was picked as a reasonable 'average' position given that there is considerable inter–specimen variation and that the rotational axis of the knee changes with flexion (Trent et al, 1976; Fuss, 1991). The functionality of LCL was maintained by stabilising the fibula with transcortical bone screws, as described in section 5.2. The portion of the fibula that was more than 10mm distal to the bone screws was removed as it could not be contained in the pot and would impinge on the clamping system.

A–P drawer tests were carried out using an Instron 1122 materials testing machine. The tibia, in its pot, was held via a four degree of freedom attachment to the crosshead of the Instron (Figure 6.1). This attachment allowed all possible degrees of freedom in response to A-P

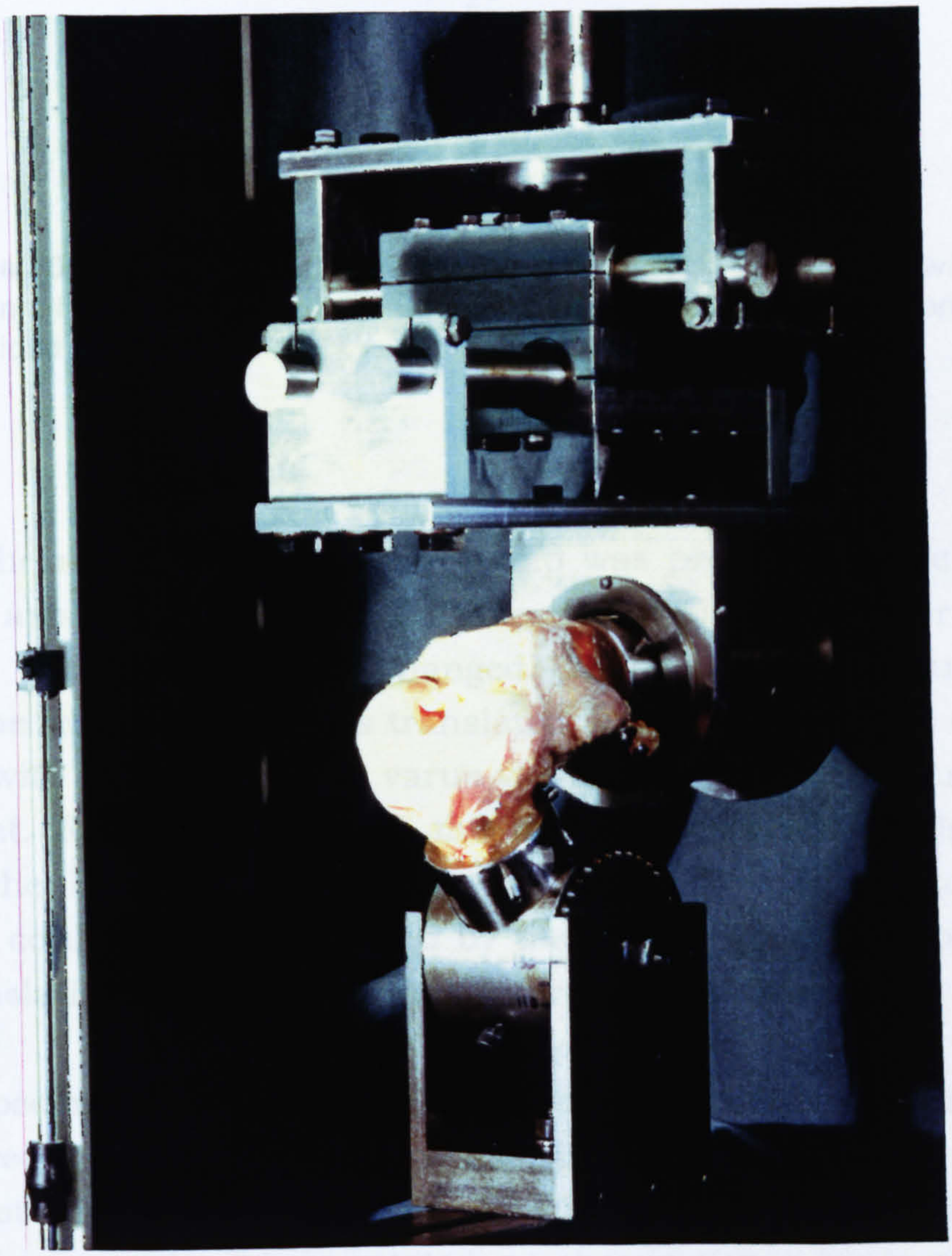


Figure 6.1 Knee mounted in the Instron at 130° of flexion. The tibia is horizontal and held in the four degree of freedom crosshead attachment.

drawer at a fixed flexion angle: internal–external rotation, varus–valgus rotation, medial–lateral translation and compression–distraction of the tibia relative to the femur. The rolling control bearings allowed the tibia to rotate and translate into a normal passive physiological relationship with respect to the femur. The Instron crosshead controlled anterior–posterior translation and the remaining degree of freedom, flexion–extension, was provided by the femoral clamp which was secured to the base of the Instron (Figure 6.1). The femoral clamp was rotated to change the flexion angle and could be locked at 10° intervals by an indexing screw engaging in conical holes.

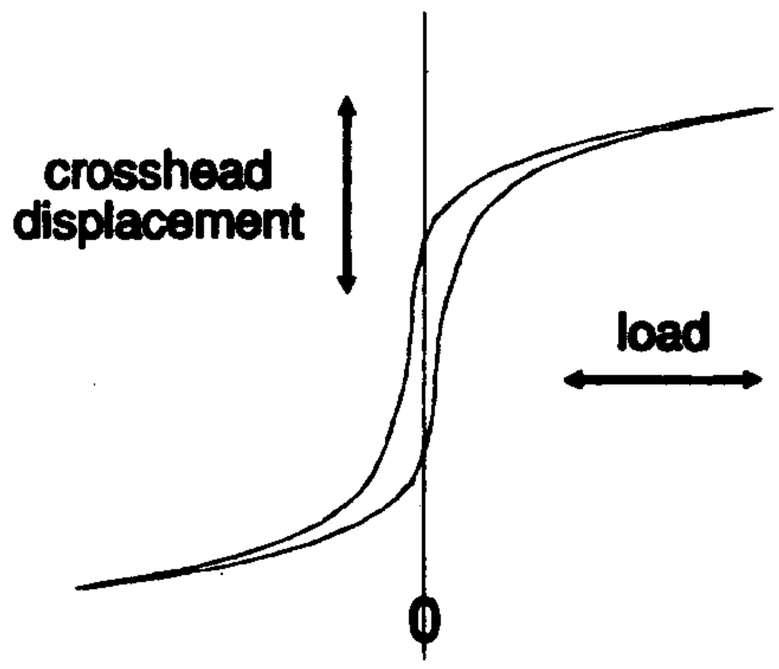


Figure 6.2 Typical load/crosshead displacement graph showing region of approximately zero stiffness and the correctly positioned zero load line.

Because the rotational axis of the clamp was proximal to the flexion–extension axis of the knee the crosshead had to be raised or lowered when the flexion angle was changed. To ensure free varus–valgus rotation the femoral clamp was translated to bring the femoral condyles into line with the axis of the varus-valgus bearing on the crosshead attachment. From this position the deviations in varus–valgus axis between the knee and the fixture with specimen and flexion angle variations could be accommodated by the medial–lateral and proximal–distal translations of the four degrees of freedom attachment

When a specimen was tested the tibia was clamped first and the femur was allowed to hang free. The crosshead was then lowered and the femoral pot slid into the lower clamp, which was positioned in 90° of flexion. The femoral pot was rotated, so that the tibia lay in the plane perpendicular to the 'flexion–extension' axis of the femoral clamp, and

then secured in place with a grub screw. For each specimen the load cell was balanced by placing the joint in 20° or 30° of flexion, which preliminary investigation showed were the flexion angles at which the joints were most lax, and imposing an A–P load cycle. The laxity was such that there was a region of approximately zero stiffness around the neutral point. The load cell balance was adjusted so that the lines of the hysteresis loop, which were approximately parallel in the minimum stiffness region, were equidistant from the zero load line (Figure 6.2).

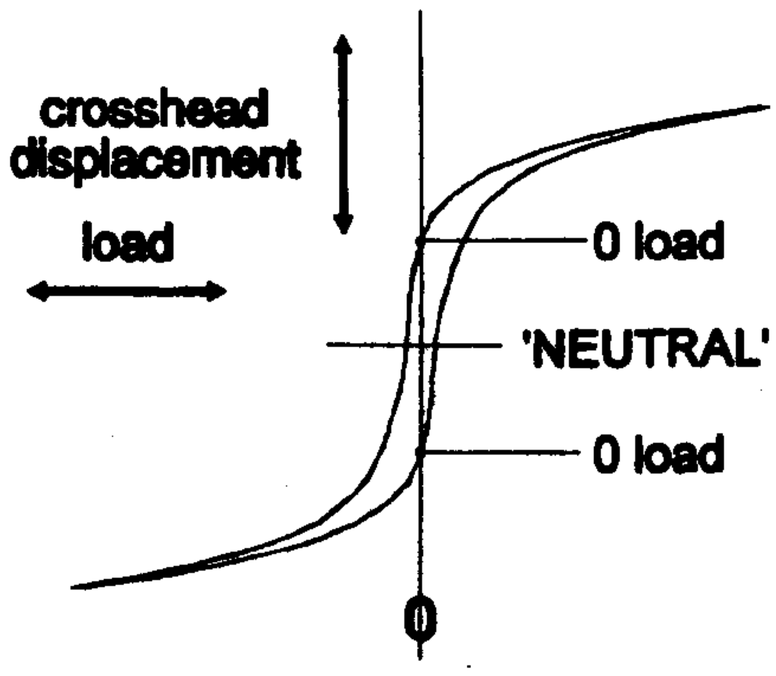


Figure 6.3 Typical load/crosshead displacement graph showing zero load points and the neutral position.

Two separate methods were used in this study to assess the role of the aPC and the pPC. Firstly, selective cutting (section 1.2.4.2) was utilised to determine percentage contributions to resisting posterior drawer and secondly, miniature pressure transducers were implanted to allow estimation of the loads imposed on the aPC and the pPC during posterior drawer. The details specific to each of these two methods are outlined separately below.

6.2.1 Selective cutting

To allow access to the femoral origin of the PCL, the patella was removed, this has been shown not to affect A–P drawer (Amis, 1989a). A neutral A–P position was established for each flexion angle by imposing a ±50N drawer cycle: neutral was defined as the position of the midpoint between the two zero load points of the load/crosshead–displacement hysteresis loop (Figure 6.3).

The Instron crosshead was then moved to this midpoint and its position was recorded via the digital readout on the Instron extension module accurate to 0.1mm. Knees were then subjected to cyclic A-P



Figure 6.4 Anterior aspect of the flexed knee. The aPC (⌓) has been cut from the femur and the pPC (◆) has been left intact.

displacements of ±6mm at 100mm/min. Load/crosshead–displacement graphs were output to a chart recorder. Eight conditioning cycles were allowed before the final graph was recorded, by which time differences between subsequent cycles could not be detected. This procedure was repeated at 10° intervals between 130° and 0° of flexion.

Then, with the knee in full flexion, the femoral attachment of the PCL was identified through the intercondylar notch and the aPC was cut from the femur along with the anterior meniscofemoral ligament when present. The separation of the bundles was carried out based on the

geometry of the femoral PCL insertion and the intercondylar notch as determined in previous studies (section 3.1, section 4.2.1 and Figure 6.4). The bundles were identified by their femoral attachments: when the aPC was cut all PCL fibres inserting into the notch roof were sectioned leaving only fibres that inserted into the lateral face of the medial epicondyle intact. The knee was then subjected to the same A–P displacements as before, based on the recorded neutral position for each flexion angle, from 130° to 0° of flexion. The repeatability of the anterior drawer data was a useful check on the consistency of the experiment, since the PCL does not resist anterior drawer. The procedure was then repeated after cutting the pPC along with the posterior meniscofemoral ligament, if present. From the reduction in required displacing force, measured from the load–displacement graphs, the percentage contribution of the aPC, pPC and combined other structures to resisting posterior drawer was calculated for 2mm, 4mm and 6mm posterior displacement.

6.2.2 Implanted pressure transducer

This study used miniature pressure transducers* similar to those used by Holden et al(1991). Four other methods have been used to estimate ligament loads; buckle transducers attached to ligament fibres (Lewis and Fraser, 1979; Jasty et al, 1982; Lew and Lewis, 1982; Ahmed et al, 1987; Lewis et al, 1982); a miniature load cell attached to the ligament insertion via a bone tunnel (Wascher et al, 1991); Hall Effect strain transducer attached to ligament fibres (Ogata et al, 1988) and strain gauges attached to bone adjacent to ligament fibre insertions (France et al, 1983) (section 1.2.5). These other methods were considered and rejected for the reasons discussed below.

Buckle transducers, including the smaller 'modified buckle transducer' (Barry and Ahmed, 1986), are bulky and could not have been fitted to the central fibres of the two bundles. Also, impingement errors have been reported for buckle transducers that require osteotomy and/or the removal of tissue to rectify (Lewis et al, 1982). A buckle transducer could, in theory, have been fitted to the whole of each bundle of the

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* Model 105S, Precision Measurement Company, P.O. Box 7676, Ann Arbor, Michigan 48107.

PCL. However, the pPC must pass from 'behind' the aPC in extension to 'in front' in full flexion (Section 3.1.3.3). Therefore, it was thought that buckle transducers may have given acceptable results for lower flexion angles but that full flexion could not have been reached without impingement errors that would have required the removal of ligament fibres to correct. Therefore, buckle transducers were considered unsuitable for the present study.

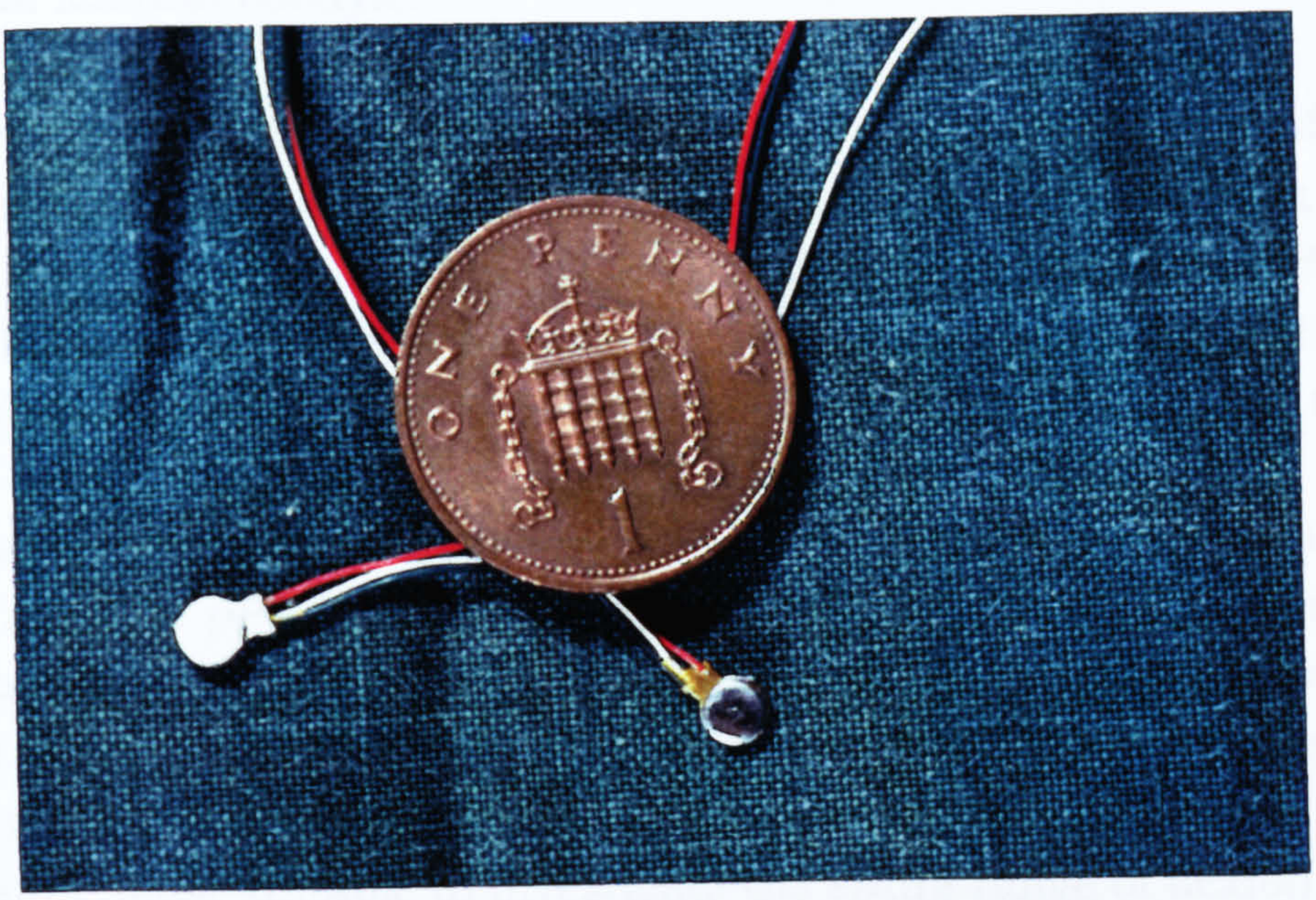


Figure 6.5 Miniature pressure transducers used in this study

The miniature load cell method (Wascher et al, 1991) could only have been used to measure the load on the whole of the PCL. This was because Wascher et al(1991) reported that a gap of at least 2mm around the bone block was necessary to prevent bone to bone impingement due to strain in the load cell and its mounting. The required gap between the bone blocks would have necessitated the destruction of at least 10% of the fibres of the PCL and may have resulted in the impairment of function of more fibres as the ligament/bone interface was disturbed. The aforementioned problem, and also the considerable technical difficulty of fitting two miniature load cells, led to the conclusion that this method was unsuitable for the present study.

Hall Effect strain transducers are limited to being mounted on the surface of a ligament bundle, due to their bulk. Ogata *et al* (1988), who fitted a Hall Effect strain transducer to the 'central' portion of the PCL, reported that only the first 90° of flexion could be observed without errors associated with impingement of the transducer. A gauge fitted to the pPC would have been even more susceptible to impingement errors since the pPC passes between the aPC and the lateral surface of the medial femoral condyle.

Bone mounted strain gauges, as used by France *et al* (1983), offered the advantage that the fibres of the ligament would not have been deviated from their natural path. However, such a method had the disadvantages that bundle load would not have been measured directly and that fitting the strain gauges would have required the removal of capsular tissue.

Miniature pressure transducers were chosen to measure the load in the aPC and the pPC because they could be implanted into the middle of the bundles with minimal disturbance to the ligamentous tissue of the knee. Their small size, and central location within the bundles, meant that they were least likely to be subject to impingement errors and were therefore most suitable to investigate the entire range of flexion.

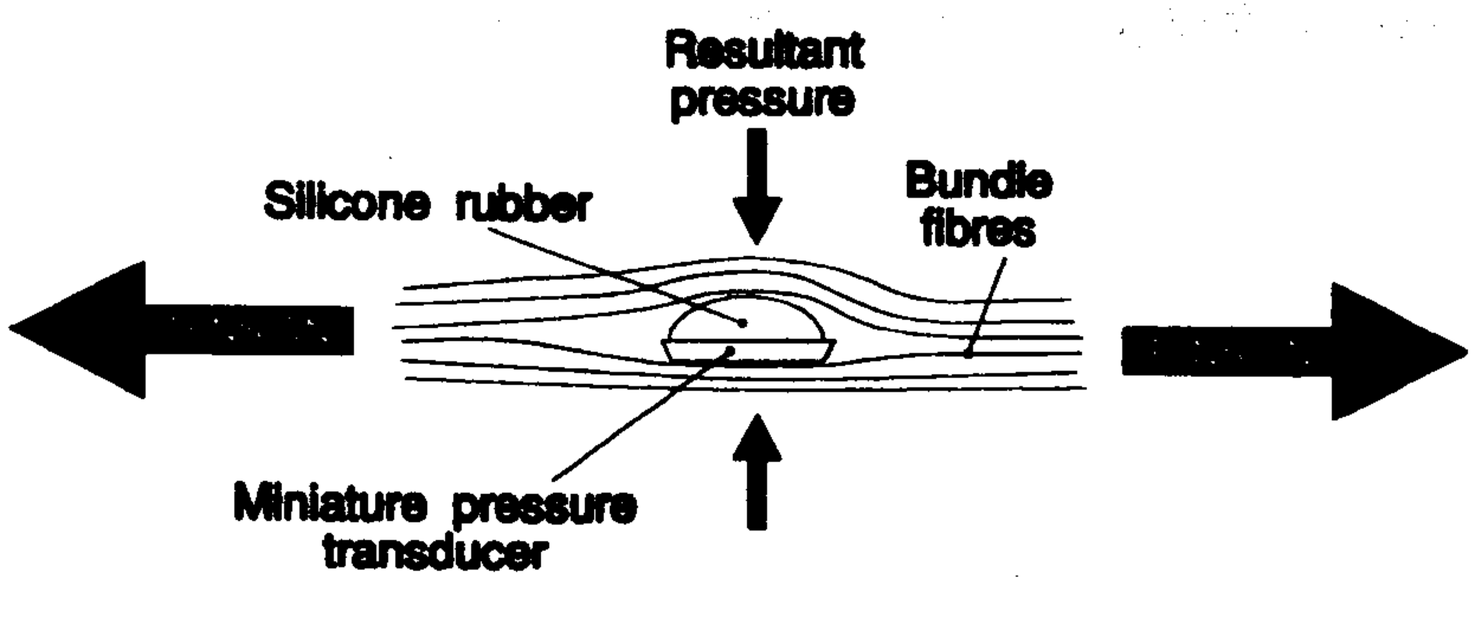


Figure 6.6 Schematic diagram of the operation of an implanted pressure transducer to measure load in a ligament bundle

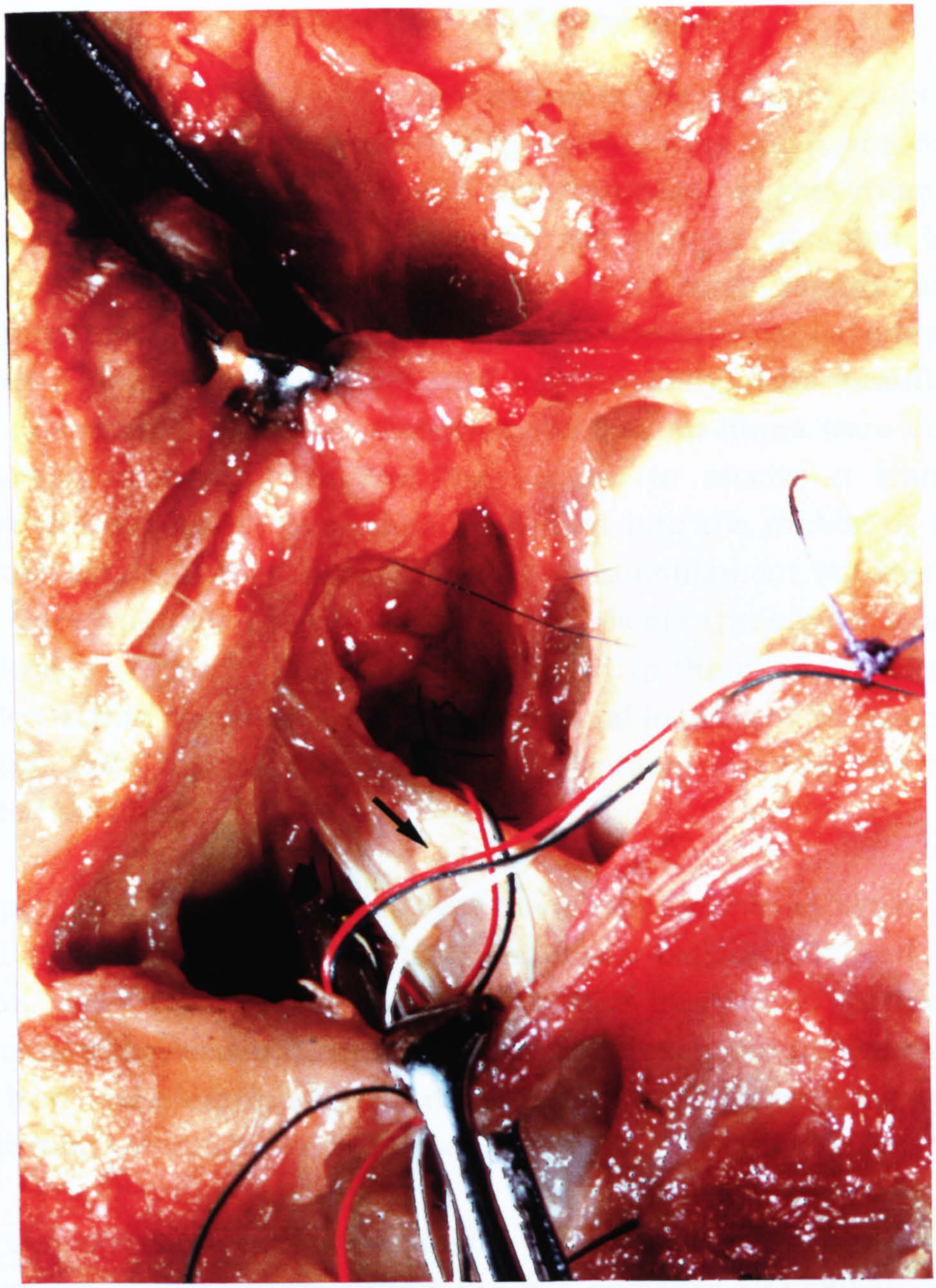


Figure 6.7 Posterior aspect of the knee showing the pressure transducers implanted into the aPC (⌓) and the pPC (⬟) prior to the posterior capsule being closed. The PCL is partially obscured by the posterior meniscofemoral ligament (⟶).

The transducers used were 3.5mm in diameter and 0.4mm thick (Figure 6.5). Only one face of the transducer was 'active': it was fitted with a straingauge in a quarter bridge configuration. The principle of operation of the pressure transducers to measure load in a ligament is illustrated in Figure 6.6. If the transducer causes ligament fibres to deviate from a straight path then tension in the ligament will result in a pressure, related to the ligament tension, on the transducer. To increase the sensitivity of the transducer–ligament system to load a drop of silicone rubber was glued onto the sensing face, which increased the thickness from 0.4mm to approximately 1.5mm and left the diameter unchanged

at 3.5mm (Figure 6.6). To gain free access to the midsections of the two bundles of the PCL the knee had to be opened from the posterior aspect (Figure 6.7). In order to minimise the disruption to the intact joint a portal was made by cutting through the oblique popliteal ligament parallel to its fibres. The aPC and the pPC were each prepared to receive the transducers by making a small cut in the synovial covering with a scalpel. The cut was then expanded into the body of the bundle in the fibre direction, using blunt dissection so that no fibres were cut, until the resulting cavity was just big enough to receive a transducer. Pressure transducers were thus implanted into the middle of the aPC and the pPC and then secured with 7-0 monofilament sutures (Figure 6.7). Care was taken not to damage the delicate transducer lead wires. The lead wires were then carefully sutured to the capsule: some slack was left within the joint to prevent artifactual loading or loosening of the transducers. Then the oblique popliteal ligament was repaired with 2–0 braided sutures.

The prepared knee specimens were then subjected to cyclic A–P loads, of ±150N at 100mm/min, at 10° intervals between 0° and 130° of flexion. After eight conditioning cycles data from three further cycles was recorded. Pressure transducer output, crosshead displacement and A–P load were captured and displayed in real time using the data acquisition system described in section 2.3.

In order to calculate the relationship between ligament bundle tension and pressure transducer output the two pressure transducer–ligament bundle systems were calibrated using the Instron 1122 materials testing machine. For precise calibration it would have been necessary to reproduce the motion of the knee with a single bundle intact and record the resulting loads with a six axis load cell. However, the sophisticated equipment required for this was not available. Because the load cell had a single degree of freedom it was necessary to align each of the bundles with the axis of the load cell when they were loaded. To this end the four degree of freedom crosshead attachment was removed and the tibia was clamped directly to the crosshead at 50° to the vertical with the anterior aspect of the tibia facing down. This angle was chosen as an reasonable 'average' position based on the observation of sectioned knees. According to the sagittal plane model of Zavatsky and O'Connor (1992) the angle of the 'neutral' PCL fibre with respect to the tibial

plateau varies between 50° and 61° over the flexion range 0°–120° (Figure 1.20 and 7.20). In the semi–flexed knee the angles of the aPC and pPC with respect to the tibial plateau were very similar (Figure 3.8). At the extremes of flexion, when the femoral insertion of the PCL was more perpendicular the 'neutral' fibre, there was a maximum difference in the angles of the aPC and pPC with respect to the tibial plateau (Figures 3.7 and 3.9). The maximum angular difference was about 30°. So, the error involved in leaving the tibia at a fixed angle was a minimum of 0° and a maximum of 25°.

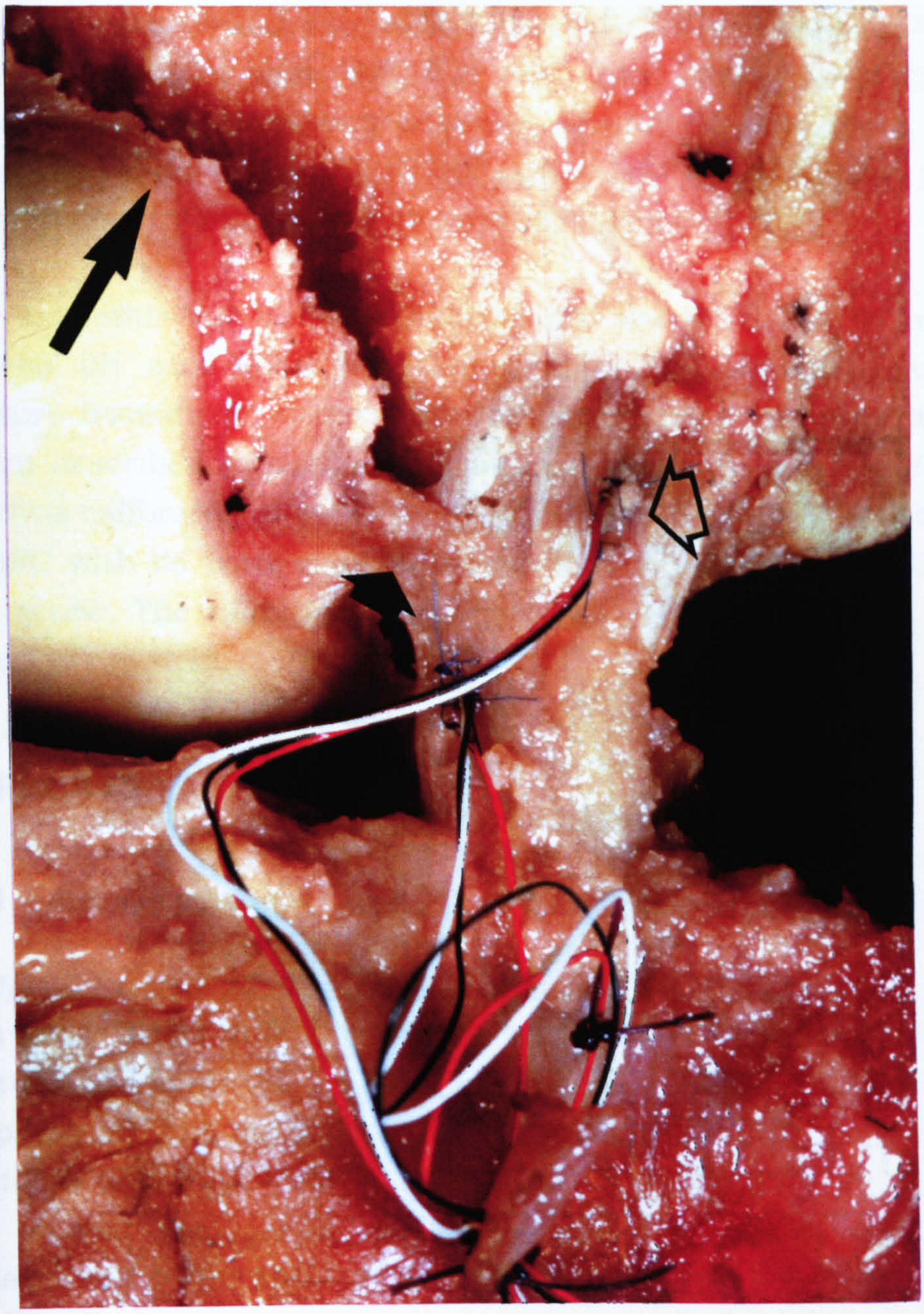


Figure 6.8 Posterior aspect of the knee, showing the separated aPC (⌂) and pPC (⬟). The femoral origin of the pPC has been separated from the femur by cutting a bone block (⟶).

The femur was reclamped, with the anterior aspect facing down, in its original holder. All soft tissue bar the PCL was then removed from the specimen. The two bundles were then separated by blunt dissection. The PCL origin was then divided by an osteotome, following the line of the blunt dissection to minimise fibre damage. A saw–cut was then made from the outside to join the split made by the osteotome. This created a femoral bone block which incorporated the origin of the pPC (Figure 6.8). The femur was then rotated to bring the aPC origin into what were observed to be the correct physiological alignment for flexion angles between 0° and 120° at 30° intervals: with respect to the femoral shaft these angles were 30°, 70°, 90°, 110° and 140° (Figure 7.20). These angles, which were close to the 'neutral' fibre angles in the model of Zavatsky and O'Connor (1992), were also used for the pPC: as a result there was a maximum error of 30° for the pPC.

After the femur had been secured the aPC was subjected to load cycling between 0N and 250N at 100mm/min. The load cell output and pressure transducer output were recorded using the data acquisition system described in section 2.3. The pressure transducer-aPC system was thus calibrated over the range of flexion. The aPC was then cut and the pPC with its bone block was restored to its normal position with a bone screw. The calibration procedure was then repeated for the pPC-transducer system.

6.3 RESULTS

6.3.1 Selective cutting

A typical series of load/crosshead displacement graphs, showing progressive drops in load as structures are cut, is shown in Figure 6.9. Figure 6.10 shows the graphs of percentage contributions of the aPC, the pPC and combined other structures to resisting posterior drawer against flexion angle for 2, 4 and 6mm of posterior displacement. Figure 6.11 shows the graph of load against flexion angle for each displacement in the intact knee. These graphs represent the average of nine knees.

Loading of the PCL in Posterior Drawer

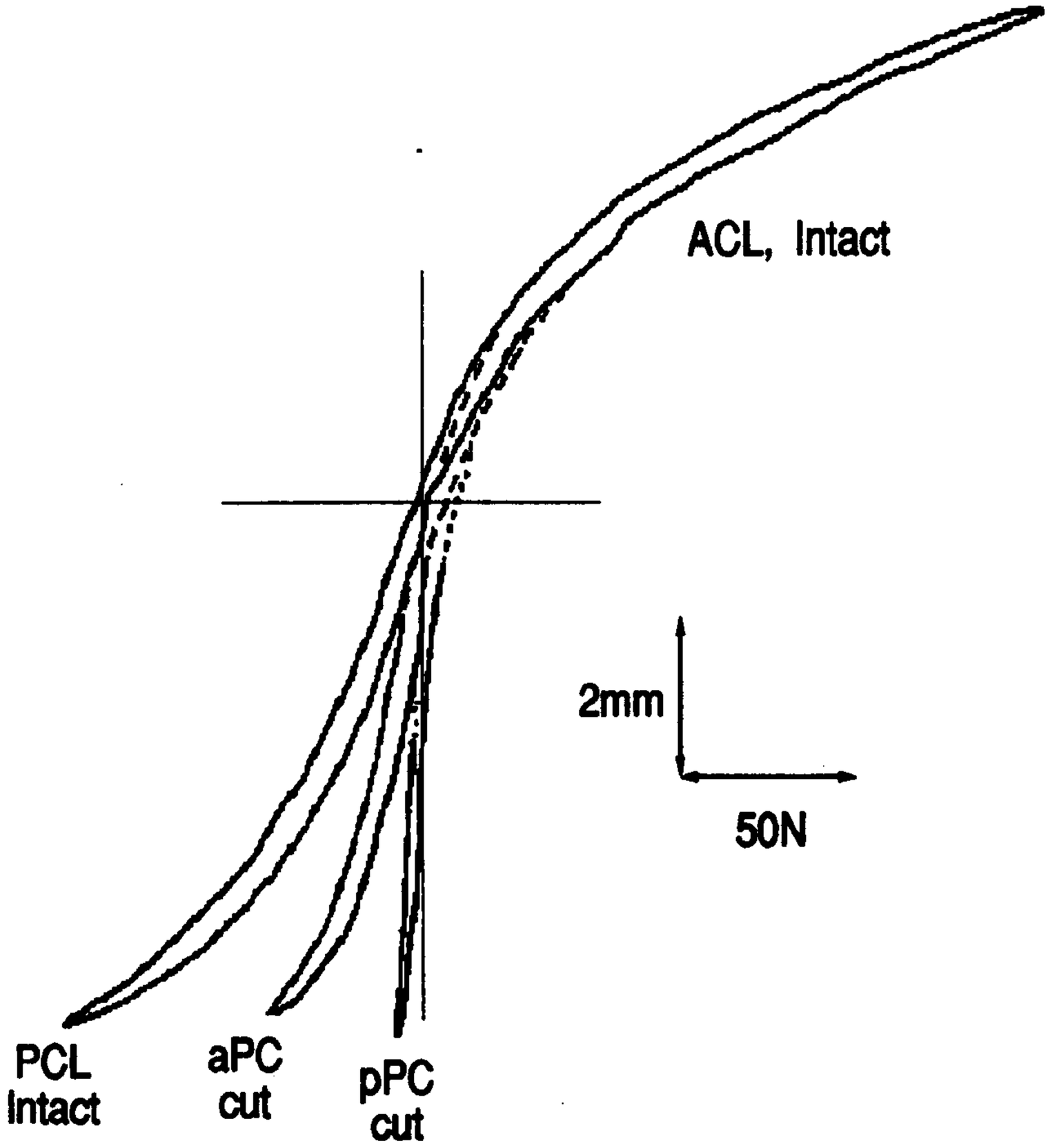
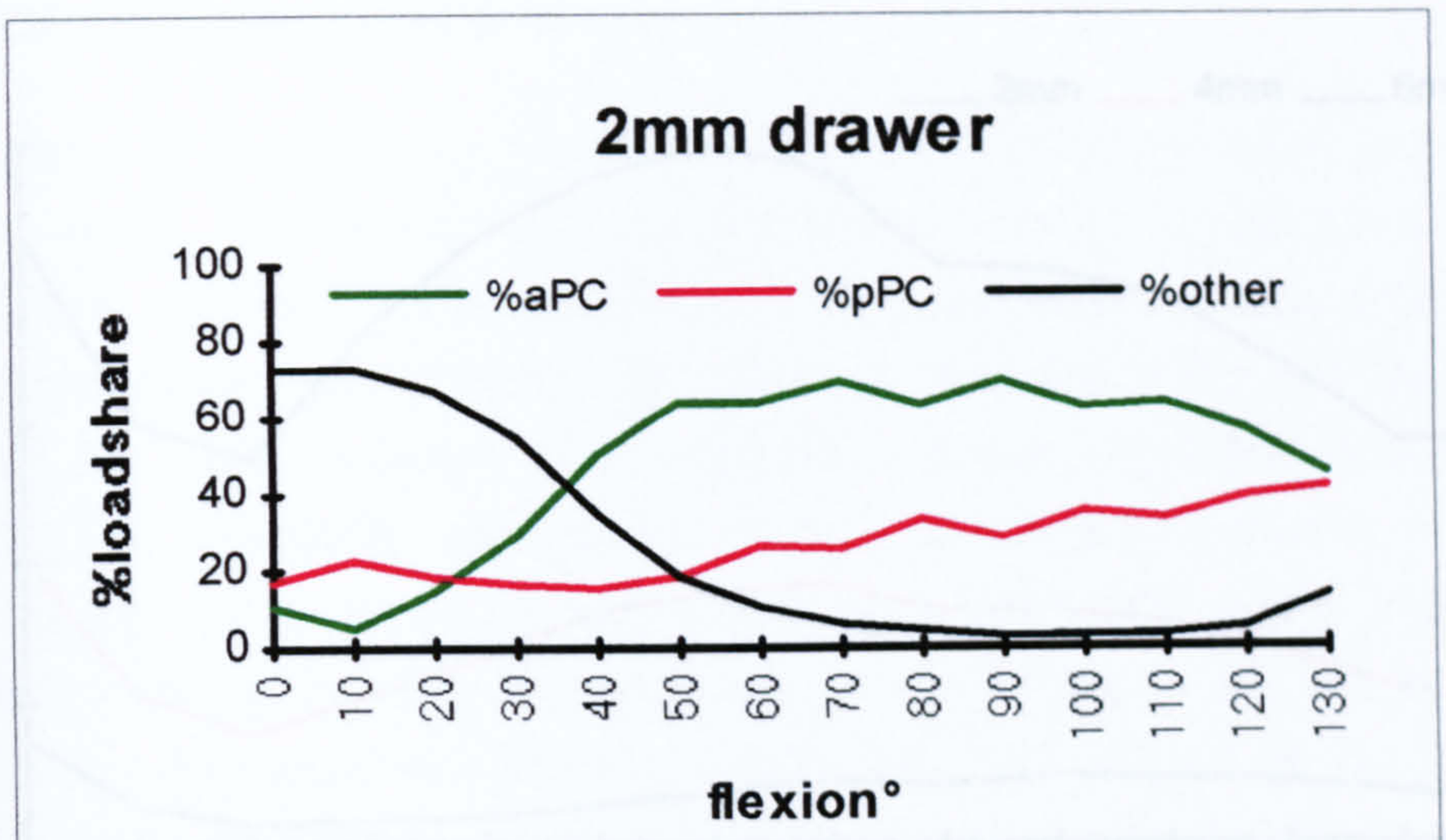
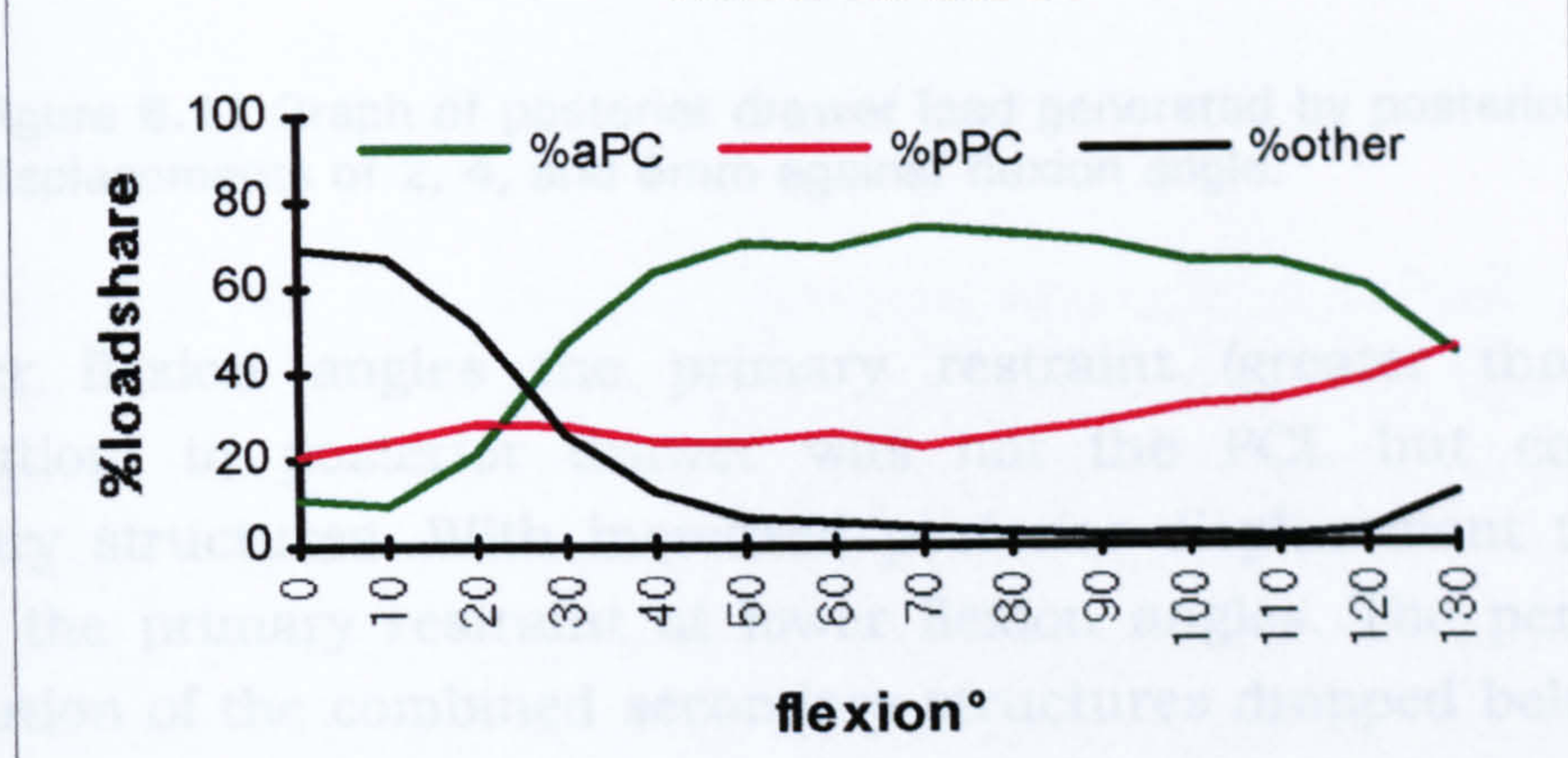


Figure 6.9 Typical overlay of the three charts of load against A–P displacement for 90° of flexion. The crossed hairlines mark the assumed A–P neutral and zero load. Note that the apparent neutral position was displaced posteriorly as the PCL was cut.

Table 6.1 Percentage contributions (± s.d) to resisting posterior drawer

2mm				
flexion°	%aPC	%pPC	%other	p(aPC<>pPC)
0	11 ± 11	17 ± 17	73 ± 19	*0.391*
10	5 ± 8	22 ± 26	73 ± 25	*0.110*
20	15 ± 16	18 ± 11	67 ± 16	*0.648*
30	29 ± 21	16 ± 14	55 ± 27	*0.144*
40	50 ± 24	15 ± 17	35 ± 26	0.012
50	64 ± 20	18 ± 15	18 ± 13	0.003
60	63 ± 19	26 ± 18	10 ± 8	0.015
70	69 ± 17	25 ± 15	6 ± 7	0.003
80	63 ± 21	33 ± 19	4 ± 4	*0.058*
90	69 ± 11	28 ± 11	3 ± 4	0.001
100	62 ± 13	35 ± 12	3 ± 3	0.012
110	64 ± 12	33 ± 9	3 ± 4	0.003
120	56 ± 23	39 ± 17	5 ± 9	*0.218*
130	45 ± 29	41 ± 18	14 ± 15	*0.815*
4mm				
flexion°	%aPC	%pPC	%other	p(aPC<>pPC)
0	11 ± 9	21 ± 13	69 ± 16	*0.090*
10	9 ± 10	24 ± 17	67 ± 18	*0.069*
20	21 ± 16	28 ± 18	51 ± 17	*0.485*
30	47 ± 23	28 ± 16	26 ± 12	*0.161*
40	63 ± 17	24 ± 13	12 ± 8	0.003
50	70 ± 16	24 ± 13	6 ± 6	0.001
60	69 ± 15	26 ± 14	5 ± 6	0.002
70	74 ± 14	23 ± 12	4 ± 4	0.000
80	72 ± 14	25 ± 12	3 ± 3	0.001
90	70 ± 12	28 ± 11	2 ± 2	0.000
100	66 ± 13	32 ± 12	2 ± 2	0.004
110	65 ± 11	33 ± 10	2 ± 2	0.002
120	59 ± 12	38 ± 10	3 ± 4	0.018
130	44 ± 21	45 ± 13	11 ± 10	*0.900*
6mm				
flexion°	%aPC	%pPC	%other	p(aPC<>pPC)
0	12 ± 9	23 ± 12	65 ± 13	*0.079*
10	16 ± 12	28 ± 15	56 ± 13	*0.143*
20	30 ± 17	34 ± 17	36 ± 10	*0.686*
30	50 ± 17	33 ± 16	17 ± 5	*0.156*
40	56 ± 18	35 ± 17	9 ± 3	*0.098*
50	61 ± 15	34 ± 15	5 ± 4	0.021
60	64 ± 13	33 ± 13	4 ± 4	0.007
70	66 ± 14	31 ± 15	3 ± 3	0.006
80	65 ± 14	33 ± 14	2 ± 2	0.009
90	61 ± 14	37 ± 13	2 ± 2	0.026
100	59 ± 14	39 ± 14	2 ± 2	*0.054*
110	57 ± 13	42 ± 13	1 ± 1	*0.119*
120	51 ± 15	47 ± 14	2 ± 2	*0.635*
130	36 ± 15	57 ± 13	8 ± 4	*0.053*





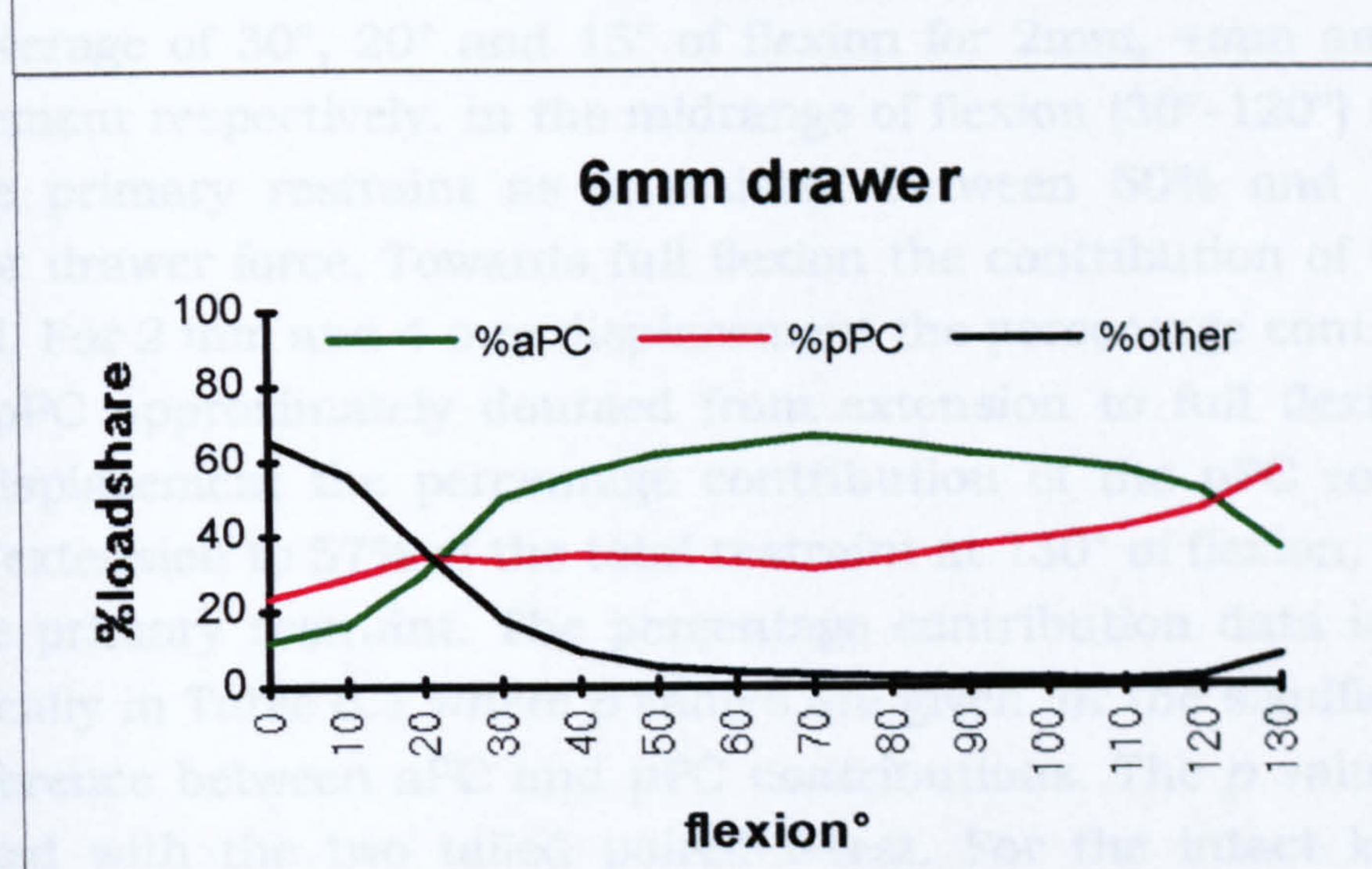


Figure 6.10 Graphs of percentage contribution to resisting posterior displacement against flexion angle for the aPC, the pPC and combined other structures.

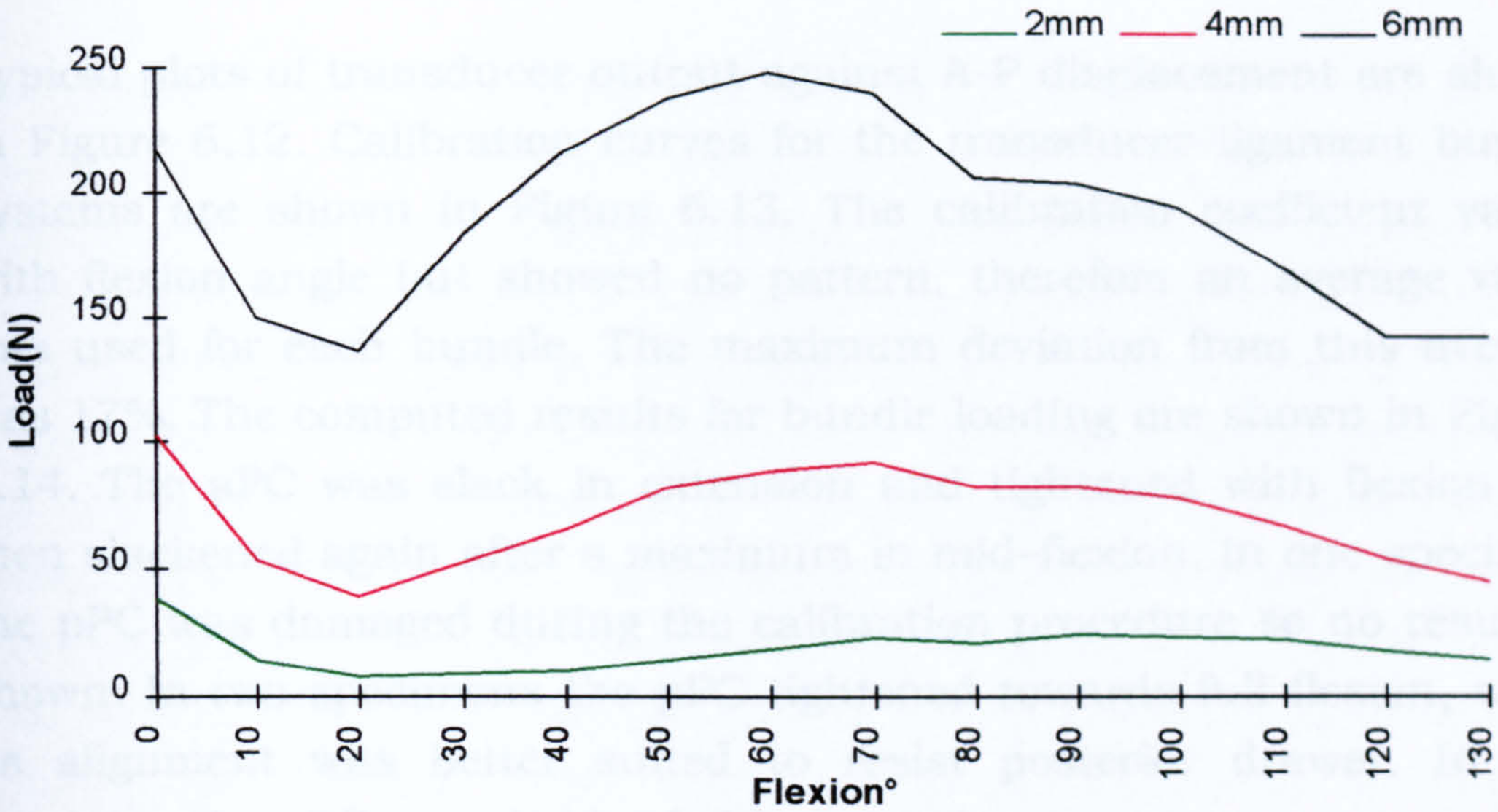


Figure 6.11 Graph of posterior drawer load generated by posterior displacements of 2, 4, and 6mm against flexion angle.

At lower flexion angles the primary restraint (greater than 50% contribution) to posterior drawer was not the PCL but combined secondary structures. With increased posterior displacement the PCL became the primary restraint at lower flexion angles. The percentage contribution of the combined secondary structures dropped below 50% at an average of 30°, 20° and 15° of flexion for 2mm, 4mm and 6mm displacement respectively. In the midrange of flexion (30°-120°) the aPC was the primary restraint as it resisted between 50% and 74% of posterior drawer force. Towards full flexion the contribution of the aPC reduced. For 2 mm and 4 mm displacement the percentage contribution of the pPC approximately doubled from extension to full flexion. For 6mm displacement the percentage contribution of the pPC rose from 23% at extension to 57% of the total restraint at 130° of flexion, when it was the primary restraint. The percentage contribution data is shown numerically in Table 6.1 where *p* values are given for the significance of the difference between aPC and pPC contributions. The *p* values were computed with the two tailed paired t–test. For the intact knee the resultant load for all posterior displacements peaked at extension and at mid–flexion (Figure 6.11).

6.3.2 Implanted pressure transducer

Typical plots of transducer output against A-P displacement are shown in Figure 6.12. Calibration curves for the transducer–ligament bundle systems are shown in Figure 6.13. The calibration coefficient varied with flexion angle but showed no pattern, therefore an average value was used for each bundle. The maximum deviation from this average was 17%. The computed results for bundle loading are shown in Figure 6.14. The aPC was slack in extension and tightened with flexion but then slackened again after a maximum in mid–flexion. In one specimen the pPC was damaged during the calibration procedure so no result is shown. In two specimens the pPC tightened towards full flexion, when its alignment was better suited to resist posterior drawer. In one specimen the pPC was also loaded in extension.

6.4 DISCUSSION

I consider that the two studies correlate well and offer the following qualitative explanation of the results. The effectiveness of a ligamentous structure in resisting A-P drawer must be related to its alignment in the sagittal plane relative to the tibial plateau. If a fibre is aligned in parallel with the tibial plateau then the fractional component of its tension that lies in the A-P direction is unity. If the fibre is normal to the tibial

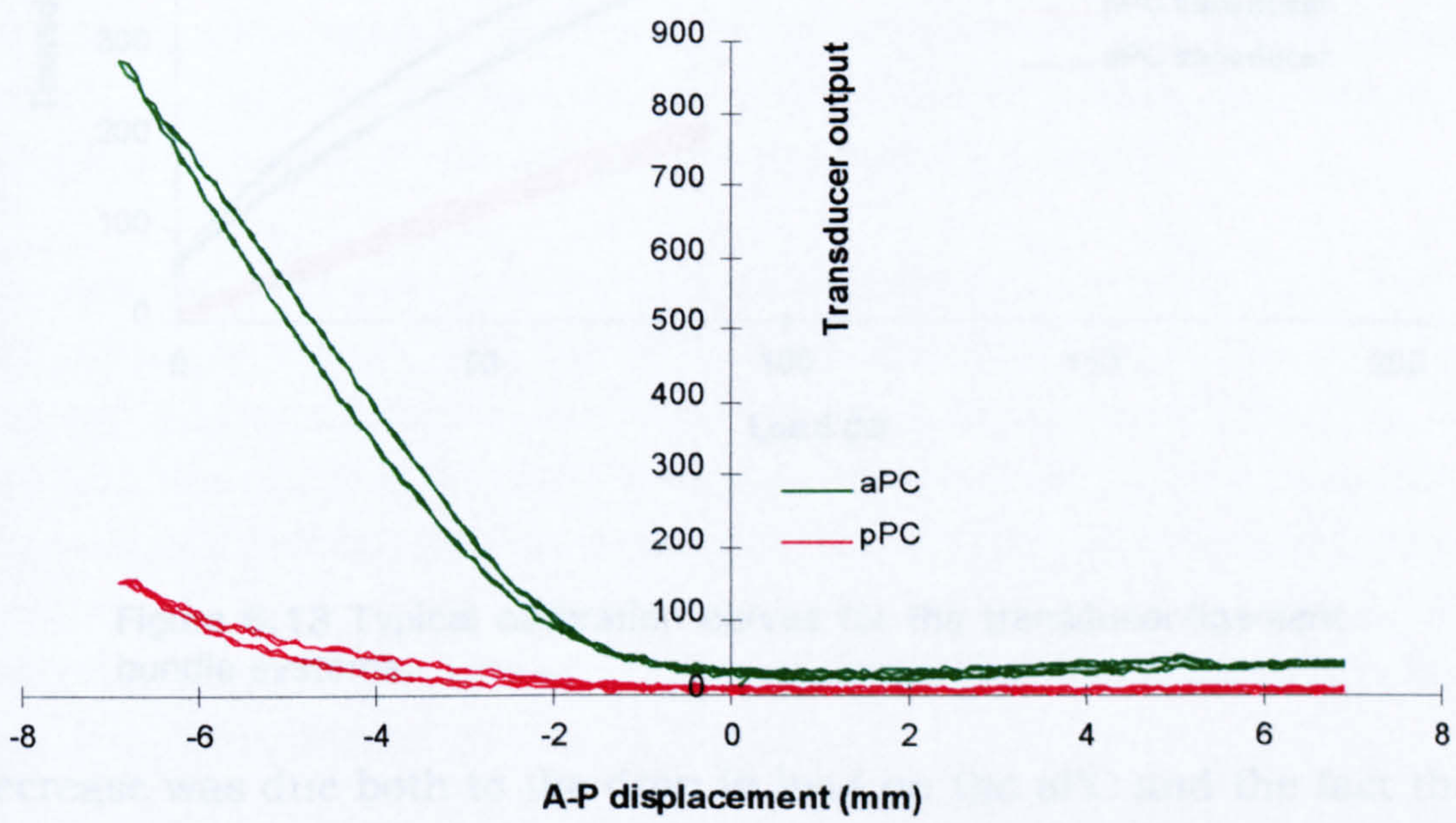


Figure 6.12 Graph of transducer output against A-P displacement for a knee at 90° of flexion. under ±150N drawer.

plateau then the A-P component of its tension is zero.

In extension the aPC carried a small load, as expected since it was visibly slack in the unloaded knee (section 3.1.3.3), but it was well aligned and therefore resisted a small percentage of the drawer force when the tibia was displaced. The pPC was visibly tight but because of its poor alignment it resisted only slightly more drawer force. As flexion progressed the aPC became tighter in the unloaded knee, so the load on the aPC for a given displacement increased as did its contribution to resisting posterior drawer, although its alignment became somewhat less efficient with increasing flexion. The load on the pPC decreased with flexion because it became slacker in the unloaded knee (section 3.1.3.3) but this was offset by its improved alignment, therefore it resisted a slightly increased percentage of drawer force. After mid–flexion the load on the aPC decreased because the pPC tightened. Also, the contribution of the aPC to resisting posterior drawer decreased: this

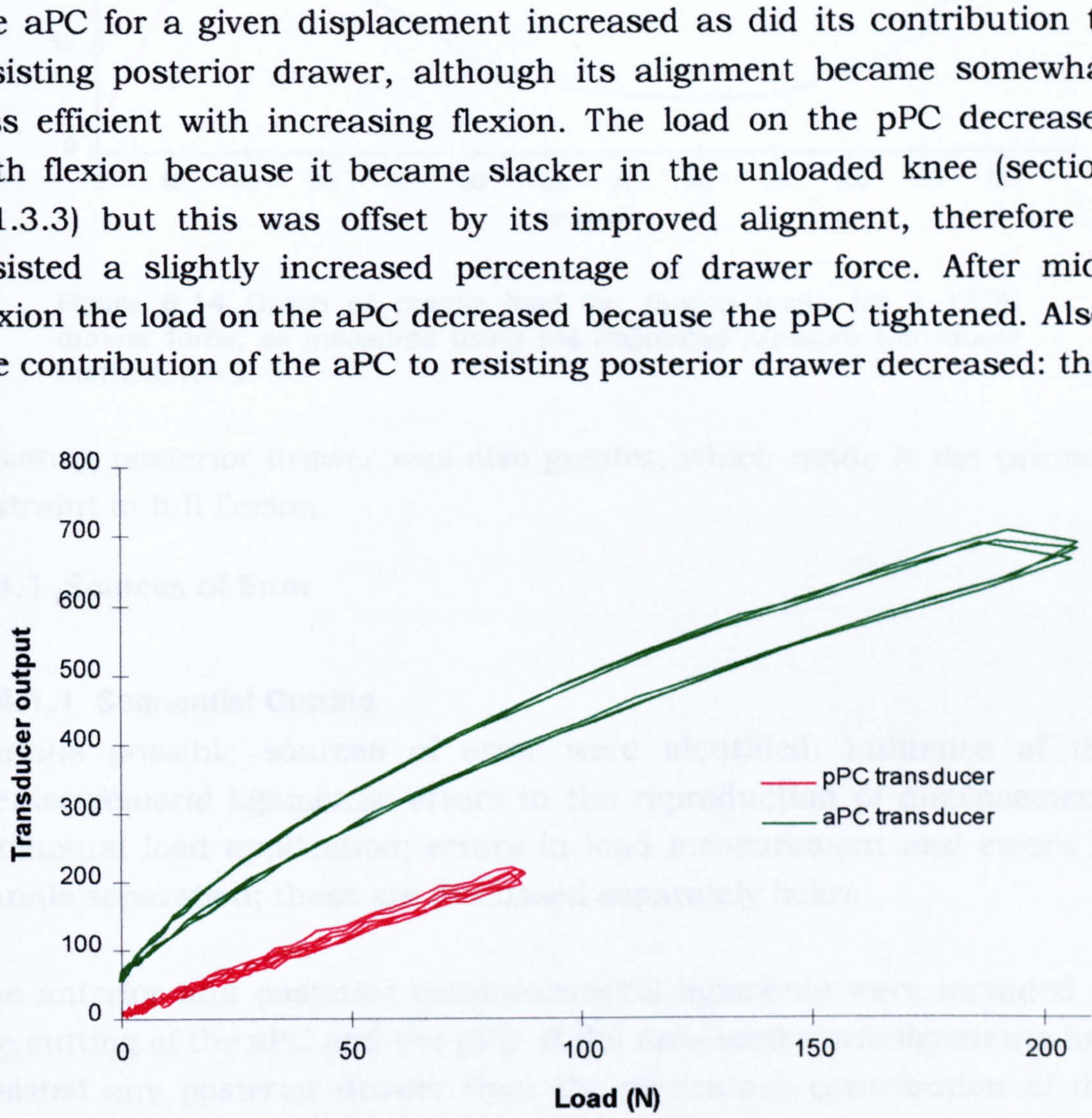


Figure 6.13 Typical calibration curves for the transducer-ligament bundle systems.

decrease was due both to the drop in load on the aPC and the fact that its orientation continued to become less efficient. The load on the pPC increased, and in full flexion it was greater than the load on the aPC. Since the pPC was well aligned in full flexion its contribution to

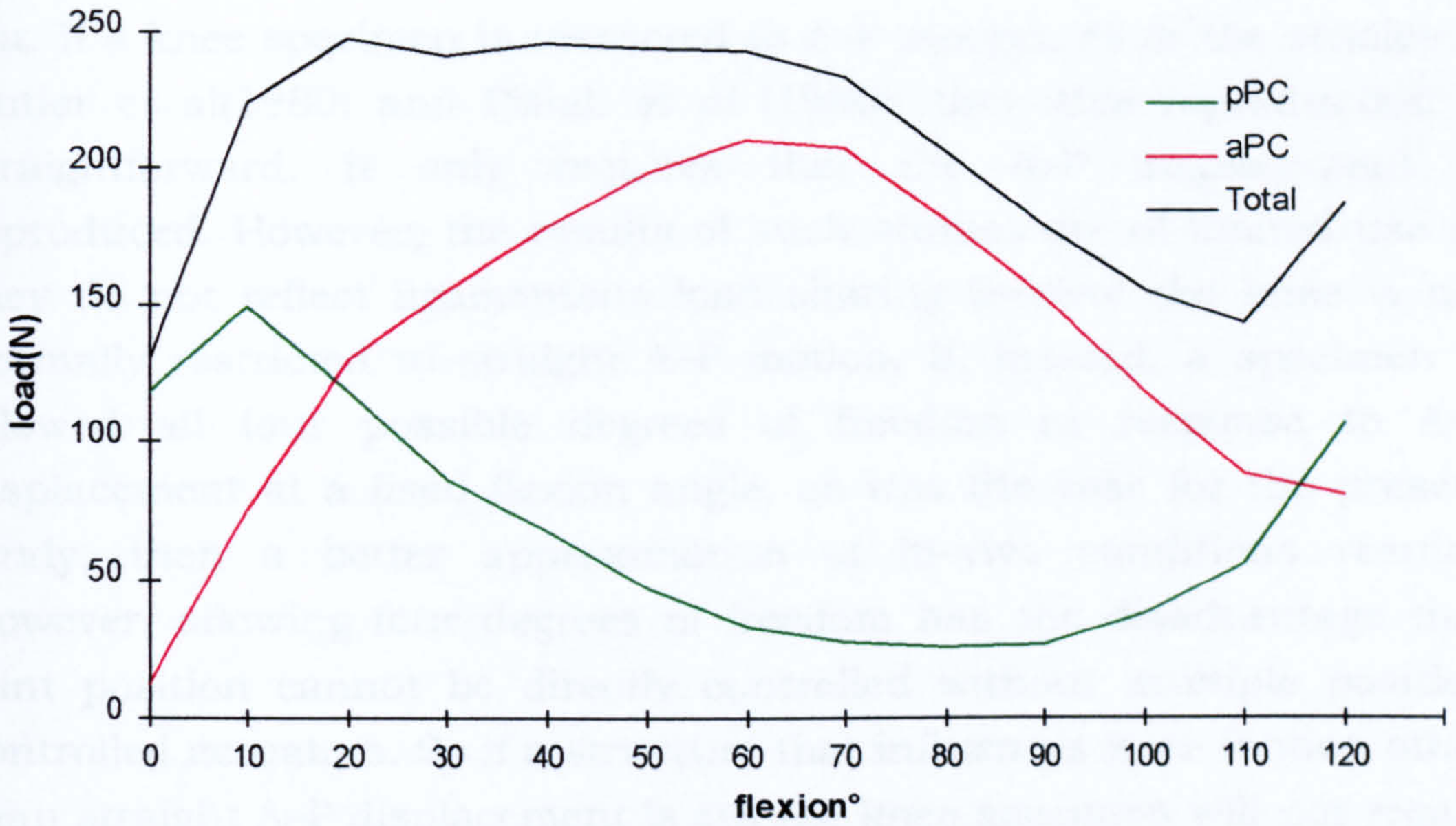


Figure 6.14 Graph of bundle load vs. flexion angle for a 150N drawer force, as measured using the implanted pressure transducer method, n = 3.

resisting posterior drawer was also greater, which made it the primary restraint in full flexion.

6.4.1 Sources of Error

6.4.1.1 Sequential Cutting

Various possible sources of error were identified: influence of the meniscofemoral ligaments; errors in the reproduction of displacement; artifactual load application; errors in load measurement and errors in bundle separation; these are discussed separately below.

The anterior and posterior meniscofemoral ligaments were included in the cutting of the aPC and the pPC. If the meniscofemoral ligaments had resisted any posterior drawer then the percentage contribution of the two bundles of the PCL would have been overestimated. However, It has been shown that lateral meniscectomy has no effect on posterior drawer (Levy et al, 1989). Since the meniscofemoral ligaments insert into the posterior horn of the lateral meniscus their effect on posterior drawer has, therefore, been shown to be negligible.

The technique of selective cutting relies on the reproduction of joint geometry, in response to a certain displacement, after each structure is

cut. If a knee specimen is restricted to A–P motion, as in the studies of Butler et al(1980) and Piziali *et al* (1980), then this reproduction is straightforward: it only requires that the A–P displacement is reproduced. However, the results of such studies are of limited use as they do not reflect ligamentous load sharing in–vivo: the knee is not normally restricted to straight A–P motion. If, instead, a specimen is allowed all four possible degrees of freedom in response to A–P displacement at a fixed flexion angle, as was the case for the present study, then a better approximation of in–vivo conditions results. However, allowing four degrees of freedom has the disadvantage that joint position cannot be directly controlled without multiple position controlled actuators. So if a structure that influences knee motion other than straight A–P displacement is cut the knee specimen will not regain its original geometry when subject to the repeated A–P displacement. It has been previously reported that cutting the PCL reduces the coupled external tibial rotation associated with posterior drawer (Fukubayashi et al, 1982; Gollehon et al, 1987; Grood et al, 1988). In the present study it was qualitatively observed that cutting the PCL reduced the coupled external tibial rotation. This error in the reproduction of geometry may have caused the percentage contribution of the combined other structures to be underestimated: if a moment had been applied to the tibia to maintain the coupled external rotation after the PCL had been sectioned then the measured load, for a certain displacement, could

In order to quantify this error the tibial holder was instrumented so that displacements could be measured to within 0.5mm and rotations to within 1°. A knee specimen was then put through an A-P drawer of ±4mm before and after section of the PCL. It was found that there were small (range 0–2.5mm) coupled medial–lateral and compression-distraction translations. Within the limits of measurement these translations were unchanged by section of the PCL. There were small (<2°) coupled adduction–abduction rotations which were also unchanged by section of the PCL. At 60°, 90° and 130° there were larger (5.5°–7°) coupled external rotations with posterior drawer. Below 90° of flexion internal–external rotation was unchanged, within the limits of measurement. At the higher flexion angles the coupled external rotation was reduced, by 40% and 70% at 90° and 130° respectively. A cord was wrapped around the cylindrical bone pot holding the tibia so that it was

possible to apply an external rotation torque to the tibia by tying weights to the cord. A torque was applied to the tibia in the PCL deficient knee to restore the coupled external rotation. The required torques were 0.06Nm and 0.03Nm for flexion angles of 90° and 130° respectively. It was not surprising that these torques were so small as the knee was hardly loaded by a 4mm posterior translation: this was true for all specimens in flexion (Table 6.1 - note '%other'). It was concluded that the errors caused by changing coupled motions were much smaller than the errors due to fixing the axis of tibial internal–external rotation, discussed below.

A–P displacements were applied via the tibia, which was free to rotate. This freedom of rotation meant that any medial–lateral misalignment between the axis of the clamp and the rotational axis of the knee resulted in a tibial torque. The potential misalignment error was estimated to be ±5mm. This would have given rise to tibial torques of ±1Nm, ±0.4Nm and ±0.1Nm due to the posterior drawer forces of approximately 200N, 75N and 20N generated by posterior displacements of 6mm, 4mm and 2mm respectively. These artifactual tibial torques would have caused tibial rotations of approximately ±7°, ±5° and ±1°. At the time of writing the author was not aware of any published data for the rotational stiffness of the knee during posterior drawer so these angles were based on the tibial internal–external torque–rotation curves stiffness given by Markolf et al(1976). It is likely that the rotational stiffness of the knee is higher when the tibia is posteriorly displaced. Misalignment would have resulted in the overestimation of posterior displacement. This is because it was the displacement of the axis of the clamp that was measured: when the clamp axis had been posteriorly displaced the rotational axis of the tibia would have lagged by a distance equal to the misalignment error times the sine of the resultant tibial rotation. So, based on the tibial rotational stiffness assumed above, the posterior displacements were: $6^{+0}_{-0.6}$mm, $4^{+0}_{-0.4}$mm and $2^{+0}_{-0.1}$mm. Any misalignment error that caused an external torque would have been constant for a given specimen and would not have affected the ratio of contributions to resisting posterior drawer, although it would have blurred any difference between the ratios at different displacements. Since the PCL caused coupled external rotation in posterior drawer it is reasonable to assume that it would have increased tibial internal rotational stiffness. Therefore, any

misalignment error that caused an internal torque would have resulted in less overestimation of posterior displacement when the PCL was intact. This would have resulted in the contribution of the combined other structures being underestimated.

The loads were measured to ±0.25N from the Instron chart recorder which was accurate to ±1N for the range used. This resulted in a probable error of ±2% in calculation of the percentage contribution. From Figure 6.9 it can be seen that 'neutral' apparently moved posteriorly as the PCL was cut. This phenomenon was most noticeable around mid–flexion when the joint was tightest (Figure 6.11): in this region the cruciates were taut in A-P neutral. When the PCL was cut the tension in the ACL was no longer balanced by a PCL tension so A-P neutral moved posteriorly to a point where the ACL was relaxed. If the percentage contributions had been calculated for a posterior displacement that did not relax the ACL then the *negative* contribution of the ACL would have been included in the contribution of the 'combined other structures'. This would have caused the positive contribution of the 'combined other structures' to be underestimated. If the percentage contributions had been calculated for a posterior displacement that did relax the ACL then the results would have been unaffected. For 4mm and 6mm posterior drawer the ACL was completely relaxed but for the 2mm posterior displacement this was not always the case. Therefore, the 'true' percentage contribution of the combined other structures was underestimated for 2mm posterior displacement.

This study tested the hypothesis that there is a difference in the function of the aPC and pPC in posterior drawer. In order to reach conclusions from the results, given possible errors in bundle separation, it is necessary to consider the effect that such errors would have had if the hypothesis were false and if it were true. If there were no difference in the function of individual fascicles then separating the PCL along any lines would have yielded load share/flexion angle curves of the same shape and proportion for each separated bundle: the height of the curve at any angle of flexion would have been directly proportional to the area of the bundle. This would also have been true for the average of these curves, no matter how variable the separation. Therefore, a false positive result was not possible. If there were a difference in the

function of the two bundles then inappropriate bundle definition could have failed to demonstrate it: if fascicles of opposite function had been grouped together then a false negative would result. Assuming that the bundles were appropriately defined, errors in bundle separation would have reduced the apparent difference between the bundles when the results were averaged. So, from the above it is safe to conclude that the positive result, showing a difference in the function of the aPC and pPC, is qualitatively valid but may underestimate the true difference.

6.4.1.2 Implanted Pressure Transducer

The miniature pressure transducers had a linearity of ±1.6%(manufacturer's data), however they were only used over about 1% of their range, so this non–linearity will have had a negligible effect. Other sources of error were due to misplacement of the transducers, cutting bone blocks and calibration. These errors are discussed below.
The transducers were implanted into the middle of each bundle so their output was a measure of the average load on the central fascicles. Variability of placement will have resulted in a reduction in average differences between bundles, following the same argument as above for errors in bundle separation.

When the bundles were separated from each other there was a loss of fibres around the cut edge of the bone block, although care was taken to minimise this. Fibres near to the edge of the cut may also have had their function impaired due to damage to their insertions. The consequences of this fibre loss depended on whether or not the lost/impaired fibres had exerted a pressure on the implanted pressure transducers when under tension.

The loss/impairment of fibres would have led to an increase in the pressures applied to the transducers for a given calibration tension: the remaining fibres, including those exerting a pressure on the transducers, would have carried a higher individual load. Therefore, the pressure transducer output for a given calibration tension would have been overestimated which would have caused the loads in the intact bundles to be underestimated. If the lost/impaired fibres had exerted a pressure on a transducer in the intact PCL then their loss would have led to a reduction in the pressure applied to the transducer for a given calibration tension. This artifactual reduction in pressure could have

been greater than the artifactual increase in pressure described above. If so, then the pressure transducer output for a given calibration tension would have been underestimated which would have caused the loads in the intact bundles to be overestimated.

The largest errors were probably associated with the accuracy of calibration. The transducer–ligament system response to load was treated as linear although this was not always the case (Figure 6.13). The direction of loading during calibration was not precise or accurate for individual knees. I did not think that greater precision in the angle of loading would have yielded more accurate results as the use of a single axis test machine to reproduce the loading caused by unconstrained posterior drawer will have introduced equal or greater errors.

If further work were to be carried out using this technique then I would recommend that a system be designed to record the relative positions of the tibia and femur during A-P drawer and then reproduce these positions during calibration.

6.4.1.3 Summary

In summary, all the errors in methodology discussed above will have caused the differences between the bundles and between the different displacements to be obscured. Errors in the reproduction of displacement may have caused the contribution of the combined other structures to be underestimated. Therefore, the observed differences between the bundles and between the different displacements are qualitatively valid but may underestimate the true difference.

The precision of the pressure transducer study was not great, but I think that it was sufficient to accurately demonstrate the gross difference that was found in the behaviour of the two bundles of the PCL over the range of flexion.

6.4.2 Clinical Relevance

This study has shown, by selective cutting, that at low flexion angles posterior drawer was primarily resisted by structures other than the PCL. This is in line with some previous studies of ligament function

which have shown that the arcuate and lateral collateral ligaments resisted posterior drawer at flexion angles below 30° (Gollehon et al, 1985; Grood et al, 1988) and that the load on the PCL was minimal in extension and rose with flexion up to 90° (Jasty et al, 1982).

I conclude that the primary function of the pPC is to resist posterior drawer in full flexion and not in extension and that of the aPC is to resist posterior drawer from partial flexion to full flexion. Figure 6.11 shows that the load for all displacements peaked at extension and in mid–flexion. These peaks imply that stability at these flexion angles was most important for correct joint function. Since posterior drawer was primarily resisted by 'secondary' structures and by the aPC in these regions I hypothesise that to enable walking and running after PCL rupture it would only be necessary to reconstruct the aPC as long as the 'secondary' structures were intact. However, if the 'secondary' structures were deficient reconstructing the aPC and/or the pPC would not restore normal joint stability near extension and a peripheral reconstruction would be required. For activities requiring stability in deep flexion, such as snow boarding, a double bundled aPC and pPC reconstruction might be required.

6.4.2.1 Isometricity

The results of this study suggest that the contributions of the PCL fibre bundles to resisting posterior drawer and the forces generated in those bundles over the range of flexion are dependent on three factors: the initial tautness of the bundle at A–P neutral, the orientation of the bundle relative to the applied drawer force and the mechanical properties of the bundles. The common clinical practice of 'isometric' reconstruction aims to ensure that the initial tautness of the graft/implant is constant over the range of flexion so that the graft/implant is not overloaded or excessively slack as the knee moves. However, this procedure only takes one of the above factors into account. Different 'isometric' reconstructions will carry different loads and have different effects on posterior drawer depending on the orientation of the graft/implant over the range of flexion. Previous studies have shown that it is possible to achieve a near–constant initial tautness over the range of flexion using a femoral drill hole in a number of positions (Sidles et al, 1988; Grood et al, 1989). However, each of these possible 'isometric' reconstructions will have a different

load/displacement/flexion angle characteristic, such as is shown for the intact PCL in Figure 6.11. Since there is no reason to suppose that any other load/displacement/flexion angle characteristic would be superior to that of the intact knee and because isometricity does not result in constant graft/implant tension in response to a drawer force there is no justification for attempting an isometric PCL reconstruction rather than an anatomic aPC and/or pPC reconstruction.

7. IMPLANT DESIGN AND DEVELOPMENT

7.1 INTRODUCTION

The restoration of normal joint function after ligamentous rupture clearly requires the restoration of normal anatomy. At some time in the future this will be achieved, probably using some combination of scaffold, tissue culture and biochemical factors. However, the restoration of normal, complex ligamentous anatomy is beyond the scope of current technology. In fact the first step of the process, defining normal anatomy and its function, is far from complete. So, ideal solutions are not yet available but patients still need treatment. All surgical intervention is based on the idea that some adjustment to the anatomy of a patient will have a net beneficial effect. There are no cases where this adjustment results in the restoration of normal anatomy. The adjustments are traditionally based on a surgeon's 'best guess'. These 'best guesses' have been improved in the light of clinical experience and, more recently, engineering analysis.

The studies reported in this thesis have shown that the PCL can usefully be divided into two functional bundles, the aPC and the pPC. It has been demonstrated that these bundles have different mechanical and material properties and different effects on posterior stability over the range of flexion. The purpose of these studies was to enable a reconstruction system to be designed to restore the A-P stability of a PCL deficient knee. The conclusions reached about reconstruction of the PCL are relevant to all reconstruction methods but particular attention has been given to the design of a synthetic replacement.

The study of the mechanical properties of the PCL reported in section 4 showed that the aPC made up the bulk of the ligament. The bundle loading study reported in section 6 showed that the function of the aPC was to resist posterior drawer from mid–flexion to full flexion. So, to enable normal joint function after isolated PCL rupture it would certainly be necessary to reconstruct the aPC. The pPC was shown to have one sixth of the strength of the aPC (section 4) and to resist posterior drawer in deep flexion (section 6). So, to completely restore A-

P stability over the range of flexion it would also be necessary to reconstruct the pPC.

Different patients put different demands on their injured joints and have different expectations from surgery. Therefore, there needs to be a range of approaches to PCL deficiency. Many people cope well with conservative treatment. This can be explained by the results of section 6 which showed that the PCL was not responsible for posterior stability at lower flexion angles: PCL–deficient limbs can be stable during weightbearing in normal gait. If posterior stability is required over a larger range of flexion, to enable running/stopping/cutting then the aPC must be reconstructed. If posterior stability is required in deep flexion then the pPC must also be reconstructed. The choice of none, single or double bundled reconstruction is controlled by the balance between patient expectations and the trauma associated with surgery: the more complete the reconstruction the more complex and invasive the surgery.

7.2 AUTOLOGOUS Vs SYNTHETIC GRAFTS

From a purely mechanical point of view there is no real difference between the sets of 'organic'(autografts/allografts) and synthetic graft types. Autologous and synthetic materials have an overlapping range of material properties which can yield grafts of various strengths and stiffnesses. If an autologous graft and a synthetic graft of matched mechanical properties were to be used to reconstruct a knee in-vitro with the same fixation and under the same tension they would give identical results. The mechanical effect of an implant is dependent on its placement, its stiffness and its initial tension: none of these parameters are connected to the *source* of the material used.

As was discussed in section 1.6, there have been several badly designed synthetic ligament implants. The basic attractions of a synthetic implant, no donor site morbidity and surgical simplicity, led to an ill–considered fashion during which quite a large number of badly designed implants were used. The inevitable avalanche of bad results led to the current, and in the author's view, equally ill–considered fashion for dismissing synthetic implants.

It is not the contention of this thesis that synthetic implants should replace autologous techniques. Rather, it is suggested that there is a place for a synthetic PCL implant in the current range of options available for the treatment of PCL insufficiency. In the future it is unlikely that large amounts of autologous tissue or non–resorbable synthetic prostheses will be used in ligament surgery as neither option is ideal, but for now these must suffice.

7.3 SINGLE Vs MULTIPLE BUNDLES

While a double bundle reconstruction may be better in theory there are potential problems. There is some experience of double bundled reconstructions for the ACL. Radford and Amis (1990) found that, in–vitro, double bundled reconstructions better restored anterior stability at 20° and 90° than a single reconstruction. In the light of these encouraging results a double bundled ACL implant was tested in a sheep model Radford et al (1994). Twenty four sheep were used to test three reconstructions.

- Single bundle 'over the top' (OTT). A tibial drill hole was used but no femoral drill hole: the implant was wrapped over the top of the lateral femoral condyle to a screw and grommet fixation on the lateral side of the distal femur. This reconstruction was tight in extension and slackened with flexion.
- Single bundle 'through the condyle' (TTC). Femoral and tibial drill holes were used. This reconstruction was tight in flexion and slackened with extension.
- Double bundle combined OTT and TTC. The two reciprocally acting single bundle reconstructions were combined, using two separate tibial drill holes, to reproduce the posterolateral and anteromedial bands of the natural ACL.

The reconstructions used Apex style implants that were matched to the natural ovine ACL. The animals were killed at six months post–op for analysis.

Radford et al (1994) reported that all groups showed tissue ingrowth and that there was no evidence of implant debris in the synovium. They did not find any instances of anchorage failure or implant failure. However, it was found that all the reconstructions slackened over the implantation period. Implant slackening appeared to be related to the

resorption of bone at the points of stress concentration where the implants entered bone tunnels. It was found that the degree of bone resorbtion increased with increasing number of bone tunnels: one tunnel OTT reconstructions faired best and three tunnel double bundled reconstructions were the worst. Radford et al (1994) also found that implant slackening correlated with joint degeneration; they therefore recommended that double bundle reconstructions not be used.

The results of the doubled bundled human PCL reconstructions of Laboureau and Dericks (1992) were much more favourable than the results of the sheep study of Radford et al (1994). It is possible that the PCL reacts better to a two bundled approach than the ACL, but I can see no obvious mechanism for this. It is also possible that the differences result from inter–species variation in the remodelling response of bone.

Although good results for double bundled reconstructions have been reported the study of Radford et al (1994) invites caution. In the case of the PCL the pPC makes only a small contribution to the stability of the knee during most activities: unlike the ACL reconstruction reported by Radford and Amis (1990) the extra PCL bundle does not 'double' the functionality of the implant. Since the addition of a pPC implant may compromise the function of an aPC implant it was thought best to establish if a single implant functioned well clinically before investigating more complex reconstructions.

7.4 BASIC LIGAMENT DESIGN

The commercially produced Apex polyester ligament had proved to be reliable in clinical trials (Harries and Amis, 1995) and appeared to encourage the growth of a neo-ligament (Figures 1.33 and 1.34). Therefore it was decided that the ligament should be a simple

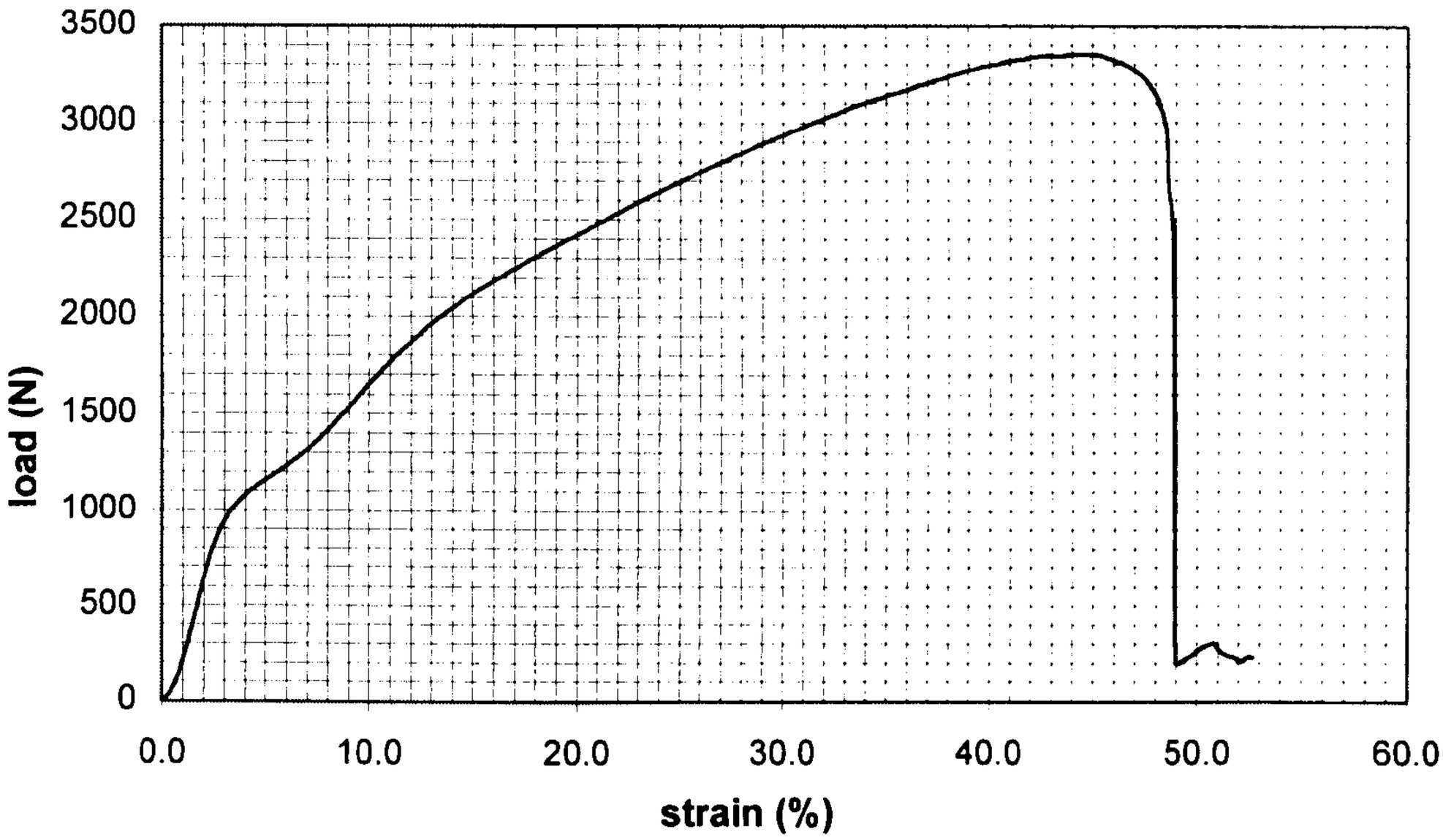


Figure 7.1 Graph of load against strain for a 1000 turn trial implant

modification of the tried and tested Apex design. To check the mechanical properties of the design a 223mm long ligament was made with 1000 turns of low modulus polyester yarn*. The yarn consisted of 20 untwisted filaments which had had the minimum of processing to ensure biocompatibility: there were no titanium oxide particles which are used in normal yarns to give a matt finish. Before mechanical testing the ligament was subjected to the autoclaving sterilisation procedure that would be required prior to implantation: steam autoclave at 121°C for 45 minutes. The ligament was then loaded to failure at 100mm/min in the Instron materials testing machine. The end loops of the implant were placed over 10mm long sections of 8mm diameter steel rod which were clamped in the jaws of the Instron. The rod was of the same diameter as the grommets that were to be used to anchor the implant.

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* ICI Bright polyester, 55fr20 type 1001

The test ligament failed at a load of 3.35kN and a strain of 44.2% (Figure 7.1). There was a non-linear 'toe' region below 1% strain. Between 1% and 3% there was a linear region. Over 3% the stiffness of the implant sample decreased until failure occurred.

The natural aPC had a 'toe' region below 6%±5% strain (data from study in section 4), behaved linearly up to 13%±6% strain and reached maximum load at a strain of 18%±5% (Table 4.2). It was not possible for the implant to exactly match the natural aPC, both because of inter–specimen variations and because of the material properties of the implant polymer. Other materials were not investigated because the required biocompatibility tests were beyond the scope of this project. Also, the Apex implant had demonstrated tissue ingrowth which was thought to be essential for the long term survival of reconstructions.

Because an exact match was not possible the implant design involved some compromise. Overengineering the implant, increasing the number of turns to increase the strength and stiffness, was not an option for two reasons. Firstly, a larger implant would require a greater insult to the bone at implantation and secondly over–restraining posterior translation would alter the balance of forces in the knee, possibly leading to the sort of degenerative changes that a ligament reconstruction seeks to prevent. Implant stiffness effects have been studied for the ACL in a sheep model by Cooke and Goodship (1986). In their study the ACL was cut and reconstructed by a 'stiff', 'matched' or 'lax' implant. It was found that the stiff and matched implants significantly reduced lameness but that the stiff implants resulted in significantly more cartilage damage after 6 months.

The results of the study in section 4 suggested that to mimic the rupture resistance of the average natural aPC the implant should generate a load of 4kN(1.6kN*2.5) at a strain of 18% (section 4.4.2.). However since patients requesting PCL reconstruction are generally more physically active than average the implant design was based on the mean plus one standard deviation. This required a load of 5.25kN at a strain of 18%. From the graph in Figure 7.1 it was calculated that to satisfy this 'failure' criterion the implant had to be made of 2283 turns of the selected yarn. The failure load could not be directly measured

because it was beyond the maximum load of the available testing equipment.

An implant of 2300 turns, to satisfy the failure criteria, would have a stiffness of 423N/mm for a length of 223mm (estimated from Figure 7.1). This length, or greater, would be required to allow for the tunnels and extra–articular anchorages, although the intra–articular portion would only be as long as the aPC which was found to be 35mm (Table 4.2). It is likely that in vivo this stiffness would increase as the active length of the implant decreased with bone tunnel ingrowth. If the implant were to be perfectly anchored in the bone tunnels then the stiffness would rise to an estimated 2.7kN/mm. The required stiffness to mimic the natural aPC at the mean plus one standard deviation was estimated to be 830N/mm (section 4.4.2). The implant would match the stiffness of the natural aPC if its active length were to reduce to about 110mm. It appeared that an implant of 2300 turns would initially be too compliant but that with time it would reach the required stiffness and possibly exceed it. Therefore the stiffness criteria demanded that the implant be no more than 2300 turns.

The remaining mechanical parameters were the 'toe' region and the linear behaviour limit. For a parallel fibre implant the extent of the 'toe' is defined by the material properties of the constituent fibres. For the polyester used in the test implant the 'toe' was 1% (Figure 7.1). The 'toe' region could have been expanded up to the 'normal' average by using a knitted or woven structure but this would have negated the advantages of the parallel fibre structure. A knitted/woven structure would have been bulkier and would not have been so amenable to tissue ingrowth: greater bulk would require the removal of more bone and a lack of ingrowth would probably compromise the long term survival of the implant. Therefore, the mismatch in the 'toe' region was accepted.

With regard to linearity, the average natural aPC was found to behave linearly up to a strain of 13%±6% (Table 4.2) and an age adjusted (section 4.4.2) load of 2.9kN±1.5kN. So, again adding one standard deviation to allow for the probable skew in patient population, the implant was required to behave linearly up to a strain of 13% and a load of 4.4kN. The data in Figure 7.1 shows that an implant of 2300 turns would not satisfy either of these conditions: the implant would

behave linearly up to a strain of 3% and a load of 2.1kN. This means that such an implant would be plastically deformed by a posterior drawer that would not permanently damage a natural ligament. Making the implant strong enough to resist a load of 4.4kN without plastically deforming would require 4900 turns. Such an implant would be excessively stiff and require larger bone tunnels. It would have been possible to design a more complex structure that would have reduced the stiffness and allowed 13% length change but this was not done for the same reasons as given in the 'toe' discussion above.

It was considered important to have an implant that was a good match to the natural aPC during normal activity. In normal walking the load on the PCL has been shown to peak, on average, at 330N (Morrison, 1970). Another study by Morrison (1969) has estimated that on descending a ramp (which was found to be the most arduous task for the PCL) the load on the PCL peaked at an average of 1.2kN. These loads are well within the calculated linear region of a 2300 turn implant, so the 2300 turn design was retained. It was accepted that the implant was less resistant to non–rupturing trauma than the natural aPC.

In order to minimise the required tunnel diameter a slight modification was made to the Apex design. Where the implant was bound it was constrained to a circular cross-section, but where it was free it could deform easily. The minimum diameter tunnel was the diameter of the implant section where all the fibres are bound together. Two diameters of half the fibres were 41% wider than one diameter of the whole implant (Figure 7.2). The bindings within the anchor loop were offset so

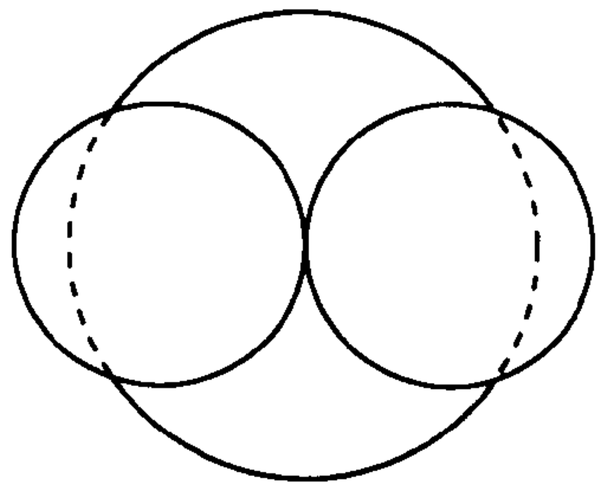


Figure 7.2 Overlay of implant cross-sections.

that they did not have to be pulled through the tunnel together. When the bound sections were adjacent to unbound fibres the loose fibres could deform to fill a circular cross-section more efficiently, so a smaller

tunnel was required. At first a 7mm diameter tunnel was tried, the implant could pass through a straight, smooth, 7mm hole easily. However, in a real bone tunnel the implant jammed so an 8mm diameter hole was used.

The length range of the implant was dependent on the tunnel placement and anchorage method. The sterilisation process also influenced the length range; it was found that the implants shortened by 7% when they were autoclaved.

7.5 TUNNEL PLACEMENT

Tunnel placement was defined by the desired implant insertions, the need to minimise bending, the implant anchorage method and surgical limitations.

7.5.1 Tibia

Given that the tunnel was to start at the anatomical insertion, minimum bending of the implant path would result from the most distal placement of the tibial drill hole. There was a practical limit to distal placement because it is advisable to avoid double piercing the posterior tibial cortex: the popliteal artery could be damaged with serious consequences (Figure 7.4).

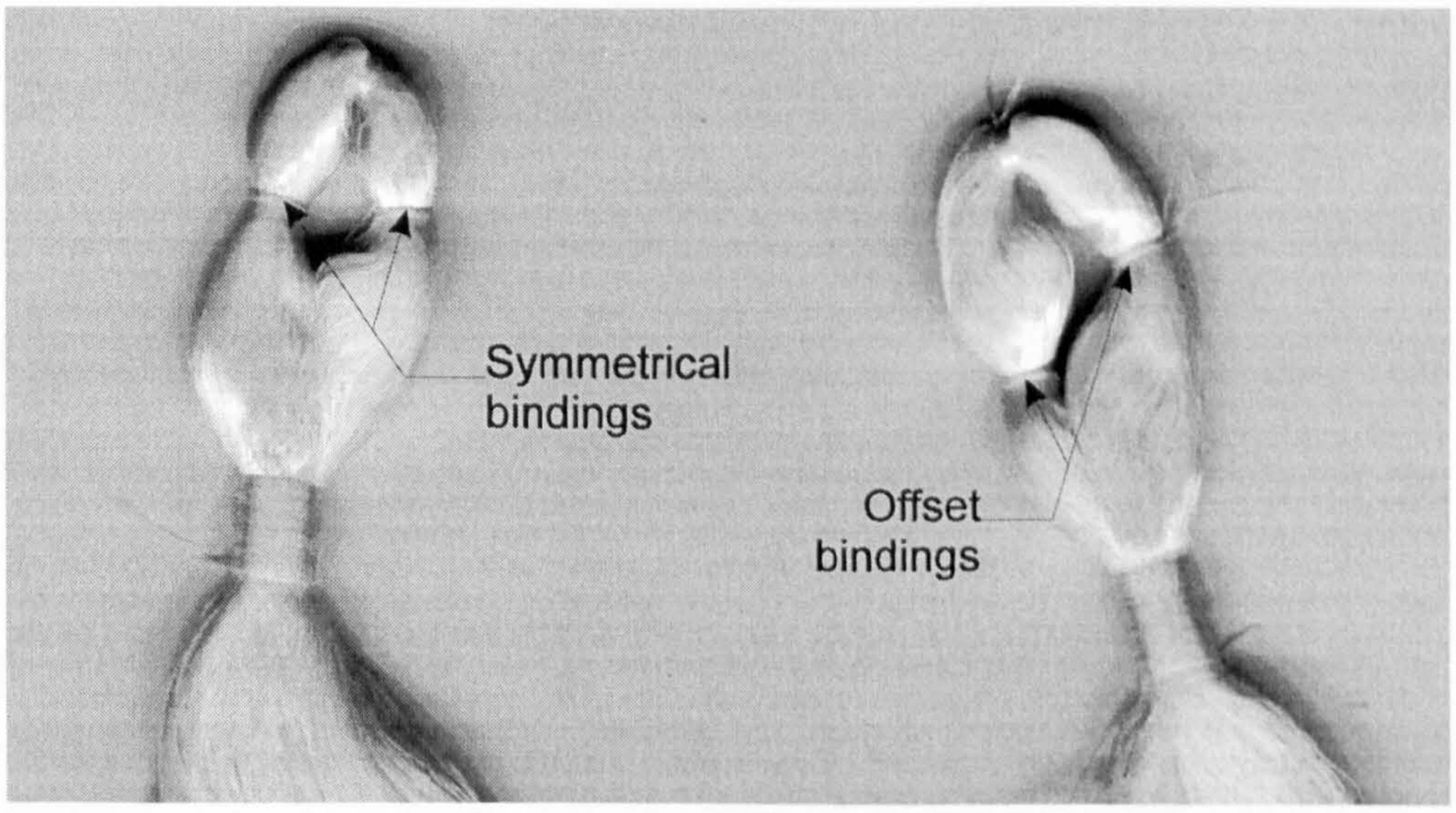


Figure 7.3 PCL implant showing left, symmetrical bindings and right, modified offset bindings.

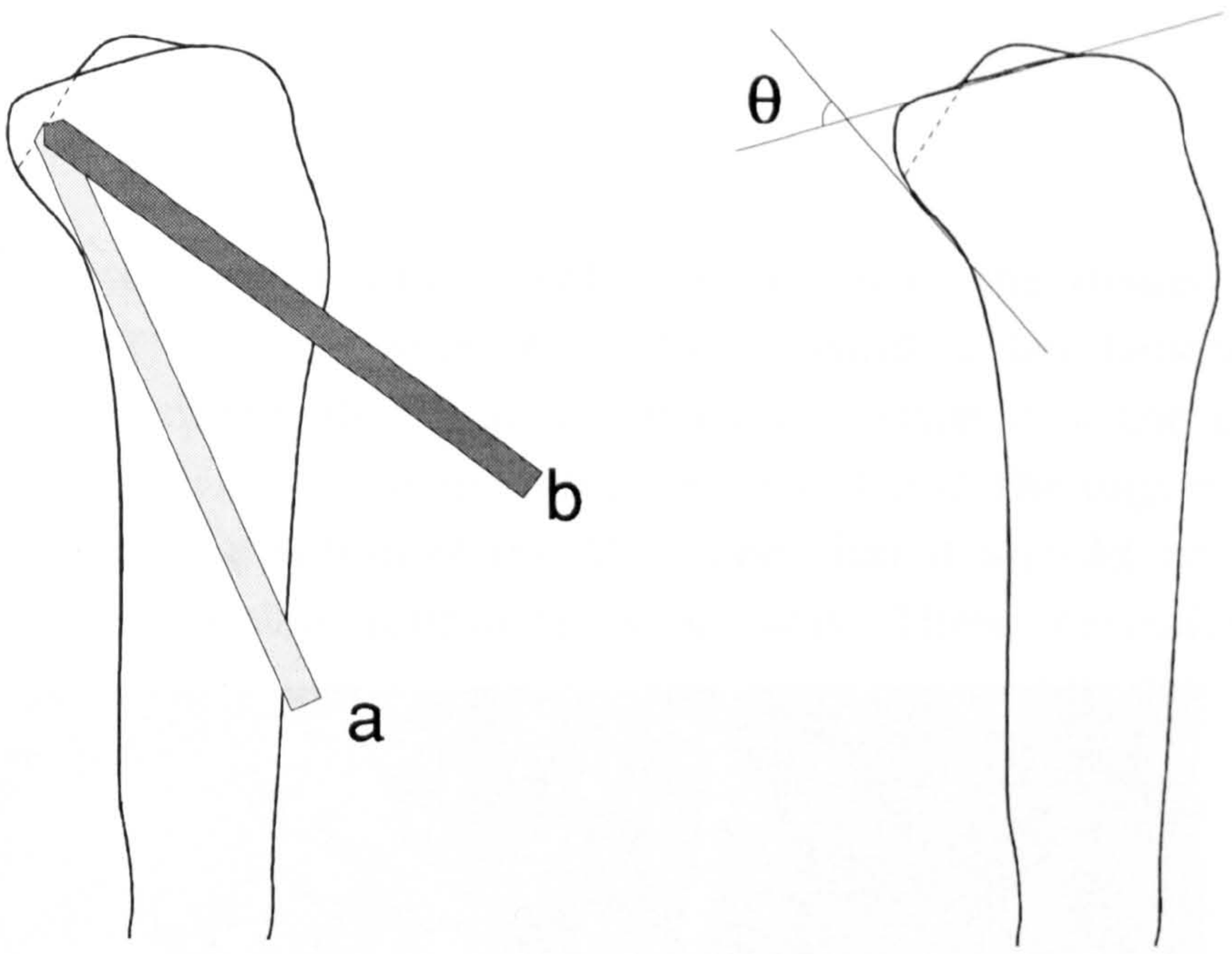


Figure 7.4 Diagrams of the proximal tibia. The figure on the left shows two possible drill holes, (a)&(b). Hole (a) would result in less implant bending but it interferes with the posterior tibial cortex. Hole (b) represents the 'fail-safe' angle chosen. The figure on the right shows the angle (θ) that was measured between the tibial plateau and the posteroproximal cortex of the tibia.

One option was to use a tunnel that was optimally adjusted for individual patients on the basis of a sagittal plane x-ray. However, to reduce the complexity and risk of the procedure it was decided that a 'fail-safe' fixed angle tunnel should be used. To this end a series of 48 sagittal plane X-rays were analysed and the angle between the tibial plateau and the posteroproximal cortex of the tibia was measured (Figure 7.4). It was found that this angle was 54°±5° (range 44°- 65°) so a 'fail-safe' tunnel angle of 45° was chosen. This tunnel placement was also optimal for an Apex transcortical screw and grommet anchorage. The performance of the screw and grommet improves with thicker cortical bone and the cortex of the tibia thickens distally.

7.5.2 Femur

Minimum bending of the implant path would be achieved if the tunnel went from the aPC insertion straight through the femur to the medial epicondyle just proximal to the edge of the articular cartilage. This tunnel placement was unsuitable for the Apex grommet anchorage so a toggle was designed.

7.6 ANCHORAGES

7.6.1 Toggle

There were a number of anatomical restrictions in the design of the femoral toggle. The target area of medial epicondyle lay between the femoral insertion of the MCL and the articular cartilage of the patellar surface of the femur. It was considered important that the toggle should not interfere with the action of the MCL and that it should not cause irritation to the patellar retinaculum or skin. These considerations



Figure 7.5. Pull through test for femoral fixation toggle. The toggle was loaded via a wire that was attached to the crosshead of an Instron. Note that the toggle does not perfectly conform to the medial epicondyle (⟶)

meant that the toggle had to have an A-P length of less than 30mm and be as flush to the bone as possible. A toggle was designed which had a sub–cortical section which would support the implant so that the top of

the anchorage loop was flush with the bone surface. The sub–cortical section was connected to two 3mm thick plates which lay on the surface of the epicondyle. The load bearing area of toggle was 140mm^2. A previous study of ligament anchorages (Amis, 1988) has shown, with old donors, that small carbon fibre toggles failed at 500N with a cortical thickness of 0.5mm. This failure load equates to a pressure of 10N/mm^2. It was therefore hoped that the PCL toggle would carry a load of 1.4kN when tested in specimens from old donors. It has been shown that cortical bone strength decreases by about 5% per decade (McCalden et al, 1993). So a toggle failing at 1.4kN in an 80 year old bone could be expected to resist a load of 1.9kN in 25 year old bone, which is where it would be implanted in clinical practice. This load

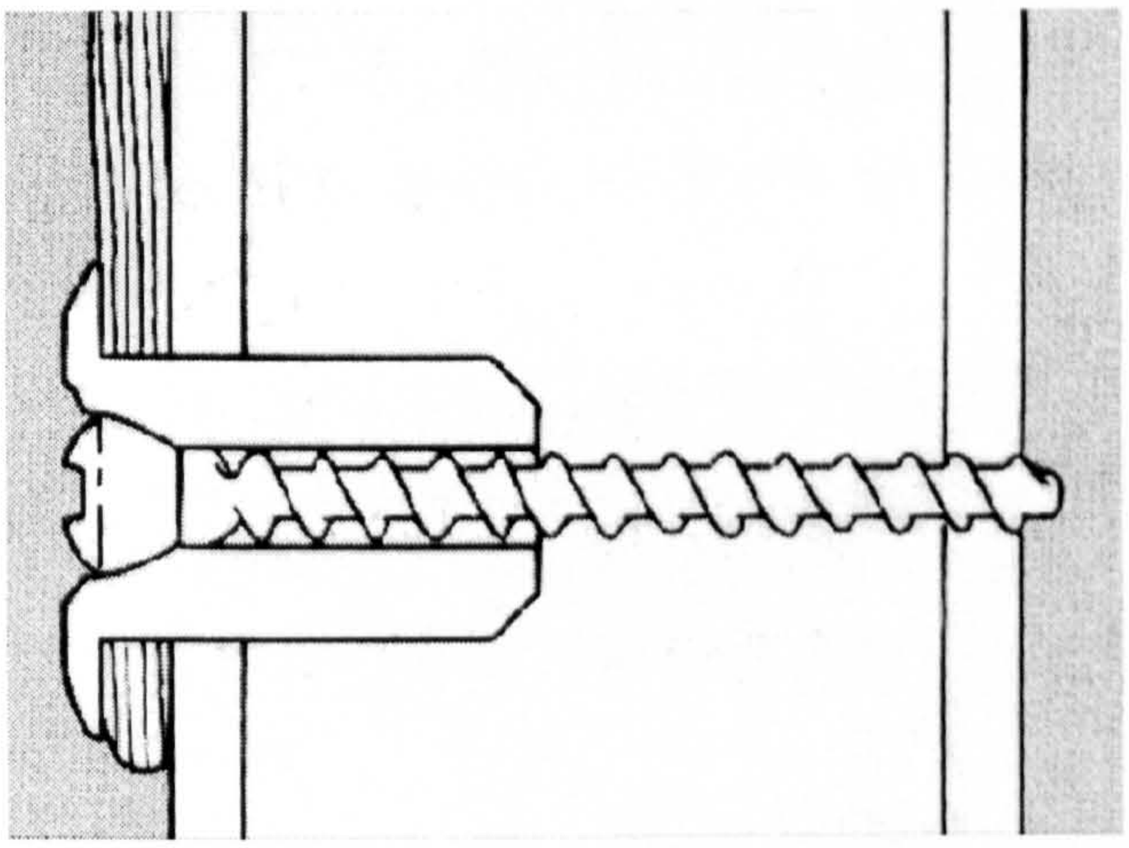


Figure 7.6 Transcortical screw and grommet

would be equivalent to the strength of a grommet and screw in 2.5mm thick cortical bone (Figure 1.35)

A prototype femoral toggle was manufactured and tested in two specimens (Figure 7.5). The results were disappointing: the bone beneath the toggle failed at 500N and 650N. The unexpectedly low failure load may have been due to two factors. Firstly, the toggle did not perfectly conform to the epicondyle and progressive failure was observed. Secondly, the cancellous bone underlying the area tested may have been less dense, or less well aligned for the applied load, than that underlying the area tested by Amis (1988). The first of these factors could have been changed, but inter–patient variation in epicondyle curvature would have meant that customised toggles would be needed. It may be that in-vivo the epicondyle would remodel so as to better support a non-conforming toggle. However, this would require a gradual

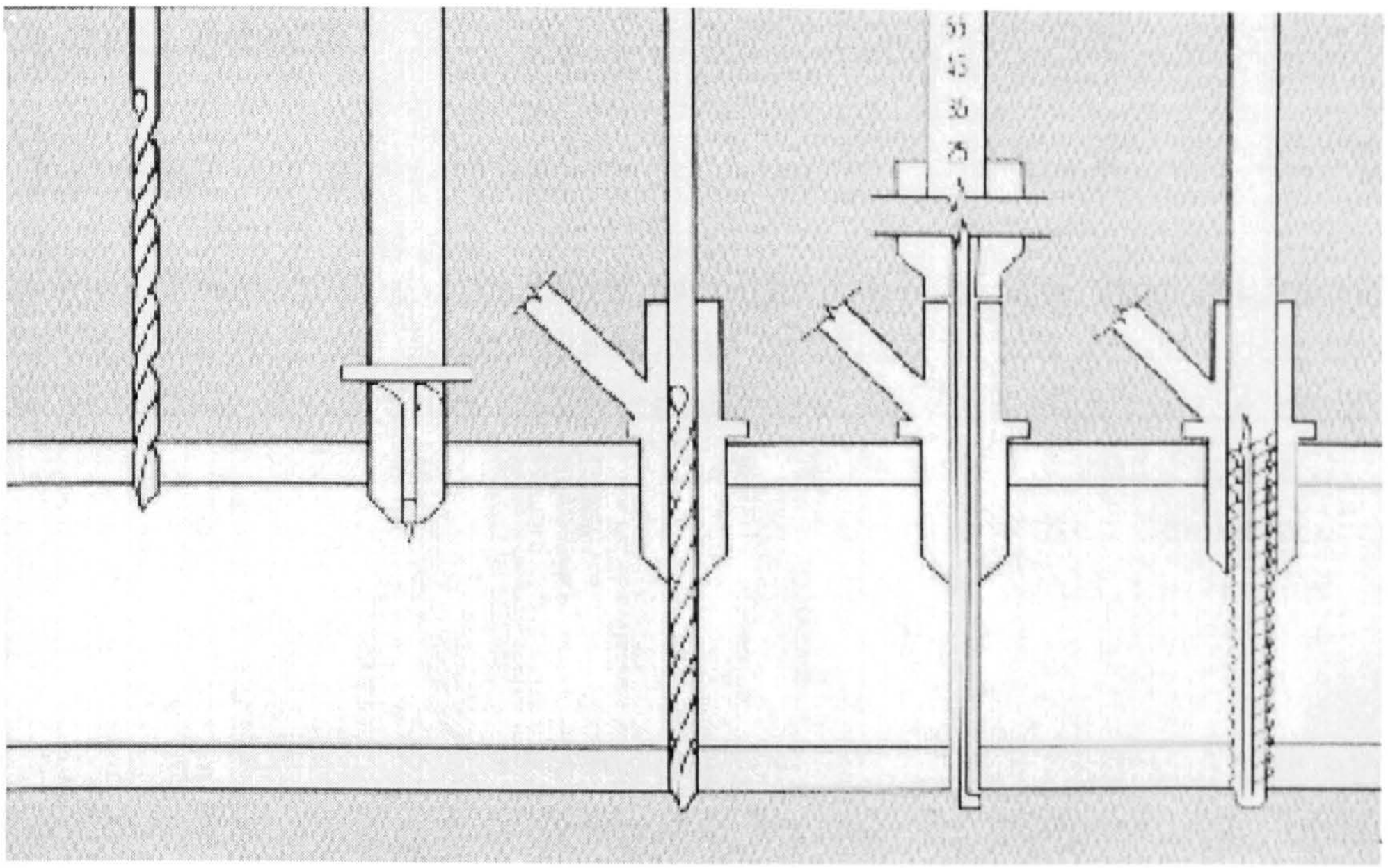


Figure 7.7 Drilling and tapping procedure for Apex transcortical screw and grommet.

increase in load bearing which would remove one of the major advantages of an artificial reconstruction: immediate secure fixation which allows an early return to activity. It was therefore decided that the femoral toggle be rejected in favour of the transcortical bone screw and grommet (Figure 7.6).

7.6.2 Grommets

The original Apex grommet (Figure 7.6), had a diameter that was designed so that overloading the implant caused the grommet to migrate through the cortex rather than leading to bone fracture (Amis, 1988). Therefore, the grommet diameter was not changed. The PCL implant had a larger diameter than the Apex, so the lip of the grommet had to be increased. The procedure for drilling, tapping and transcortical screw selection was the same as for the Apex system (Figure 7.7).

7.7 DRILL GUIDES

7.7.1 Tibia

Existing tibial drill guides hook over the back of the tibia (Surgicraft ABC, LARS, Acufex). A drill guide was designed which would hook over the posterior of the tibial condyles and rest in the notch on the medial side of the lateral tibial condyle (Figure 7.8, Figure 7.9 and Figure D.1). In practice it was found that the hooked drill guide could not be placed over the back of the tibia because the capsule could not easily be released.

It was also realised that placing the implant in the centre of the aPC tibial insertion may not be advisable because of the possibility of the implant cutting through the bone. In section 3.2 it was shown that the

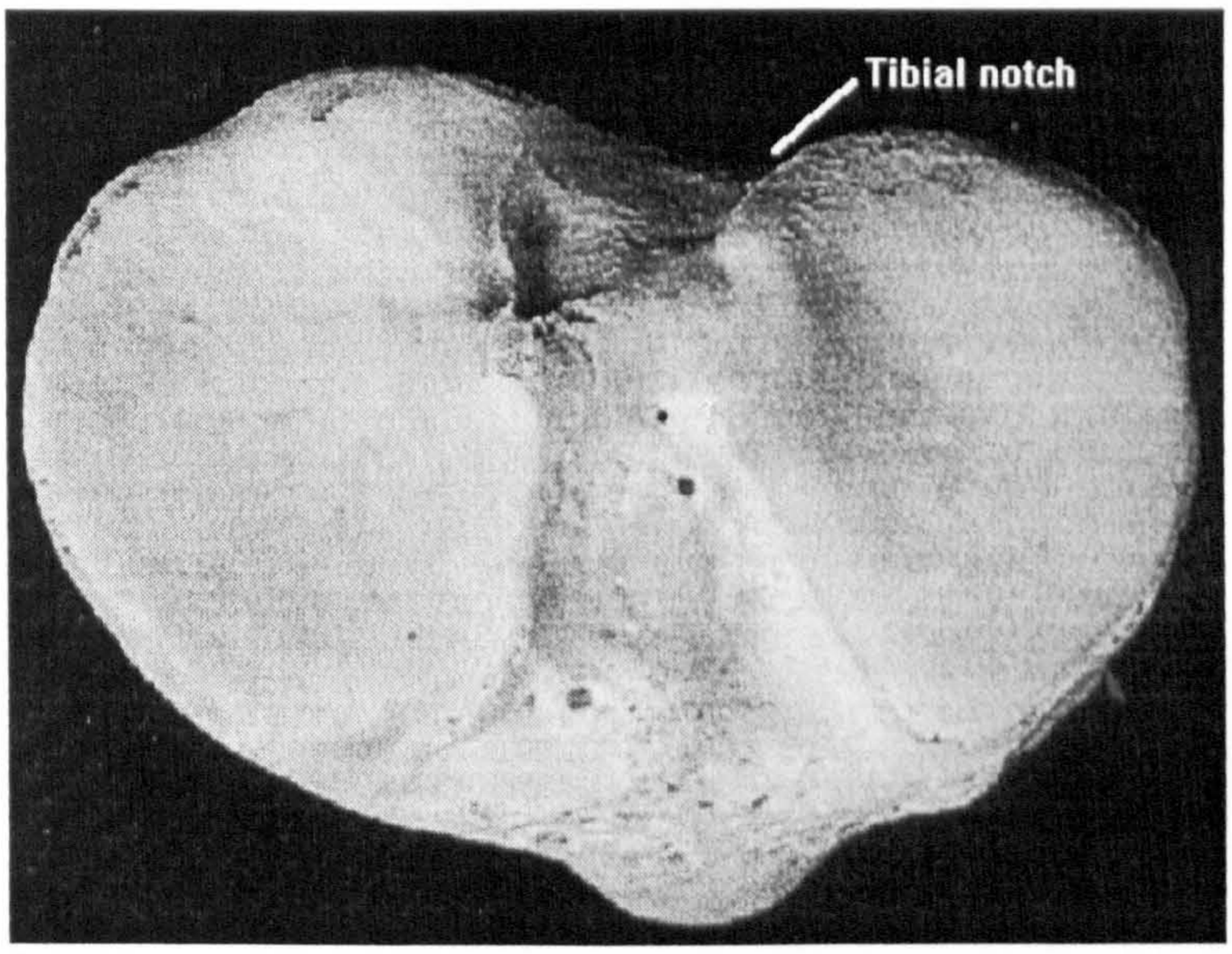


Figure 7.8 Proximal aspect of the tibia showing the notch aimed for by the initial tibial drill guide

cortex of the tibial insertion of the PCL was only 1.3mm thick. It was thought that the implant might migrate through this thin cortex until it came up against the posterior insertions of the menisci which bear onto the intercondylar eminence. This process would effectively slacken the implant by 2-3mm(6-8%) and thus compromise its function. It was thought better to start with the implant at the end of its possible migration. Figure 7.22 and Figure 7.27 show the desired implant insertions.

If the implant was to be placed so that it abutted the insertions of the menisci then it was important that the A-P position of the tunnel was well controlled. If the tunnel was too posterior then implant migration might occur and if it was too anterior then the posterior insertions of the menisci would be damaged. The second drill guide was designed so



Figure 7.9 Tibial drill guide mark one. The hook (⬟) was designed to locate in the posterolateral tibial notch (Figure 7.8). The guide–wire (⟶), held in the guide–wire barrel (⟶), was prevented from excess travel by a specially designed drill stop (⌂).

that the tunnel position was defined by the posterior meniscal insertions and the condyles of the tibia. The end of the drill guide formed a ring of the same diameter as the tunnel (Figure 7.10, Figure D.10). The guide was designed to be inserted through an anteromedial portal and pushed posteriorly and distally before being drawn anteriorly until it rested in the tibial fovea, held between the meniscal insertions and the tibial condyles.



Figure 7.10 Tibial drill guide mark two. Fitted with the guide–wire barrel (⬟), held in position by the thumbwheel (⟶), and a guide–wire (⌂) held in the drill stop (➞) at maximum travel. A close up of the ring at the end of the drill guide is shown inset bottom left.

7.7.1.1 Guide Wire Stop

The danger of overdrilling the guide–wire into the posterior soft tissues was averted by fitting a drill stop (Figure 7.10, Figures D.4-D.6). The drill stop was designed to hold the guide–wire at a fixed position and then release it after drilling. Special guide–wires were manufactured with a depression at the chuck end (Figure D.3). This depression engaged with a stainless steel ball which in turn engaged with the drill stop core. The ball was held in position by a spring loaded outer sleeve. The core of the drill stop ended with a 6mm diameter shaft: this diameter was chosen as the common size for all chuck driven tools to save time when changing tools over. (Figure 7.11 and D.4)

The drill stop was designed so that the guide–wire would stop with its tip just below the cortex. This ensured that the guide–wire would remain in place while it was over–drilled: if it were free then the cannulated drill could push it further, causing damage. The cannulated drill was stopped by the circular tip of the drill guide (Figure 7.12).

7.7.1.2 Leader Passer

For the first drill guide it was envisaged that the implant leader tape would be passed through the tibial hole with arthroscopic forceps. It was thought that this would be feasible because the tibial tunnel was between the condyles of the tibia and not 'over the back' of the tibia, as in some other reconstruction protocols. However, it was suggested by surgeons that this might be more difficult than it sounded, so the

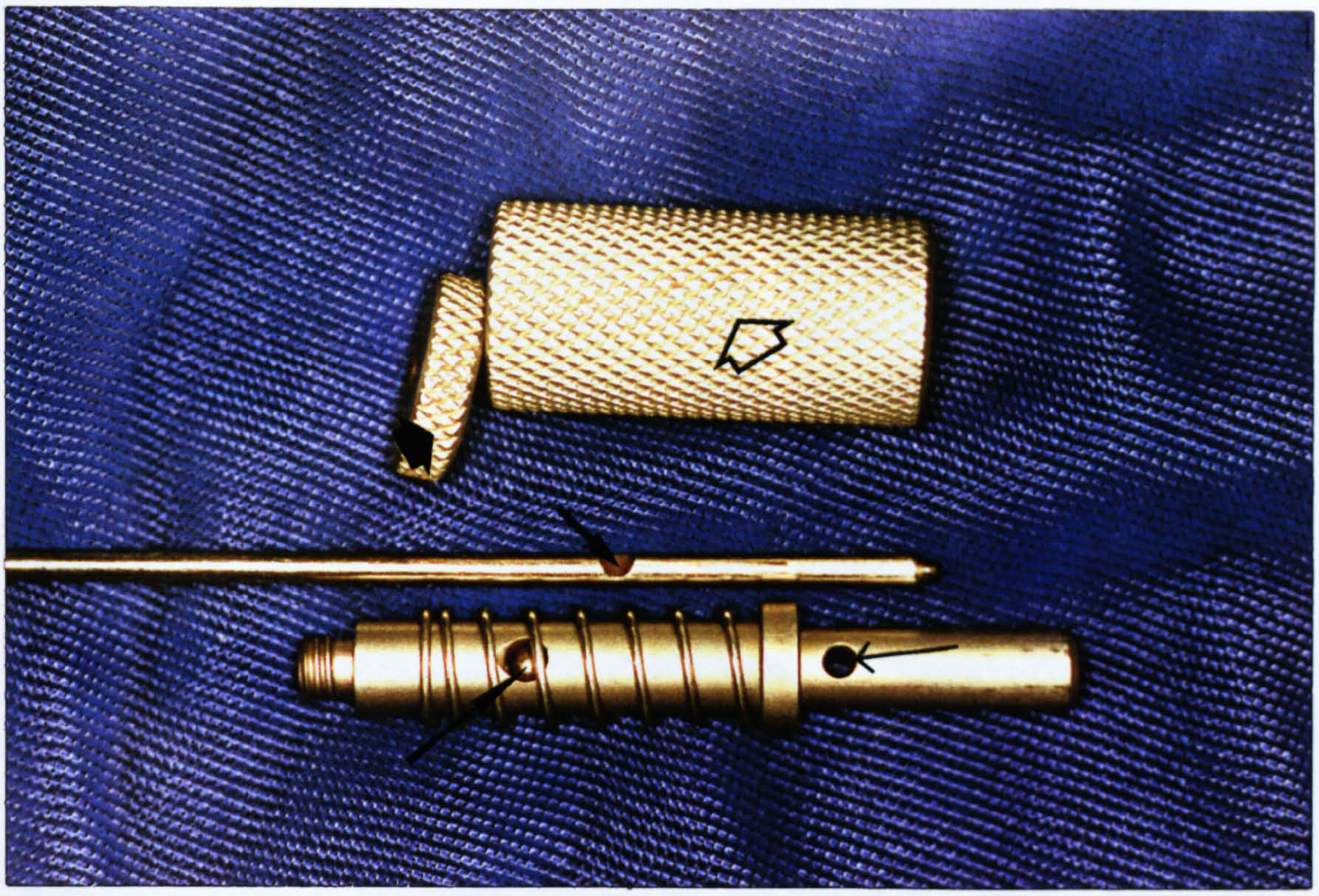


Figure 7.11 Guide–wire stop, disassembled. The spring–loaded outer sleeve (⌂) forces the steel ball (⟶) to engage in the depression on the guide–wire (⟶). The unit is held together by the lock–nut (⬟) which can be tightened by using the guide–wire as a lever through the hole in the shaft (⟶).

second drill guide was designed with the facility to pass an implant leader through the joint.

Various solutions to the PCL leader passing problem have been devised:

- The Corin APS system used a bead on a suture which screwed into the tibial drill guide. This required the surgeon to thread the bead and then thread the suture through an introducer which seemed unnecessarily complex.
- The Surgicraft ABC system used a wire with a sprung loop at the end which would lock into a hole through the tibial drill guide. The author has been told of several cases of the end loop breaking off and becoming lost in the posterior compartment of the knee.
- The LARS Laboureau system used a hollow tibial drill guide which bent lead wires back through the joint after they were passed up through the tibial tunnel: This solution did not even sound simple



Figure 7.12 Tibial drill guide mark two. Fitted with the drill barrel (◆) and a cannulated drill (⌂). A close up of the drill at the end of its travel is shown inset top right.

Since none of the above solutions appeared to be optimal a new design was developed. A plastic bead was designed which was shaped so that it would lock into the ring of the tibial drill guide and allow the suture leader to be pulled through the joint. A leader passer was designed that did not require the suture to be threaded through it; instead it was run along a slot (Figure 7.13, Figure D.14). The leader passer was the same diameter as the drill used to make the tibial tunnel so it was accurately located by the drill guide barrel. The suture was used to pull through a measuring tape which was in turn used to pull through the correctly sized implant. It was hoped that this solution would be cheap, since the bead could be formed onto the suture, and simple.

It was thought that there may be a problem of impingement when extracting the drill guide and bead from the joint in a young, 'tight' knee. To minimise this potential problem the bead and leader passer were designed so that the projection of the bead was minimal. A method of reducing the distal projection of the bead to zero was devised (Figure 7.13, D.12 and D.13) but this required the bead to be held in place by tension in the suture. Clinical experience proved that while this method appeared to work in the hands of the author it was not robust enough for general use. Fortunately it was also found that impingement was not a problem. The latter finding meant that the bead and passing tool could be redesigned to be more secure, at the expense of greater bulk (Figure 7.14, Figure D.15).



Figure 7.13 Suture leader passing tool (◆) holding the bead (➡) via a suture running along the length of the passing tool in a slot (⟶). Bottom left, close-up of bead approaching ring, note that the tip of the passing tool is of the same diameter as the bead. Bottom right, bead engaged in the ring of the drill guide, note that the bead is flush with the bottom of the ring to minimise impingement when extracting the drill guide.

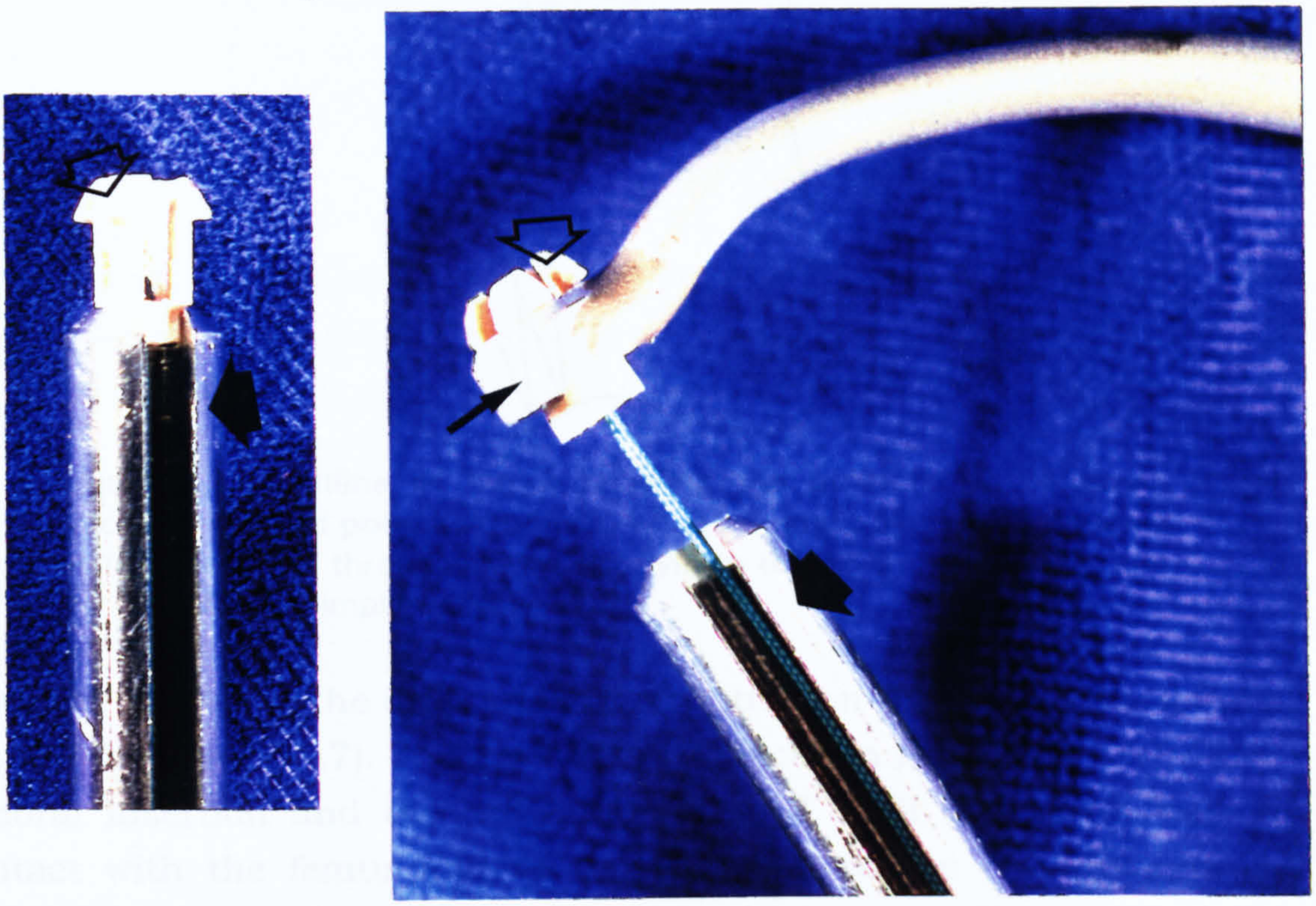


Figure 7.14 Modified bead (⌂) and leader passing tool.(⬟). Left, bead held in passing tool. Right, bead engaged in drill guide ring (⟶), note that the modified bead projects distally.

7.7.2 Femur

As stated in section 7.5.2, minimum bending of the implant path would be achieved if the tunnel went from the aPC insertion straight through the femur to the medial epicondyle just proximal to the edge of the articular cartilage. However, this would not be ideal for screw and grommet implant fixation. The area between the proximal end of the femoral tunnel and the grommet was identified as a potential site for bone resorption leading to implant loosening: If the path between the distal femoral tunnel and the grommet were not straight, then the loss of cancellous bone within the condyle would cause implant loosening (Figure 7.15). Therefore, the femoral drill guide was designed to place the femoral tunnel as near parallel to the medial femoral shaft as

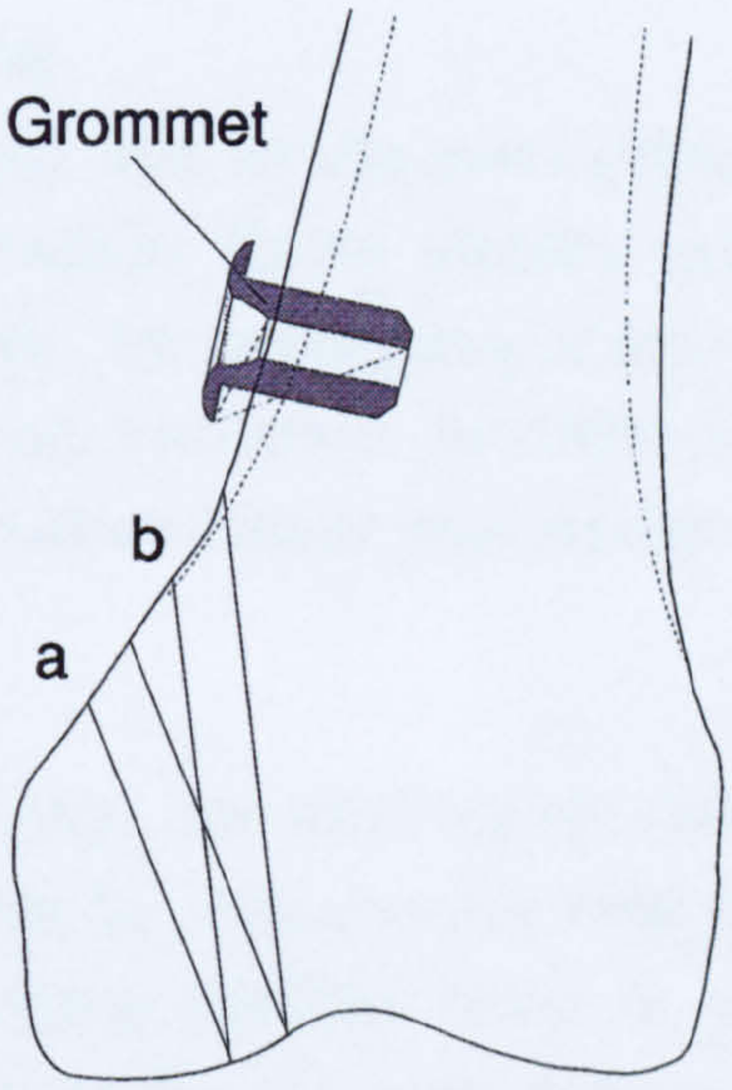


Figure 7.15 Outline of femur showing the grommet in proximal, thick, cortex and potential tunnels. It was thought likely that a tunnel at (a) would cut through the epicondyle to (b), so it was best to aim for tunnel (b) at implantation.

possible. The tip of the drill guide was centred on the axis of the handle (Figure 7.16 and D.17). The tip was to be placed in the centre of the aPC femoral insertion and the drill guide rotated until the barrel was in contact with the femur and as near parallel to the femoral axis as possible. It was recognised that this would increase the angle of the bend in the femur and that the tunnel edge would have to be carefully radiused.

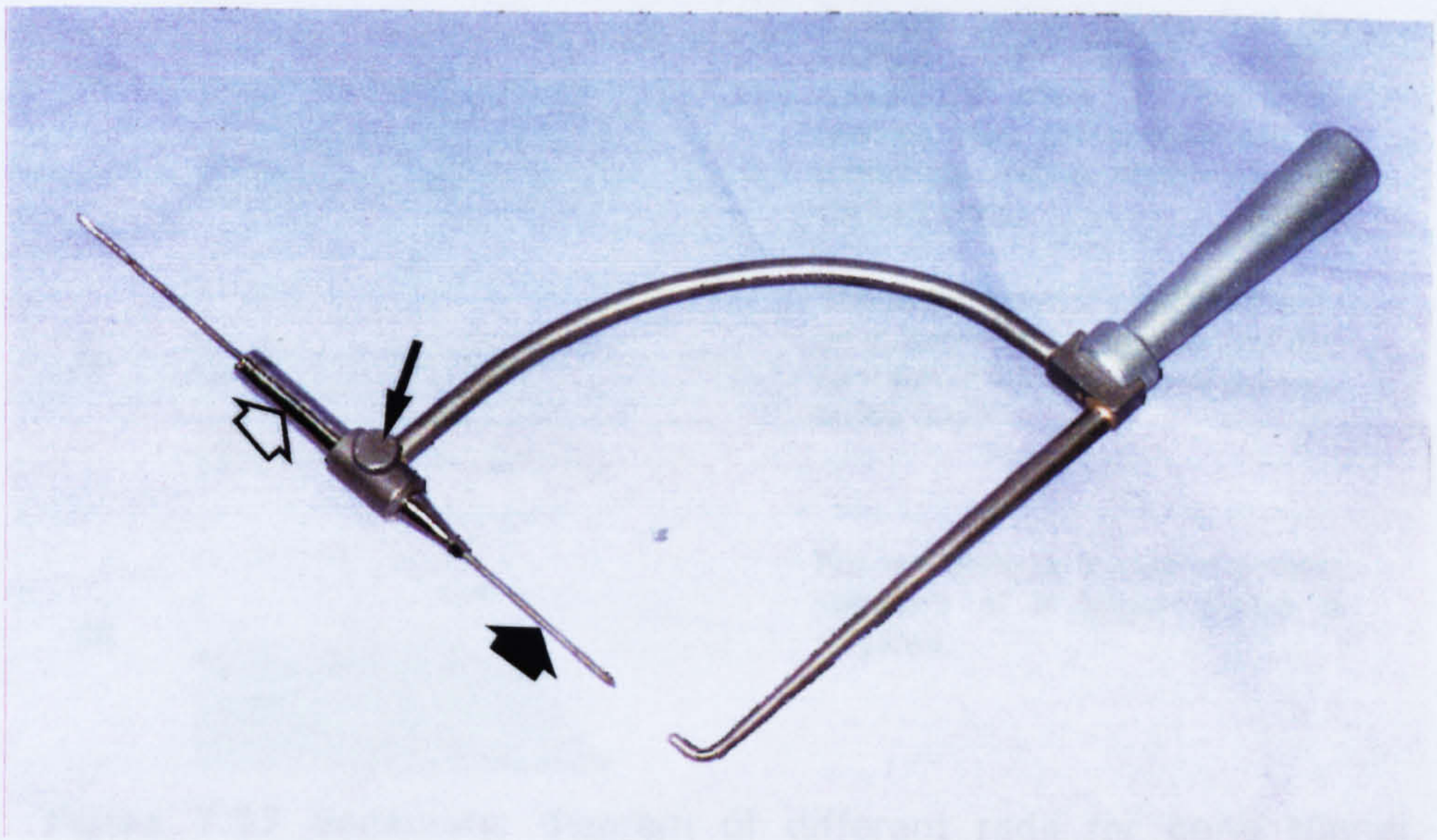


Figure 7.16 Femoral drill guide, with a guide wire (⬟) held in the guide–wire barrel (⌂) which is secured in place by the thumbwheel (⟶).

7.7.2.1 Back radius cutter

Implant failure can occur due to abrasion/stress concentration/cutting at the bone tunnel entrance. These factors can all be minimised, for a certain tunnel placement, by producing a smooth radius of the largest possible size at the tunnel entrance. In order to minimise the wear rate of the implant a back radius cutter was designed (Figure 7.18, D.7 and D.8).

It is important to note that an improperly designed radius cutter can make the situation worse by introducing new sharp edges (Figure 7.17) or by incidentally damaging healthy bone or soft tissue. The optimum magnitude of the radius to be cut was defined by the thickness of the cortex at the femoral insertion of the aPC (Figure 7.17): in the study presented in section 3.2 this was found to be 2.5±0.3mm.

The domed head was the same diameter as the drill hole. When the radius was complete the shaft of the cutter rested on the side of the tunnel preventing the cutter from extending the exit hole in the joint.

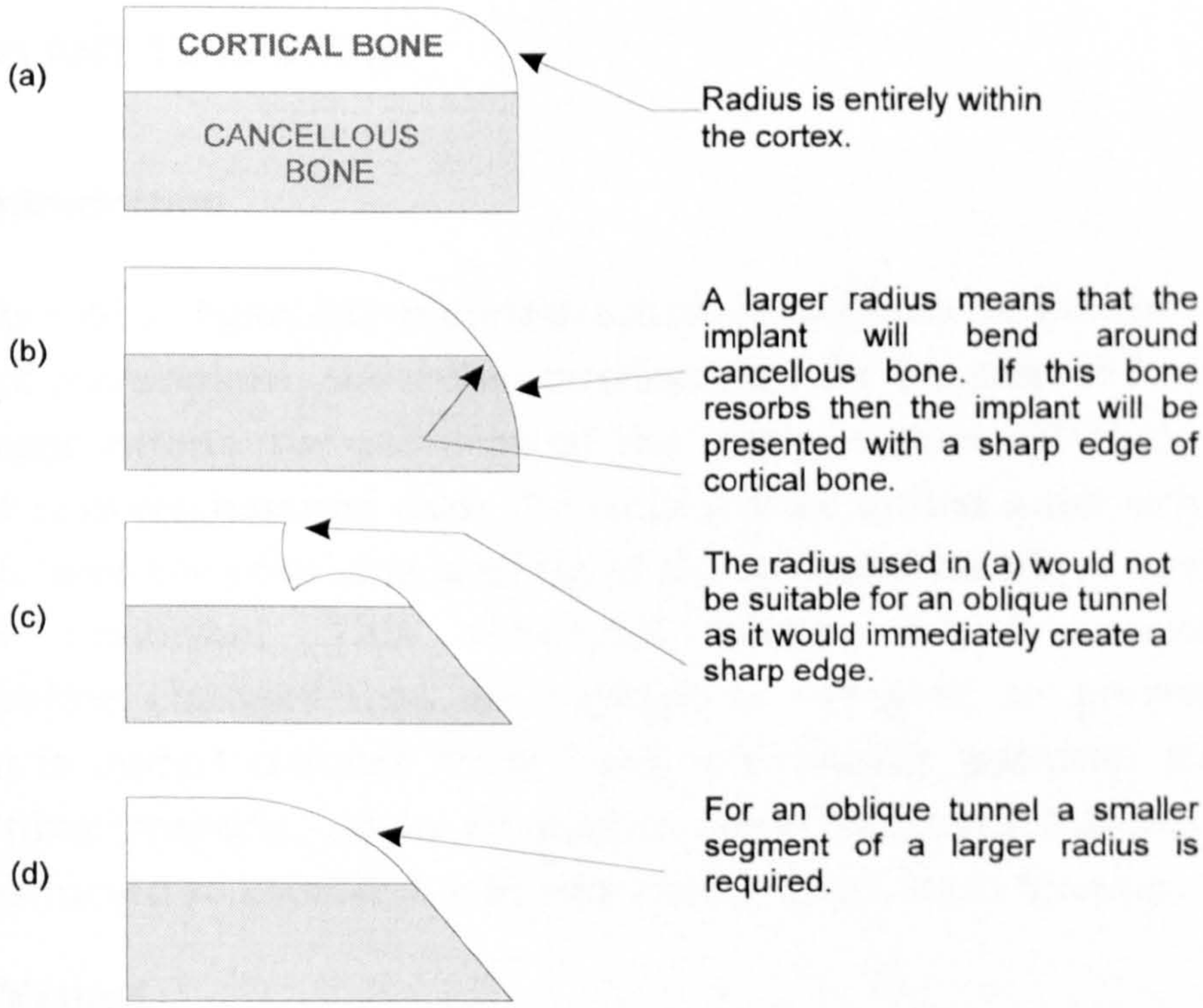


Figure 7.17 Schematic diagram of different radii for bone tunnel entrances. (a) and (b) represent tunnels perpendicular to the surface and (c) and (d) tunnels oblique to the surface.



Figure 7.18 Back radius cutter. The blunt tip prevents accidental tissue damage.

7.8 IMPLANT TENSIONING

7.8.1 Introduction

The effect of a ligament reconstruction is dependent on the insertion points of the implant, the initial tension that the implant is fixed at and, to a lesser extent, the stiffness of the implant (Amis 1989a). If a PCL implant is overtensioned then the tibia will be pulled anteriorly, loading the ACL, and the resulting loading of the articular cartilage and menisci will be abnormal. This abnormal loading could accelerate the degenerative changes that an implant is designed to prevent. If the implant is undertensioned then it will not restrain posterior translation of the tibia properly, again leading to abnormal joint loading. A study was conducted to establish a reasonable implantation tension.

7.8.2 Method

Two knees were used to asses the influence of implant tension on the restoration of normal posterior drawer characteristics. The knee was potted as described in section 2.2. Then the knee was mounted in the Instron via the four degree of freedom rig described in section 6.2. The

A-P motion of the intact joint in response to ±150N A-P drawer force was measured at 30° intervals between 0° and 120° of flexion. A straight posterior laxity was then induced by preventing tibial rotation and applying progressively larger posterior displacements, at 1000mm/min, until the PCL completely ruptured. The knee was then removed from the Instron and the PCL was reconstructed using the polyester ligament and tunnel placement described in sections 7.4 and 7.7. The implant was anchored at the tibia, with screw and grommet, but left free at the femoral end. The knee was then remounted in the Instron and the femoral end of the implant was attached to a specially designed tensiometer that was clamped to the femoral pot. It was considered appropriate to tension the implant from the femoral end because there was a much lower angle of wrap at the femoral insertion and therefore less resistance to adjustment of the intraarticular portion of the implant.

The total A-P laxity was measured rather than just posterior laxity because over–tensioning the PCL graft could have altered the anterior drawer characteristic by preloading the ACL. This overloading would have caused the tibia to be displaced anteriorly from its normal range. In PCL reconstruction, as in all surgical intervention, it is important to check that a 'cure' does not induce a new chronic condition.

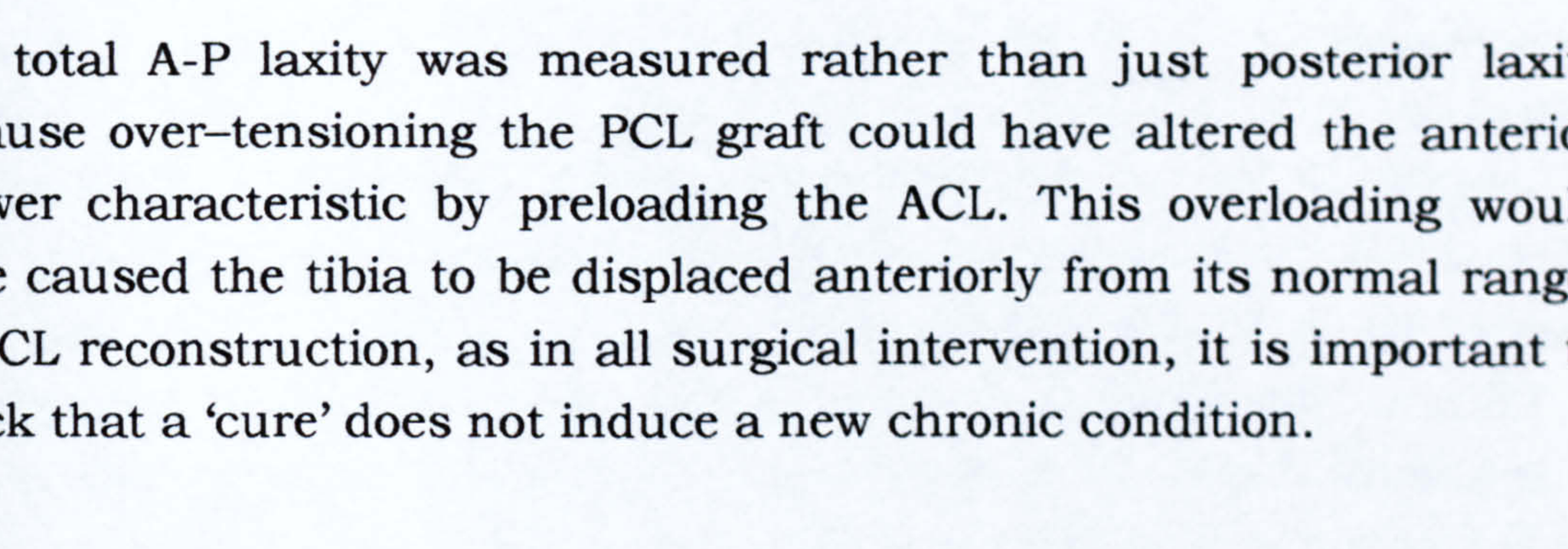


Figure 7.19 Graph of relative A-P laxity for ±150N drawer force with an implant tensioned to 10, 20 and 30N at 45° of flexion.

7.8.3 Results And Discussion

On the basis of the results of the studies reported in earlier sections it was thought that an implant based on the aPC insertions should be just unloaded in A-P neutral at 30° of flexion. So, tensioning was first carried out at 30° of knee flexion with 1N. It was found that, at 30° of flexion, tensions of 1N and 10N were 30% under– and over–restraining, respectively. The fact that a small difference in tension led to a large difference to the A-P drawer characteristic made tensioning at 30° of flexion difficult to control. It was thought that tensioning at a higher flexion angle, when the aPC would normally be taut, would be less sensitive to small changes in tension. Although a higher tension requirement would be less sensitive to small changes, too high a tension requirement would be awkward for the surgeon to apply. A compromise of 45° of flexion was decided upon, and the second knee was reconstructed and tensioned to 10N, 20N and 30N. The results of these tests are shown in Figure 7.19. It was found that an implantation tension of 20N restored a normal A-P drawer characteristic, to within 6% (≅0.6mm of posterior drawer), from extension to 90° of flexion. At 120° of flexion a 20N implantation tension under–restrained the knee by 29% (2.6mm of posterior drawer). The finding that the aPC–based reconstruction did not restore the normal A-P characteristic at 120° of flexion was expected: the bundle loading study in section 6 showed that the contribution of the aPC to resisting posterior drawer reduced from mid–flexion to full–flexion.

7.9 ABRASION/FATIGUE RESISTANCE

7.9.1 Introduction

A study was set up to test the effectiveness of the back radius cutter (Figure 7.18) in reducing implant wear and fatigue. For this study an abrasion test rig designed for the ACL was adapted. The rig was designed to test the femoral insertion of the implant, as this insertion is subject to more rotation (Figure 7.20) and would fail by abrasion before the tibial insertion. The rig was designed to cyclically move the femur about a medial–lateral axis that passed through the implant insertion. The controlled variables were the angle between the implant and the

femur and the implant load. It was decided to simulate the loads and motions of the PCL femoral insertion due to normal walking.

7.9.2 Method

The angle between the implant and the femoral and tibial axis over the range of flexion was determined as follows. A reconstruction was performed on a knee that had had the PCL excised. The lateral femoral condyle, incorporating the femoral insertions of the ACL and LCL, was then released by sagittal and transverse saw cuts. The lateral femoral condyle could be replaced while the joint was flexed and then removed to allow a sagittal plane view of the PCL implant. The tibia was held vertically in a vice so that the lateral aspect of the knee was presented to a camera. The sagittal plane position of the tibial insertion of the implant was marked on the lateral aspect of the tibia with Indian Ink. The position of the laterally projected tibial insertion was determined using an adjustable pointing device. The knee was then photographed over the range of flexion. When the knee was moved the lateral femoral condyle was held in its anatomical position and a compressive force applied: the motion of the knee was controlled by the intact MCL, the implant and by the geometry of the condyles and menisci.

The resulting transparencies were then projected onto paper and the position of the tibial and femoral implant insertions recorded along with reference points for the axis of the tibia and femur. The angle between the implant, the femur and the tibia could then be directly measured for each flexion angle. Over the flexion range of normal gait (0°–60°) the angle between the implant and the tibial plateau varied by 10° whereas the angle between the implant and the femoral axis varied by 60°. This result showed reasonable agreement with the values calculated using the sagittal plane models of O'Connor et al (1990) and Zavatsky and O'Connor (1992a) (Figure 7.20).

Data for PCL loads during level walking were acquired from the study of Morrison (1970). This load data was given plotted against percentage time in the gait cycle so it was necessary to translate the percentage time into flexion angle, this was done using data from Lafortune et al (1992). So, the load on the PCL was calculated in terms of flexion angle over the gait cycle. Then the data for implant/femur angle over the

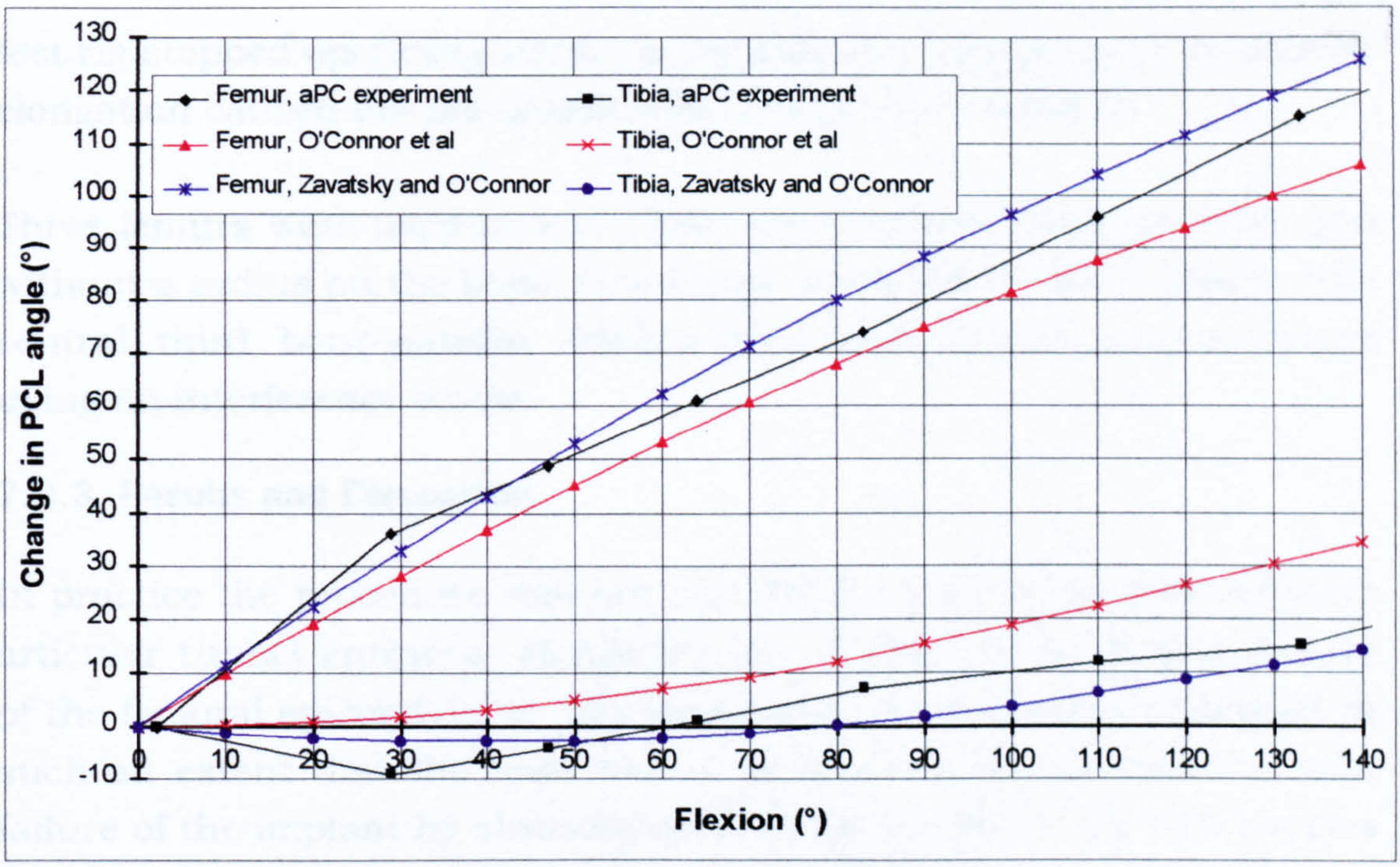


Figure 7.20 Graph showing the change in angle between the PCL implant, the femoral axis and the tibial plateau over the range of flexion for an in-vitro implant and for the neutral fibres of the models of O'Connor et al (1990) and Zavatsky and O'Connor (1992a).

range of flexion, described above, was used to establish the implant/femur angles over which load should be applied.

In the abrasion test rig loads were applied to the implant by a pneumatic cylinder which was activated by a microswitch running on a cam fixed to the drive shaft. Rotation of the femur was controlled by a four bar linkage from the drive shaft to the shaft holding the femoral clamp. The four bar linkage was adjusted to give the 60° range of implant/femur angle that was determined as described above. A graphical construction based on the four bar linkage was then used to design a cam to trigger the load at the appropriate implant/femur angles (Figures D.18 and D.19). The loads reported by Morrison (1970) varied by a factor of three over the gait cycle, but since the rig was only designed to give one load the peak value was used.

This system produced a sinusoidal variation in the angle between the implant and the femoral axis, so the system did not accurately reproduce the gait cycle in time but did allow the correct range of implant/femur motion and load the implant at the correct angles. The

test rig stopped operating when the implant had elongated by 5mm: the elongation caused the pneumatic piston to actuate a switch.

Three femurs were used to test 2300 turn synthetic implants with and without a radius on the bone. One femur was used to test a 10mm wide central third bone–patellar tendon–bone graft which was anchored using an interference screw.

7.9.3 Results and Discussion

In practice the procedure was not suitable for testing the femoral intra articular tunnel entrance: elongation was dominated by fatigue failure of the femoral epicondyle. In two specimens the epicondyle collapsed to such an extent that the tests had to be abandoned. In the third trial failure of the implant by abrasion occurred at 26,000 and 29,000 cycles for un–radiused and radiused holes respectively.

For the bone–patellar tendon–bone graft failure occurred at 50 cycles. The interference screw migrated through the cancellous bone causing enough elongation to stop the test rig. The rig was then retightened. Subsequently the bone block in the femur broke in two, with some fibres attached to each fragment. The distal fragment came out of the drill hole and the remaining fibres peeled off the proximal fragment.

Both of these results appear to be disastrous: Seedhom et al (1973) have shown that the average person takes around 5,000 steps with each limb per day. So, according to this experiment a synthetic implant would fail after 5 to 6 days and a bone–patellar tendon–bone graft would fail almost as soon as it was loaded. Neither of these results correlate with clinical experience. An ACL reconstruction using a femoral drill hole is subject to a similar range of motion relative the femoral tunnel entrance as a PCL reconstruction. Therefore, the abrasion/fatigue response of an ACL reconstruction should not be too dissimilar to that of a PCL reconstruction. Bone–patellar tendon–bone grafts are the gold standard for ACL reconstructions and are also used successfully in PCL reconstructions for far more than 50 cycles. When the Apex ACL ligament was used with a femoral drill hole in a sheep model there were no instances of implant failure over the six–month trial (Radford et al, 1994).

Clearly, this experiment was not a good simulation of in–vivo conditions, which appear to be more benign. The failure of the bone–patellar tendon–bone anchorage and the collapse of the femoral epicondyle in two of the synthetic implant tests can be explained by the poor quality of the cancellous bone, which came from older donors. The fact that both reconstructions have been shown to be more robust in–vivo suggests that biological reaction and tissue ingrowth is an important factor for both autogenous and synthetic implants. Autogenous grafts rely on tissue ingrowth for fixation. Implant abrasion occurs at points of high stress and motion; these conditions can be expected to cause some inflammatory reaction. It is possible that the tissue remodelling at these points of stress and motion around bone tunnel entrances results in a more benign environment for implants: this could explain the implant survival found in the sheep study of Radford et al (1994).

7.10 OUTLINE OF SURGICAL PROTOCOL

7.10.1 Indications

Functional impairment, due to pain or 'giving–way', associated with symptomatic posterior instability in flexion; demonstrated by the posterior sag or posterior drawer sign, with the absence of tibial avulsion. If there is posterolateral or posteromedial laxity or both, demonstrated by a posterior drawer sign in extension, then additional extra–articular reconstruction will be necessary. The implant was

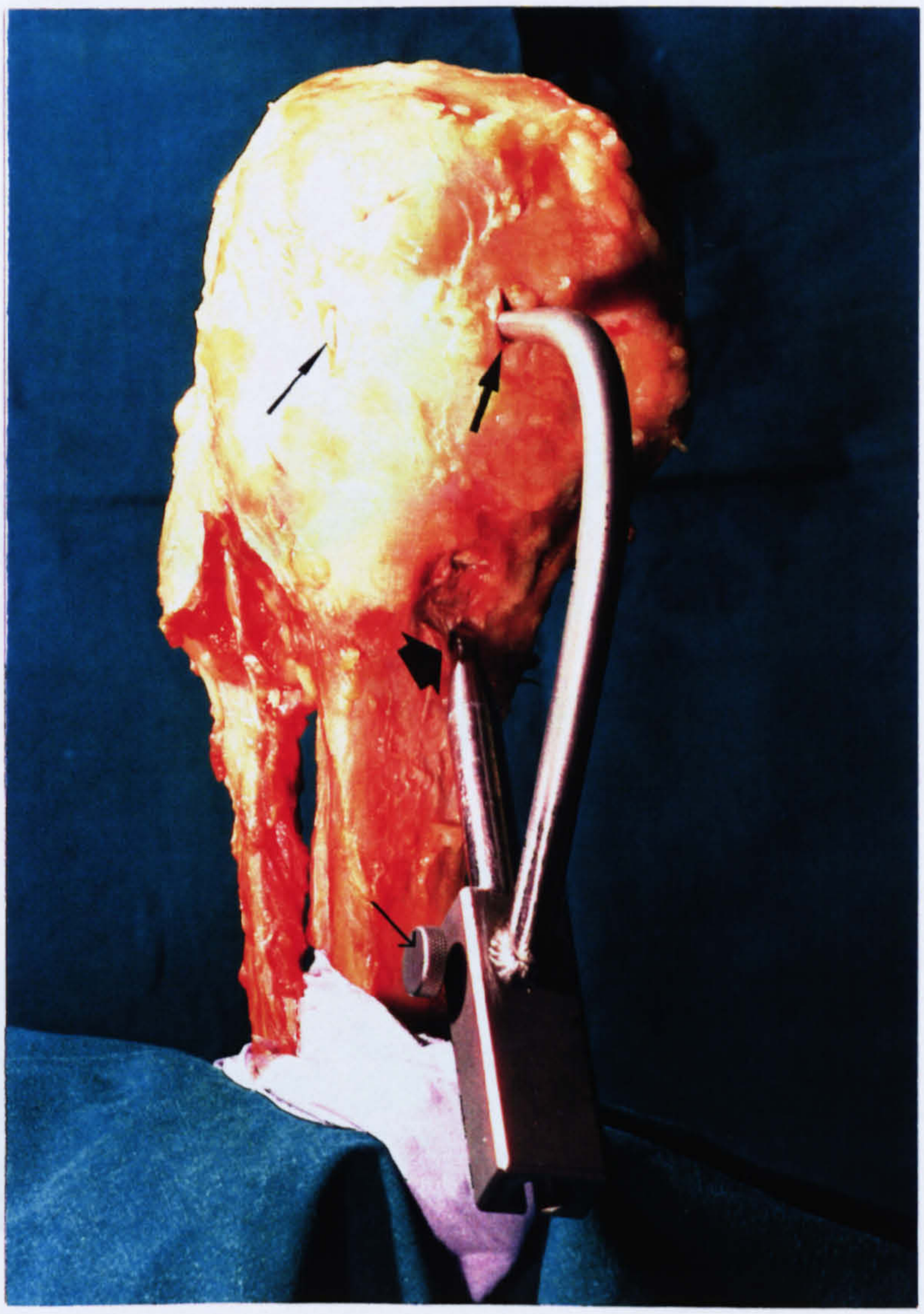


Figure 7.21 Anterior aspect of the flexed knee. The tibial drill–guide has been inserted through an antero–medial portal (➞). The guide–wire barrel (➧) has been advanced into the central part of the medial facet of the tibia and locked in place with the thumbwheel (⟶). An arthroscope may be placed in an antero–lateral portal (⟶).

designed to serve the function of the natural aPC which resists posterior drawer in flexion but does *not* resist posterior drawer in extension (section 6.4.2). So, if there is instability in extension the implant alone will not correct it and functional stability will *not* be restored.

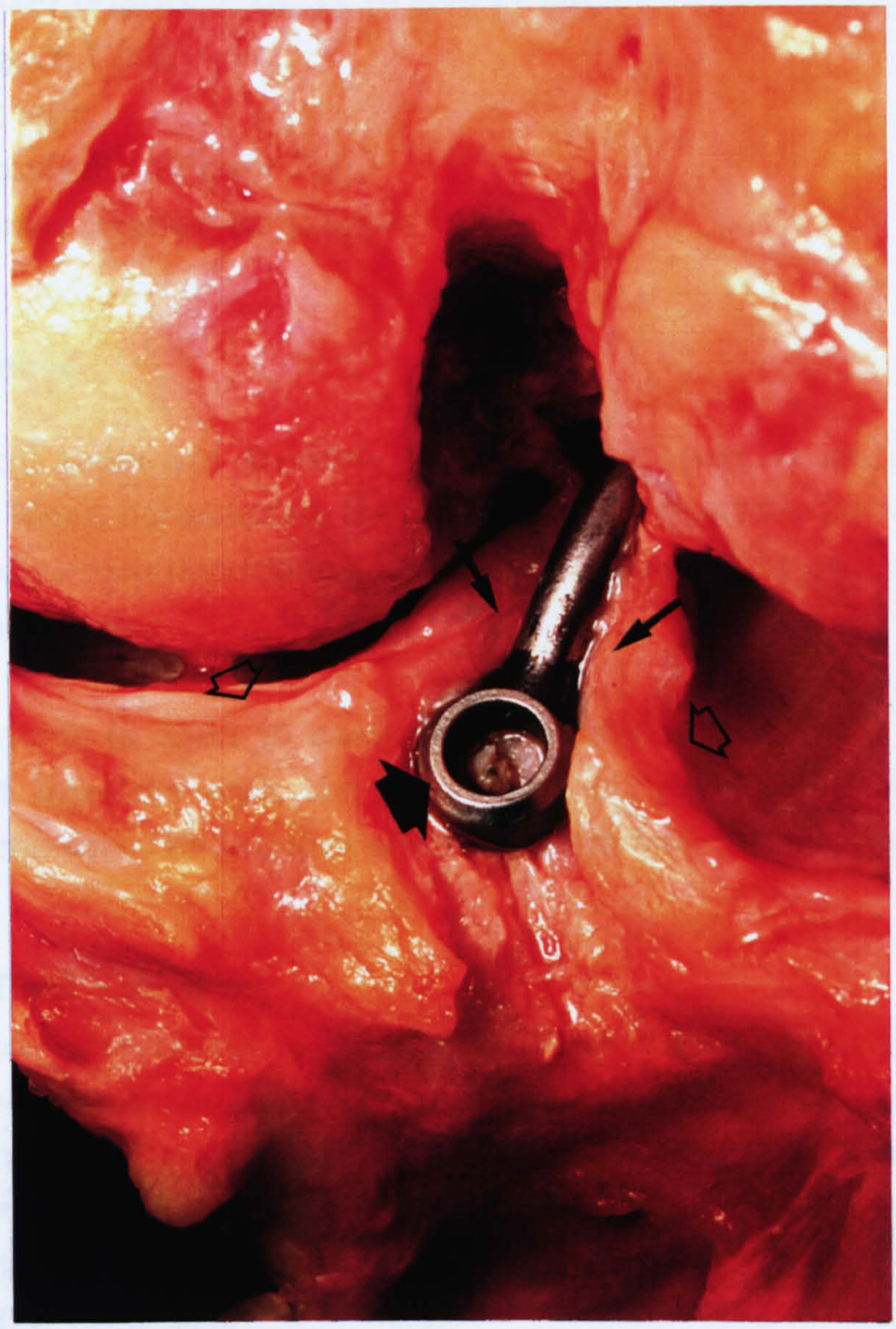


Figure 7.22 Posterior aspect of the extended knee with capsule removed. The tip of the tibial drill–guide (⬟) is correctly positioned in the anterior portion of the PCL insertion wedged between the tibial condyles (⌂) and the meniscal insertions (⟶).

7.10.2 Equipment

Tibial drill–guide (Figure 7.10, D.10 and D.11)
Femoral drill–guide (Figure 7.16, and D.17)
Guide–wires (Figure D.3)
Drill–stop (Figure 7.10, and D.6)
8mm cannulated drill
Back radius cutter (Figure 7.18, D.7 and D.8)
Leader passer (Figure 7.13, D.14 and D.16)
Leader (Figure 7.13)
Measuring tape
Range of implants (Figure 7.3)
Apex grommet fixation kit (Figure 7.7)

7.10.3 Method

The surgical tools are suitable for either an open or an arthroscopic approach. When considering the approach the need for posterolateral or posteromedial reconstructions should be addressed in order to optimise the skin incisions.

When the joint is open to examination any scar tissue or osteophytes that might interfere with the placement of the drill–guides or cause impingement with the implant should be checked for and removed.

7.10.3.1 Tibial Tunnel

Fit the guide–wire barrel to the tibial drill–guide and then insert the tip of the drill–guide through an anteromedial arthrotomy just medial to the patellar tendon (Figure 7.21). Push the drill–guide tip posteriorly and distally then pull it anteriorly, maintaining some distal pressure, until the tip catches in the fovea of the tibial posterior intercondylar area (Figure 7.22).

Align the drill–guide so that the straight portions of the bent bar, which connects the barrel holder to the tip, are approximately parallel to the tibial plateau and the anterior edge of the tibia. Then advance the guide–wire barrel to the skin in the central region of the medial facet of the tibia. Make an incision through to the bone extending about 10mm proximally and 30mm distally from where the guide–wire barrel tip

contacts the skin. Lock the drill–guide in position by pushing the spikes on the tip of the guide–wire barrel into the surface of the tibia and tightening the thumbwheel on the drill–guide (Figure 7.21). The guide–wire barrel can be conveniently pushed using the leader passing tool.



Figure 7.23 anterior aspect of the flexed knee with capsule and patella removed for clarity. The tibial drill–guide is fitted with the drill barrel (⌓). The leader passing tool (⬟) has been passed through the tibial bone tunnel so that the tip of the passing tool is in contact with the tip of the drill–guide. The tool is used to ream the bone tunnel and then to pass the bead and suture leader through the tibia.

If the tip of the drill–guide cannot be visualised then its correct placement should be verified by a medial–lateral view with an image intensifier.

When the drill–guide is correctly placed fit a guide–wire to the drill–stop and drill it into place (Figure 7.10). Verify the correct placement of the guide-wire by a medial–lateral view with an image intensifier: The guide–wire should not pass within 4mm of the posterior tibial cortex and the tip should be just below the cortex at the PCL insertion. The

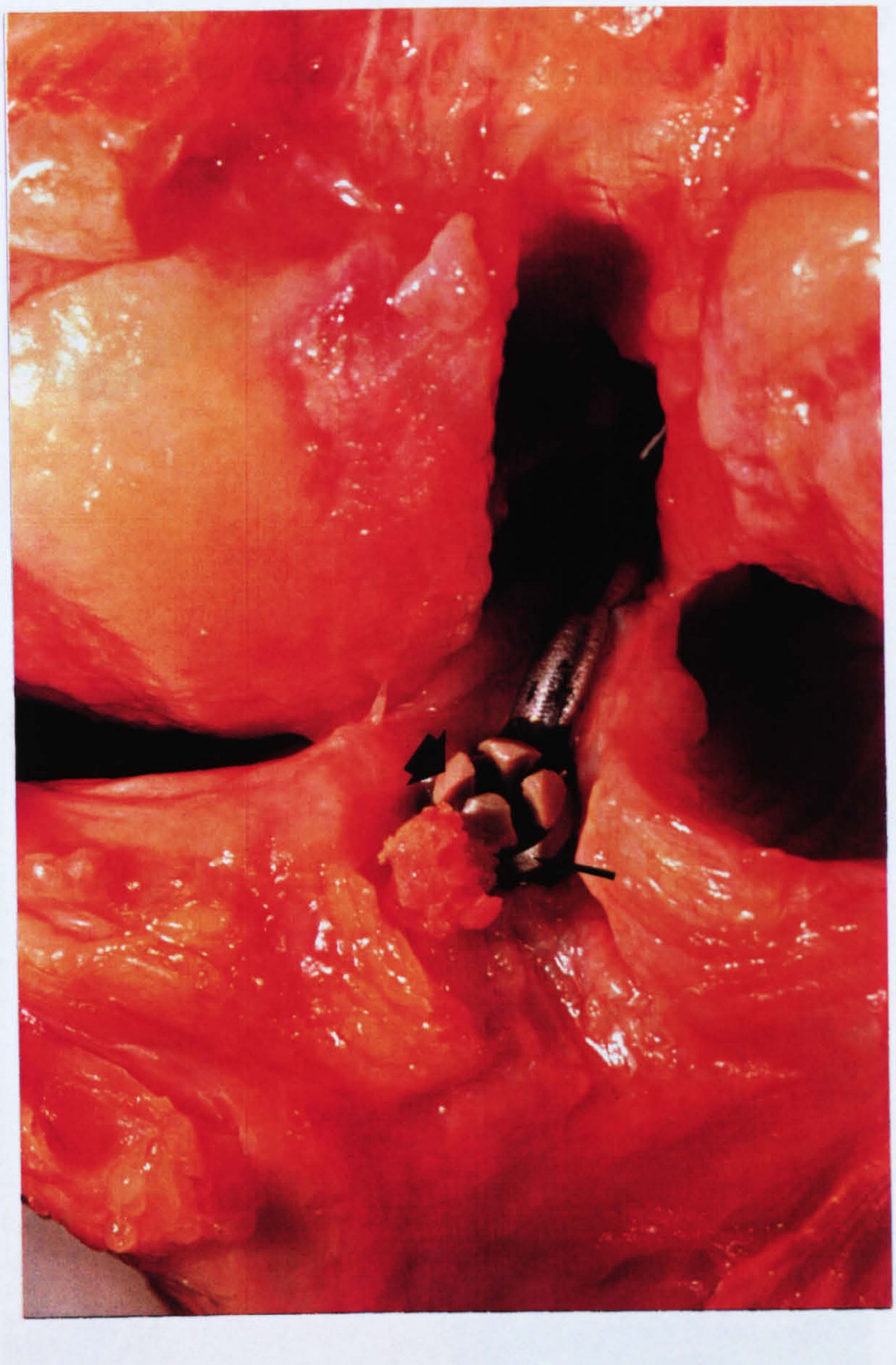


Figure 7.24 Posterior aspect of the extended knee with capsule removed. The leader passing bead (⬟) is engaged in the tip of the tibial drill–guide (⟶)

first criterion will ensure that the drill does not pierce or unnecessarily damage the posterior tibial cortex and the second will ensure that the guide–wire will not be advanced by the drill into the neurovascular structures of the popliteal fossa. If all is well then remove the guide–wire barrel from the drill–guide and insert the barrel for the cannulated drill. Overdrill the guide–wire until the drill makes positive contact with the drill–guide tip. Then remove the drill and guide–wire, leaving the drill–guide in place.

Push the leader passing tool through the drill–guide and into the tunnel with a twisting motion to clear the tunnel of debris (Figure 7.23). Verify that the path is clear by feeling for positive contact with the drill–guide

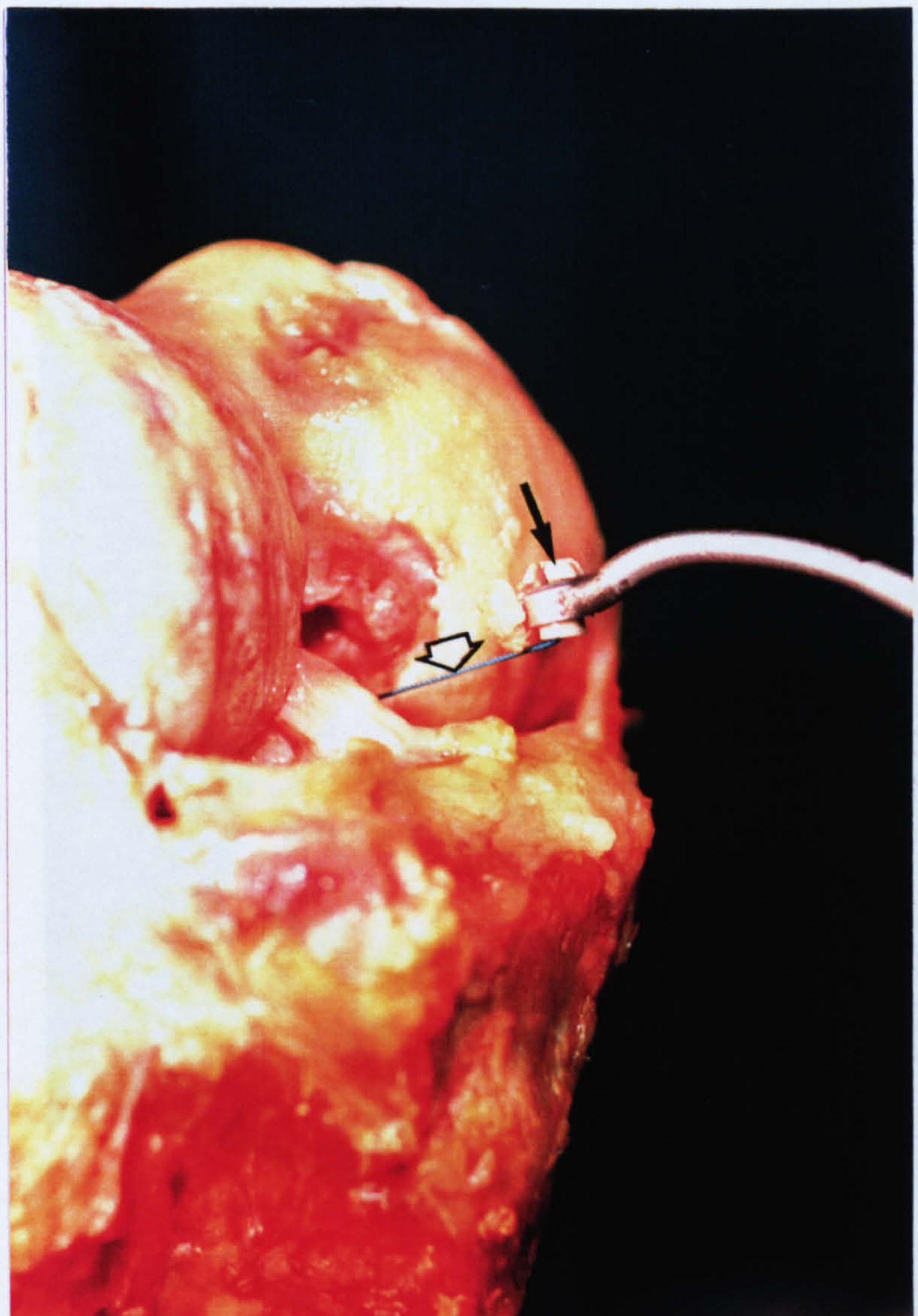


Figure 7.25 Anterolateral aspect of the flexed knee. The tibial drill–guide is shown pulling the bead (⟶) and suture leader (⌂) through the joint

tip. If there is impinging tissue then clear it using the drill. When the bone tunnel is clear, wipe the debris from the slot of the leader passing tool and fit the bead and suture leader (Figure 7.1). Then pass the leader through the drill–guide and the tibia (Figure 7.23). When the bead engages with the ring at the tip of the drill–guide (Figure 7.24) a positive 'click' will be felt. At this point the leader passing tool and the drill barrel can be removed from the drill–guide, leaving the suture leader in the tunnel.

Clamp the free distal end of the suture leader, to prevent pulling it all the way out, and remove the drill–guide from the joint (Figure 7.25). Then remove the bead and clamp the ends of the suture leader together.

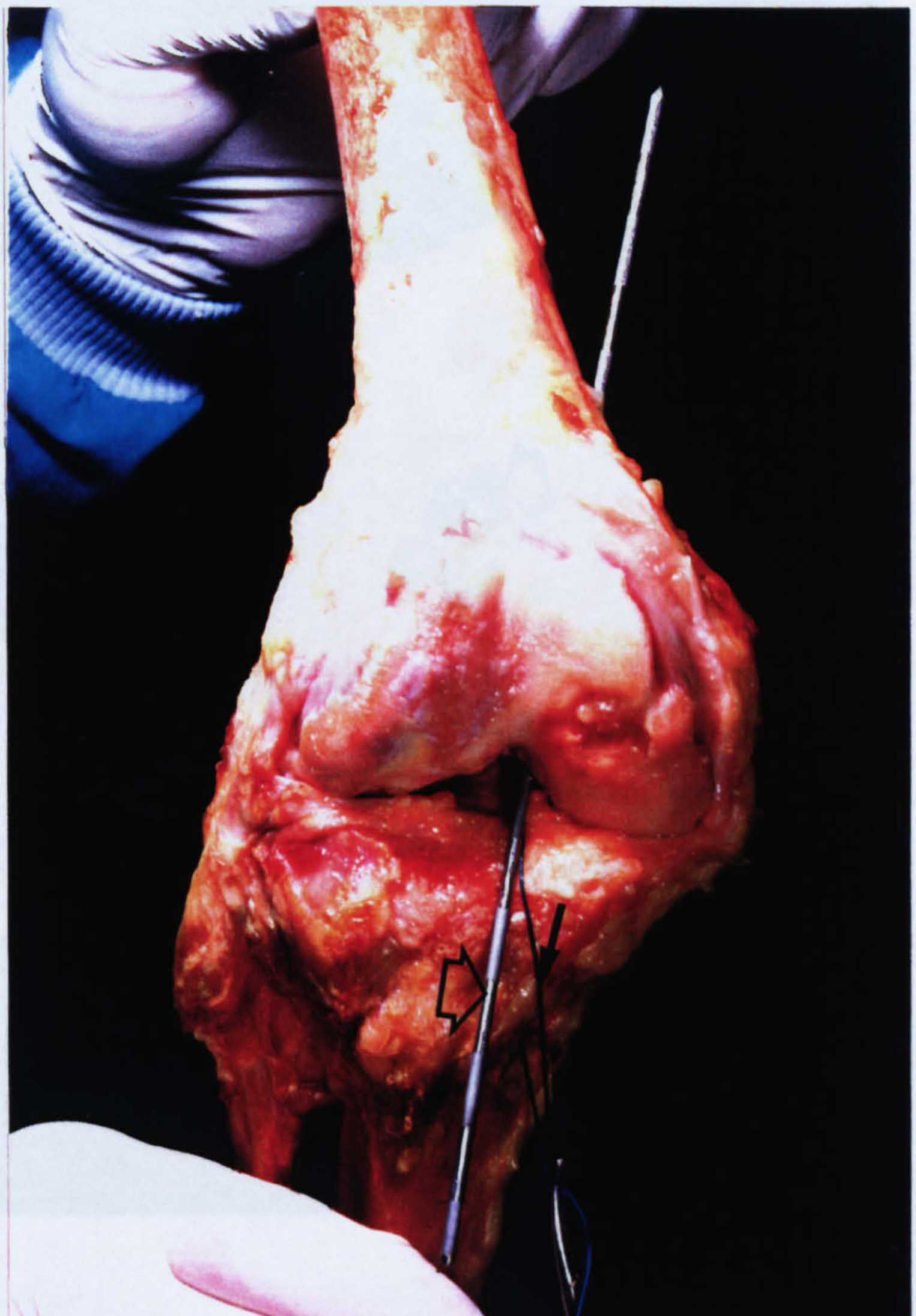


Figure 7.26 Anterior aspect of the knee showing a guide–wire in the correct orientation for the femoral bone tunnel (⌂). Note the suture leader passing through the tibia and held in a clamp (⟶).

Finally, smooth the distal portion of the extra–articular tibial tunnel entrance with a rasp. The intra-articular tunnel entrance is not rasped in order to protect the meniscal insertions which should be at the edge of the tunnel entrance (Figure 7.24). The tibial tunnel placement was designed so that this mass of soft tissue would provide a 'radius' over which the implant would wrap.

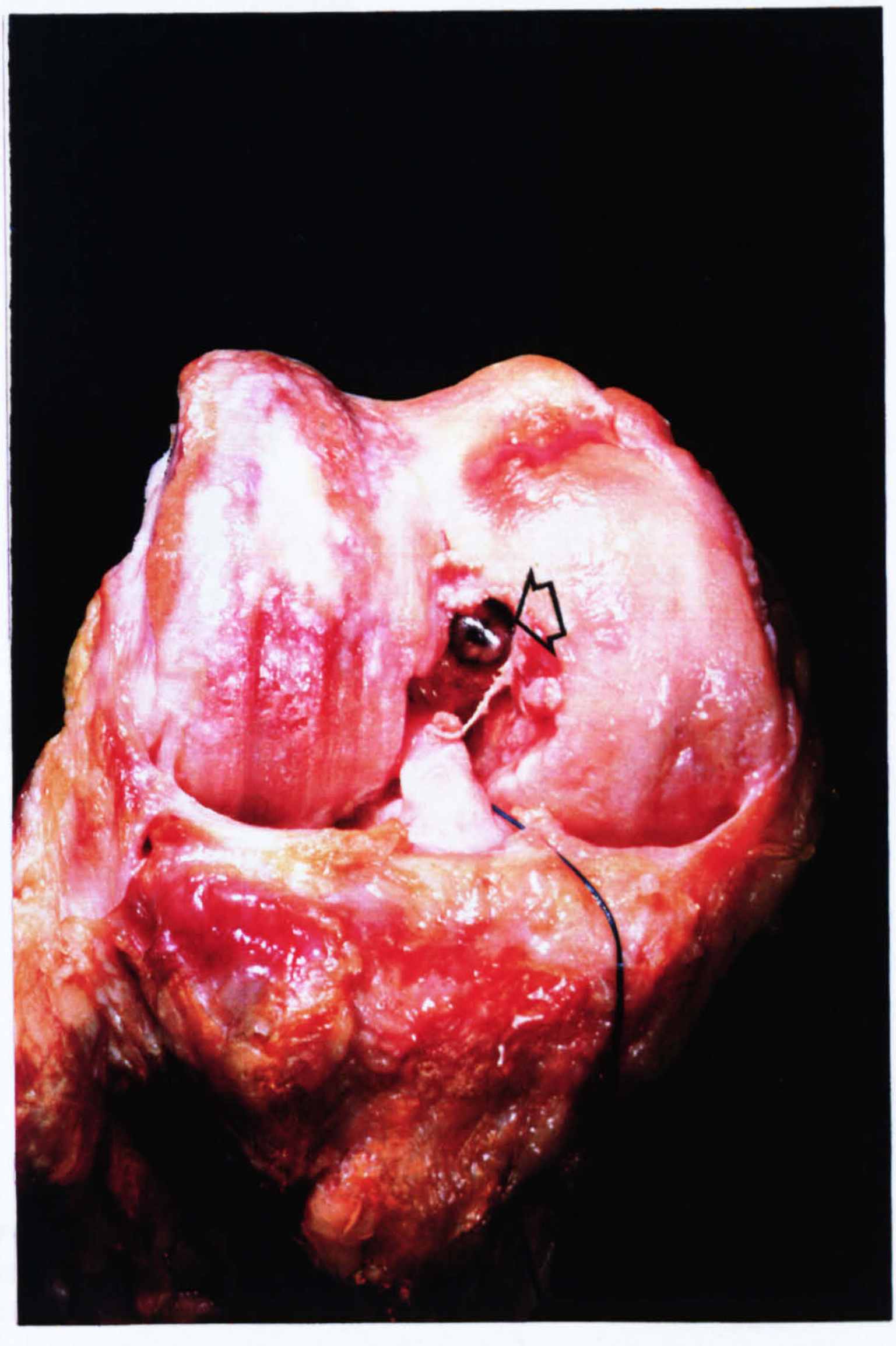


Figure 7.27 Anterior aspect of the flexed knee. The back radius cutter is shown in its cutting position (⌂).

7.10.3.2 Femoral Tunnel

Make a medial incision, starting just proximal to the femoral condyles and extending proximally by about 50mm. Open a path to the medial side of the femoral shaft by blunt dissection through the Vastus Medialis and release the muscle from the bone to enable placement of the drill guide. Place the tip of the femoral drill–guide into the centre of the femoral insertion of the aPC. This should be on the medial side of

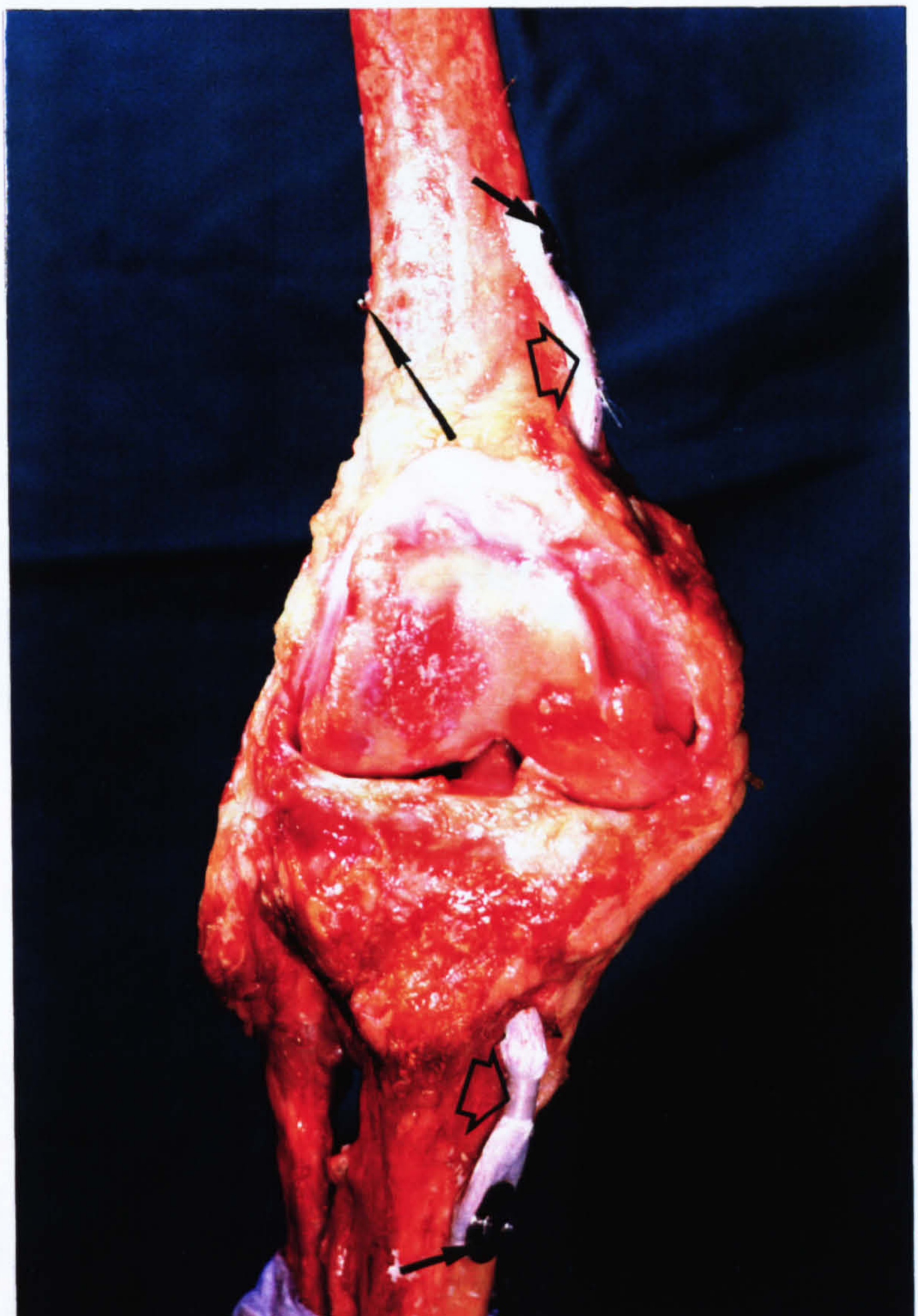


Figure 7.28 Anterior aspect of the extended knee showing the implant (⇦) and grommets (⟶). Note that the AO screw just pierces the opposite cortex (⟶).

the roof of the notch and 6-7mm from the articular cartilage. Rotate the drill–guide so that the extra–articular portion rests on the shaft of the femur. Lock the drill–guide in place by pushing the barrel into the femur. Then drill a guide–wire through the femur (Figure 7.26) and

verify that it is in the centre of the aPC insertion. Remove the drill–guide and overdrill the guide–wire with the cannulated drill. Then radius the intra–articular femoral tunnel entrance using the back radius cutter (Figure 7.27), concentrating on the proximal edge where the implant will wrap around. Finally, smooth the extra–articular femoral tunnel entrance with a rasp.

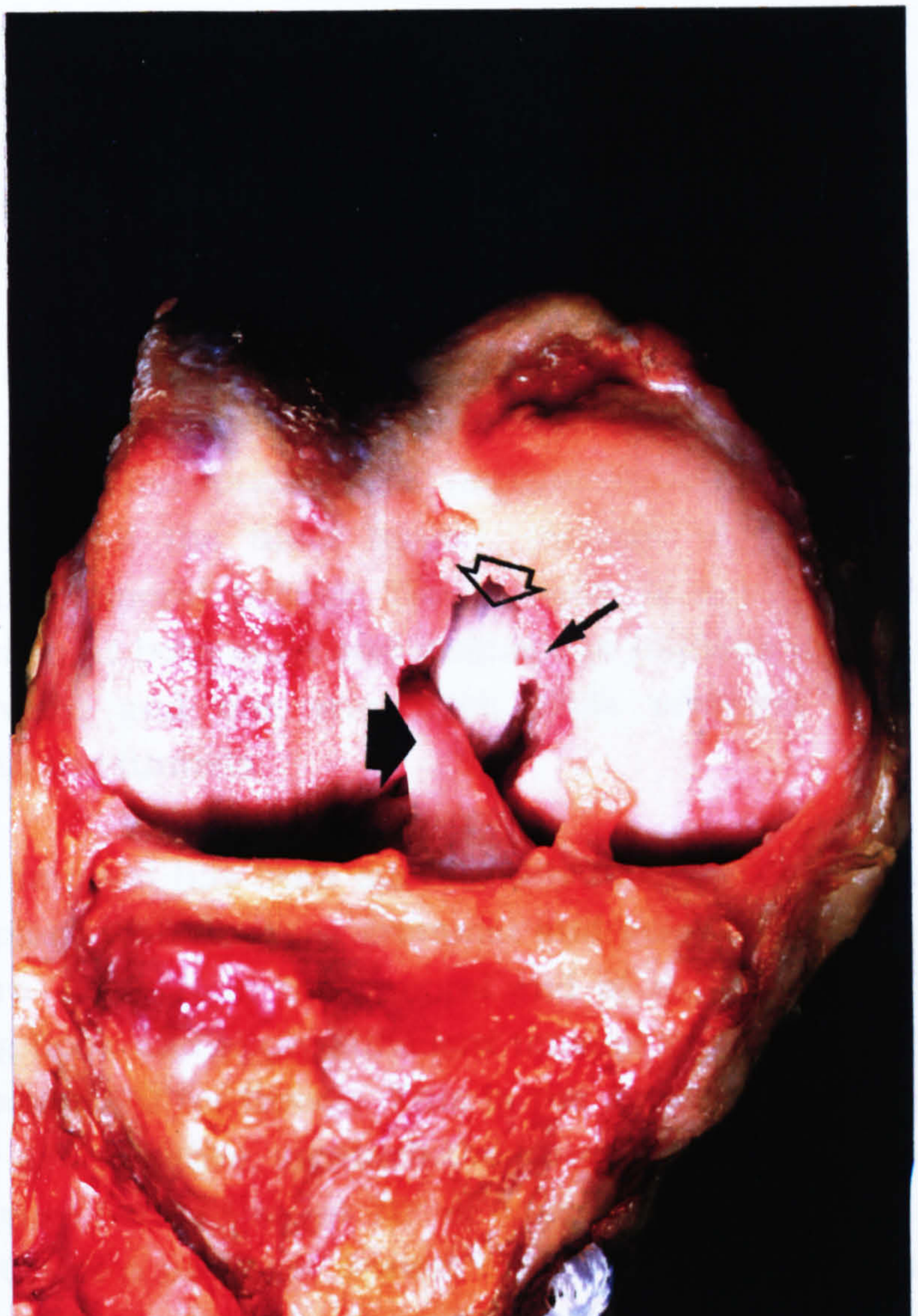


Figure 7.29 Anterior aspect of the flexed knee showing the implant crossing the ACL (◆) and inserting into the aPC region of the PCL's femoral insertion (◇). The posterolateral fibres of the PCL are absent (→).

7.10.3.3 Fitting The Implant

Pass arthroscopic forceps or a 'crochet hook' into the joint via the femoral tunnel and grasp the suture leader. Release the intra-articular end of the leader from the clamp and pull the suture leader through the femoral tunnel. Once the leader is through the joint hold it in a clamp. Then tie the measuring tape to the distal end of the suture leader and pull it through the tibial tunnel, through the joint and then out through the femoral tunnel. If the tape and implant were to be passed in a proximal to distal direction it is more likely that they would jam due to soft tissue being drawn into the tibial tunnel. Measure the correct size of implant: this is the tunnel entrance to tunnel entrance distance plus 4cm, which allows for a 2cm gap between the tunnel entrance and the grommet. Tie the implant to the distal end of the measuring tape and swap the clamp from the tape to the implant. It is convenient to tie the tape onto the implant with a bowline knot which creates a loop that can be easily cut after pulling the implant through the joint. Pass the implant through the tibial tunnel, through the joint and then out through the femoral tunnel. This process is made easier by keeping some tension at both ends and pulling the implant back and forth, two steps forward and one step back. This prevents soft tissue from jamming in the tunnels.

When the implant has been successfully installed anchor the tibial end of the implant with an Apex grommet and screw (Figure 7.6, Figure 7.7 and Figure 7.28). Hold the femoral end of the implant with the Apex tensioning hook and apply sufficient tension to restore normal posterior laxity at about 70° of flexion. This is the angle of flexion, in the region where the aPC is active, at which the knee should be stiffest in posterior drawer (figure 6.11). Also, at 70° of flexion the PCL is required to resist at least 90% of a posterior drawer force (table 6.1), so this is the most sensitive angle to check implant function. It is easier to apply tension with the knee in extension, when the aPC is slack, and then move to 70° of flexion to check for the posterior draw sign, repeating as necessary. When the drawer sign has been eliminated flex and extend the knee 10 times, holding the tensioning hook spike in the bone, to allow the implant to 'bed in'. Then check for posterior laxity again and, if necessary, re-apply tension to eliminate the posterior drawer sign. Mark the required grommet position and fix the implant with another Apex

grommet and screw. Figure 7.28 and Figure 7.29 show the implant in place.

7.10.3.4 Extra–Articular Procedures

If a posterolateral/posteromedial instability was detected it is essential that some extra–articular procedure is performed to correct it. As an example the popliteus bypass of Müller, a posterolateral repair, is illustrated below. There are many varieties of posterolateral and posteromedial reconstruction but there has been little biomechanical analysis of their efficacy. It is not possible to recommend any particular one of them without further study.

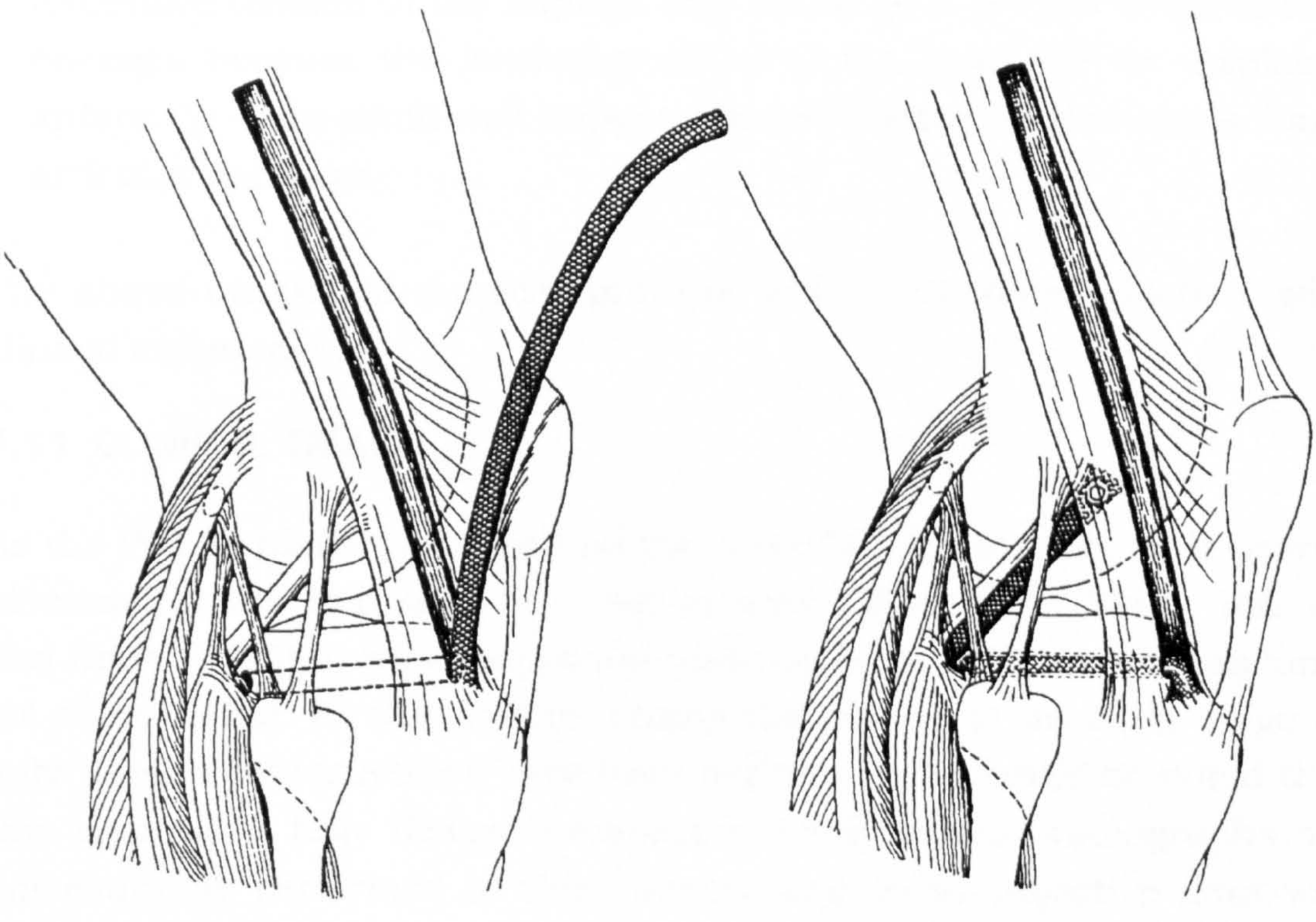


Figure 7.30 Lateral aspect of the knee showing the basis of the Müller popliteus bypass for posterolateral reconstruction. A strip of illiotibial tract is passed posteriorly through a tibial tunnel to follow the line of the natural popliteus tendon from the posterolateral corner or the tibia to its insertion on the epicondyle of the lateral femoral condyle. Taken from Jakob and Warner (1990)

7.10.3.5 Errors And Their Consequences

- Failure to remove impinging osteophytes will probably result in implant rupture.
- Placing the proximal tibial tunnel entrance too far posteriorly may result in implant slackening as the implant cuts/resorbs through the thin cortex of the tibial insertion of the PCL.
- Failure to smooth the femoral insertion tunnel entrance will probably result in implant rupture. The implant is forced to bend round an angle which will cycle between about 30° and 90° during normal walking (Figure 7.20). If a sharp edge is left then cutting will occur and fibres will fail by fatigue as they bend sharply resulting in high strains, and implant failure is likely to be rapid.
- Placing the proximal femoral tunnel entrance too far medially may result in implant slackening as the implant cuts into the thin cortex of the epicondyle
- Insufficient tension in the implant may fail to correct the functional impairment of the knee.
- Excessive tension in the implant may cause pain and/or degenerative changes because the neutral position of the knee will be displaced anteriorly. This could well impose excessive loads on the menisci and articular cartilage.

The above outline of surgical protocol will be developed further with clinical experience.

7.11 CLINICAL TRIALS

As the PCL implant was based on the Apex ACL prosthesis, with proven biocompatibility and lack of complications (Amis et al, 1992; Harries and Amis. 1995), it was considered reasonable to produce PCL implants for clinical trial. At the time of writing the trial is at an early stage as only three reconstructions have been performed. It should be noted that this was not a fully fledged prospective study. Stress radiographs are not routinely performed in this country and so no objective data was available. The reconstructions were fitted in to the surgeon's normal practice and were not controlled by the author. The author attended all the operations and supplied the implant, surgical tools and advice.

The first two operations were performed by Mr Nigel Tubbs of Birmingham General Hospital. Two patients with 'problem' knees who

had persistently requested a reconstruction and who were aware of the experimental nature of the procedure were selected by Mr Tubbs. The operations were carried out in August and November 1992. The first was an open procedure and the second was performed arthroscopically.

No objective data was available so the results can only be reported in terms of the subjective opinions of the surgeon and patients. Mr Tubbs was satisfied at the time of operation that the posterior subluxation had been reduced and that there was a full range of motion. The first patient moved from the area and was not seen for some time by Mr Tubbs. He has subsequently found out that the patient was satisfied with the result of the reconstruction and was able to return to playing rugby league. Unfortunately the patient then reinjured the knee in a road traffic accident. The records of the second patient showed a subjectively poor result with no improvement in anterior knee pain. This may be connected with low compliance to physiotherapy.

The third operation was performed by Mr Robert Mackenney of Watford General Hospital in October 1995. The patient had dislocated his knee, fracturing the medial tibial condyle and rupturing the PCL and LCL. A lateral reconstruction using fascia lata had failed to give satisfactory function. There was severe posterior and posterolateral instability. The PCL was reconstructed using the PCL implant, which eliminated the straight posterior laxity in flexion. It was then found that the previous LCL reconstruction had adhered to the capsule and not to bone. Posterolateral stability was restored by anchoring the LCL reconstruction tissue in a trough in the epicondyle using a screw and bone block.

8. CONCLUSIONS AND SUGGESTIONS FOR FUTURE WORK

The studies reported in this thesis have advanced the understanding of the natural PCL. The mechanical and material properties of the aPC and pPC have been measured and the functions of the aPC and pPC in resisting posterior drawer have been established. Clinical recommendations have been given that should enhance the success of reconstructive surgery for PCL deficiency. On the basis of these recommendations a synthetic implant system has been designed and tested. The system will be further refined with clinical experience.

It has been shown that in the PCL–deficient knee aPC reconstruction using a synthetic implant can restore normal A-P stability below full flexion: the hypothesis that an additional pPC reconstruction would restore A-P stability in full flexion has yet to be tested. It would also be useful, given the present antipathy towards synthetic implants, to test the efficacy of aPC/pPC reconstructions using autogenous material.

The investigation of the function of the PCL in posterior drawer highlighted the importance of 'secondary' structures in resisting posterior drawer near extension. Further work is required to examine the effects of combined injury and reconstruction involving the PCL and the 'secondary' structures of the posterolateral and posteromedial corners.

The accuracy and subtlety of analysis of the forces and motions of the knee would be enhanced if it were possible to allow and measure true unconstrained motion in response to an input force or displacement. It is not possible to produce a passive mechanical system that will allow the axis of rotation of a joint unconstrained movement; an active system is needed. The system would require actuator control of all degrees of freedom combined with position and force feedback. Such a system would enable definitive selective cutting studies to be carried out. It would also be useful in simulating complex injury mechanisms and determining the envelope of motion and axis of rotation allowed by a repair/reconstruction/prosthesis.

Appendix A : ANATOMICAL CONVENTIONS

DEFINITION OF TERMS

The general co-ordinate system for the anatomical descriptions in this thesis is shown in Figure A.1. All descriptions are based on the normal anatomical position, that is with the knee extended. The motion of the knee is described by three translations; anterior–posterior drawer, medial–lateral drawer, and compression–distraction, and three tibial rotations; internal–external rotation, adduction–abduction rotation and flexion–extension rotation (Figure A.2). In this thesis 'flexion' and 'extension' refer to flexion and extension of the knee unless specifically stated otherwise.

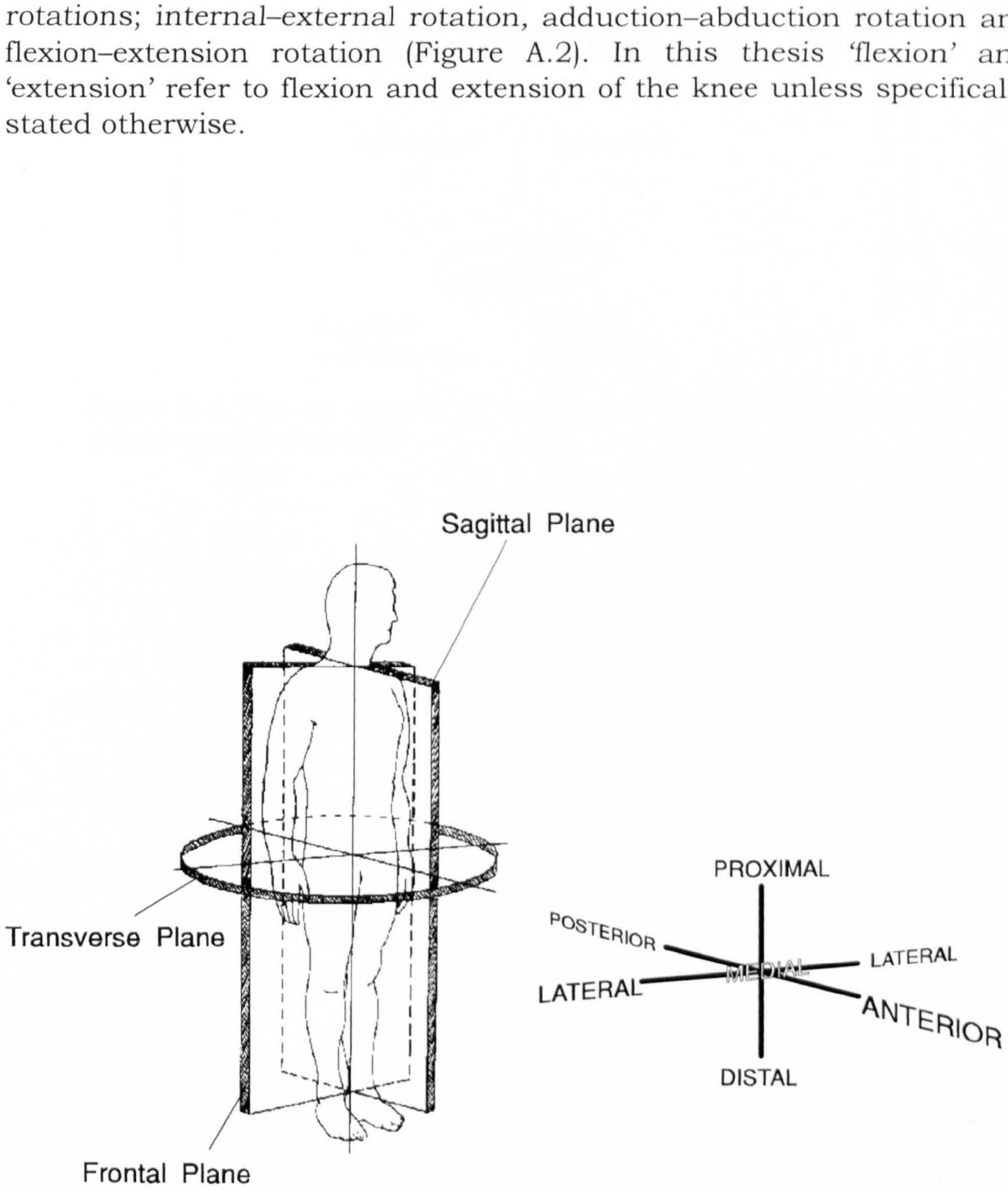


Figure A.1 Schematic diagrams showing the anatomical co-ordinate system.

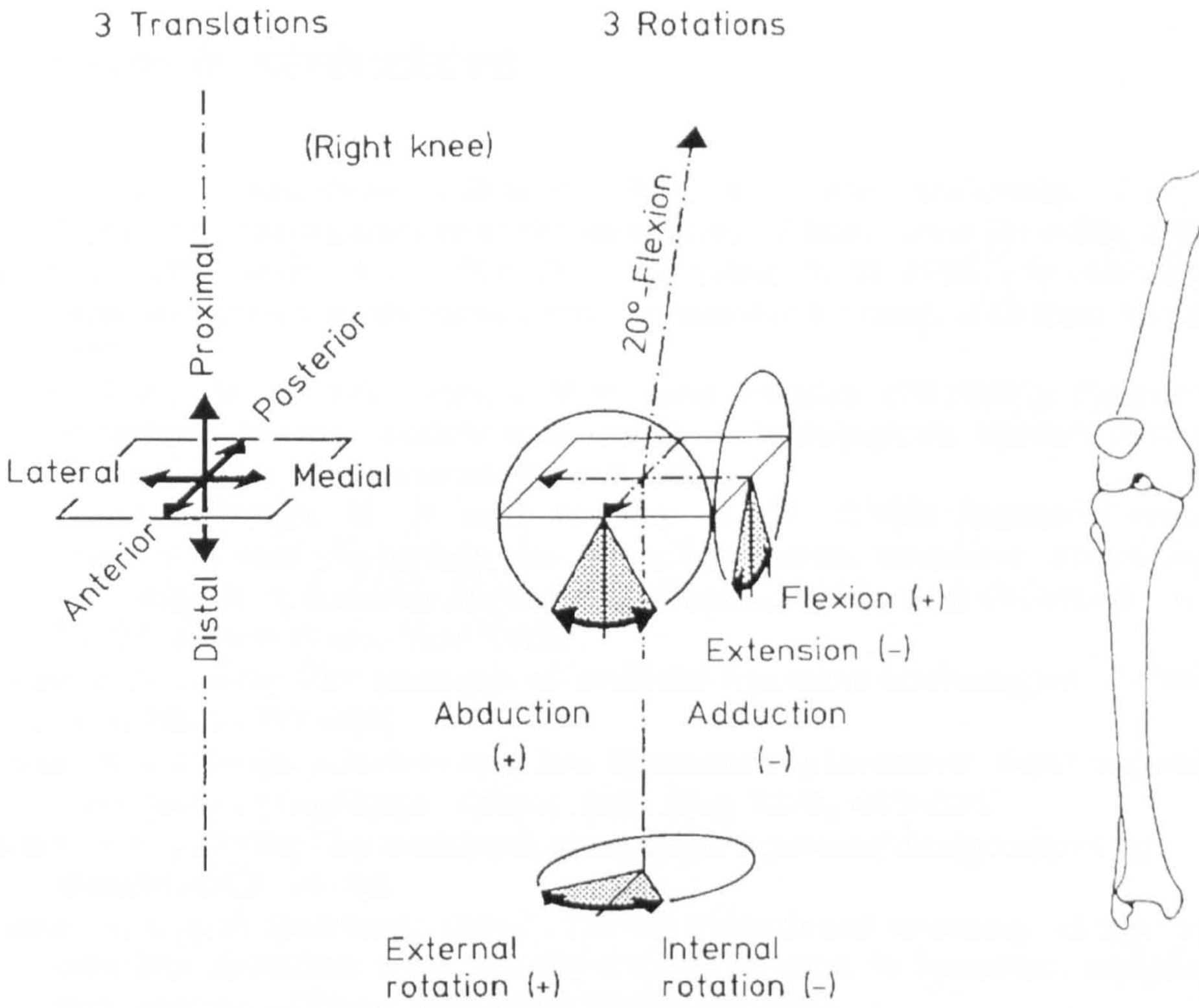


Figure A.2 The six degrees of freedom of the knee. Taken from Stäubli and Jakob (1990)

Appendix B :REFERENCES

Abbott, L.C., Saunders, J.B.D.M., Bost, F.C. and Anderson, C.E. (1944) Injuries to the ligaments of the knee joint. *J Bone Joint Surg* **26,** 503-521.

Ahmed, A.M., Hyder, A., Burke, D.L. and Chan, K. H. (1987) In-vitro ligament tension pattern in the flexed knee in passive loading. *J Orthop Res* **5,** 217-230.

Allen, P.R., Amis, A.A., Jones, M.M. and Heatley FW.(1987) Evaluation of preserved bovine tendon xenografts: a histological, biomechanical and clinical study. *Biomaterials* **8,** 146-152.

Amiel, D., Billings, E. Jr and Akeson, W. H. (1990) Ligament structure, chemistry and physiology. In- *Knee Ligaments: Structure, Function, Injury and Repair.* (Edited by Daniel, D., Akeson, W.H., and O'Conner, J.J.) pp. 77-94. Raven Press, New York.

Amis, A.A. (1988) The strength of artificial ligament anchorages. *J Bone Joint Surg* **70-B,** 397-403.

Amis, A.A. (1989a) Anterior cruciate ligament replacement. Knee stability and the effects of implants. *J Bone Joint Surg* **71-B,** 819-824.

Amis, A.A. (1989b) The evolution of artificial ligament design strategies. *I Mech E* **c384/063,** 91-96.

Amis, A.A. and Dawkins, G.P.C. (1991) Functional anatomy of the anterior cruciate ligament: fibre bundle actions related to ligament replacements and injuries. *J Bone Joint Surg* **73-B,** 260-267.

Amis, A.A., Camburn, M., Kempson, S.A. *Radford WJ, and Stead AC. (1992)* Anterior cruciate ligament replacement with polyester fibre. A long-term study of tissue reactions and joint stability in sheep [published erratum appears in J Bone Joint Surg Br 1993 Jan;75(1): 169]. *J Bone Joint Surg Br* **74,** 605-613.

Amis, A.A., Kempson, S.A., Campbell, J.R and Miller JH., (1988) Anterior cruciate ligament replacement. Biocompatibility and biomechanics of polyester and carbon fibre in rabbits. *J Bone Joint Surg Br* **70,** 628-634.

Andersen, H.N., Frandsen, P.A., and Sondergaard Petersen, P.E. (1991) [Genucom knee analysis after introduction of Dacron prostheses in knees with isolated rupture of the anterior cruciate ligament. 44 consecutive cases]. *Ugeskr Laeger* **153,** 1278-1281.

Anderson, A.F., Snyder, R.B., Federspiel, C.F. and Lipscomb AB. (1992) Instrumented evaluation of knee laxity: a comparison of five arthrometers. *Am J Sports Med* **20,** 135-140.

Andriacchi, T. P., R. P. Mikosz, S. J. Hampton, and J. O. Galante. (1983). Model Studies of the Stiffness Characteristics of the Human Knee Joint. *J. Biomechanics* **16**, 23-29.

Appel, M. and Gradinger, R. (1989) Die Architektur des Kreuzbandaufbaus. *Praktische Sport-Traumatologie und Sportmedizin* **1,** 12-16.

Arms, S.W. and Butler, D.L. (1989) Cruciate ligament fiber bundle recruitment: a mathematical model. *Trans ORS* **14,** 190

Arnauw, G., Verdonk, R., Harth, A. et al., (1991) Prosthetic versus tendon allograft replacement of ACL-deficient knees. *Acta Orthop Belg* **57 Suppl 2,** 67-74.

Bach, B. R., D. J. Daluga, R. Mikosz, T. P. Andriacchi, and R. Seidl. (1992). Force displacement characteristics of the posterior cruciate ligament. *Am. J. Sports Med.* **20**, 67-71.

Baker, C.L., Jr., Norwood, L.A., and Hughston, J.C. (1984) Acute combined posterior cruciate and posterolateral instability of the knee. *Am J Sports Med* **12,** 204-208.

Barfod, B. (1971) Posterior cruciate ligament--reconstruction by transposition of the popliteal tendon. *Acta Orthop Scand* **42,** 438

Barrack, R. L. and Skinner, H. B. (1990) The sensory function of the knee ligaments. In- *Knee Ligaments: Structure, Function, Injury and Repair.* (Edited by Daniel, D., Akeson, W.H., and O'Conner, J.J.) pp. 95-114. Raven Press, New York.

Barrett, G.R., Line, L.L., Jr., Shelton, W.R. Manning JO, and Phelps R. (1993) The Dacron ligament prosthesis in anterior cruciate ligament reconstruction. A four-year review. *Am J Sports Med* **21,** 367-373.

Barry, D. and Ahmed, A.M. (1986) Design and performance of a modified buckle transtucer for the measurement of ligament tension. *J Biomech Eng* **108,** 149-152.

Bartolozzi, P., Salvi, M., and Velluti, C. (1990) Long-term follow-up of 53 cases of chronic lesion of the anterior cruciate ligament treated with an artificial Dacron Stryker ligament. *Ital J Orthop Traumatol* **16,** 467-480.

Barton, T.M., Torg, J.S., and Das, M. (1984) Posterior cruciate ligament insufficiency. A review of the literature. *Sports Med* **1,** 419-430.

Bassett, L.W., Grover, J.S., and Seeger, L.L. (1990) Magnetic resonance imaging of knee trauma. *Skeletal Radiol* **19,** 401-405.

Battle, W.H. (1900) A case after Open Section of the Knee-joint for Irreducible Traumatic Dislocation. *Clin Soc Trans* **XXXIII,** 232-233.

Beauchamp, P., Laurin, C.A., and Bailon, J.P. (1979) [A study of the tensile strength of cruciate ligaments with regard to the possibilities of prosthetic replacement (author's transl)] Etude des proprietes mecaniques des ligaments croises en vue de leur remplacement prothetique. *Rev Chir Orthop* **65,** 197-207.

Berry, J.L., Berg, W.S., Stahurski, T.M. et al., (1988) Evaluation of Dacron-covered and plain bovine xenografts as replacements for the anterior cruciate ligament. *Clin Orthop* **236**, 270-278.

Bianchi, M. (1983) Acute tears of the posterior cruciate ligament: Clinical study and results of 27 cases. *Am J Sports Med* **11,** 308-314.

Blankevoort, L., Huiskes, R., and de Lange, A. (1991) Recruitment of knee joint ligaments. *J Biomech Eng* **113,** 94-103.

Bolton, C.W. and Bruchman, W.C. (1985) The GORE-TEX expanded polytetrafluoroethylene prosthetic ligament. An in vitro and in vivo evaluation. *Clin Orthop* **196**, 202-213.

Bradley, J., J. W. Goodfellow, and J. J. O'Connor. (1987). A radiographic study of bearing movement in unicompartmental Oxford knee replacements. *J. Bone Joint Surg. Br.* **69**, 598-601.

Brantigan, O.C. and Voshell, A.F. (1941) The mechanics of the ligamens and menisci of the knee joint. *J Bone Joint Surg* **23,** 44-66.

Bray, R.C., Flanagan, J.P., and Dandy, D.J. (1988) Reconstruction for chronic anterior cruciate instability: a comparison of two methods after six years. *J Bone Joint Surg* **70-B,** 100-105.

Brennan, J.J. (1960) Avulsion injuries of the posterior cruciate ligaments. *Clin Orthop* **18,** 157-163.

Bruckner, H. and Gutschmidt, P. (1980) [Replacement of the posterior cruciate ligament of the knee by the simultaneously destroyed meniscus (author's transl)] Der Ersatz des hinteren Kreuzbandes durch den mitverletzten Meniskus. *Zentralbl Chir* **105,** 454-459.

Burks, R.T. (1990) Gross Anatomy. In- *Knee Ligaments: Structure, Function, Injury and Repair.* (Edited by Daniel, D., Akeson, W.H., and O'Conner, J.J.) pp. 59-76. Raven Press, New York.

Burks, R.T. and Schaffer, J.J. (1990) A simplified approach to the tibial attachment of the posterior cruciate ligament. *Clin Orthop* **254,** 216-219.

Butel, J., Borgi, R., Mignot, P. and Faure C. (1978) [Surgery of retrospinal disinsertion of the posterior cruciate ligament of the knee (author's transl)] Le traitement chirugical de la desinsertion retrospinale du ligament croise posterieur du genou. *J Chir (Paris)* **115,** 93-96.

Butler, D.L. (1987) Evaluation of fixation methods in cruciate ligament replacement. *A A O S Instructional Course Lectures* **36,** 173-178.

Butler, D.L., Grood, E.S., Noyes, F.R. and Sodd A.N. (1989) Mechanical properties of primate vascularized vs. nonvascularized patellar tendon grafts; changes over time. *J Orthop Res* **7,** 68-79.

Butler, D.L., Kay, M.D., and Stouffer, D.C. (1986) Comparison of material properties in fascicle-bone units from human patellar tendon and knee ligaments. *J Biomechanics* **19,** 425-432.

Butler, D.L., Noyes, F.R., and Grood, E.S. (1980) Ligamentous Restraints to Anterior-Posterior Drawer in the Human Knee. A Biomechanical Study. *J Bone Joint Surg* **62-A,** 259-270.

Butler, D.L., Sheh, M.Y., Stouffer, D.C. Samaranayake V.A., and Levy M.S. (1990) Surface strain variation in human patellar tendon and knee cruciate ligaments. *J Biomech Eng* **112,** 38-45.

Cabaud, H.E. (1983) Biomechanics of the anterior cruciate ligament. *Clin Orthop* **172,** 26-31.

Cain, T.E. and Schwab, G.H. (1981) Performance of an athlete with straight posterior knee instability. *Am J Sports Med* **9,** 203-208.

Campbell, W.C. (1939) Reconstruction of the ligaments of the knee. *Am J Surg* **XLIII,** 473-480.

Chan, K. and Ahmed, A.M. (1991) Polymethylmethacrylate.*Joint Replacement Arthroplasty.* (Edited by Morrey, B.F.) , pp. 23-36. Churchill Livingstone, New York.

Chen, E.H. and Black, J. (1980) Materials design analysis of the prosthetic anterior cruciate ligament. *J Biomed Mater Res* **14,** 567-586.

Chiu, F.Y., Wu, J.J., Hsu, H.C. Lin L., and Lo W.H. (1994a) Management of insufficiency of posterior cruciate ligaments. *Chung Hua I Hsueh Tsa Chih Taipei* **53,** 282-287.

Chiu, F.Y., Wu, J.J., Hsu, H.C. Lin L., and Lo W.H. (1994b) Management of avulsion injury of the PCL with reattachment. *Injury* **25,** 293-295.

Christen, B. and Jakob, R.P. (1992) Fractures associated with patellar ligament grafts in cruciate ligament surgery. *J Bone Joint Surg* **74-B,** 617-619.

Claes, L., Burri, C., Neugebauer, R. Wolter D., and Rose P. (1983) The influence of various carbon fibre braiding techniques and methods of fixation on the extensibility of ligament prostheses. *Biomaterials* **4,** 134-136.

Claes, L., Durselen, L., Kiefer, H. and Mohr W. (1987) The combined anterior cruciate and medial collateral ligament replacement by various materials: a comparative animal study. *J Biomed Mater Res* **21,** 319-343.

Claes, L.E. and Durselen, L. (1990) Biomechanical considerations in the design and use of ligament prostheses.*Current perspectives on implantable devices*. (Edited by Williams, D.F.), pp. 1-63. JAI Press,

Clancy, W.G., Jr., Shelbourne, K.D., Zoellner, G.B. Keene J.S., Reider B., and Rosenberg T.D. (1983) Treatment of knee joint instability secondary to rupture of the posterior cruciate ligament. *J Bone Joint Surg* **65-A,** 310-322.

Clark, F. J., Horch, K. W., Bach, S. M , Larson, G. F. (1979) Contributions of cutaneous and joint receptors to static knee-position sense in man. *J-Neurophysiol* May; **42**(3), 877-88

Cooke, P.H. and Goodship, A.E. (1986) Biomechanical compatibility of cruciate ligament prostheses. *Engineering in Medicine* **15,** 95-98.

Covey, D. C., A. A. Sapega, G. M. Sherman, and J. S. Torg. (1992). Testing for "Isometry" during Posterior Cruciate Ligament reconstruction. *Trans. Orthop. Res. Soc.* **38**, 665

Cross, M.J. and Powell, J.F. (1984) Long-term followup of posterior cruciate ligament rupture: a study of 116 cases. *Am J Sports Med* **12,** 292-297.

Cubbins, W.R., Callahan, J.J., and Scuderi, S.S. (1939) Cruciate ligaments: A résumé of operative attacks and results obtained. *Am J Surg* **XLIII,** 481-485.

Dandy, D.J. and Pusey, R.J. (1982) The long-term results of unrepaired tears of the posterior cruciate ligament. *J Bone Joint Surg* **64-B,** 92-94.

Daniel, D.M. (1991) Assessing the limits of knee motion. *Am J Sports Med* **19,** 139-147.

DeHaven, K.E. (1980) Diagnosis of acute knee injuries with hemarthrosis. *Am J Sports Med* **8,** 9-14.

Dejour, H. and Walch, G. (1987) [Chronic posterior instabilities] Die chronischen hinteren Instabilitaten. *Orthopade* **16,** 149-156.

Dejour, H., Walch, G., Peyrot, J., and Eberhard P. (1988) [The natural history of rupture of the posterior cruciate ligament] Histoire naturelle de la rupture du ligament croise posterieur. *Rev Chir Orthop* **74,** 35-43.

Drucker, M.M. and Wynne, G.F. (1975) Avulsion of the posterior cruciate ligament from its femoral attachment: an isolated ligamentous injury. *J Trauma* **15,** 616-617.

Dunn, M.G., Tria, A.J., Kato, Y.P. et al., (1992) Anterior cruciate ligament reconstruction using a composite collagenous prosthesis. A biomechanical and histologic study in rabbits. *Am J Sports Med* **20,** 507-515.

Edwards, R.G., Lafferty, J.F., and Lange, K.O. (1970) Ligament strain in the human knee joint. *Journal of Basic engineering* 131-136.

Ellis, D.G. (1969) Cross-sectional area measurements for tendon specimens: A comparison of several methods. *J Biomechanics* **2,** 175-186.

Eriksson, E., Haggmark, T., and Johnson, R.J. (1986) Reconstruction of the posterior cruciate ligament. *Orthopedics* **9,** 217-220.

Feder, S.M., Rasmussen, T.J., Butler, D.L., Noyes F.R., and Ratcliffe A. (1994) Biomechanical and Biochemical Changes in Human Tendon Allografts Due to 4Mrad of Gamma Irradiation. *Trans Orthop Res Soc* **19,** 40-

Ficat, P., Cuzacq, J.P., and Ricci, A. (1975) [Surgical repair of chronic instabilities of the cruciate ligaments of the knee] Chirurgie reparatrice des laxites chroniques des ligaments croises du genou. *Rev Chir Orthop* **61,** 89-100.

Fleming, R.E., Jr., Blatz, D.J., and McCarroll, J.R. (1981) Posterior problems in the knee. Posterior cruciate insufficiency and posterolateral rotatory insufficiency. *Am J Sports Med* **9,** 107-113.

Fowler, P.J. and Messieh, S.S. (1987) Isolated posterior cruciate ligament injuries in athletes. *Am J Sports Med* **15,** 553-557.

France, E.P., Daniels, A.U., Goble, E.M. and Dunn H.K. (1983) Simultaneous quantitation of knee ligament forces. *J Biomechanics* **16,** 553-564.

Friederich, N.F., Muller, W., and O'Brien, W.R. (1992) [Clinical application of biomechanic and functional anatomical findings of the knee joint] Klinische Anwendung biomechanischer und funktionell anatomischer Daten am Kniegelenk. *Orthopade* **21,** 41-50.

Fukubayashi, T., Torzilli, P.A., Sherman, M.F. and Warren R.F. (1982) An in vitro biomechanical evaluation of anterior-posterior motion of the knee. Tibial displacement, rotation, and torque. *J Bone Joint Surg* **64,** 258-264.

Fuss, F.K. (1989) Anatomy of the cruciate ligaments and their function in extension and flexion of the human knee joint. *Am J Anat* **184,** 165-176.

Fuss, F.K. (1991) Voluntary rotation in the human knee joint. *J Anat* **179,** 115-125.

Galle, P. (1979) Reconstruction of osseous rupture of posterior cruciate ligament. *Arch Orthop Trauma Surg* **95,** 241-243.

Gallie, W.E. and LeMesurier, A.B. (1927) The repair of injuries to the posterior crucial ligament of the knee-joint. *Ann Surg* **85,** 592-598.

Gallimore, G.W., Jr. and Harms, S.E. (1986) Knee injuries: high-resolution MR imaging. *Radiology* **160,** 457-461.

Geissler, W.B. and Whipple, T.L. (1993) Intraarticular abnormalities in association with posterior cruciate ligament injuries. *Am J Sports Med* **21,** 846-849.

Gillquist, J., Hagberg, G., and Oretorp, N. (1979) Arthroscopic examination of the posteromedial compartment of the knee joint. *Int Orthop* **3,** 13-18.

Girgis, F.G., Marshall, J.L., and Al Monajem, A.R.S. (1975) The cruciate ligaments of the knee joint. Anatomical, functional and experimental analysis. *Clin Orthop* **106,** 216-231.

Gollehon, D.L., Torzilli, P.A., and Warren, R.F. (1985) The role of posterolateral and cruciate ligaments in human knee stability (a biomechanical study) . *Trans ORS* **10,** 270

Gollehon, D.L., Torzilli, P.A., and Warren, R.F. (1987) The role of the posterolateral and cruciate ligaments in the stability of the human knee. A biomechanical study. *J Bone Joint Surg* **69,** 233-242.

Goodship, A.E., Wilcock, S.A., and Shah, J.S. (1985) The development of tissue around various prosthetic implants used as replacements for ligaments and tendons. *Clin Orthop* **196,** 61-68.

Gray, H. 1989. Gray's Anatomy. Churchill Livingstone, Edinburgh.

Grood, E.S. and Noyes, F.R. (1976) Cruciate ligament prosthesis: strength, creep, and fatigue properties. *J Bone Joint Surg Am* **58,** 1083-1088.

Grood, E.S., Hefzy, M.S., and Lindenfield, T.N. (1989) Factors affecting the region of most isometric femoral attachments. Part I: The posterior cruciate ligament. *Am J Sports Med* **17,** 197-207.

Grood, E.S., Stowers, S.F., and Noyes, F.R. (1988) Limits of movement in the human knee. Effect of sectioning the posterior cruciate ligament and posterolateral structures. *J Bone Joint Surg* **70-A,** 88-97.

Grover, J.S., Bassett, L.W., Gross, M.L. Seeger L.L., and Finerman G.A. (1990) Posterior cruciate ligament: MR imaging. *Radiology* **174,** 527-530.

Harries, W.J. and Amis A.A. (1995) The Apex polyester fibre anterior cruciate ligament implant: operative procedure and early results. *The Knee* **1,** 209-215.

Hazen, R. (1910) Traumatic affections of the knee-joint. *Long Island Med J* **March,** 79-89.

Hefzy, M.S. and Grood, E.S. (1985) Effect of femoral attachment location on deformation patterns of the anterior and posterior cruciate ligaments. *ASME Biomechanics Symposium* 89-92.

Hefzy, M.S., Grood, E.S., and Lindenfeld, T.L. (1986) The posterior cruciate ligament: a new look at length patterns. *Trans ORS* **11,** 128

Helfet, A.J. (1948) Function of the crucialte ligaments of the knee-joint. *Lancet* **May 1,** 665-667.

Herzog, W., Read, L. J. (1993) Lines of action and moment arms of the major force-carrying structures crossing the human knee joint. *J-Anat.* 1993 Apr; **182 (Pt 2)**, 213-30

Hey Groves, E.W. (1917) Operation for the repair of the crucial ligaments. *Lancet* **Nov 3rd,** 674-675.

Hey Groves, E.W. (1920) The crucial ligaments of the knee-joint: their function, rupture, and the operative treatment of the same. *Brit J Surg* **VII,** 505-515.

Holden, J.P., Grood, E.S., and Cummings, J.F. (1991) The effect of flexion angle on measurement of anteromedial band force in the goat ACL. *Trans ORS* **16,** 588

Hollis, J.M., Lyon, R.M., Marcin, J.P. Horibe S., Lee E.B., and Woo S.L. (1988) Effect of age and loading axis on the failure properties of the human ACL. *Trans ORS* **13,** 81

Holzach, P., Dietl, G., and Gachter, A. (1985) [Incidence, treatment and late results of fresh and old injuries of posterior cruciate ligaments] Inzidenz, Behandlung und Spatresultate frischer und alter hinterer Kreuzbandlasionen. *Helv Chir Acta* **52,** 189-194.

Horwitz, M.T. (1938) An investigation of the surgical anatomy of the ligaments of the knee joint. *Surg ,Gynec and Obst* **57,** 287-291.

Hsieh, H. and Walker, P.S. (1976) Stabilizing Mechanisms of the Loaded and Unloaded Knee Joint. *J Bone Joint Surg* **58-A,** 87-93.

Hughston, J.C. (1969) The posterior cruciate ligament in knee-joint stability. *J Bone Joint Surg* **51-A,** 1045-1046.

Hughston, J.C. and Degenhardt, T.C. (1982) Reconstruction of the posterior cruciate ligament. *Clin Orthop* **164**, 59-77.

Hughston, J.C., Bowden, J.A., Andrews, J.R. and Norwood L.A. (1980) Acute tears of the posterior cruciate ligament. Results of operative treatment. *J Bone Joint Surg* **62,** 438-450.

Insall, J.N. and Hood, R.W. (1982) Bone-block transfer of the medial head of the gastrocnemius for posterior cruciate insufficiency. *J Bone Joint Surg* **64,** 691-699.

Itokazu, M., Yamane, T., and Shoen, S. (1990) Incomplete avulsion of the femoral attachment of the posterior cruciate ligament with an osteochondral fragment in a twelve-year-old boy. *Arch Orthop Trauma Surg* **110,** 55-57.

Jackson, D.W., Grood, E.S., Goldstein, J., Rosen, M.A., Kurzweil, P.R. and Simon, T.M. (1991) Anterior cruciate ligament reconstruction using patella tendon autograft and allograft -an experimental study in goats. *Trans Orthop Res Soc* **16,** 208-

Jacobsen, K. (1977) Stress radiographical measurements of post-traumatic knee instability. A clinical study. *Acta Orthop Scand* **48,** 301-310.

Jakob, R.P. and Warner, J.P. (1990) Lateral and Posterolateral Rotatory Instability of the Knee. In- The Knee and the Cruciate Ligaments. (Edited by Jakob, R.P. and Stäubli, H.-U.) pp. 464-494. Springer–Verlag, Berlin.

James, S.L., Woods, G.W., Homsy, C.A. Prewitt J.M., and Slocum D.B. (1979) Cruciate ligament stents in reconstruction of the unstable knee. A preliminary report. Clin Orthop 143, 90-96.

Jasty, M., Lew, W.D., and Lewis, J.L. (1982) In vitro ligament forces in the normal knee using buckle transducers. *Trans ORS* **7,** 241

Jenkins, D.H. (1978) The repair of cruciate ligaments with flexible carbon fibre. A longer term study of the induction of new ligaments and of the fate of the implanted carbon. *J Bone Joint Surg Br* **60-B,** 520-522.

Jenkins, D.H., Forster, I.W., McKibbin, B. and Ralis Z.A. (1977) Induction of tendon and ligament formation by carbon implants. *J Bone Joint Surg Br* **59,** 53-57.

Jenny, J.Y., Jenny, G., and Daubresse, F. (1991) [Intermediate results of the replacement of the anterior cruciate ligament with 3 types of dacron prostheses]. *Int Orthop* **15,** 23-28.

Johner, R., Noesberger, B., and Ganz, R. (1979) [The combination of fractures of the lower extremity and lesion of the posterior cruciate ligament] Die Kombination von Schaftfraktur der unteren Extremitat und hinterer Kreuzbandlasion. *Helv Chir Acta* **45,** 797-802.

Jones, R. and Smith, S.A. (1914) On rupture of the crucial ligaments of the knee and on fractures of the spine of the tibia. *Brit J Surg* **1,** 70-89.

Kannus, P. and Jarvinen, M. (1990) Nonoperative treatment of acute knee ligament injuries. A review with special reference to indications and methods. *Sports Med* **9,** 244-260.

Kannus, P., Bergfeld, J., Järvinen, M. et al., (1991) Injuries to the Posterior Cruciate Ligament of the Knee. *Sports Med* **12,** 110-131.

Kapandji, I.A. (1987) The Knee. In: *The Physiology of the Joints*. pp. 64-146. Churchill Linvingstone, London.

Kennedy, J.C. and Galpin, R.D. (1982) The use of the medial head of the gastrocnemius muscle in the posterior cruciate-deficient knee. Indications--technique--results. *Am J Sports Med* **10,** 63-74.

Kennedy, J.C. and Grainger, R.W. (1967) The posterior cruciate ligament. *J Trauma* **7,** 367-377.

Kennedy, J.C., Hawkins, R.J., Willis, R.B. and Danylchuck K.D. (1976) Tension studies of human knee ligaments. Yield point, ultimate failure, and disruption of the cruciate and tibial collateral ligaments. *J Bone Joint Surg* **58,** 350-355.

Klein, W. and Jensen, K.U. (1992) Synovitis and artificial ligaments. *Arthroscopy* **8,** 116-124.

Kurosawa, H., Yamakoshi, K., Yasuda, K. and Sasaki T. (1987) Simultaneous mesaurement of strain distributions in anterior and posterior cruciate ligaments. *Trans ORS* **12,** 125

Kurosawa, H., Yamakoshi, K., Yasuda, K. and Sasaki T. (1991) Simultaneous measurement of changes in length of the cruciate ligaments during knee motion. *Clin Orthop* **265,** 233-240.

Laboureau, J. P. and Dericks, G. H. (1992) Two-bundle prosthetic plasty for reconstruction of the posterior cruciate ligament: 11-year follow up. *J.Bone Joint Surg. Br* **74-B** SUPP III, 258 (Abstract)

Lafortune, M.A., Cavanagh, P.R., Sommer, H.J., III and Kalenak A. (1992) Three-dimensional kinematics of the human knee during walking. *J Biomechanics* **25,** 347-357.

Langrana, N.A. and Bronfeld, J. (1985) Computer-assisted analysis of ligament constraints in the knee. *Clin Orthop* **196,** 42-50.

Last, R.J. (1948) Some anatomical details fo the knee-joint. *J Bone Joint Surg* **30-B,** 683-688.

Law, J.K., Parsons, J.R., Silver, F.H. and Weiss A.B. (1989) An evaluation of purified reconstituted type 1 collagen fibers. *J Biomed Mater Res* **23,** 961-977.

Lee, H.G. (1937) Avulsion fracture of the tibial attachments of the crucial ligaments: treatment by operative reduction. *J Bone Joint Surg* **XIX,** 460-468.

Lee, T.Q. and Woo, S.L. (1988) A new method for determining cross-sectional shape and area of soft tissues. *J Biomed Eng* **110,** 110-114.

Lew, W.D. and Lewis, J.L. (1982) The effect of knee-prosthesis geometry on cruciate ligament mechanics during flexion. *J Bone Joint Surg* **64,** 734-739.

Lewis, J.L. and Fraser, G.A. (1979) On the use of buckle transducers to measure knee ligament forces. *ASME Biomechanics Symposium* 71-74.

Lewis, J.L., Lew, W.D., and Schmidt, J. (1982) A note on the application and evaluation of the buckle transducer for knee ligament force measurement. *J Biomech Eng* **104,** 125-128.

Leyshon, R.L., Channon, G.M., Jenkins, D.H. and Ralis Z.A. (1984) Flexible carbon fibre in late ligamentous reconstruction for instability of the knee. *J Bone Joint Surg Br* **66,** 196-200.

Lindström, N. (1959) Cruciate ligament plastics with meniscus. *Acta Orthop Scand* **29,** 150-152.

Lipscomb, A.B., Johnston, R.K., and Snyder, R.B. (1981) The technique of cruciate ligament reconstruction. *Am J Sports Med* **9,** 77-81.

Loos, W.C., Fox, J.M., Blazina, M.E. Del Pizzo, W., and Friedman, M.J. (1981) Acute posterior cruciate ligament injuries. *Am J Sports Med* **9,** 86-92.

Lopez Vazquez, E., Juan, J.A., Vila, E. et al., (1991) Reconstruction of the anterior cruciate ligament with a Dacron prosthesis. *J Bone Joint Surg* **73,** 1294-1300.

Lysholm, J. and Gillquist, J. (1981) Arthroscopic examination of the posterior cruciate ligament. *J Bone Joint Surg* **63,** 363-366.

Manaster, B.J., Remley, K., Newman, A.P. and Mann, F.A. (1988) Knee ligament reconstruction: plain film analysis. *AJR Am J Roentgenol* **150,** 337-342.

Marinozzi, G., Pappalardo, S., and Steindler, R. (1983) Human Knee Ligaments: Mechanical Tests and Ultrastructural Observations. *Ital J Orthop Traumatol* **9,** 231-240.

Markolf, K.L., Mensch, J.S., and Amstutz, H.C. (1976) Stiffness and laxity of the knee-the contributions of the supporting structures. A quantitative in vitro study. *J Bone Joint Surg* **58,** 583-594.

McCalden, R.W., McGeough, J.A., Barker, M.B. et al, (1993) Age–Related Changes in the Tensile Properties of Cortical Bone. *J Bone Joint Surg* **75-A,** 1193-1205.

McCormick, W.C., Bagg, R.J., Kennedy, C.W., Jr. and Leukens C.A. (1976) Reconstruction of the posterior cruciate ligament: preliminary report of a new procedure. *Clin Orthop* 30-31.

McMaster, W.C. (1975) Isolated posterior cruciate ligament injury: literature review and case reports. *J Trauma* **15,** 1025-1029.

Mears, D.C. (1975) The use of dissimilar metals in surgery. *J Biomed Mater Res Symposium* 133-148.

Meyers, M.H. (1975) Isolated avulsion of the tibial attachment of the posterior cruciate ligament of the knee. *J Bone Joint Surg* **57,** 669-672.

Moore, H.A. and Larson, R.L. (1980) Posterior cruciate ligament injuries. Results of early surgical repair. *Am J Sports Med* **8,** 68-78.

Morrison, J.B. (1969) Function of the knee joint in various activities. *Biomed Eng* **4,** 573-580.

Morrison, J.B. (1970) The Mechanics of the Knee Joint in relation to Normal Walking. *J Biomechanics* **3,** 51-61.

Mott, H.W. (1983) Semitendinosus Anatomic Reconstruction for Cruciate Ligament Insufficiency. *Clin Orthop* **172,** 90-92.

Nielsen, S. and Helmig, P. (1986) Posterior instability of the knee joint. An experimental study. *Arch Orthop Trauma Surg* **105,** 121-125.

Nielsen, S., Ovesen, J., and Rasmussen, O. (1985) The posterior cruciate ligament and rotatory knee instability. An experimental study. *Arch Orthop Trauma Surg* **104,** 53-56.

Noyes, F.R. and Grood, E.S. (1976) The strength of the anterior cruciate ligament in humans and rhesus monkeys. *J Bone Joint Surg* **58-A,** 1074-1082.

Noyes, F.R., Butler, D.L., Grood, E.S. et al., (1984) Biomechanical Analysis of Human Ligament Grafts used in Knee-Ligament Repairs and Reconstructions. *J Bone Joint Surg* **66-A,** 344-352.

Noyes, F.R., DeLucas, J.L., and Torvik, P.J. (1974) Biomechanics of anterior cruciate ligament failure: an analysis of strain-rate sensitivity and mechanisms of failure in primates. *J Bone Joint Surg* **56-A,** 236-253.

O'Brien, W.R., Friederich, N.F., Muller, W. and Leukens C.A. (In Press) Functional anatomy of the cruciate ligaments. *Acta Orthop Scand Supl*

O'Conner, J.J., Shercliff, T., FitzPatrick, D., Bradley, J., Daniel, D.M., Biden, E. and Goodfellow, J. (1990) Geometry of the Knee. In- Knee Ligaments: Structure, Function, Injury and Repair. (Edited by Daniel, D., Akeson, W.H., and O'Conner, J.J.) pp. 77-94. Raven Press, New York.

Ogata, K., McCarthy, J.A., Dunlap, J. and Leukens C.A. (1988) Pathomechanics of posterior sag of the tibia in posterior cruciate deficient knees. An experimental study. Am J Sports Med 16, 630-636.

Palmer, I. (1938) On the injuries to the ligaments of the knee joint. *Acta Orthop Scand Suppl* 5-283.

Parolie, J.M. and Bergfeld, J.A. (1986) Long-term results of nonoperative treatment of isolated posterior cruciate ligament injuries in the athlete. *Am J Sports Med* **14,** 35-38.

Passariello, R., Trecco, F., De Paulis, F. Fascetti, E., Bonanni G., and Beomonte Zobel B. (1986) CT demonstration of capsuloligamentous lesions of the knee joint. *J Comput Assist Tomogr* **10,** 450-456.

Passler, J., Fellinger, M., and Seggl, W. (1989) [Post-traumatic hemarthrosis of the knee joint--an indication for arthroscopy] Der posttraumatische Hamarthros des Kniegelenkes--eine Indikation zur Arthroskopie. *Aktuel Traumatol* **19,** 135-138.

Paulos, L.E., Rosenberg, T.D., Grewe, S.R. Tearse, D.S., and Beck, C.L. (1992) The GORE-TEX anterior cruciate ligament prosthesis. A long-term followup. *Am J Sports Med* **20,** 246-252.

Pavlov, H. and Freiberger, R.H. (1978) An easy method to demonstrate the cruciate ligaments by double contrast arthrography. *Radiology* **126,** 817-818.

Pavlov, H., Hirschy, J.C., and Torg, J.S. (1979) Computed tomography of the cruciate ligaments. *Radiology* **132,** 389-393.

Pickett, J.C. and Altizer, T.J. (1971) Injuries of the Ligaments of the Knee: a study of the types of injury and treatment in 129 patients. *Clin Orthop* **76,** 27-32.

Piziali, R.L., Seering, W.P., Nagel, D.A. and Schurman, D.J. (1980) The function of the primary ligaments of the knee in anterior-posterior and medial-lateral motions. *J Biomechanics* **13,** 777-784.

Polly, D.W., Jr., Callaghan, J.J., Sikes, R.A., McCabe, J.M., McMahon, K., and Savory, C.G. (1988) The accuracy of selective magnetic resonance imaging compared with the findings of arthroscopy of the knee. *J Bone Joint Surg* **70-A,** 192-198.

Pournaras, J. and Symeonides, P.P. (1991) The results of surgical repair of acute tears of the posterior cruciate ligament. *Clin Orthop* **267,** 103-107.

Prietto, M.P., Bain, J.R., Stonebrook, S.N. et al., (1988) Tensile strength of the human posterior cruciate ligament *(PCL) . [Abstract] Trans ORS* 195

Pringle, J.H. (1907) Avulsion of the spine of the tibia. *Ann Surg* **XLVI,** 169-178.

Radford, W. J. P., Amis, A. A., Kempson, S. A., Stead, A. C. and Camburn, M. (1994) A comparative study of single and double bundle ACL reconstructions in sheep. *Knee surg. sports traumat. arth.* **2**(2), 94-99

Radford, W.J. and Amis, A.A. (1990) Biomechanics of a double prosthetic ligament in the anterior cruciate deficient knee. *J Bone Joint Surg Br* **72,** 1038-1043.

Reicher, M.A., Hartzman, S., Bassett, L.W., Mandelbaum, B., Duckwiler, G., and Gold, R.H. (1987) MR imaging of the knee. Part I. Traumatic disorders. *Radiology* **162,** 547-551.

Richmond, J.C., Manseau, C.J., Patz, R. and McConville O. (1992) Anterior cruciate reconstruction using a Dacron ligament prosthesis. A long-term study. *Am J Sports Med* **20,** 24-28.

Rong, G.W. and Wang, Y.C. (1987) The role of cruciate ligaments in maintaining knee joint stability. *Clin Orthop* **215,** 65-71.

Roth, J.H., Bray, R.C., Best, T.M. et al., (1988) Posterior cruciate ligament reconstruction by transfer of the medial gastrocnemius tendon. *Am J Sports Med* **16,** 21-28.

Rovick, J.S., Reuben, J.D., Robertson, D.D. et al., (1987) Control of knee kinematics by ligaments. [Abstract] *Trans ORS* 126

Satku, K., Chew, C.N., and Seow, H. (1984) Posterior cruciate ligament injuries. *Acta Orthop Scand* **55,** 26-29.

Sayles, R.S., Thomas, T.R., Anderson, J. Haslock, I., and Unsworth, A. (1979) Measurement ofhte surface microgeometry of articular cartalage. *J Biomechanics* **12,** 257-267.

Scharling, M. (1981) Replacement of the anterior cruciate ligament with a polyethylene prosthetic ligament. *Acta Orthop Scand* **52,** 575-578.

Schreiber, S.N. (1991) Arthroscopy update #9. Posterior compartment observation and instrumentation in the knee using anteromedial and anterolateral portals and an interchangeable cannula system. *Orthop Rev* **20,** 67-8, 73, 76-.

Seedhom, B. B., Dowson, D. and Wright, V. (1973) Wear of solid phase formed high density polyethylene in relation to the life of artificial hips and knees. *Wear* **24**, 35-51

Selvik, G. (1979) Roentgen Stereophotogrammetric methods for the evaluation of the three dimensional kinematic behaviour and cruciate ligament length patterns of the human knee joint. [Abstract] *J Biomechanics* 12:727-31.

Sepulchre, P. and Blaimont, P. (1986) [Biomechanics of the posterior cruciate ligament of the knee] Biomecanique du ligament croise posterieur du genou. *Acta Orthop Belg* **52,** 437-448.

Sidles, J.A., Larson, R.V., Garbini, J.L. Downey, D.J., and Matsen, F.A. (1988) Ligament length relationships in the moving knee. *J Orthop Res* **6,** 593-610.

Smith, S.A. (1918) The diagnosis and treatment of injuries to the crucial ligaments. *Brit J Surg* **VI,** 176-189.

Southmayd, W.W. and Rubin, B.D. (1980) Reconstruction of the posterior cruciate ligament using the semimembranosus tendon. *Clin Orthop* **150**, 196-197.

Stanish, W.D., Rubinovich, M., Armason, T. and Lapenskie G. (1986) Posterior cruciate ligament tears in wrestlers. *Can J Appl Sport Sci* **11,** 173-177.

Strasser, H. (1917) Lehrbuch der Muskel und gelenkmechanick. Springer, Berlin.

Stäubli, H.-U. and Jakob, R.P. (1990) Terms, Definitions, and Glossaries. In- The Knee and the Cruciate Ligaments. (Edited by Jakob, R.P. and Stäubli, H.-U.) pp. 2-21. Springer–Verlag, Berlin.

Strover, A.E. (1992) The Surgicraft® ABC ligament for chronic anterior cruciate ligament insufficiency. *Knee Surgery Current Practice.* (Edited by Aichroth, P.M. and Cannon, W.D. Jr.), pp. 281-297. Martin Dunitz, London.

Strover, A.E. and Firer, P. (1985) The use of carbon fiber implants in anterior cruciate ligament surgery. *Clin Orthop* **196,** 88-98.

Tesmoingt, P. and Dossa, J. (1982) [Rupture of the posterior cruciate ligament. Palliative treatment by transposition of the internal gastrocnemius mucle into the intercondyloid notch (author's transl)] Ruptures du ligament croise posterieur. Traitement palliatif par transposition du jumeau interne dans l'echancrure intercondylienne. *Nouv Presse Med* **11,** 201-202.

Tibone, J.E., Antich, T.J., Perry, J. and Moynes D. (1988) Functional analysis of untreated and reconstructed posterior cruciate ligament injuries. *Am J Sports Med* **16,** 217-223.

Tkaczuk, H. (1968) Tensile properties of human lumbar longitudinal ligaments. *Acta Orthop Scand Suppl* **115,** 5-69.

Torg, J.S., Barton, T.M., Pavlov, H. and Stine R. (1989) Natural history of the posterior cruciate ligament-deficient knee. *Clin Orthop* **246,** 208-216.

Torisu, T. (1977) Isolated avulsion fracture of the tibial attachment of the posterior cruciate ligament. *J Bone Joint Surg* **59,** 68-72.

Torisu, T. (1979) Avulsion fracture of the tibial attachment of the posterior cruciate ligament. Indications and results of delayed repair. *Clin Orthop* **143,** 107-114.

Trent, P.S., Walker, P.S., and Wolf, B. (1976) Ligament length patterns, strength, and rotational axes of the knee joint. *Clin Orthop* **117,** 263-270.

Trickey, E.L. (1968) Rupture of the posterior cruciate ligament of the knee. *J Bone Joint Surg* **50,** 334-341.

Trickey, E.L. (1980) Injuries to the posterior cruciate ligament: diagnosis and treatment of early injuries and reconstruction of late instability. *Clin Orthop* **147,** 76-81.

Turner, D.A., Prodromos, C.C., Petasnick, J.P. and Clark, J.W. (1985) Acute injury of the ligaments of the knee: magnetic resonance evaluation. *Radiology* **154,** 717-722.

Turner, I.G. and Thomas, N.P. (1990) Comparative analysis of four types of synthetic anterior cruciate ligament replacement in the goat: in vivo histological and mechanical findings. *Biomaterials* **11,** 321-329.

Van Dommelen, B.A. and Fowler, P.J. (1989) Anatomy of the posterior cruciate ligament. A review. *Am J Sports Med* **17,** 24-29.

Verdonk, R., Vandendriessche, G., De Smet, L., Van Overbeke, J, Vandekerckhove, B, and Claessens, H. (1986) Free patellar tendon graft for reconstruction of old posterior cruciate ligament ruptures. *Acta Orthop Belg* **52,** 554-560.

Viidik, A., Sandqvist, L., and Magi, M. (1965) Influence of post-mortal storage on tensile strength characteristics and histology of ligaments. *Acta Orthop Scand Suppl* **79,** 1-35.

Wang, C. and Walker, P.S. (1974) Rotatory Laxity of the Human Knee Joint. *J Bone Joint Surg* **56-A,** 161-170.

Wang, C., Walker, P.S., and Wolf, B. (1973) The effects of flexion and rotation on the length patterns of the ligaments of the knee. *J Biomechanics* **6,** 587-596.

Wascher, D.C., Markolf, K.L., and Finerman, G.A.M. (1991) Direct in vitro measurement of total PCL force: new insights into injury mechanisms. [Abstract] *Trans ORS* 160

Whipple, T.L. and Ellis, F.D. (1991) Posterior Cruciate Ligament Injuries. *Clin Sports Med* **10,** 515-527.

Wilk, R.M. and Richmond, J.C. (1993) Dacron ligament reconstruction for chronic anterior cruciate ligament insufficiency. *Am J Sports Med* **21,** 374-379.

Wirth, C.J. (1989) [Cruciate ligament injuries of the knee joint] Kreuzbandverletzungen des Kniegelenkes. *Orthopade* **18,** 302-13; discus.

Wirth, C.J. and Jager, M. (1984) Dynamic double tendon replacement of the posterior cruciate ligament. *Am J Sports Med* **12,** 39-43.

Wismans, J., Veldpaus, F. and Janssen J. (1980) A Three-Dimensional Mathematical Model Of the Knee-Joint. *J. Biomechanics* **13**, 677-685.

Woo, S.L. (1982) Mechanical properties of tendons and ligaments:I. Quasi-static and nonlinear viscoelastic properties. *Biorheology* **19,** 385-396.

Woo, S.L., Danto, M.I., Ohland, K.J., Lee, T.Q. and Newton, P.O. (1990) The use of a laser micrometer system to determine the cross-sectional shape and area of ligaments: a comparative study with two existing methods. *J Biomech Eng* **112,** 426-431.

Yerys, P. (1991) Arthroscopic posterior cruciate ligament repair. Arthroscopy 7, 111-114.

Zavatsky, A. B. 1993. The Functional Architecture of Human Knee Ligaments. Phd Thesis Oxford University, Hertford College.

Zavatsky, A. B. and J. J. O'Connor. (1992a). A Model of Human Knee Ligaments in the Sagittal Plane Part 1: Response to Passive Flexion. Proc. Inst. Mech. Eng. H. 206, 125-134.

Zavatsky, A. B. and J. J. O'Connor. (1992b). A Model of Human Knee *Ligaments in the Sagittal* Plane Part 2: Fibre Recruitment under Load. Proc. Inst. Mech. Eng. H. 206, 135-145.

Zoghi, M., Hefzy, M.S., Fu, K. C. (1992) Jackson-WT A three-dimensional morphometrical study of the distal human femur. Proc-Inst-Mech-Eng-H. 206, 147-57

Appendix C :PUBLICATIONS

Race, A. And Amis, A.A. (1992). Mechanical properties of the two bundles of the human posterior cruciate ligament *Trans. O.R.S.* **17**, 124. (Abstract)

Race, A. And Amis, A.A. (1993) Mechanical properties and loading of the two bundles of the posterior cruciate ligament: a new perspective on PCL function. *J. Bone Joint Surg.* **75-B** SUPP I, 54. (Abstract)

Race, A. And Amis, A.A. (1993) Loading of the two bundles of the posterior cruciate ligament: an analysis of bundle function in A-P drawer. *Trans. E.O.R.S.* **3**, 67. (Abstract)

Race, A. And Amis, A.A. (1994) Loading of the two bundles of the posterior cruciate ligament: an analysis of bundle function in A-P drawer. *Trans. O.R.S.* **19**, 606. (Abstract)

Race, A. And Amis, A.A. (1994) A moulding method to find cross–sections of soft tissue bundles with complex shapes. *Trans. O.R.S.* **19**, 783. (Abstract)

Race, A. And Amis, A.A. (1994). Mechanical properties of the two bundles of the human posterior cruciate ligament. *J. Biomec* **27**, 13–23

Race, A. And Amis, A.A. (1996) Loading of the two bundles of the posterior cruciate ligament: an analysis of bundle function in A-P drawer. *J. Biomec* (In Press)

Race, A. And Amis, A.A. (1996) Cross–sectional area measurement of soft tissue – a new casting method. (Technical Note) *J. Biomec* (In Press)

Appendix D :DRAWINGS

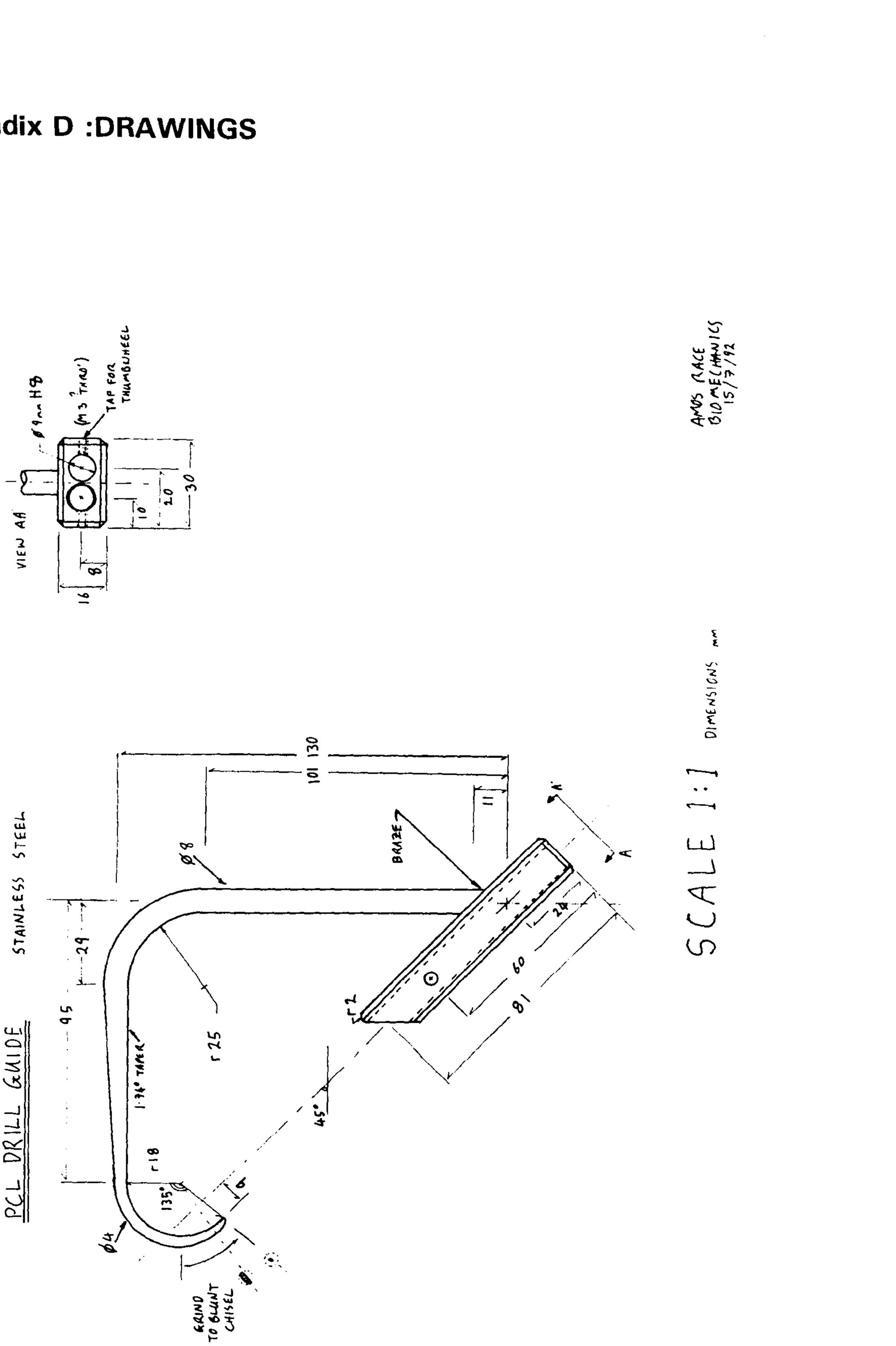


Figure D.1 Tibial drill guide mk1.

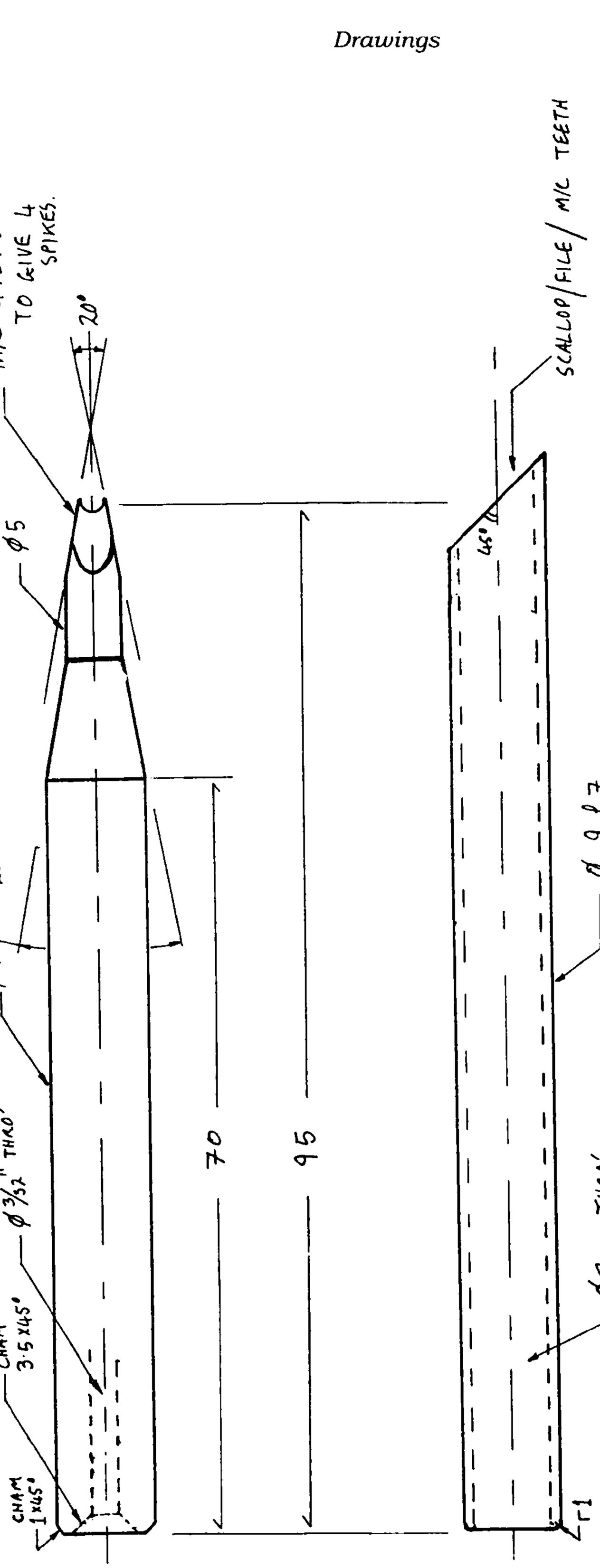


Figure D.2 Barrels to hold guide wire and 7mm drill for tibial drill guide mk1.

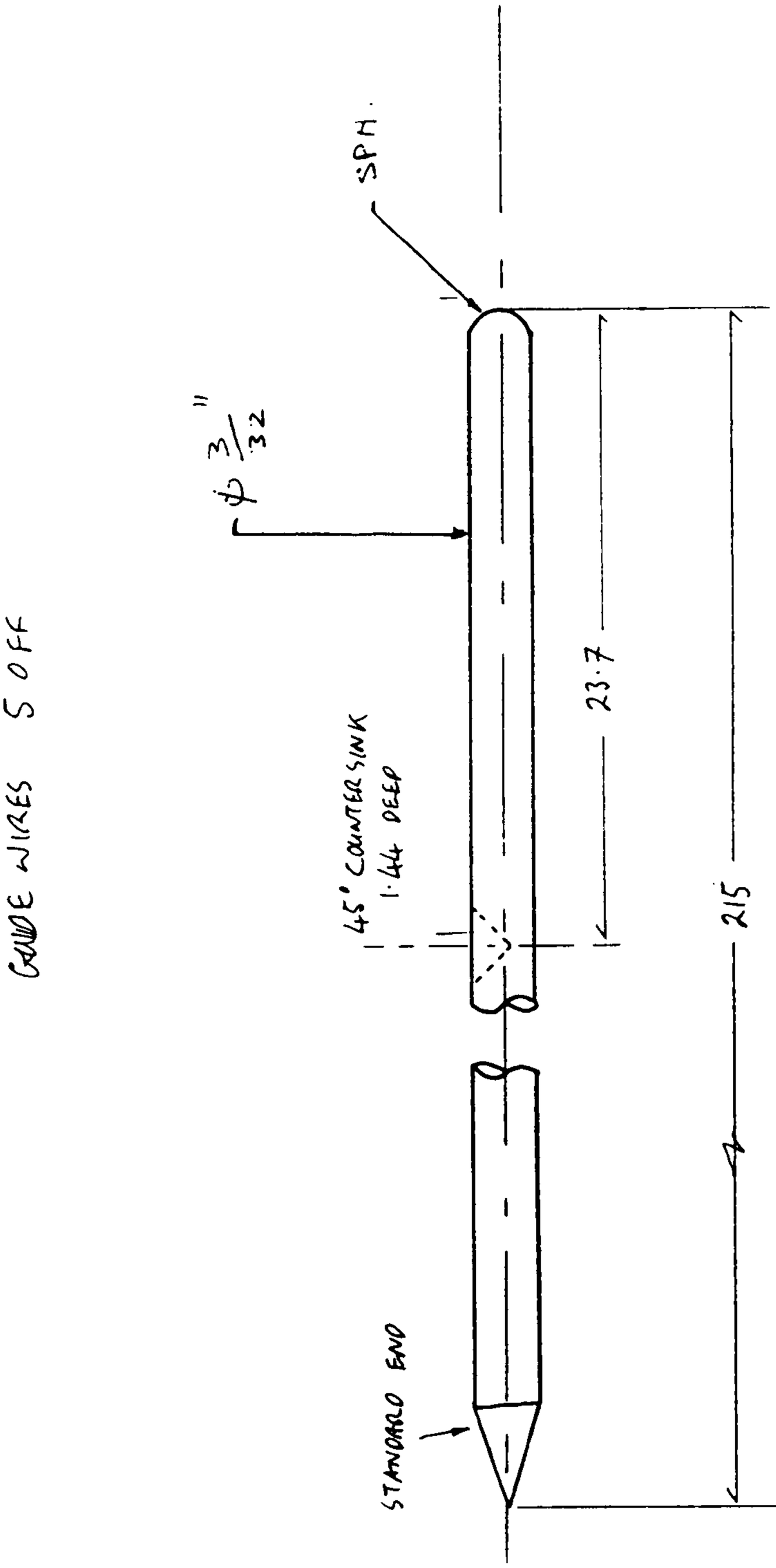


Figure D.3 Guide wire, modified to fit into guide wire holder.

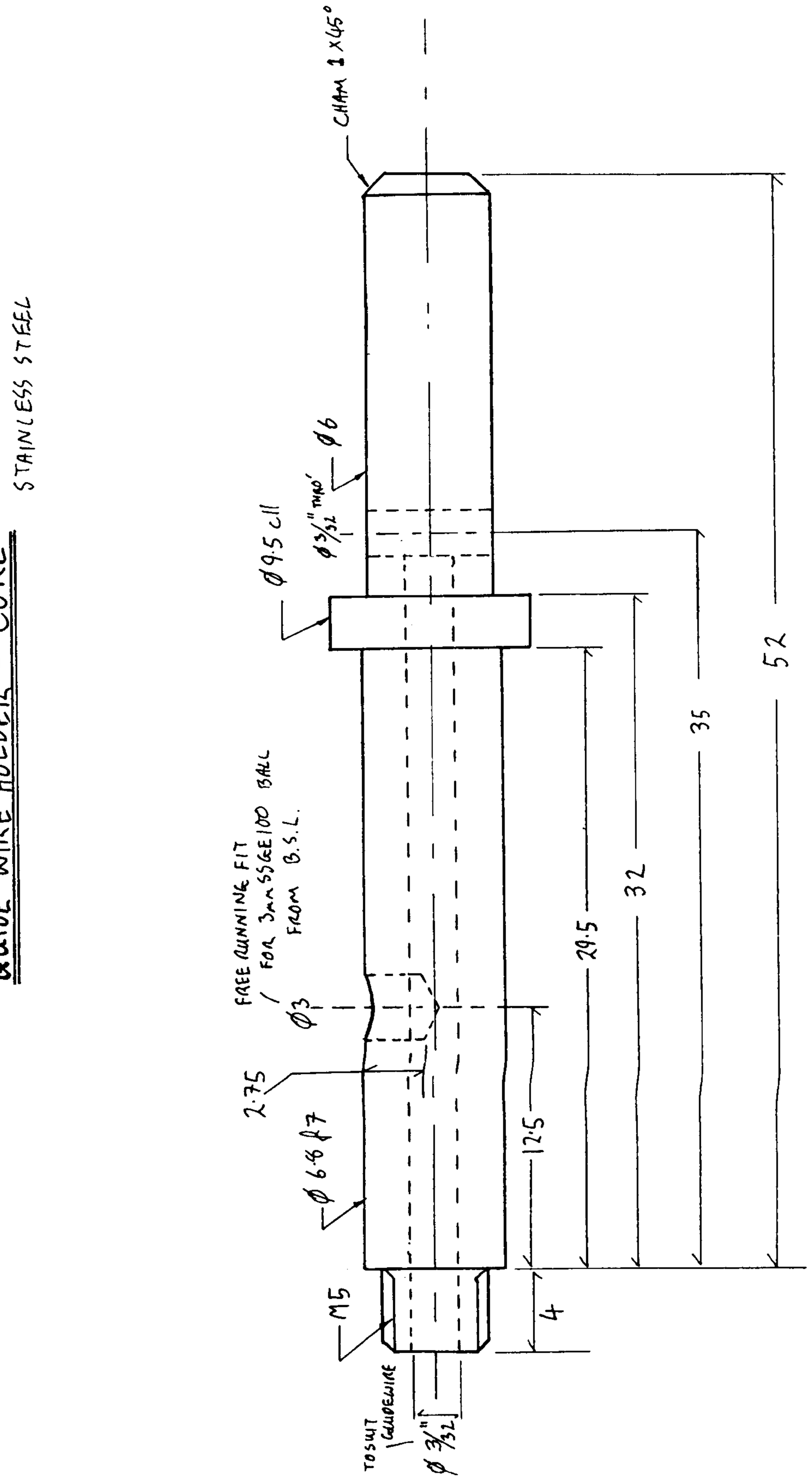


Figure D.4 Core of guide wire holder.

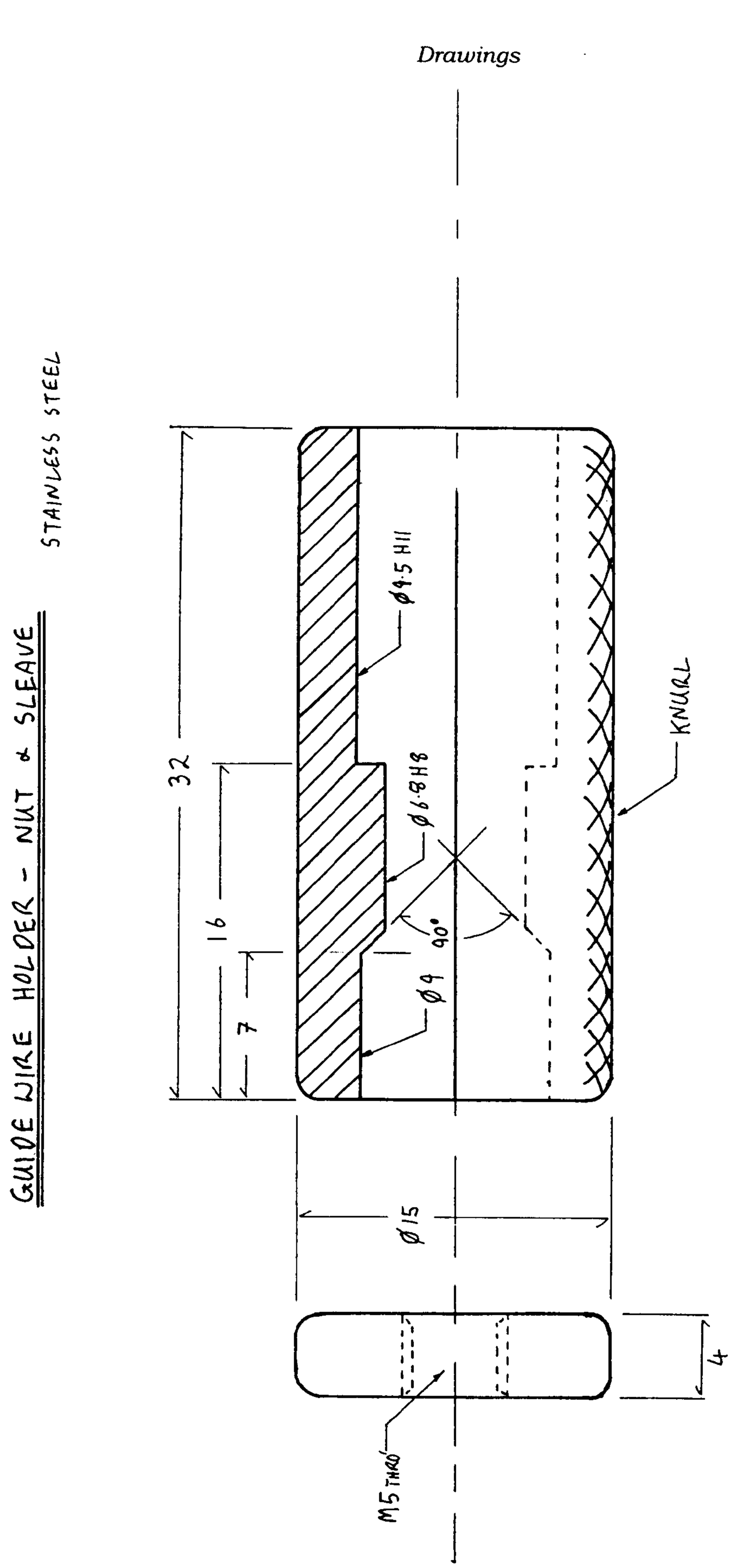


Figure D.5 Outer sleave of guide wire holder.

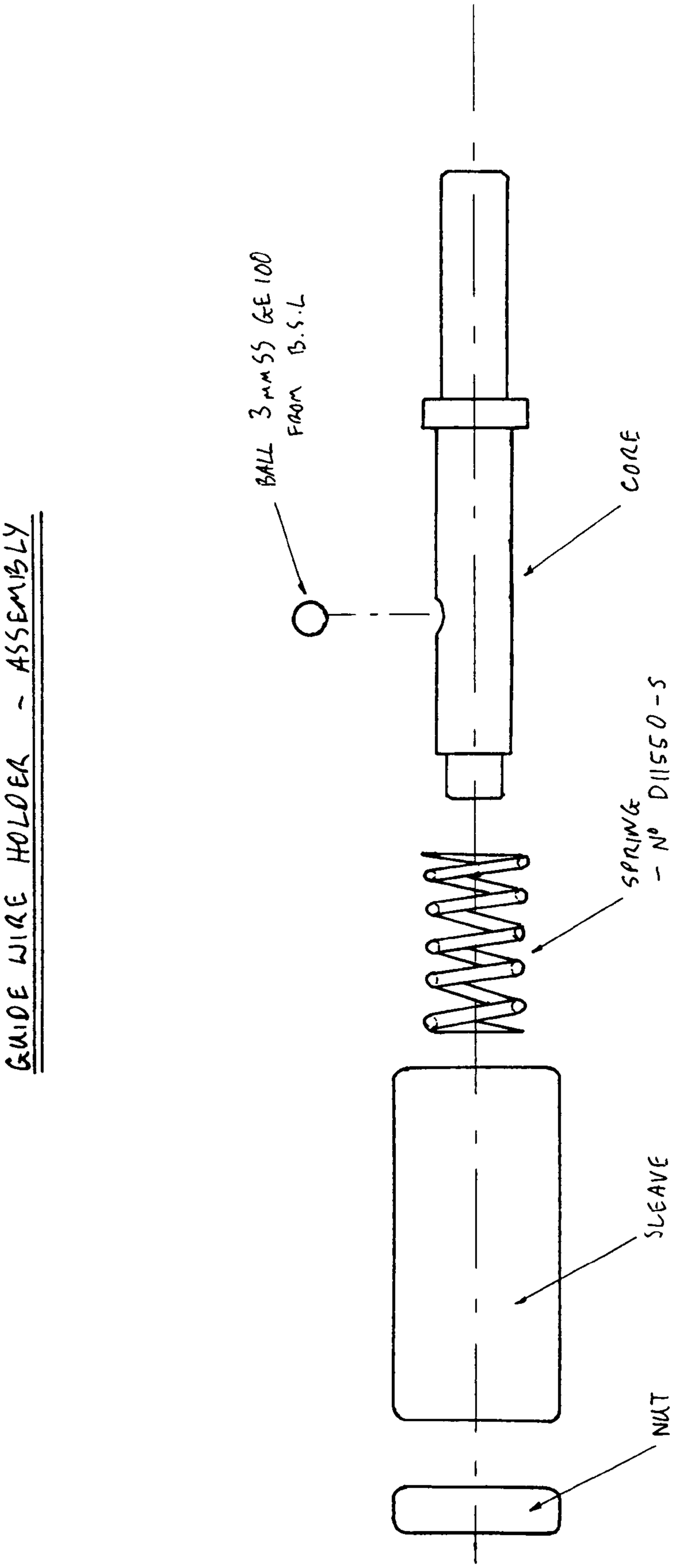


Figure D.6 Guide wire holder assembly.

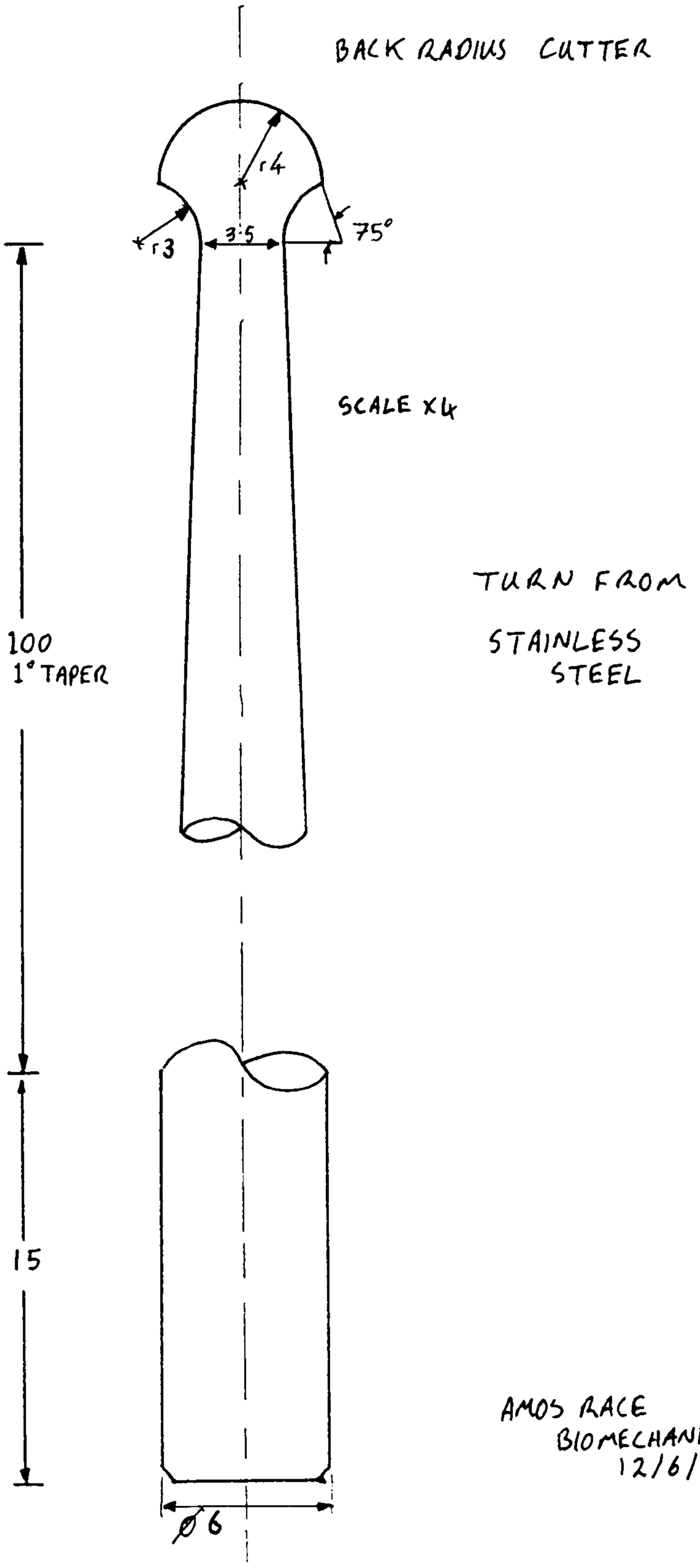
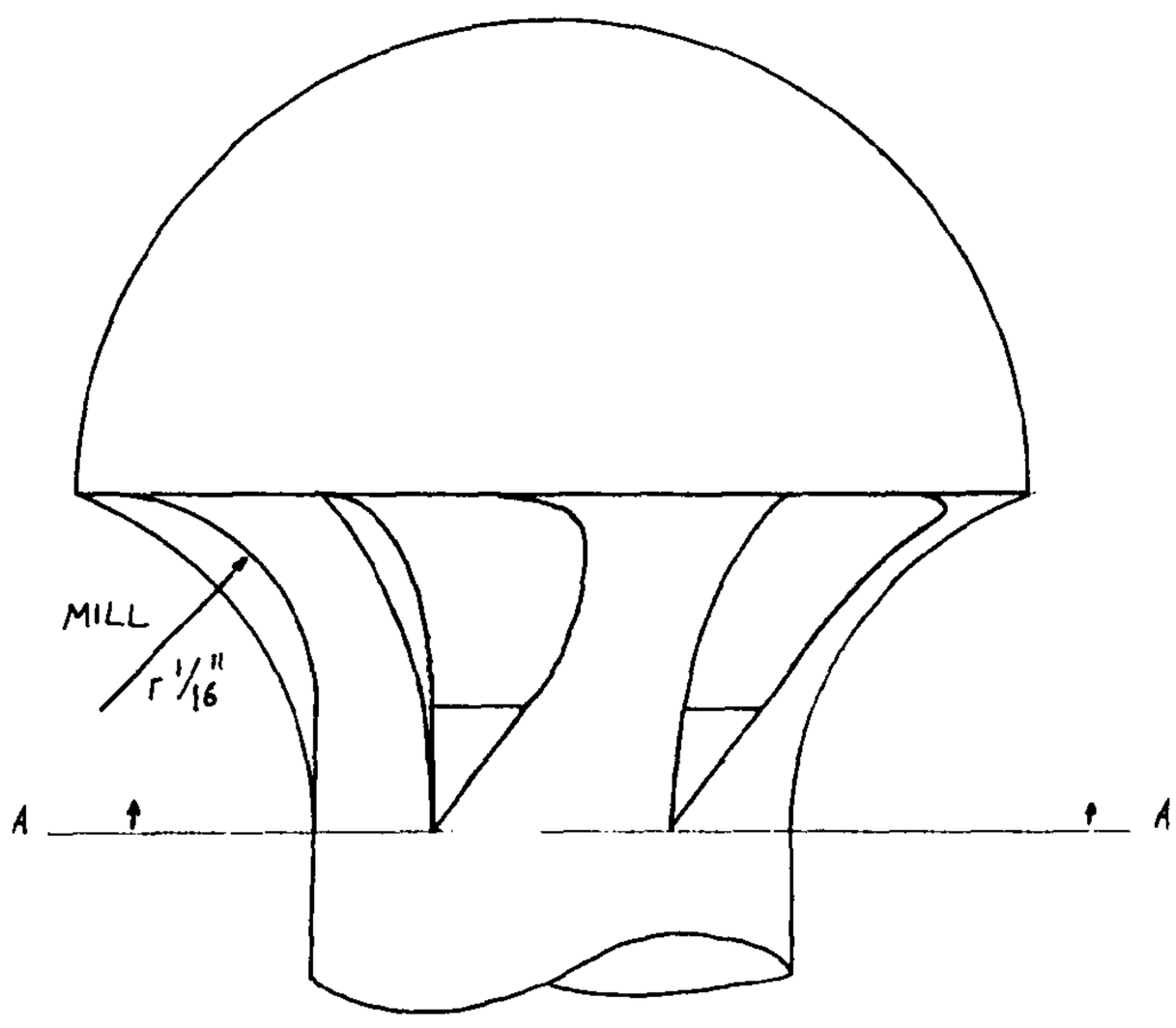
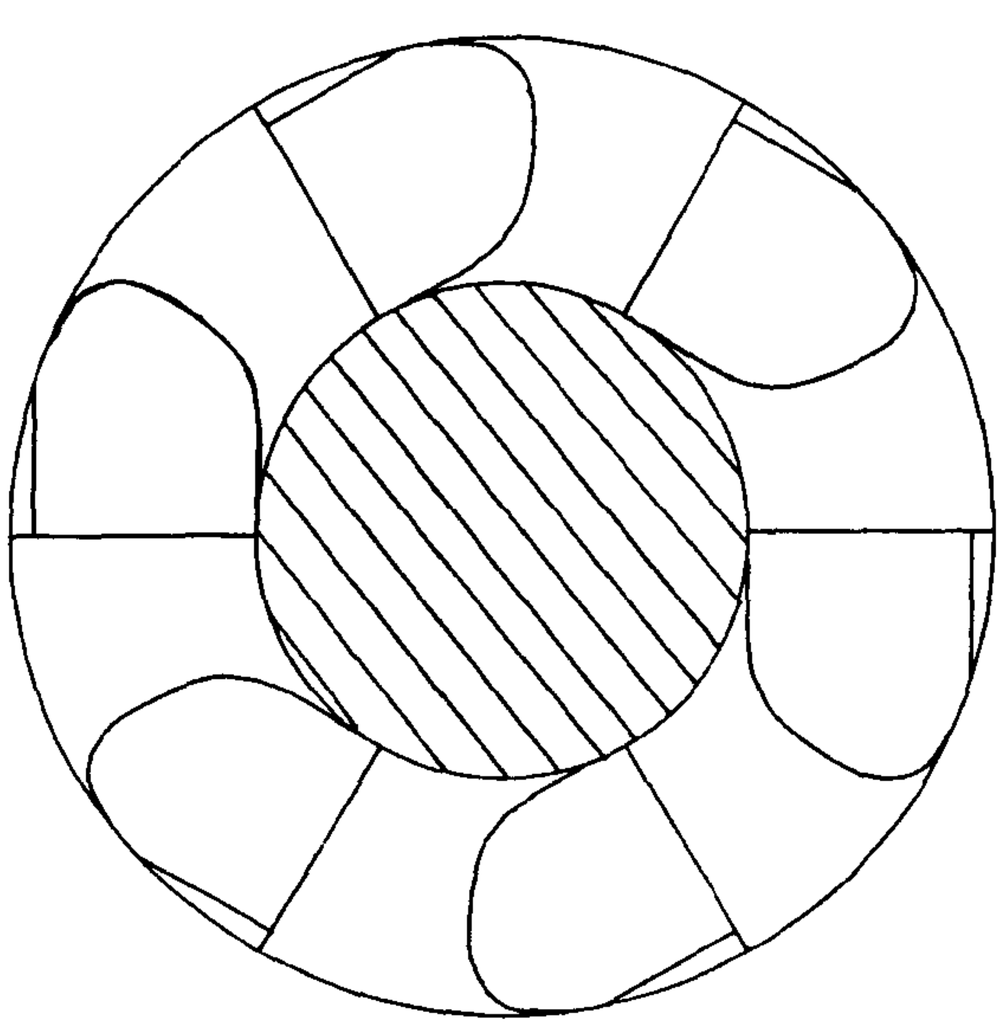


Figure D.7 Back radius cutter.





20:1

SECTION AA

AMOS RACE

BIOMECHANICS

12/6/92

Figure D.8 Detail of cutting head for back radius cutter.

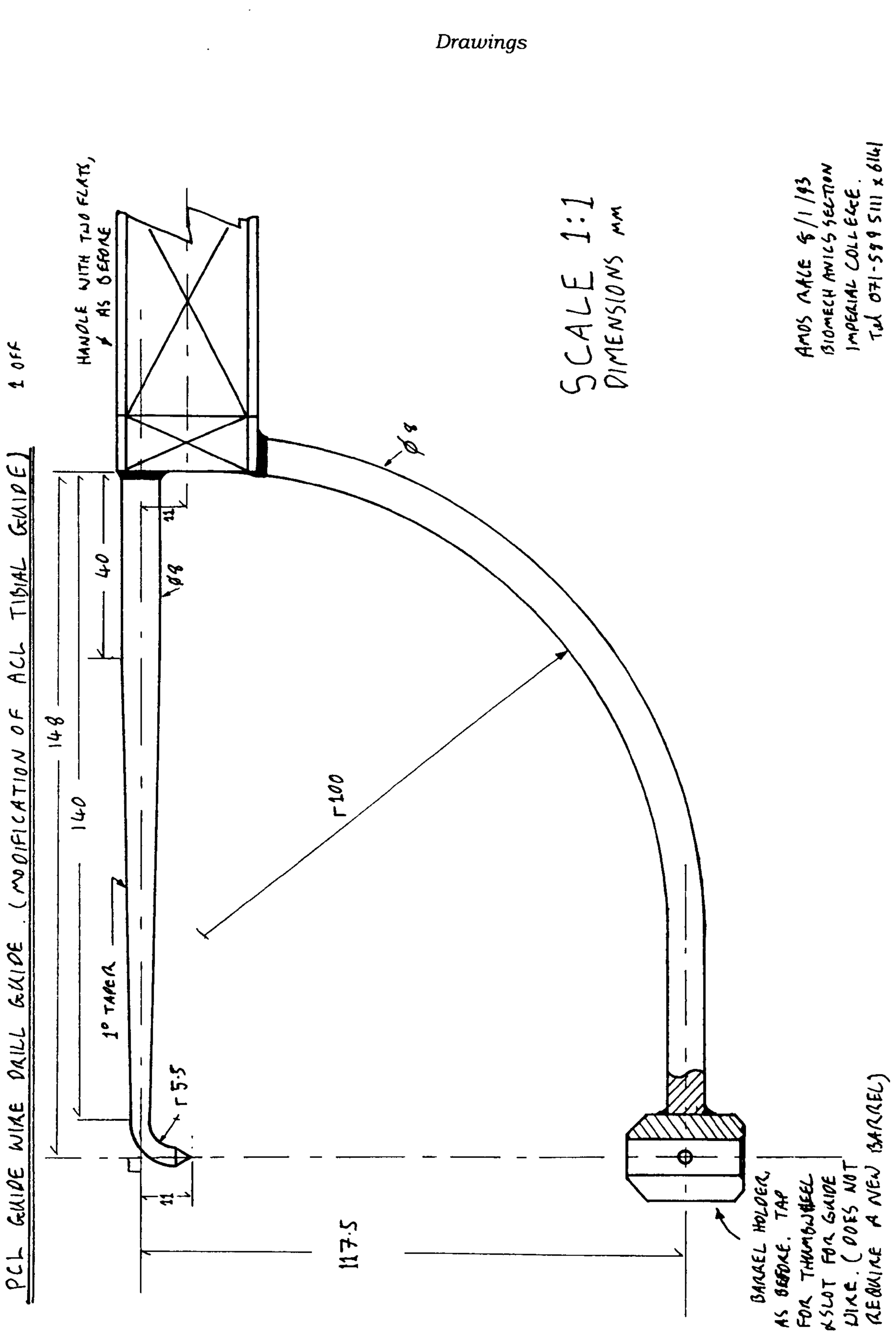


Figure D.9 Femoral guide wire drill guide.

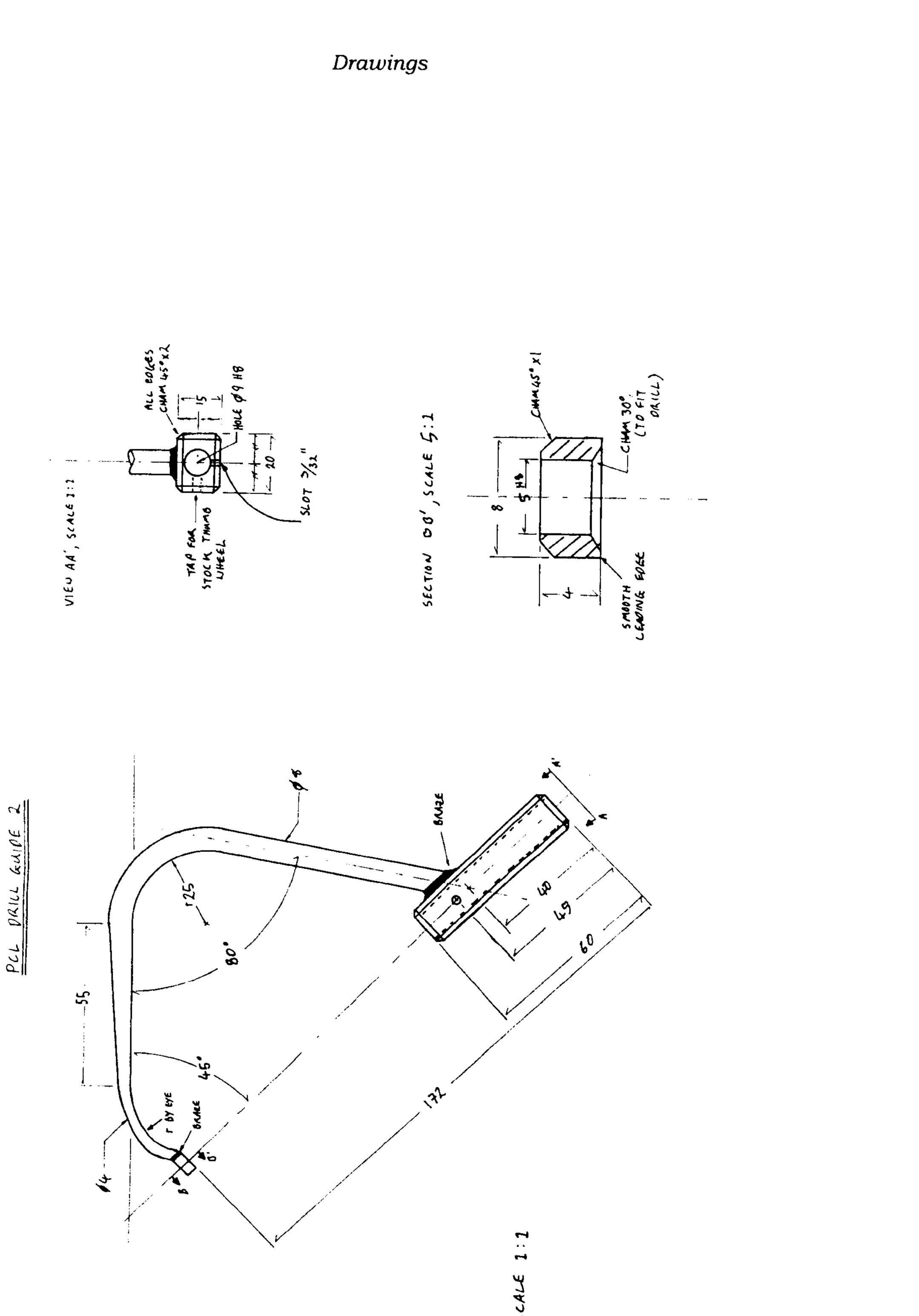


Figure D.10 Tibial drill guide mk2.

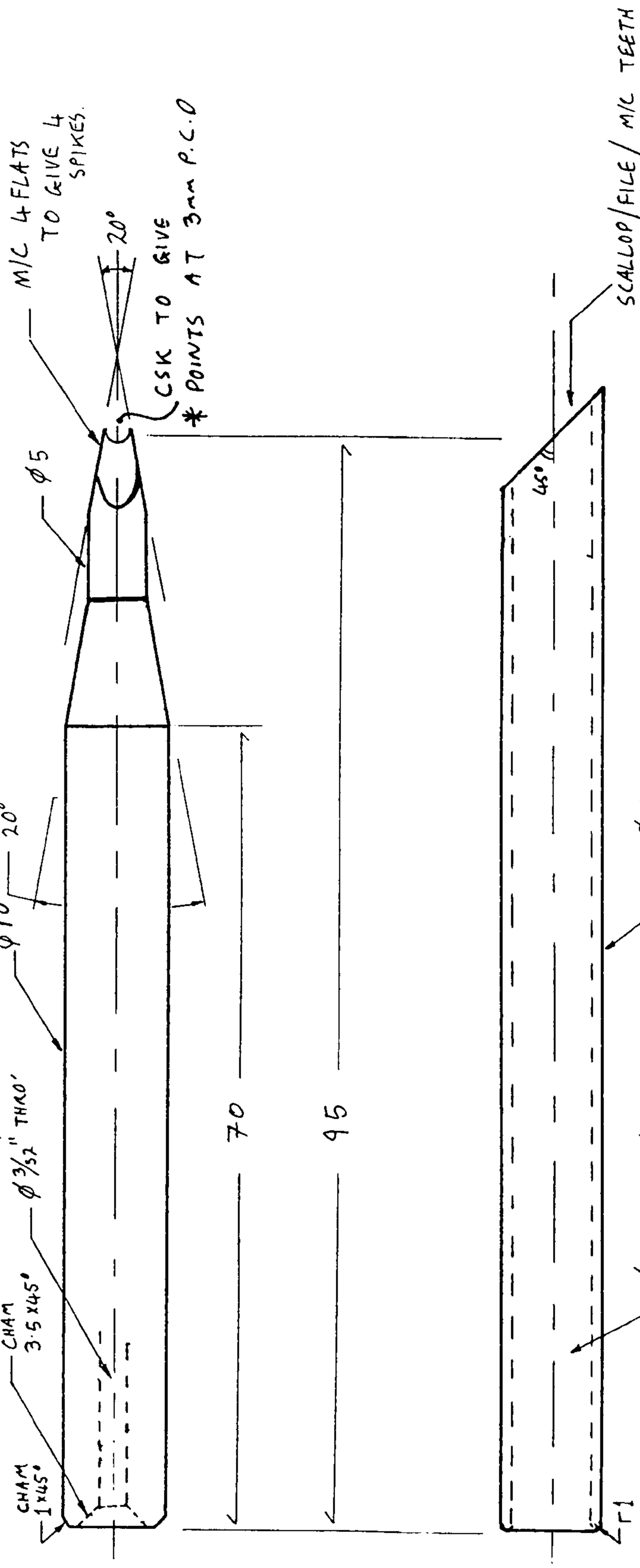


Figure D.11 Barrels to hold guide wire and 7mm drill for tibial drill guide mk2.

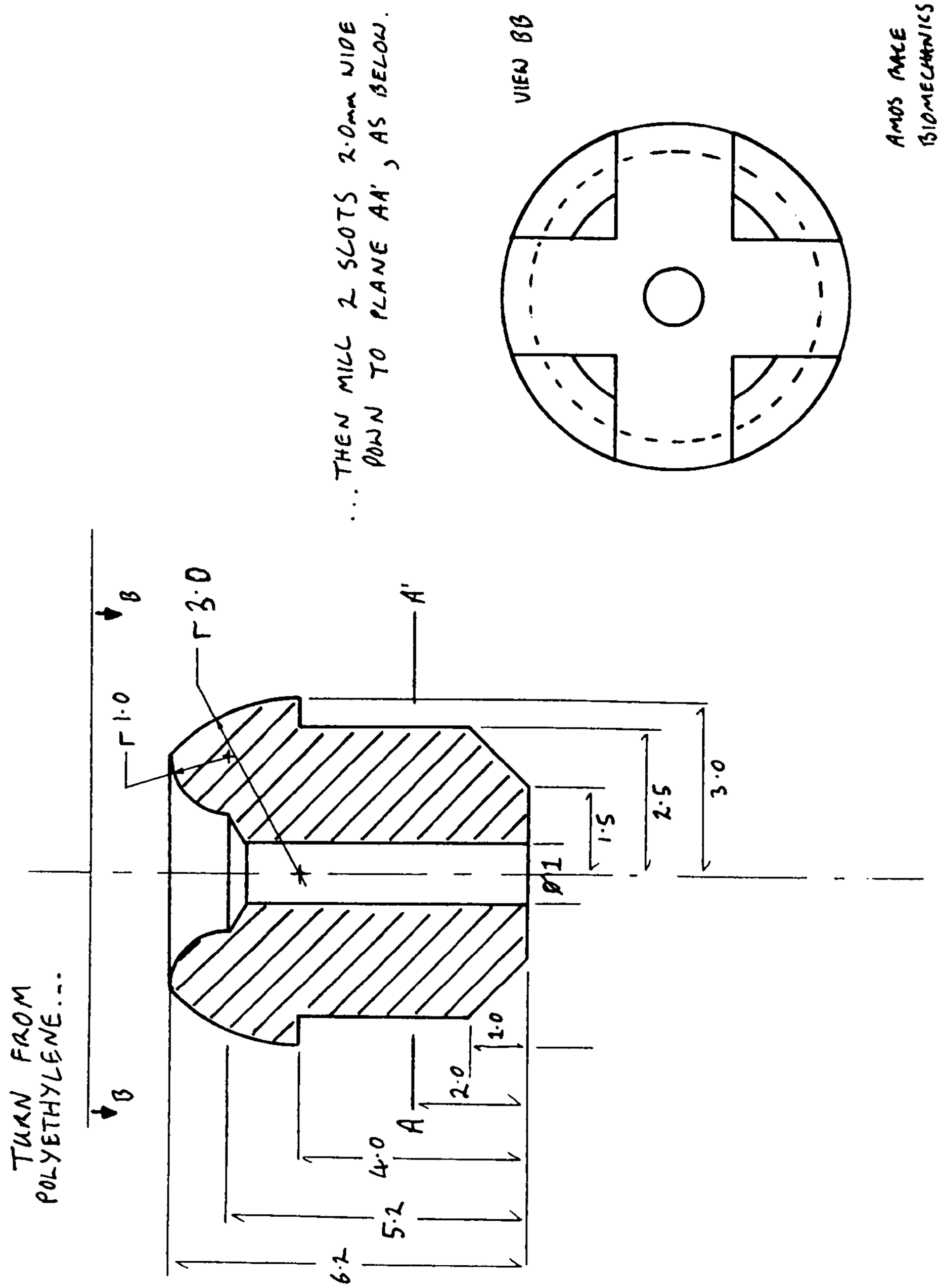


Figure D.12 Suture leader bead.

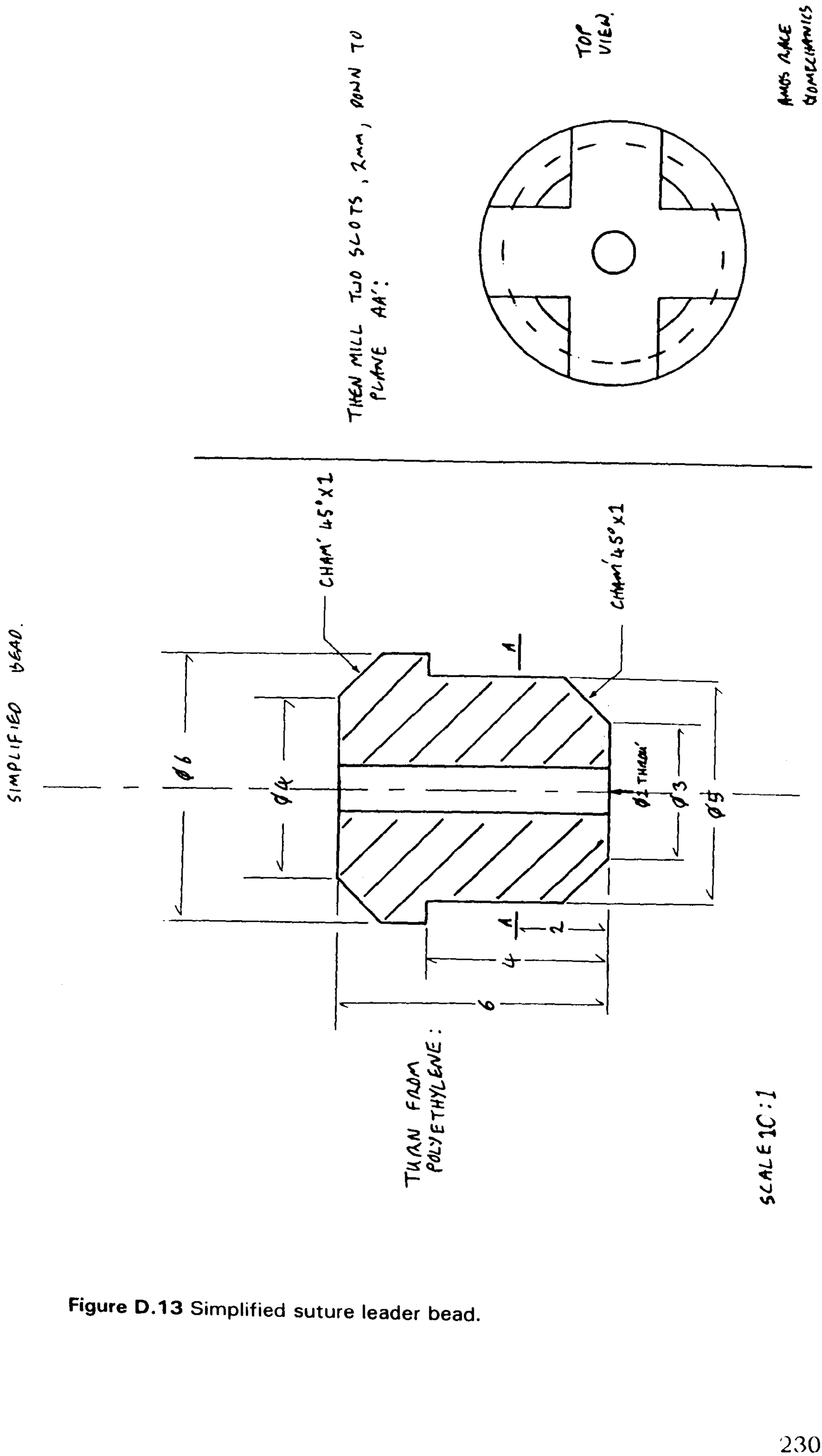


Figure D.13 Simplified suture leader bead.

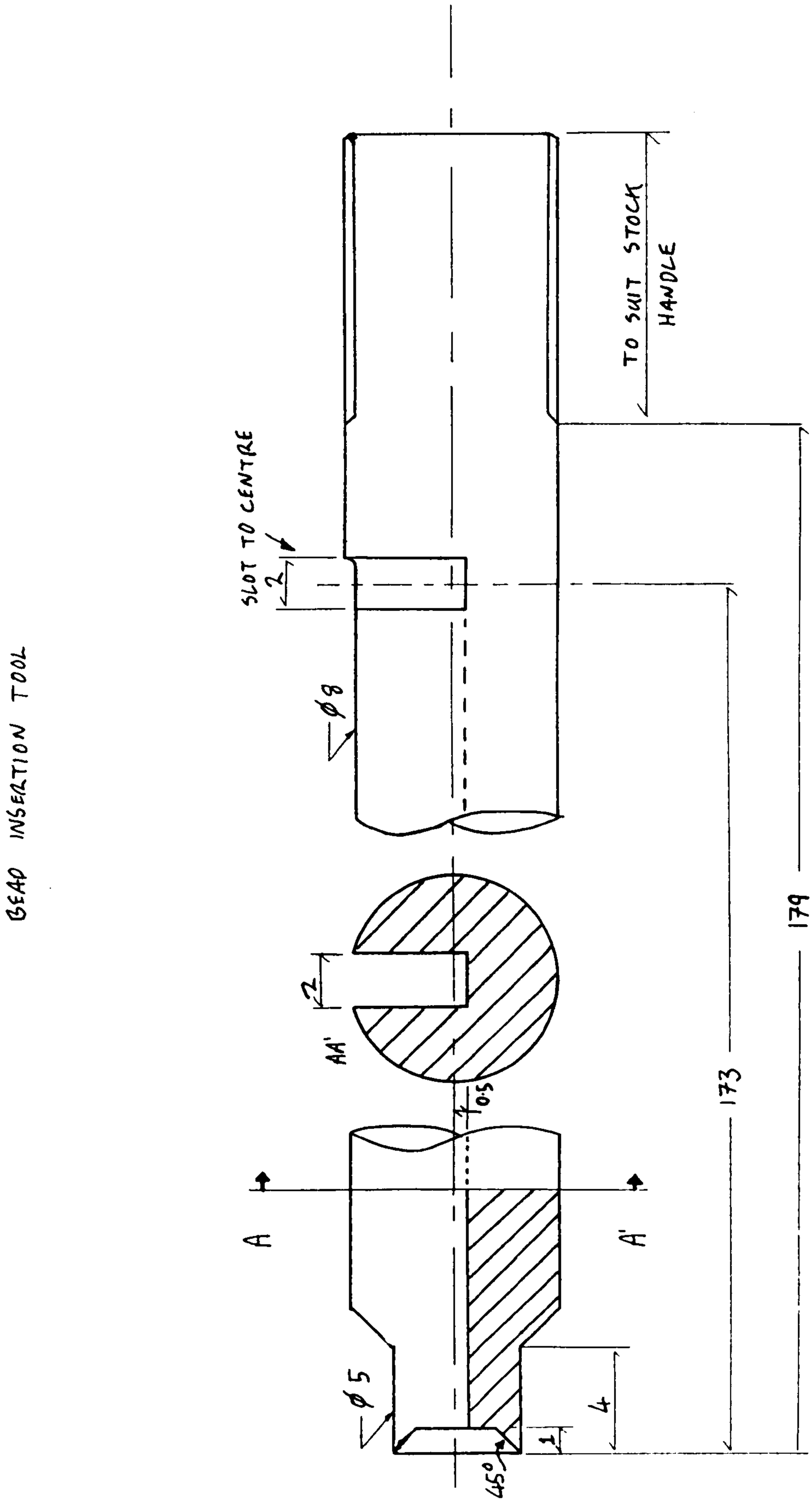


Figure D.14 Suture leader passing tool/reamer.

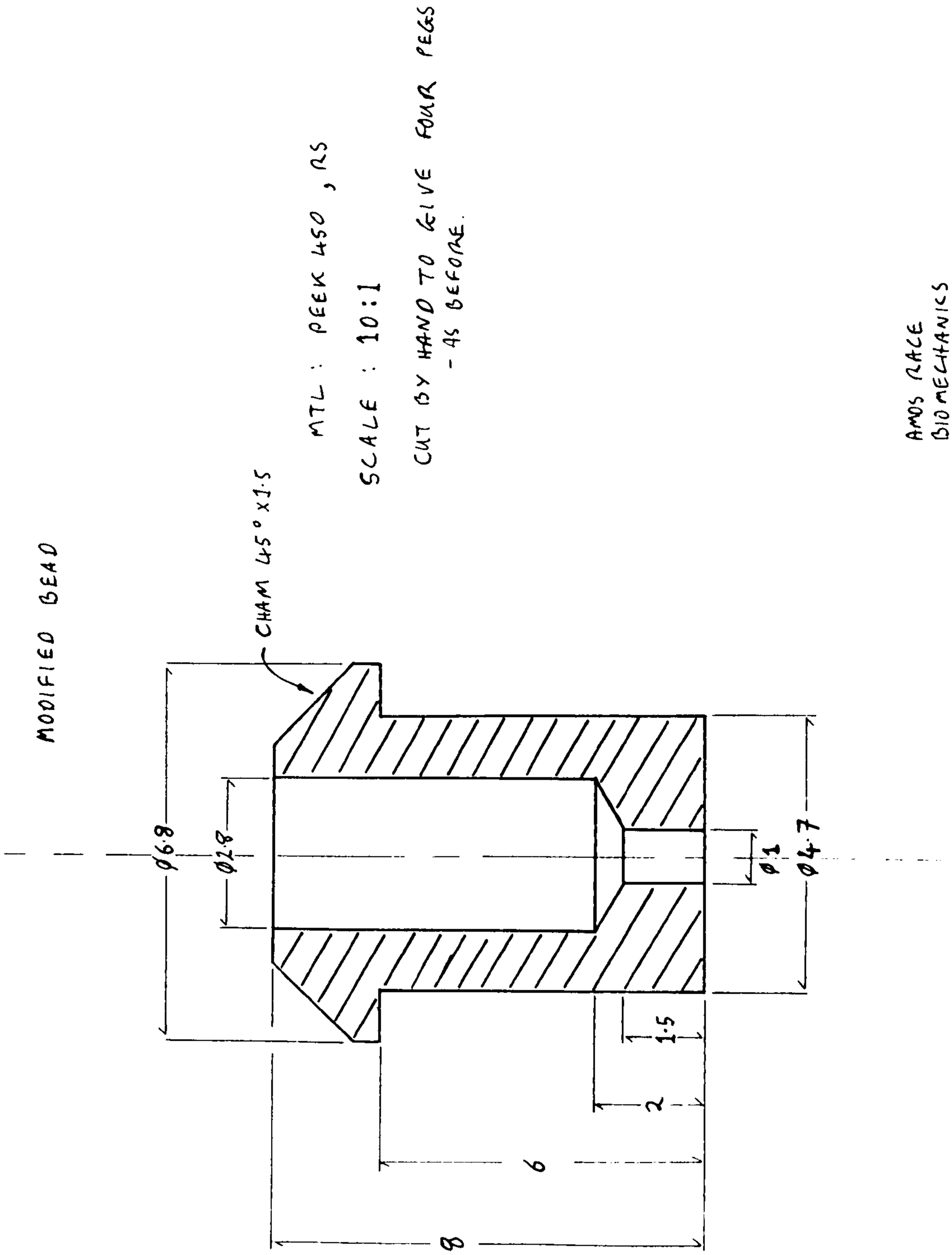


Figure D.15 Modified suture leader bead

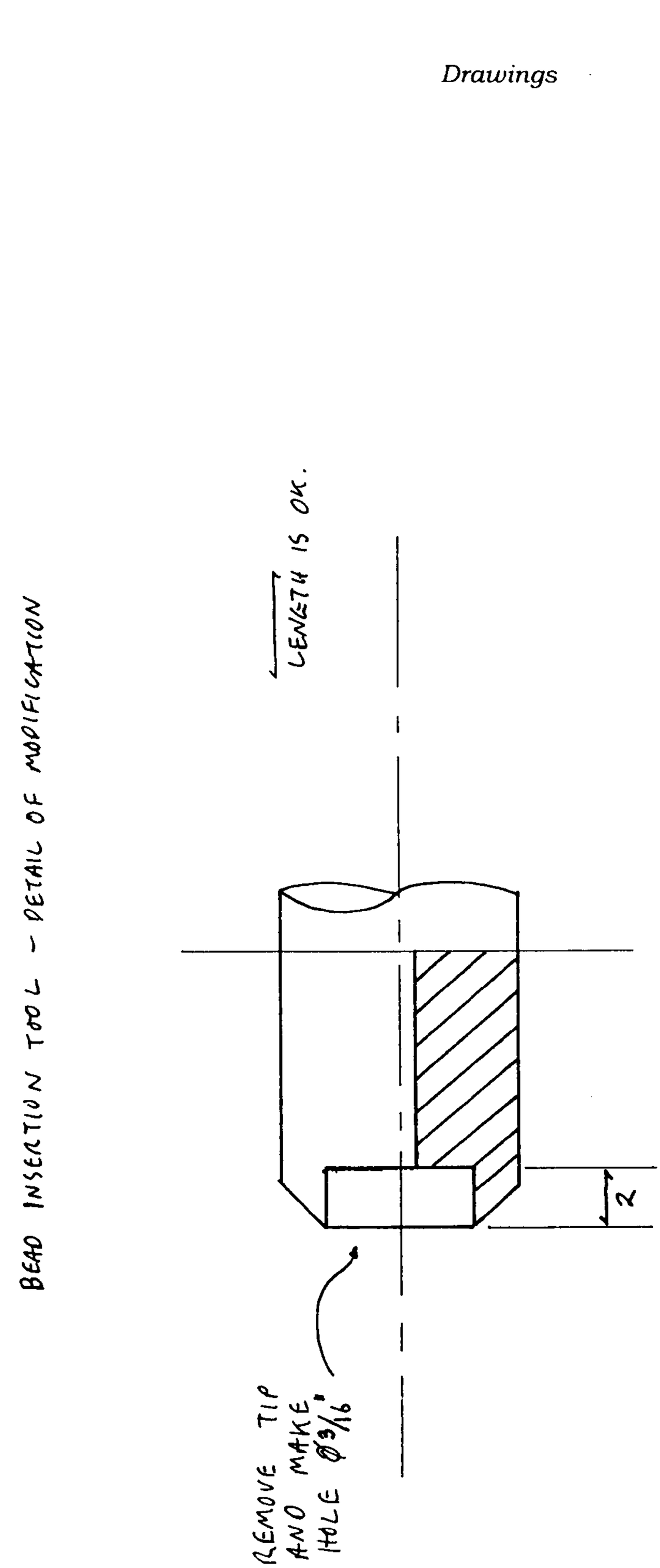


Figure D.16 Modification of the tip of the suture leader passing tool/reamer

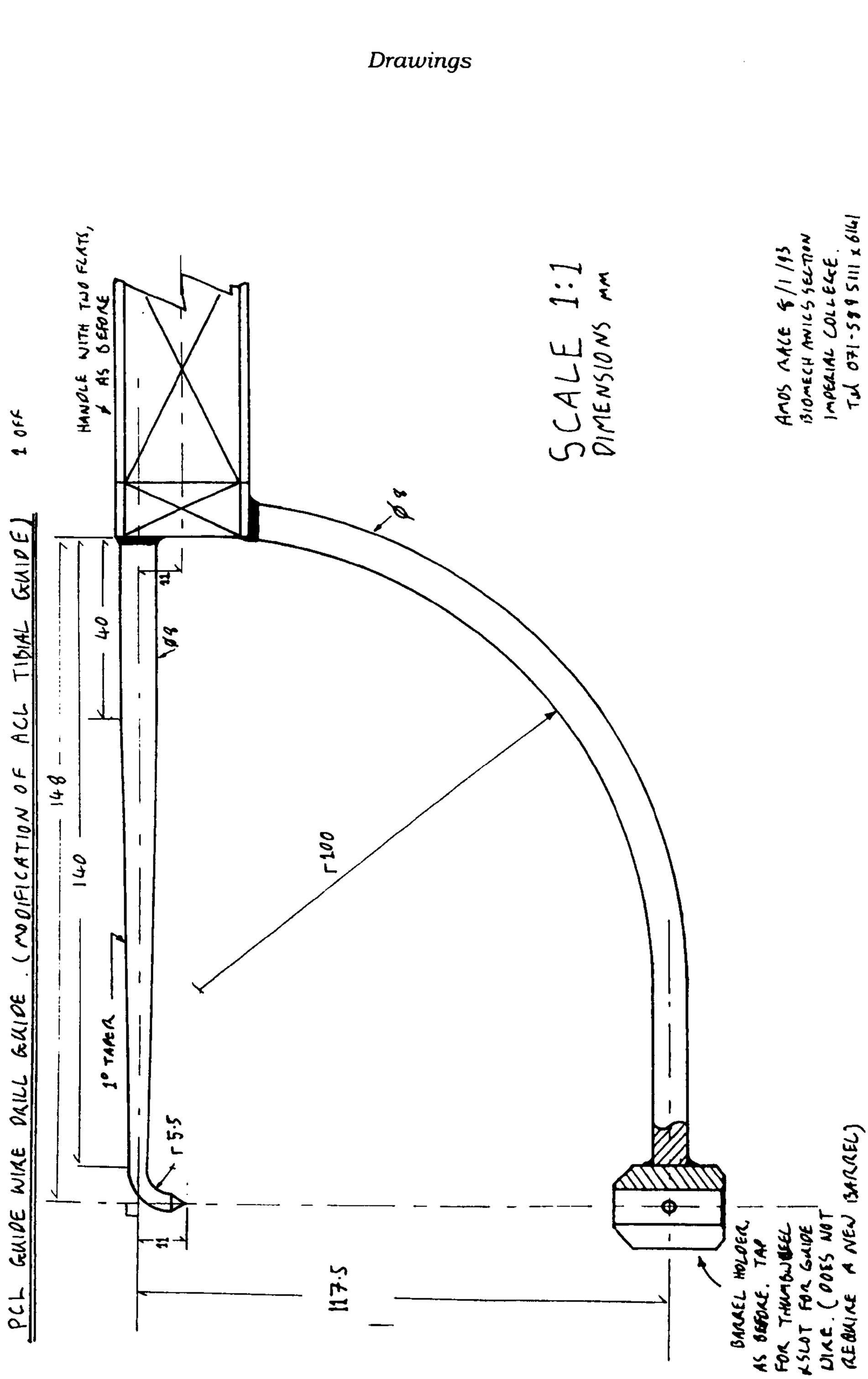


Figure D.17 Femoral drill guide.

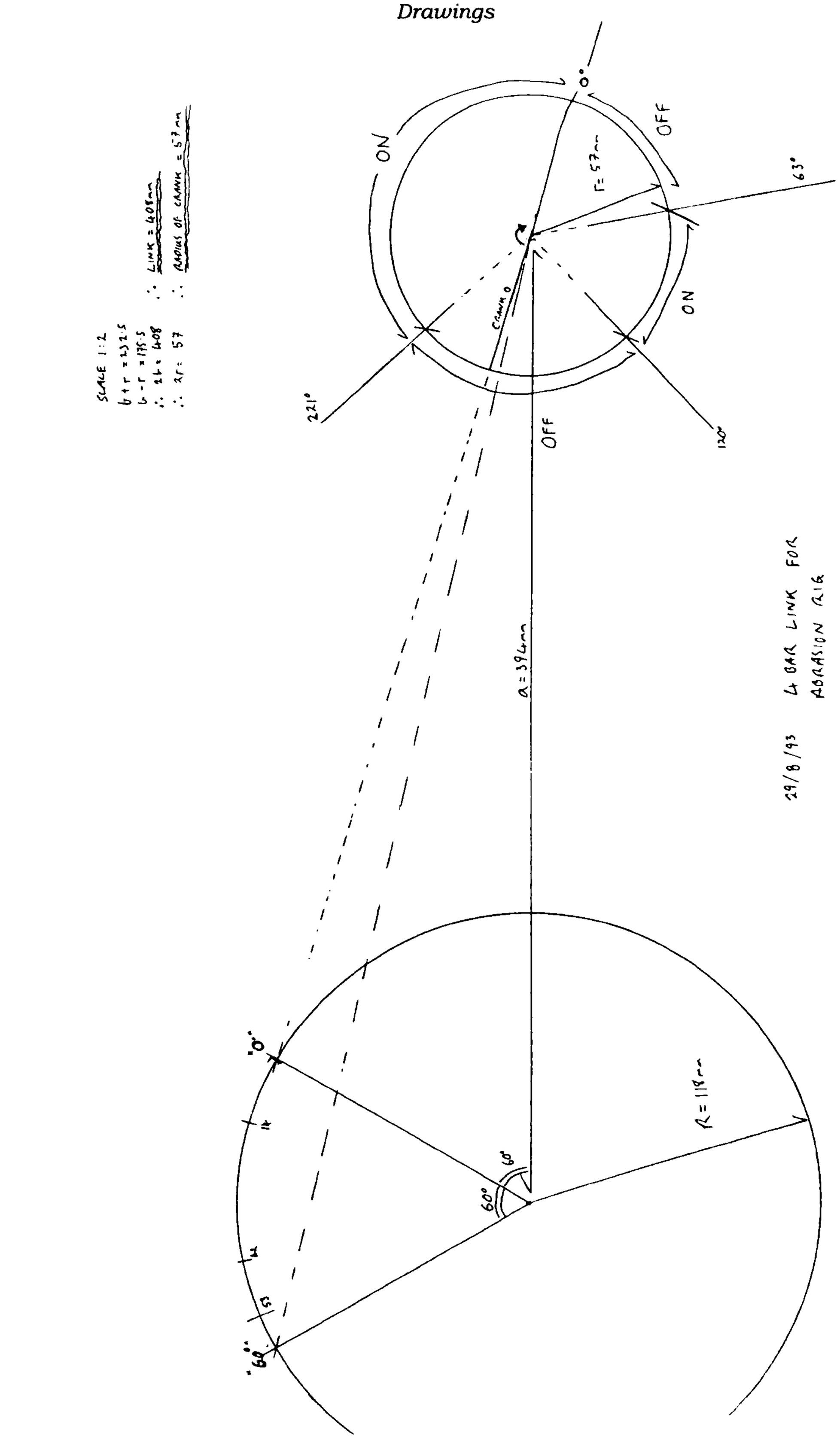


Figure D.18 Graphical solution of the four bar link for the abrasion test rig.

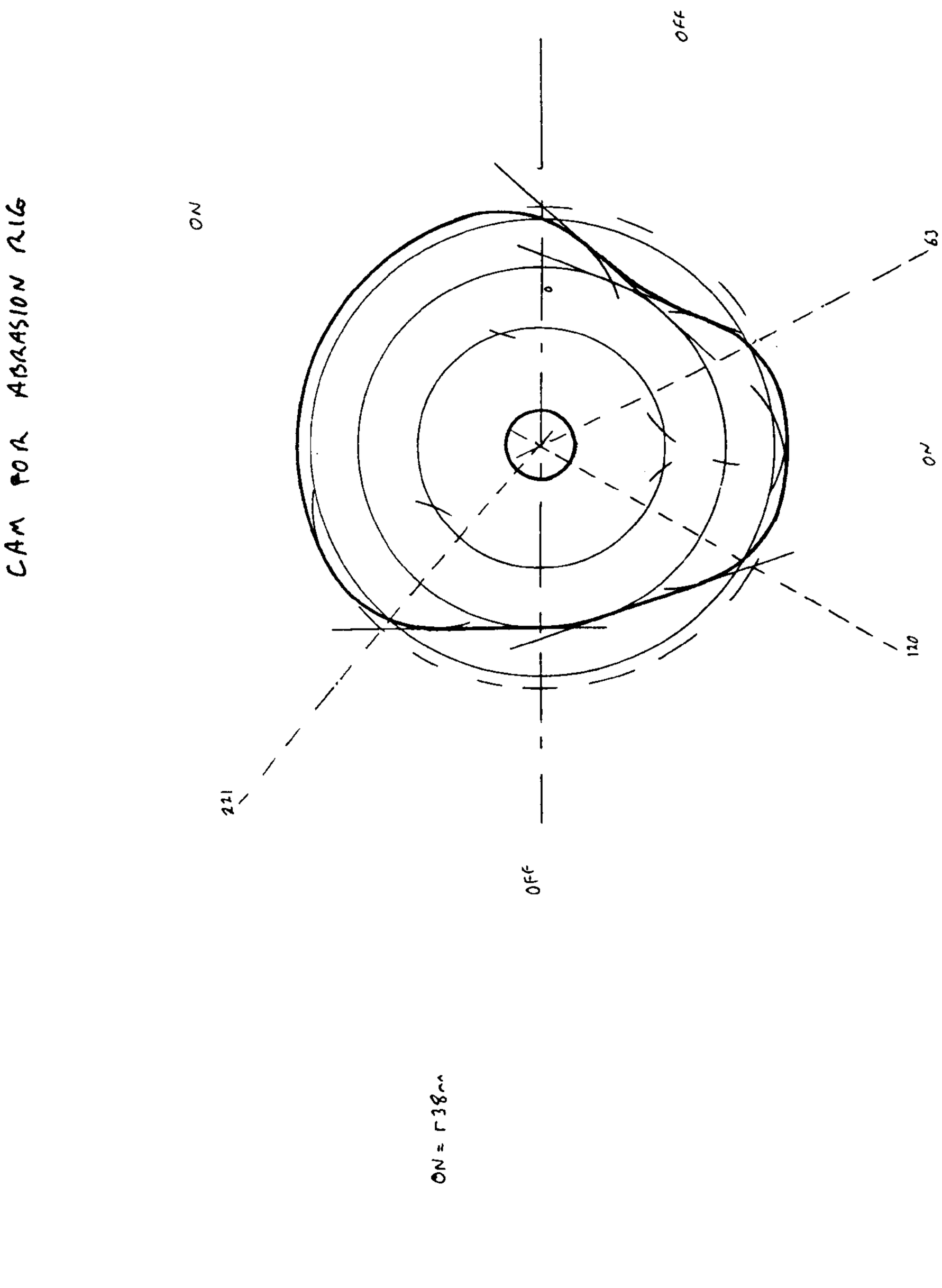


Figure D.19 Cam design for abrasion test rig.

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