CHRONIC MALFUNCTION OF THE PATELLOFEMORAL JOINT: THE
CAUSAL ROLE OF ABNORMALITIES OF QUADRICEPS
ACTION AND POOR TRACKING

by

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A thesis submitted in partial fulfilment of the
requirements for the Degree of Doctor of Philosophy
in the University of London

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ABSTRACT

Malalignment of the patella has been implicated in patellofemoral problems, but the aetiology of the condition is not well understood. Efforts have been made in this thesis to study the underlying abnormalities, both biomechanical and of muscle function, which might contribute to this syndrome. The function of 72 patients with chronic anterior knee pain, where the onset has been insidious rather than a direct response to trauma, has been studied and compared with groups of normal subjects. Many of the experiments undertaken relate to the muscle activity, in the portions of the quadriceps thought to affect the position of the patella. The changes which have been seen, have then been correlated with other abnormalities, in order to understand their place in the pathology of this condition. Other experiments were done only with groups of normal subjects, and relate to the normal functioning of the quadriceps, in different conditions.

Particular areas of study have been:-

1. Signs and symptoms have been recorded in the patient group, and correlated with other abnormalities seen. The intensity of pain and extent of disability have been recorded by means of a questionnaire given to each patient.

2. The quadriceps strength of both lower limbs has been assessed by means of a maximal voluntary extensor effort, isometrically performed, and compared with the predicted strength based on the height of the individual.

3. EMG activity of the oblique fibres of vastus medialis and vastus lateralis as well as rectus femoris, was compared with extensor force at different knee angles, isometrically, isokinetically, and dynamically. Evidence of reflex inhibition due to pain and other factors has been sought. Effort has been made to understand the changes in
muscle activation of the patient group, by studying the quadriceps activation of a
group of normal subjects in different knee positions and at different velocities.

4. The biomechanics of the whole of the lower limb has been studied at rest, by
surface markings and range of movement of the various joints. The effect of this on
the gait pattern has been studied by video recording of the patient group, as well as
normal subjects.

5. A study of the position of the patella by computerised tomography, with respect
to its articulation with the lower end of the femur, in a small group of patients and also
with normal subjects.

6. A small group of patients was tested before and after 6 weeks of conservative
treatment, in order to ascertain which parameters were capable of change. From the
questionnaire, the changes noted were correlated with the extent of disability and
intensity of pain.

7. A study of the normal anatomy of the joint in one cadaver has been made, so that
the effects of the observed biomechanical factors could be more fully understood.

Not only was there a considerable loss of quadriceps strength in the patient group, but
the EMG studies showed abnormalities of quadriceps activation, which were
particularly evident in the oblique fibres of the vasti. There were also differences in the
alignment of the long lower limb bones in these individuals, which were not apparent in
the normal subjects. Correlation among different results did not show direct
correlation of one pathological finding with another. They suggest that this syndrome
is multifactorial. The studies on normal subjects show that in some conditions,
changes in rotation of the hip and knee do alter the way in which the vasti are activated.
ACKNOWLEDGEMENTS

I would like to express my sincere thanks to the following people:-
my supervisor, Professor Roger Woledge, for all his help, patience and advice. His
exceptional breadth of knowledge, both scientific and mathematical, has been
invaluable. I would also like to thank other members of the staff of the Institute of
Human Performance, particularly Andrew Pinder, Suzanne Phillips, and Deirdre Birtles
and Tony Christopher.
I also record my thanks to my previous supervisor Professor David Jones for his help
in setting up and starting the project, also Paul Sacco, whose help was invaluable in
mastering the equipment, and to Dave McKintyre for his computer expertise,
to Geoffrey Cusick, who heads the Medical Instrumentation Services of the Middlesex
Hospital, for designing equipment,
to the doctors who sent me patients, and the physiotherapy staff of the Middlesex
Hospital and Royal National Orthopaedic Hospital,
to the Sir Jules Thorn Charitable Trust who financed the project,
to all the patients and all the normal subjects who gave up so much time for the
project,
and lastly, but certainly not least, to my husband, John, for his love and support.
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CHAPTER I

INTRODUCTION AND LITERATURE REVIEW

1.1. General View of Patellofemoral Disorders

The disorders of the patellofemoral joint which have been included in this study are characterised by retropatellar, or in some cases peripatellar pain which is brought on by climbing or descending stairs and athletic pursuits which involve taking weight on the bent knee, and frequently also by prolonged sitting. Symptoms may last for some hours after being exacerbated, and are specific enough for this condition to be regarded as a syndrome. The onset of patellofemoral symptoms is sometimes insidious, and sometimes an acute response to trauma; however in many cases, the existence of underlying abnormalities make the patient prone to trauma. (When choosing a sample of patients for this study, a criterion for inclusion was that the onset was insidious.) The label “Anterior knee pain” was thought by many authorities not to be specific enough and to describe insufficiently the underlying factors. These disorders used to be described as chondromalacia patellae, but arthroscopy studies found that only about 50% of these patients had cartilage changes. Bentley and Dowd (1984) reported 51%. Many patients who were operated upon for other reasons were found to have quite severe cartilage abnormalities and yet were not complaining of anterior knee pain, so this terminology fell into disrepute, except when it was a description of the state of the cartilage. The anatomy and function of the patellofemoral joint has been extensively studied for well
over half a century, using a large variety of techniques, in order to increase understanding of its dysfunction. Although much progress has been made, the aetiology of the disorders of this joint is still not well understood and, due to lack of understanding the treatments are not satisfactory and patients experience prolonged problems.

Knee disorders are very common, and various estimates of the percentages of these patients whose problems are associated with the patellofemoral joint have been made over the years. For example, Dehaven and Lintner (1986), in their study found that in 18% of the male patients, and in 33% of the females, the problem was associated with the patellofemoral joint. Another survey showed that 25% of athletes had patellofemoral joint dysfunction at some time in their athletic career. Dysfunction is also prevalent in other groups of adolescents and young adults, and may affect both those who are athletically inclined, and those who are not so physically active. Aleman (1928) reported chondromalacia in 33% of arthrotomies performed on young soldiers. The reported proportion of female to male patients with these problems has been rather variable (3:2 Goodfellow et al. 1976, 2:3 Dehaven et al. 1979). The condition is sometimes complicated by reflex sympathetic dystrophy, which is an exaggerated sympathetic response to injury or operation. However, patients with this abnormality find that everything that they do is painful and not just specific activities and movements. They also show an exaggerated response to temperature and touch.
Medical treatment of disorders of this joint is either operative or conservative, but differential diagnosis is not easy and frequently, in the past they were mistaken for meniscal or ligamentous disorders, or bursitis, or loose bodies. Suprapatellar, or medial inflamed or scarred plicae have also escaped detection. Operative techniques have been developed to (a) realign the patella proximally (b) effect distal realignment, (c) release tight lateral structures or (d) shave the articular cartilage which lines the patellar surface. If the new tracking made is accurate then the prognosis should be good. Mankin (1971) considered that healing of cartilage was poor in superficial cartilage, but considerably improved in deeper layers, where chondrification takes place. Goodfellow et al. (1976) considered that true chondromalacia takes place in the deeper layer. They made the distinction between this and surface "flakiness", which they regarded as a separate entity. However, the effectiveness of some of these operative measures has been questioned. With lateral release, probably the most frequently performed of the operative measures, initial good success rates have been demonstrated, but in one study (Osborne and Fulford 1982), it was found that after 3 years these good outcomes had declined to 37%. Shellock et al. (1990) in their MRI study showed that in a proportion of cases medial subluxation can take place after lateral release. Terry (1984) also thinks that this procedure adversely affects lateral stability.

The operative procedures summarised as a) and b) were introduced to alter the force vectors on the patella.

a) Insall et al. (1979) instituted a procedure whereby vastus lateralis was moved medially and the medial and lateral quadriceps expansions joined. This procedure
was allied to a lateral release. Hughston and Walsh (1979) effected a proximal realignment by a vastus medialis advancement. They reported that 76% of the long term results were excellent or good. Medial reefing or plication is also performed. b) The first person who performed distal realignment for recurrent dislocation of the patella was Roux (1888), who moved the distal end of the patellar tendon medially. Hauser (1938) and Trillat et al. (1964) used similar techniques for the same condition. In the Goldthwaite-Roux procedure, described by Aichroth (1979), there is an extensive lateral release, medial transfer of the lateral two-thirds of the patellar tendon, and medial reefing. Chrisman et al. (1979) reported a 72% success rate for the Hauser technique and a 93% success rate for the Goldthwaite-Roux. The Hauser medialisation has also been used to lower the patellofemoral contact force or patellofemoral joint reaction force (P.F.J.R.F.), based on Ficat’s theory of lateral hyperpression (1970). Maquet (1979) and Bandi (1976) advocated ventralisation to lower the P.F.J.R.F. and calculated a reduction of 20%. Ferguson et al. (1979) reported good results if the tibial tubercle was not raised more than 2 cm. Fulkerson (1983) advocated anteromedialization for a lowering of the pressure and realignment. However, Hehne (1990), simulated both ventralisation (by 1 cm) and medialisation on cadavers and measured the interarticular pressures. His findings showed no change in the pressure on ventralisation, and an increase of 25% in the average pressure both laterally and medially, on medialisation. This, he considered, detracted from any benefit these techniques might have, when used for the purpose of relief of hyperpression.
Conservative measures in the past have concentrated on improving the function of the quadriceps. However, this approach has been supplemented by a series of techniques which have been developed mainly by McConnell (1986); these measures have aimed at stretching tight structures and improving the tracking of the patella. Non-steroidal anti-inflammatory agents have also been extensively used. It has been claimed that the success rate of some conservative measures is 75% (Dehaven and Lintner, 1986). However, Devereaux and Lachman (1984) reported that 70% of 137 conservatively treated patients were still symptomatic a year after treatment. The patients had received different conservative treatments: 100% had undergone quadriceps strengthening exercises, 23% faradic stimulation to vastus medialis, 41% non-steroidal anti-inflammatory drugs; and other pain relieving measures had also been taken. A high proportion of the patients in this study had received previous conservative treatment, and were still symptomatic, but the variety of chosen modalities was bewilderingly wide.

Extensive studies have been made to increase understanding of the anatomy, the way in which the patella articulates with the femur at different knee angles, and the biomechanical forces to which the joint is subjected. The action of the quadriceps is linked to these factors, and its control of the patella is the area which is least well understood. Much of this study is aimed at increasing this understanding, and at discovering in what ways the quadriceps muscle function (and its relationship to the action of the hamstrings) of the patients differs from that of normal subjects.
1.2. Anatomy and Normal Function of the Patellofemoral Joint

The patella is the largest sesamoid bone in the body; its size is very variable. Wiberg (1941) described four different shapes of the patella, and thought that there was a preponderance of certain shapes in chondromalacia patellae, but Hehne (1990) did not consider that differences in shape had any bearing on pathology. He postulated that the development of shape was dependent on the individual way in which the patella articulates with the femur. The posterior surface of the patella is divided longitudinally by 2 ridges into 3 facets, the largest of which is the lateral facet. Some authors have also gone on to subdivide the patellar transversally. The facets articulate with the trochlear surface of the femur. This surface takes the form of a shallow groove which is deeper distally. When the knee is fully extended, the only part of the patella to make contact with the femur is a small extension facet at the lower end of the articular cartilage, the proximal part of the patella being in contact with a sub-synovial pad of fat (Wiberg 1941, Hungerford and Barry 1979). As the knee is progressively flexed, the patella moves downwards, into this shallow groove, or trochlea, at about 20° of knee flexion (the exact angle is dependent on the level of the patella in the extended position). After 90° of flexion it makes contact with the femoral condyles (articular surface which forms part of the femoro-tibial articulation in the extended position, made possible by displacement, on flexion, of the point of contact between the femoral and tibial condyles). This has been demonstrated by Goodfellow et al. (1976). The areas of the patella then in contact are the odd facet (in the Wiberg Type II patella, described by Wiberg in
1941), the most medial of the three facets, and a somewhat larger area of the lateral facet.

Fig. 1.1 Right Patella (posterior aspect)

a) at 60° of knee flexion  

b) at 120° of knee flexion

Fig. 1.2. Cross section of patella and femur to show articulation

As the patella makes its downward path during flexion, the band of articulation changes and the area increases (Goodfellow et al. 1976). Both the change in articulation and the increasing area of the articulating surface are important to the correct functioning of the joint. The former enables the joint to be lubricated. In the adult joint the synovial fluid diffuses through the different layers of cartilage. This is important for adequate nutrition (James 1982), since the cartilage of the patella is the thickest in the body in order for it to withstand the large forces to which it is
subjected (Bandi 1972). As the leading edge of the articulating surface passes downwards, the compression causes the fluid to be squeezed out on to the surface. The increasingly large area of contact is one of the mechanisms by which the potentially high patellofemoral contact force is offset on flexion so that the pressure per unit area remains approximately the same, irrespective of angle (Hehne 1990).

Fig. 1.3. Schematic representation of patellofemoral contact (from Muller, 1983)

Fig. 1.4. Contact Areas of the Patella at Different Angles of Flexion
1 is at 20°, 2 is at 45°, 3 is at 90°, 4 is at 135°
(after Goodfellow et al. 1976)

Although the patella is mobile at rest with the knee extended, the extent of passive movement possible, both laterally and medially, is generally less than half the total width of the patella (Carson et al. 1984). Mobility greater than this is regarded as hypermobility and is usually associated with symptoms. Decreased
mobility may be associated with patellar tilt and areas of abnormally high pressure (Ficat et al. 1977). In its downward path as the knee is bent the patella should follow a smooth midline trajectory. This is achieved by a variety of factors. At certain knee angles the shape of the bone more easily controls this, but at other angles the poor congruity probably has to be compensated for by soft tissue restraints, since there are no obvious bony ones. These may be static or dynamic. When the knee approaches full extension the soft tissue restraints are thought to be very important, as they are also after 90° of flexion when the patella has left the trochlear groove and is moving between the condyles. Once the patella has articulated with the trochlea, the lateral condyle which is more prominent than the medial condyle, helps to prevent it from dislocating laterally.

The articular cartilage also extends more proximally on the lateral side. In some cases the lateral condyle does not develop properly and the trochlea is abnormally shallow, (that is, the sulcus angle is very large on X-Ray), as described by Brattstrom (1964) and Merchant et al. (1974). It is considered pathological if the trochlear sulcus angle is greater than 145° (Ficat and Hungerford 1977). This is an important cause of lateral patellar dislocation. Indices of trochlear depth can also be used. Dejour et al. (1994) demonstrated that a high percentage of patients with patellar instability had trochlear dysplasia (85%). This was characterised by a trochlear bump of 3mm or more (66%), and a trochlear depth of 4mm or less. They also listed other factors, which contribute to patellar instability, viz laterality of the tibial tuberosity compared with the trochlear groove, and patella alta which is discussed in 1.3.
At angles approaching 90° of knee flexion the knee joint allows considerable rotation of the tibia. The extent of possible internal rotation is 30°, and that of external rotation is 40° (Kapandji, 1971). As Muller (1983) points out there are many sports, which involve considerable rotation both at the hip and knee, for example football or hurdling. Were the position of the patella not to be readjusted to compensate for this rotation the articulation would clearly be insufficient for the patellofemoral joint contact pressure to be applied in the normal manner. For example, in changes in tibial rotation, it would appear that the different angulation of the patellar tendon causes a corresponding adaptation in quadriceps action (Muller 1983, Hehne 1990). External rotation of the tibia rotates the patella laterally in a frontal or coronal plane (see the Biomechanics section below). While the depth of bony restraints provide a degree of stability, these movements must to be compensated by changes in muscle action. Since many sportsmen perform these movements over long periods without problems developing, this compensatory mechanism is normally effective, but how it is achieved is not understood. The area of articulation has been shown to be disrupted in simulated deformities of the tibia using cadavers, for example external tibial torsion (Lee et al. 1994) and also by tibial varus (Fujikawa et al. 1983).

Terry (1989) has described the layered soft tissue orientation which provides the joint with stability. Most superficial is the arciform layer which covers the retinacula. The intermediate layer is formed by the quadriceps muscles proximally, some portions of which insert into the quadriceps tendon, which is attached to the base of the patella, while other fibres continue over the patella, joining the patellar tendon which inserts into the tibial tubercle. The retinacula
are fascial expansions of the tendons of the vasti. The longer, stronger fibres spread downwards and ventrally, parallel to the patella, to be inserted on the ventral surface of the tibia. There are oblique condensations of the retinacula, which are sometimes referred to as medial and lateral patellotibial ligaments (Hughston et al. 1984). The retinacula are contiguous with the joint capsule and the iliotibial tract blends with the lateral retinaculum. Soft tissue restraints in the deep layer are the medial and lateral patellofemoral ligaments which appear to be transverse condensations of the retinacula; they are frequently thin because their dynamic counterparts (the vasti) are more important to stability (Terry 1989).

The retinacula are considered very important in the pathogenesis of patellofemoral joint problems and any loss of integrity, particularly on the medial side, whether it is caused by trauma or iatrogenically, is a frequently observed cause of problems. Although the retinacula are generally regarded as static restraints they can be dynamised by action of the vasti. Their strongest fibres are the ones inserted ventrally on to the tibia, and according to Hehne (1990), these are the ones that are dynamised by the vasti during a greater range of movement, because of the favourable direction of these fibres; he regards them as tendons of the vasti. The transverse fibres of the retinacula are tightened on full flexion, both passively as they transverse the protruding condyles, and actively at that angle by the vasti, because the application of force becomes favourable.

The iliotibial tract or band (ITB) also has a direct attachment to the patella (iliopatellar band) and there is also a menisco-patellar restraint. There has been
considerable controversy over the function of the ITB. It used to be thought that its main function was to decrease the bending stress on the femur (Pauwels 1948), however, more recently doubt has been cast on the assumptions of the early work (Takahashi and Endo 1982). It would appear that its function is more complex than was hitherto thought, and may have more to do with knee joint stability.

Anterolateral stability and limitation of anterior displacement of the tibia is in part provided by the anterior cruciate ligament (ACL) but also the ITB tract (Hassler and Jakob 1981, Terry 1986, and Terry et al. 1993). Hassler and Jakob showed that the pivot shift sign was only positive when the ITB was intact, and reduced or absent when it was transected. Because of this finding they made a detailed anatomical and physiological study of the structure of the ITB. The reason for the pivot shift being masked on severence of the ITB was elucidated by Matsuo (1990); on further flexion the knee remains internally rotated, because the ITB is responsible for the relocation of the subluxed tibia. It is likely that the iliopatellar band prevents medial hypermobility of the patella but probably has an indirect effect via the lateral retinaculum.

1.3 Movements of the Patellofemoral Joint

Extensive studies have been undertaken in order to understand the normal movement of the patella and the way in which the movement differs in joint pathology. Some of these studies have been invasive and have centred around markers actually being placed in the patella. Some of these have been static imaging studies, either X-Ray, computerised tomography (CT) or magnetic
resonance imaging (MRI), measuring the congruence angle, lateral
patellofemoral angle, and the patellar tilt angle.

The congruence angle (Merchant, 1974) is ascertained by bisecting the sulcus
angle (BAC) by a line (AO); another line is drawn through the deepest part of the
trochlea groove and the vertical ridge of the patella, (AD). The angle between
the two is then measured (see Fig. 1.5.1). Positive values show a lateral shift of
the patella.

The Laurin (1978) lateral patellofemoral angle, taken with the knee flexed to
only 20°, is found by drawing one line from the medial to lateral condyle, (AA')
and another from the ridge to the lateral border; (BB'), it is extended if
necessary. A very acute angle, or even an angle pointing in the other direction
highlights a tilted patella (Fig. 1.5.2). Another method of measuring tilt is by a
measuring the angle made by a line drawn through the lowest point of the
posterior aspects of the condyles (AA'), and another joining this along the lateral
facet (AB) (Schutzer with CT, 1986). This is termed the patellar tilt angle
(Fig. 1.5.3.).

X-Ray imaging of these, although cheaper, is currently not as popular as CT or
MRI because of the difficulty of positioning between the angles of 0-40° of knee
flexion. It is considered by most of the authors that it is important to visualise
the position between these angles. Another factor is that one cannot measure
the patellar tilt angle without CT.
There are constraints associated with both the invasive and non-invasive methods of investigation of the movement of the patella during knee flexion, and there is not complete agreement over the direction of this movement. The constraints of the former method are self evident, due to the invasiveness of the methods. The constraints of the latter are because two dimensional images (2-D) cannot effectively reflect three dimensional (3-D) events. However, techniques for the improvement of 3-D visualisation are being developed. Another difficulty is because the positioning is frequently recumbent, and cannot
reflect what happens when the individual is upright and subject to the pull of muscles. Another constraint is the difficulty of allowing for anatomical differences, which may or may not be pathological (Shellock et al. 1990). What is abundantly clear is that there are differences in patellar tracking between normal subjects and patients suffering from patellofemoral dysfunction.

Schutzer et al. (1986) found three patterns of malalignment: lateral subluxation without tilt, tilt without subluxation and subluxation with tilt. What causes abnormal tracking and its relationship to patellofemoral disorders has not been elucidated.

There is a relationship between a high riding patella (patella alta) and patellofemoral problems (about 50% of patients with the latter have a high riding patella). It is frequently idiopathic in origin. Caton et al. (1990) thought that the cause might be short quadriceps or perhaps genu recurvatum. Micheli et al. (1986) studied 19 children during an adolescent growth spurt to see how many developed this abnormality, but only two did. Their conclusion was that rapid growth can cause patella alta, but it cannot be the only factor. The position of the patella can be seen by examination of a lateral X-Ray (Insall et al. 1976). The ratio of the patellar tendon compared with the height of the patella, should not exceed 1.2:1. Patella alta is also suspected from an antero-posterior X-Ray with the knee at 0° of knee flexion, if the tip of the patella is more than 20 mm from the lowest point of the femoral condyles (Brattstrom 1970), but this relationship only holds good with the knee fully extended. The advantage of the former technique is that the relationship holds good irrespective of the knee.
flexion angle at which the X-ray is taken. A disadvantage is that the outline of the tibial tubercle does not always show up on X-Ray which makes the estimation difficult. This difficulty is overcome by the estimation of patellar height advocated by Blackburne and Peel (1977). In this method the perpendicular height of the articular surface of the patellar is compared with the perpendicular height of the lower end of this from the tibial plateau. They found that the former, in normal subjects, was approximately 0.8 of the latter. Another method which has been used is the Blumensaat line, taken at 30° (Blumensaat 1938).

One instance of the lack of agreement of the movement of the patella mentioned above, is the tracking of the patella from 0-30° of knee flexion. In the non-invasive studies, the authors usually report a medial shift and a slight medial tilt, and in the invasive studies a lateral shift and tilt. Schutzer et al. (1986), who performed CT studies, reported the tendency for a medial shift to occur, and Kujala et al. (1989), in an MRI study found a medial shift and medial tilt. Taking the majority of findings as the norm, it appears that not only does the patella track downwards, but it also follows the path of a shallow U in a frontal plane, going from lateral to medial from 0-50° of knee flexion, and then lateral again from 90° to full flexion. Little is known about the movements which occur at angles greater than 90°, it is believed by the author that it has only been studied in cadavers by Hehne (1990). He reports that from 90-120° there is a lateral shift of the patella and a medial tilt, in order for it to be accommodated between the condyles.
In summary, we are accustomed to think of the patella moving downwards with respect to the femur, but it would appear that it rotates about a vertical axis (y axis), i.e. "patellar tilt", moves medially and laterally in a coronal plane (patellar shift), and rotates about a sagittal axis (z axis). The last mentioned also occurs in a coronal or frontal plane. As well as these movements, it also in its downward path, rotates through 90° as the knee flexes, moving more posteriorly due to "roll back" movement of the femur on the tibia. This rotation occurs about an axis (x axis) which is perpendicular to the sagittal plane (Van Kampen and Huiskes 1990). Van Kampen and Huiskes (1990) studied the three dimensional tracking pattern of the patella by the reconstruction of the co-ordinates of markers implanted into the bones of cadavers, throughout knee flexion and during tibial rotation. They concluded that these patterns of movement were highly influenced by tibial rotation, which caused greater tilt and shift in the first part of knee flexion and patellar rotation (in a frontal or coronal plane) on greater flexion.
Frontal or coronal plane

Rotation around x axis

Rotation around y axis

Rotation around z axis

Fig 1.6. Movements of the patella in three dimensions which occur with tibio-femoral movements. (x axis, horizontal, y axis, vertical, z axis sagittal,) (after Van Kampen and Huiskes, 1990).

1.4. Muscles Acting on the Patella

The patellar movements which have just been described must in part be controlled by muscles, but there are large gaps in existing knowledge and understanding of how this is achieved. The largest contribution must be made by the quadriceps, since they are inserted directly on to the patella. These can be divided into four parts which are anatomically distinct. Vastus intermedius (V.I.) makes the largest contribution to extensor moment (Narici et al. 1992). This portion of the quadriceps is parallel to the plane of the anterior surface of the femur; the others have a more lateral or medial orientation (Lieb and Perry 1968). The aponeurosis of V.I. forms the deep
layer of the quadriceps tendon. On top of this muscle lies rectus femoris (R.F.), which is the only bi-articular muscle of the quadriceps group, acting on the hip as well as being a knee extensor. It is a secondary hip flexor working with psoas major, and when the thigh is fixed helps to flex the trunk. It can also flex the hip and extend the knee simultaneously. The insertion of R.F. forms the superficial layer of the quadriceps tendon, and most of these fibres do not insert into the base of the patella but pass over the patella and form the superficial layer of the patellar tendon (Javadpour et al. 1991).

Each of the two vasti has been seen to be comprised of two parts. The fact that vastus medialis is divided into two parts, with two possible different functions, has been accepted since the key paper of Lieb and Perry (1968). The long fibres (V.M.L), which have their origin mainly from the shaft and intertrochanteric line of the femur, are inserted into the middle lamina of the quadriceps tendon, interdigitating with fibres from vastus lateralis (Javadpour et al. 1991). The oblique fibres (V.M.O.) arise mainly from the tendon of adductor magnus (Bose et al. 1980), and are inserted into the medial border of the patella. The lowest of these fibres are almost horizontal and the higher ones at an angle of 55° to the shaft of the femur. Many authors have remarked on the presence of a fascial plane between the two parts of the muscle. Javadpour et al. (1991) found in all cases that this contained a branch of the femoral nerve, however both Terry (1989) and Lieb and Perry (1968) reported that in some cases there was a separate nerve to the belly of V.M.O. Recent work has now elucidated that vastus lateralis is arranged in much the same way (Javadpour et al. 1991). The long fibres of vastus lateralis (V.L.L.) arise from
the shaft of the femur and interdigitate with V.M.L. to form the middle lamina but a few fibres from V.M.L. and V.L.L. pass over the top of the patella to fuse with the retinacula. The posterolateral fibres of V.L. arise from the deep layer of the iliotibial band and are inserted into the supero-lateral and lateral margins of the patella. The angle of these fibres are 30-40° to the shaft of the femur and can be called V.L.O. (Javadpour et al. 1991). In these dissections there was a fascial plane seen between the two sets of fibres, but no nerve was seen.

Abnormalities of the anatomy of the insertion of V.M.O. have been observed by several authors in patients with patellofemoral problems, for instance Fox (1975). He spoke of a dysplasia, in which the fibres are not inserted as far down the medial side of the patella as is usually seen, and remarked upon the presence of a groove between the fibres of V.M.O. and the medial border of the patella. He regarded this abnormality as genetic. The high insertion would impair the function of V.M.O. in counterbalancing the lateral pull of the rest of the quadriceps.

The individual contributions made by the different parts of the quadriceps to knee function have been a source of controversy for decades. It was originally thought that vastus medialis selectively controlled the last 15° of knee extension (Smillie 1949), but later work proved this to be untrue. However, it is generally accepted that the main function of V.M.O. is to pull the patella medially, but how the mechanism is controlled is unknown. Lieb and Perry (1968) suspended weights attached to cables and hooks through the individual cut portions of the quadriceps group in amputated limbs in the direction of their fibre axes, and then in various
combinations. They then saw how much weight was necessary to effect knee extension. In the case of V.M.O. no matter how much was suspended, they could not effect extension, but weights suspended from V.M.O. decreased the weight necessary for V.L. by nearly 20%. They concluded from this that the main function of V.M.O. was to maintain patellar alignment.

There have been many electromyographic studies undertaken on normal subjects to increase understanding of the action of the quadriceps in a variety of positions, including standing and non-weight bearing activities. Some of these compared the activity of the muscles of patients with patellofemoral problems with normal groups. Reynolds et al. (1983) found that there was no difference between the activity in V.M.O. and V.L. in normal subjects in the last 30° in standing. Moller et al. (1987) found no difference between the amplitude of the EMG recording from V.M.O. and V.L. in patients; however, Mariani and Caruso (1979) found that EMG activity was lowered in subluxation of the patella, which then became normal after operation. But it is only recently that it has been appreciated that V.M.O. and V.L.O. might work together to control the positioning of the patella, and in this study recordings have specifically been made from these portions of the muscles.

Articularis genu is sometimes described as a fifth member of the quadriceps, and is important to the extensor mechanism in that it retracts the suprapatellar pouch and a plica of synovial membrane, the suprapatellar plica, and keeps it in position throughout flexion and extension. If the synovial membrane becomes thickened or scarred, the articularis genu cannot keep it in the suprapatellar pouch and the patient complains of variable symptoms, including snapping and pseudo-locking, and pain on
knee movements (Yates and Grana 1990). There is also a medial plica which is deep to VMO and has much the same orientation. This can also impinge during knee movements. The medial plica is likely to impinge after 30° of knee flexion (Matsusue et al. 1993) whereas the suprapatellar plica is likely to impinge after 70° (Strover et al. 1991). Strover and Dvir (1995) demonstrated a loss of quadriceps strength and pain with plicae which was reversed after excision. The variability of the symptoms produced leads to frequent misdiagnosis which was shown on subsequent arthroscopy by Matsusue et al (1993).

The hamstrings are considered clinically to have an important effect on patellar function. Many authors have noted the high incidence of hamstring shortness in patellar dysfunction (for example Henry 1989, and Brunet and Stewart 1989). Various reasons have been put forward for this. It is considered by some authors that short hamstrings resist quadriceps extension, requiring higher quadriceps force and thus causing greater patellofemoral contact force (for example Jacobson and Flandry, 1989). It has also been claimed that they contribute to inflamed plicae (Jacobson and Flandry 1989, and Aprin et al. 1984). If the shortness is confined to the medial hamstrings only, medial rotation of the tibia could occur, since they are inserted medially on the tibia, spreading out to form the pes aserinus. This could cause a change in pull of the patellar tendon and articulation of the patella. Likewise shortness of biceps femoris could cause an external rotation of the tibia. Biceps femoris is also attached to the iliobial band, and so could exert a lateral pull to the patella. Sports which involve a crouching posture or prolonged sitting may encourage hamstring shortness.
The maintenance of quadriceps/hamstring ratio is considered important to good knee function. The force exerted by the hamstrings is about 40% of that exerted by the quadriceps, but in some highly trained individuals it may be as much as 60% (Grace et al. 1984). Read and Bellamy (1990) tested the hamstring/quadriceps ratio in elite athletes with various specialities isokinetically at 90°/s and found it to be between 60-80% (~5%-10% should be deducted for a decrease in velocity of 30°/s).

Stafford and Grana (1984) tested football players isokinetically and found that at slower speeds of contraction there was not a large difference between the the athletes and a normal population (a previous study had given a ratio of about 50% in a normal population, regardless of velocity). However, at high velocities the athletic subjects had very much higher ratios which approached unity (180°/s-300°/s). They concluded from this that the hamstrings play a greater role in muscular balance at high velocities. Another muscle which affects the knee joint is popliteus, which is important in knee joint stability. It medially rotates the tibia.

1.5. Biomechanical Effects on the Joint

1.5.1 Normal Function

An understanding of the normal function of the patella is essential, because only then, as has been frequently stated, can patellofemoral joint pathology be understood.

Hungerford and Barry (1979) have enumerated the functions of the patella:-

1. To hold the quadriceps away from the lower end of the femur and so maximise the mechanical advantage of the pull of the muscles when the knee is nearly straight: this means that the length of the fulcrum is increased, and thus
the lever arm. This maintenance of sagittal depth has been termed the "tent pole" effect of the patella by Terry (1989).

2. To enable the divergent individual muscles of the quadriceps group to be inserted in the way which has already been described, so that their pull is centralised.

3. It acts as a bony shield, protecting the knee joint from surface contusions.

4. When the knee is flexed, it allows the transmission of force round an angle without undue loss due to friction. The apposition of two areas of cartilage is a much more efficient load bearer than tendon on bone. It, therefore, avoids tendon wear and tear, and it also permits that force to be spread over a reasonably large area.

1.5.2. Patellofemoral Contact Pressure

The patellofemoral contact pressures or the patellofemoral joint reaction forces have been extensively studied, because it has been thought that increased pressures are a causative factor in the pathogenesis of the chondral changes frequently seen. These have been studied in a number of ways, and the majority of these have been in cadavers. However, Reilly and Martens (1972) calculated the patellofemoral joint reaction force (P.F.J.R.F.) in vivo for various activities, from the floor reaction force as measured by a force plate. Another in vivo experiment involved the estimation of P.F.J.R.F. from the maximum isometric extensor force (MacDonald et al. 1989).

Ahmed et al. (1983) estimated the alteration in pressures in cadavers, caused by the variation of loads applied to the four heads of the quadriceps.
The earlier cadaveric studies depended on the estimation of patellofemoral joint pressures from the contact areas (e.g. Seedhom et al. 1977, Matthews et al. 1977, Fujikawa et al. 1983). Matthews et al. used a dye to visualise the contact area, Seedhom et al. inserted a casting material between the cartilage surfaces, Fujikawa et al. took slices of bone through the patella and femur. Another method used by Seedhom et al. was an estimation of the permeability to light. Some years later however the introduction of pressure sensitive film enabled the pressures to be directly measured, and this was done by Huberti and Hayes (1984) and Hehne (1990). Huberti and Hayes found that the contact pressure varied from 2 MPa at 20° of knee flexion to about 4 MPa at 90° of knee flexion when subjected to one third of the maximum isometric quadriceps moments. The contact area varied from 2.6 square centimetres at 20° to 4.1 square centimetres at 90°. Hehne, on the other hand found that there was very little change in contact pressure from 30-90° of knee flexion. He found that there was considerable deformation of cartilage, increasing the area of contact, in response to changes in the pulling force. At maximum pulling force the maximum area of contact was 5.4 square centimetres. He thought that the pressures at slight angles of flexion were likely to be high, because V.M.O. is working hard and because of the obliqueness of the lateral condyle.

The mathematical means by which the P.F.J.R.F. for various daily activities has been worked out, has altered, and become more complicated, as it has been realised that the mechanics are much more complex than was previously thought. Bandi (1972) used the equation:-
P.F.J.R.F. = 2 * Fq * \cos(y/2)

(where Fq is the quadriceps force in Newtons, and y is the angle in degrees between the quadriceps tendon and the patella tendon, that is the patellar mechanism angle), to find the P.F.J.R.F. for flexed standing, and found it to be 3.9 and 7.8 times the body weight, with and without hip flexion, at 90° (he used the terminology “patellofemoral contact force”). But there were inaccuracies in this formula that were subsequently recognised. One of these is that the quadriceps force does not equal the patellar tendon force at all angles, because the two forces work at different distances from the centre of the pulley at some angles (Ellis et al. 1980). Reilly and Martens (1972) used the formula given above, and found that for stairclimbing and descending that the P.F.J.R.F. is up to 3.3 times body weight at 60° and up to 7.8 times body weight for deep knee bends. Matthews et al. (1977) used the same formula and had similar results. All of them, and also MacDonald et al. (1989), mention the patellar mechanism angle, and not the angle of knee flexion, although only MacDonald et al. state how this was found. This is more accurate, because the angle of pull of the quadriceps is not parallel to the shaft of femur, and in the same way the patellar tendon makes an angle with the tibial shaft, which varies with the extent of knee flexion. The variation of this with knee flexion angle was worked out by Nisell (1985).

The work of the authors who found the patellar contact pressures by estimation from the P.F.J.R.F. are not likely to be as accurate as direct measurements of the pressures, because there is another factor which minimises the potentially high
loading at flexed angles that they did not account for. When the knee is flexed, the quadriceps tendon becomes loadbearing on the femur at 60° (Huberti and Hayes 1984, Hehne 1990). Huberti and Hayes measured the tendofemoral contact areas, and also the tendofemoral contact pressures, and found that they equalled 50% of the P.F.J.R.F. at 120°. Also, although the P.F.J.R.F. is dependent on extensor quadriceps force, at 90° of knee flexion Hehne (1990) considered that this is minimised by the high P.F.J.R.F. pushing the tibia dorsally on the femur. He considered that the long fibres of the vasti in this position do not contribute to the extensor force because the angle of their action is too far dorsal and the only parts of the quadriceps which do contribute to extensor force in this position are the ones which terminate in the patellar tendon. In contrast, he considered that the action of the vasti is to work with the flexors in preventing excessive strain being placed on the ligaments and posterior capsule. However, Javadpour et al. (1991) demonstrated that while the oblique fibres are inserted into the patella itself, the long fibres are inserted into the quadriceps tendon, and thus terminate in the patellar ligament. This indicates that it is likely the long fibres do contribute to extensor force.

It is generally considered that an alteration of the Q angle is a big factor in the pathogenesis of the condition (e.g. Insall et al. 1976). They described the Q angle in this way:—“This is the angle formed by the line of pull of the quadriceps muscle and the patellar ligament”. It is measured clinically by drawing a line from the anterior superior iliac spine to the centre of the patella and another line from the centre of the patella to the centre of the tibial tubercle. In practice a goniometer with an extended
arm is often used. This angle is about 10° in the male, and about 15° in the female, because of the wider pelvis; 20° and above is regarded as pathological. The normal Q angle is at a maximum when the knee is extended, because in the last 30° of flexion the the tibia is outwardly rotated. When the subject is seated and the knees flexed to 90°, in neutral rotation of the tibia it is zero.

Insall et al. (1976) found evidence of malalignment or malposition in the majority of 105 patients arthrotomised for chondromalacia, as shown by increase in the Q angle. When Huberti and Hayes (1984) studied the effect of alterations of the Q angle on patellofemoral contact pressures, they found considerable changes in the areas of contact, with both a decrease and increase in Q angle. This was associated with increased peak patellofemoral contact pressures and unpredictable patterns of cartilage unloading. Deformities of the proximal end of the tibia alter the angle of the patellar tendon and thus the Q angle. Fujikawa et al. (1983) performed high tibial osteotomies on 8 cadavers and found that at 15° of tibial varus, in 4 out of the 6 specimens, the contact areas were completely broken up with unpredictable patterns of pressures. The pressures are also altered, not only by varus deformities and presumably valgus deformities, but also changes in rotation which will have an effect on the Q angle and therefore the pressures.

Femoral anteversion decreases the Q angle, whereas femoral retroversion increases it. External tibial torsion also increases it. Lee et al. (1994) studied the influence of fixed rotational deformities of the femur on the patellofemoral
The height of the patella would also alter the patellar tendon force. The work of Ellis et al (1980) showed that:

\[ Q_t = P_t \frac{k}{k} \]

(where \( Q_t \) is the quadriceps tendon force and \( P_t \) is the patellar tendon force)

at 90° \( k = 0.65 \)

at 30° \( k = 1 \)

If the patella is high, at 90° \( k \) would be higher and therefore \( P_t \) would be greater and more equal to \( Q_t \). Thus the P.F.J.R.F. would be increased. The converse of patella alta is patella baja, which is where the patella is lower than the norm. In this case the contact area from 20° to ~80° would be greater than normal for the equivalent angle of knee flexion, and \( P_t \) would be less compared with \( Q_t \). This shows that patella baja does not have an adverse effect on the patellofemoral contact pressures.
contact pressures in human cadaver knees. The experiment was performed with the initial isometric tension of 200N, and the pressures were measured with the pressure sensitive film. At 20° of rotational deformity there was a slight increase of pressure on the contralateral facets of the patella, at 30° this was significant. The greatest increase was at 30° and 60° of knee flexion. When the tibia was rotated on the femur there was an increased pressure on the ipsilateral facet.

Patella alta also has an effect on the patellofemoral contact pressures, because the contact areas are decreased at a comparable knee flexion angle with a normally placed patella, and thus the pressures are raised. It has also been suggested by Bourne et al. (1988) that patella alta might have an adverse effect on the stability of the joint.

Huberti and Hayes (1988) in a later study, measured the pressure in the same way as before, on cadavers with patellar cartilage damage. However, the frozen, fresh knee joints were from ten adults aged 61-84, which is not the age range of classic chondromalacia, and Bentley and Dowd (1984) do not consider that the majority of patients progress to become osteoarthritic. Huberti and Hayes (1988) found that there was a lowered pressure within the areas of damage and an increased pressure around the edges of these areas. In the cadavers with grade I or II lesions they attributed this to loss of stiffness, and those with grade III or IV lesions with loss of height of the cartilage. They also performed capsular reconstructive procedures on
the knees. Medial plication increased the pressure on the medial facet, and lateral plication on the lateral facet. Lateral release had a very variable result.

Most researchers have emphasised the correlation of high contact pressures with chondromalacia, however, Morscher (1978) linked it with insufficient pressure; yet another hypothesis is that it is the variability of pressure associated with everyday activities that causes the high frequency of cartilage changes (Seedhom et al. 1977). Ficat et al. (1979) related chondromalacia of the lateral facet, and also centrally located chondromalacia, to hyperpression.

Chondromalacia of the medial facet, they considered, was frequently due to localised incongruity of the medial patellofemoral compartment. However, Hehne, after an exhaustive examination of the biomechanics of the joint, came to the conclusion that chondromalacia cannot be explained by biomechanical arguments alone and that it may indicate that the adaptation to erect walking is incomplete.

1.5.3. Biomechanical Effects of Long Bone Alignment

The effect of alignment on patellofemoral contact pressures has already been mentioned. When considering the biomechanics of the patellofemoral joint, one should not just consider knee biomechanics, but the biomechanical effect of the entire lower limb. In fact there is a case to be made that trunk movements and trunk muscle action could have an effect on patellofemoral joint function. The abnormally large range of external hip rotation necessary in ballet dancing can be a cause of patellofemoral problems. In a survey (Reid 1988), 14-20% of
Presenting problems were at the knee and 50% of these were patellofemoral. There was also a marked lowering of range of internal hip rotation.

Femoral anteversion is a frequently observed finding in patellofemoral pain. External tibial torsion is similarly implicated. These may or may not occur together, and when they do they cause the orientation of the patella to appear to squint. The effect that this has on the patellofemoral contact pressures has already be mentioned, but there has been work which has shown the correlation between torsion and patellofemoral problems.

Torsion in long bones takes place between the epiphysis and the diaphysis. This means that in the femur it causes change in angle between the shaft and the head (Grays Anatomy 36th Edition p392, Eckhoff et al. 1994), so that the neck is carried forward on the head. This causes the transverse axis of the lower end of the femur to make an angle with the transverse axis of the head. When there is increased femoral anteversion the shaft is twisted medially. On the other hand there may be posterior femoral version, although this is not nearly as common. Fabry et al. (1994) found that in a group of children, anterior femoral torsion was associated with internal tibial torsion in 59.7% of them, and 40.3% with external tibial torsion.

Le Damany (translation of the original work 1994) has traced the normal development of tibial torsion. Fabry et al. (1994) discussed normal and abnormal torsional development in children. We are born with marked femoral
anteversion which slowly derotates throughout childhood. In a correction group with intoeing, in 20.5% of the children the anterior torsion of the femur decreased, but 62.9% developed compensatory external tibial torsion. The neonate has a perfectly neutrally aligned tibia which gradually externally rotates throughout childhood. This shows that slight external tibial torsion is normal. All the factors which cause the femur not to derotate enough, or the tibia to rotate too much, are not known.

External tibial torsion may or may not be associated with proximal tibia vara, sometimes called the bayonet sign, because the tibial alignment resembles a bayonet. This accentuates the laterality of pull of the patellar tendon.

1.5.4. Iliotibial Band or Tract

Although there have not, to the knowledge of the author, been quantitative studies made, the presence of a tight iliotibial band (ITB) has frequently been observed clinically with patellofemoral pain and dysfunction. It must have some effect on the symptoms experienced but what the biomechanical effects are has not been elucidated.

It has been demonstrated in an in vivo experiment that the ITB is tensioned at the part of the gait cycle when gluteus medius, vastus lateralis, and tensor fascia lata are active, that is early and mid stance (Huggler and Jacob 1983). Pare et al (1981) showed in cadavers that action of tensor fasciae latae could be transmitted to the knee via the ITB. They investigated the action of these muscles by EMG, in certain exercises and everyday activities. They found that
the posterolateral fibres were active in internal hip rotation. In walking these fibres were active near "heel strike", and the anteromedial fibres were active in jogging, running, and sprinting near "toe off". However, gluteus maximus is also inserted into the ITB, and is a large and powerful muscle. Although its main action is to balance the trunk, it can also externally rotate the hip as well as extend the hip. In the propulsive part of the stance phase of walking, plantarflexion occurs without much electrical activity occurring in the gastrocnemii (Sunderland et al 1980). Forward propulsion is brought about by the forward fall of the trunk (Gage 1990). Gluteus maximus is silent during this phase of normal walking (Pare et al. 1981), but when the speed of progression is increased it becomes active.

Although Huggler and Jacob (1983) demonstrated an increase in the tension when these muscles were active, and the presence of tightness in the structure clinically in some individuals suggests a cause which might be muscular, there is a body of opinion which considers that the tension has a purely passive cause (Kaplan 1977, and Hassler and Jakob 1981). These authors consider that changes in knee angle alone cause changes in tension of the ITB.

1.5. 5. Lower Leg and Foot Alignment

Carson et al. (1984) wrote two papers on evaluation of patellofemoral disorders. Part I consisted of a scheme of physical examination in patients, and in Part II there was a resume of radiographic examination. The physical examination included, not only a physical examination of the knee, but also a series of tests
and measurements of the whole lower limb. There are many clinicians who would use this approach.

These authors believe that the common denominator of malalignment appears to be abnormal pronation of the subtalar joint, which causes a compensatory internal rotation of the tibia, and that the increased rotatatory stress is absorbed through the peripatellar soft tissues of the knee. They summarised this abnormal subtalar pronation in the following way:-

- *Genu vara* = genu varum

**Fig 1.7. Causes of primary and secondary excessive sub-talar pronation (from Carson et al. 1984)**

One cause of subtalar pronation is the presence of tight plantarflexors. This may be due to tightness of the gastrocnemii alone or of the deep plantarflexors as well (Root, 1977). These two factors may be differentiated by the persistence of
limited plantarflexion when the knee is bent, if the deep flexors are tight. The minimum dorsiflexion which is necessary in midstance is 10° beyond the right angle (Root 1977). If there is not sufficient movement possible, either there is premature heel raise, or a compensatory pronation.

Inman et al. in their classic book Human Walking (1981, 2nd ed. 1993), explain the concomitant sub-talar pronation, plantarflexion, and internal tibial rotation, in early stance phase of walking. They assert that the maximal sub-talar pronation in normal function is 4°, and in mid and late stance phase there is gradual external rotation of the tibia.

1.6. Pathology of the Disorder

The origin of the pain has not been elucidated, and there are many unexplained anomalies. Cartilage is aneural, so the site of pain cannot be there. It has frequently been said that the disrupted cartilage causes changes in the underlying bone, however studies have shown that in only about 50% of patients with anterior knee pain can chondromalacic changes be seen at arthroscopy (Bentley and Dowd 1984, Shellock et al. 1990), and in many instances cartilage damage has been seen at autopsy and other examinations, in patients who have never complained of pain. Insall et al. (1979) explain the pain mechanism in this way. The vertical crest, which is convex, is covered by a thick layer of relatively soft articular cartilage which is sensitive to sideways loading, which would happen when normal stresses are applied in an abnormal direction. The pain threshold of the underlying bone, according to their hypothesis, is thus exceeded. Their hypothesis is borne out by what is seen in MRI scanning. It has also been said
that, in many cases, the pain originates in the surrounding soft tissues and that perhaps the biomechanical abnormalities in the whole of the limb cause the weight to be transmitted through the knee incorrectly. Bentley and Dowd (1984) in their study of 78 patients, however, reported that nearly all of them had persistent retropatellar pain.

Cartilage damage has been divided into four grades, to indicate the extent of the involvement, Grade I (less than 0.5 cms) - Grade IV (> 2 cm). Goodfellow et al. (1976) thought that the changes in chondromalacia occurred initially in the deeper layers of cartilage (Zone II), and that this basal degeneration, where there is fasciculation of collagen, caused a secondary superficial involvement. They found evidence of it around the ridge separating the odd from the medial facet. Surface flakiness they regarded as not significant. They found little evidence to suggest that chondromalacia develops into patellofemoral arthritis. Bentley and Dowd (1984), on the other hand, found that this was the pathogenesis in only a small number of patients, and more usually there was superficial chondrocyte damage, leading to splitting of the proteoglycan chains, causing loss of cartilage resilience and fibrillation. They also found the same primary site as Goodfellow et al. (1976), and again stated that there was no evidence of progression to osteoarthritis.

It has been suggested by several authors that the abnormalities of the cartilage cause changes in the sub-chondral bone, and MRI again shows this. Bentley and Dowd (1984) reported increased uptake of T99 in some patients with
chondromalacia, Darracott and Vernon-Roberts (1971) showed that 7 out of 11 patients showed decreased uptake of strontium 87M, and 2 showed increased uptake. These findings point to disordered calcium metabolism in the underlying bone. When Darracott and Vernon-Roberts (1971) examined excised patellae they found evidence of hyperplasia of the chondrocytes and advancing ossification into the deep zone of the articular cartilage, thinning of the subchondral osseous plate, and severe osteoporosis of the underlying bone. However, it is possible that the last two changes could be explained as being due to disuse. Goodfellow et al. (1976), Bentley and Dowd (1984), and Darracott and Vernon-Roberts (1971) all agree that chondromalacia is not the same pathologically as osteoarthrosis. Bjorkstrom et al. (1980) reported changes in the intramedullary circulation, causing high intramedullary pressures, which they suspected as a cause of pain. Vernon-Roberts and Pirie (1973) reported the presence of microcallus in underlying bone where there is degeneration of articular cartilage, and thought that these might be buttresses of the trabeculae of cancellous bone. Blackburn et al. (1991) demonstrated that the microcallus material was as hard as trabeculae.

Townsend et al. (1977) found that the known trabecular patterns of the subchondral bone closely matched directions of motion of the patella. This finding would support Insall’s hypothesis as to the origin of the pain (1979). The former authors reported that experimentally found joint forces during stair climbing showed a concentration of shear forces on the medial facet. These
forces matched the disorganisation of underlying bone of this region, so that the
cartilage changes could be spared and limited.
CHAPTER H

METHODS USED IN THESE EXPERIMENTS

2.1. CHOICE OF SUBJECTS

Seventy two patients with chronic patellofemoral pain were studied. Of these patients ~10% did not complete all of the tests, having withdrawn from the study at different stages for various reasons. Also the early EMG work was not acceptably accurate due to difficulties with the equipment, but 49 patients gave useful EMG data.

The patients studied were initially approached during their consultation at the orthopaedic out-patient clinic, or subsequently. They were then asked again by the author if they wished to participate in the study. If they agreed they were asked to return with the signed consent form. They were, therefore, examined by an orthopaedic surgeon, or a consultant in Sports Medicine, and as part of the examination had routine X-Rays taken, many with sky-line views. However, not all of them had had MRI. Thus fractures and other abnormalities were excluded, and the routine clinical orthopaedic tests to aid differential diagnosis in the knee had already been performed.

When they first attended for these tests they were asked to give a full history, and had a full clinical examination. The routine clinical examinations for meniscal problems and ligamentous laxity were repeated. Also the site of pain was very
carefully elicited. They filled in a questionnaire, which included a pain scale relating
to each activity which exacerbated the pain, and they were also asked to list any
activities which they were prevented from doing because of the pain. The main
criterion for choice was retropatellar pain, but peripatellar pain was included if it was
close to the patella. This was because the cause of pain has not been elucidated and
it was felt that the patients might not specifically be able to identify the exact site
of the pain. Another criterion was that the onset of pain was between 13-30 years of
age, because it was wished to exclude patellofemoral osteoarthritis. Tests were
made to elicit retropatellar tenderness, however any tenderness elicited at 0° of
flexion was discounted because the synovium can be trapped at that angle.

Questionnaire

The questionnaire was devised following the findings of Jensen et al. (1986)
The main pain intensity score which was used was the visual analogue scale (0-10),
where 0=no pain, and 10=pain as bad as pain can be. This was explained verbally to
the patients. They either ticked along the line or filled in a number, either was felt to
be acceptable. They filled in the level of pain for a variety of activities known to
cause problems in patients with patellofemoral problems.

0. ............ Squatting
0. ............ Climbing stairs
0. ............ Descending stairs
0. ............ Sitting for long periods
0. ............ Standing for long periods
Walking

Cycling

Running

When analysing the scores they were added together, and then divided by 10 to make the figures easier to handle. The maximum score possible was thus 8.

The second question related to any activities they were prevented from doing because of the pain. They specified each activity and then ticked under the appropriate heading whether they were occasionally prevented from participating or severely incapacitated.

Severely Occasionally Activity

...............................(1)

..............................(2) ................. (3) etc.

A behavioural rating scale was also used to identify the behaviour of the pain over the previous week. The appropriate box was ticked.

( ) no pain at all
( ) some of the time
( ) most of the time

The last question related to whether any analgesics were used and how frequently.

Exclusion Criteria

Exclusion criteria were:-

1) Bipartite patella
2) Femoral trochlear fracture
3) Osteochondritis dissecans
4) Osgood Schlatters disease
5) Sinding-Larsen-Johanson disease
6) Scarred or inflamed plicae
7) Meniscal or ligamental pathology
8) Reflex sympathetic dystrophy
9) Gross effusion
10) Patellar tendinitis
11) Muscle tear
13) Recent knee operation but not including arthroscopy.
14) Patients whose symptoms were an acute response to trauma, (therefore another criterion for choice was pain of insidious onset).

Attempts were made to exclude inflamed plicae on the basis of medial condylar palpation, and the active extension and flexion of the knee in sitting. However, it should be noted that a proportion of patients with a medial plica have lateral symptoms, and these may not have been diagnosed. Patients were tested for sensitivity to light touch (one of the symptoms of reflex sympathetic dystrophy) by stroking with a finger or a wisp of cotton wool. They were also closely questioned as to sensitivity to heat and cold. There was a visual inspection of the whole lower limb in lying, sitting, and standing, and also walking, before the individual tests and measurements were undertaken.
Fifty three patients had bilateral symptoms (74%), and nineteen unilateral symptoms (26%). Twenty eight of the patients were men (39%). The age range was 19 to 39 (median age = 25). All the patients experienced pain either going up or down stairs, and running; sports involving deep flexion brought on the pain in all the patients. 21% found that sitting for long periods caused symptoms. Many (57%) were quite specific that it was taking weight on the bent knee either ascending or descending stairs that caused pain. 55% rated going down stairs more painful than going up. The duration of symptoms ranged from 4 months to 20 years.

The signs were:-

1. There was either no effusion or only slight effusion.
2. There was frequently hypermobility or hypomobility of the patella, but because the author was advised that there might be a difficulty of quantifying these findings, the results are not specifically presented.
3. The iliotibial band was also often seen to be tight on performing a modified Ober’s test (69% of those tested, n=44, had a positive result).
4. Retro-patellar tenderness

Groups of normal subjects who were symptom-free, were recruited as necessary for the individual experiments. They voluntarily gave their informed consent to perform the experiments. The age range of the normal subjects was 20-33 (median age was 61
24). The same percentage of each sex as the patient group, was maintained for each experiment. They were closely questioned to ensure that they had not had any knee symptoms. They were also questioned as to their accustomed physical activity, so that the range of these was similar in the patient and control groups.

The scale used was modified from a Tegner Activity Scale (Tegner and Lysholm, 1985) to make it applicable to a British way of life.
Table 2.1. Activity scale (adapted from Tegner and Lysholm 1985)

<table>
<thead>
<tr>
<th>Activity Scale</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>10. Competitive sports</td>
<td>Soccer - national and international elite</td>
</tr>
<tr>
<td>9. Competitive sports</td>
<td>Soccer, lower divisions</td>
</tr>
<tr>
<td></td>
<td>Ice hockey</td>
</tr>
<tr>
<td></td>
<td>Wrestling</td>
</tr>
<tr>
<td></td>
<td>Gymnastics</td>
</tr>
<tr>
<td>8. Competitive sports</td>
<td>Tennis</td>
</tr>
<tr>
<td></td>
<td>Athletics (running)</td>
</tr>
<tr>
<td></td>
<td>Motorcross, speedway</td>
</tr>
<tr>
<td></td>
<td>Handball</td>
</tr>
<tr>
<td></td>
<td>Basketball</td>
</tr>
<tr>
<td>7. Recreational sports</td>
<td>Soccer</td>
</tr>
<tr>
<td></td>
<td>Rugby</td>
</tr>
<tr>
<td></td>
<td>Bandy and ice hockey</td>
</tr>
<tr>
<td></td>
<td>Squash</td>
</tr>
<tr>
<td></td>
<td>Athletics (jumping)</td>
</tr>
<tr>
<td></td>
<td>Cross-country track both</td>
</tr>
<tr>
<td></td>
<td>recreational and competitive</td>
</tr>
<tr>
<td>6. Recreational sports</td>
<td>Tennis and badminton</td>
</tr>
<tr>
<td></td>
<td>Handball</td>
</tr>
<tr>
<td></td>
<td>Basketball</td>
</tr>
<tr>
<td></td>
<td>Downhill skiing</td>
</tr>
<tr>
<td></td>
<td>Jogging, at least five times per week</td>
</tr>
<tr>
<td>5. Work</td>
<td>Heavy labour (eg building, forestry)</td>
</tr>
<tr>
<td></td>
<td>Dancing</td>
</tr>
<tr>
<td></td>
<td>Competitive sports</td>
</tr>
<tr>
<td></td>
<td>Cycling</td>
</tr>
<tr>
<td></td>
<td>Cross-country skiing</td>
</tr>
<tr>
<td></td>
<td>Rowing</td>
</tr>
<tr>
<td>4. Work</td>
<td>Moderately heavy labour (eg truck driving, heavy domest. work)</td>
</tr>
<tr>
<td></td>
<td>Recreational sports</td>
</tr>
<tr>
<td></td>
<td>Cycling</td>
</tr>
<tr>
<td></td>
<td>Cross-country skiing</td>
</tr>
<tr>
<td></td>
<td>Jogging on even ground at least twice weekly</td>
</tr>
<tr>
<td>3. Work</td>
<td>Light labour (eg nursing)</td>
</tr>
<tr>
<td></td>
<td>Competitive and recreational sports</td>
</tr>
<tr>
<td></td>
<td>Swimming</td>
</tr>
<tr>
<td></td>
<td>Walking in forest</td>
</tr>
<tr>
<td>2. Work</td>
<td>Light labour</td>
</tr>
<tr>
<td></td>
<td>Walking on uneven ground possible but impossible to walk in forest (or country)</td>
</tr>
<tr>
<td>1. Work</td>
<td>Sedentary work</td>
</tr>
<tr>
<td></td>
<td>Walking on even ground possible</td>
</tr>
<tr>
<td>0.</td>
<td>Sick leave, disability pension because of knee probs.</td>
</tr>
</tbody>
</table>

Table 2.2. Results of Activity Scale

<table>
<thead>
<tr>
<th></th>
<th>Patients</th>
<th>Normal subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>number</td>
<td>70</td>
<td>25</td>
</tr>
<tr>
<td>mean score</td>
<td>3.53</td>
<td>4.25</td>
</tr>
<tr>
<td>s.d.</td>
<td>1.34</td>
<td>1.07</td>
</tr>
<tr>
<td>s.e.m. +/-</td>
<td>0.16</td>
<td>0.214</td>
</tr>
</tbody>
</table>

Student's t patients/normal subjects = 2.694 (p<0.05)

However the disparity of the mean scores for the two groups (patients/normal subjects) is not large and it is not anticipated that this factor will greatly influence the results of the experiments.
The activity scale was used for the normal subjects undergoing the muscle tests, but not for the measurements of hip rotation or hamstring length or dorsiflexion. However all the subjects were symptom-free.

2.2. EMG RECORDING

This method, which will now be described, was used to obtain the electromyographic signals (EMG) from the muscles concerned in all the experiments involving this procedure, both on the patients and the normal subjects. The parts of the quadriceps of interest in the experiments were the long fibres of vastus medialis (V.M.L.), and the oblique fibres (V.M.O.), the long fibres of vastus lateralis (V.L.L.) and the oblique fibres (V.L.O.), and rectus femoris (R.F.). Gray's Anatomy states that the nerves to vastus medialis and vastus lateralis enter the two muscles midway along the length, which gives some indication of the motor points of the long fibres of the vasti. However, it was not known where to find the best position to record the EMG from the oblique fibres of the vasti (V.M.O. and V.L.O.). It is generally considered that it is best to record over motor points because the signal is at a maximum at this point. To facilitate this, the motor points were first found on two subjects and since it was thought that the position in the muscle belly would not vary greatly from subject to subject, the surface electrodes were positioned over the motor points on all the subjects.
2.2.1. Method of Finding the Motor Points.

A flexible pad, manufactured for the purpose of muscle stimulation was well gelled with electrode jelly and placed over the femoral nerve in the femoral triangle. The femoral nerve supplies all the quadriceps. With the anode at this point it does not obscure any of the quadriceps muscles. The cathode was a small button electrode which was covered with lint and was also well gelled. The anode and the cathode were connected to a muscle stimulator which supplied square wave pulses of 100 µsecond duration at 1Hz intervals. The cathode was placed over V.M.L. and the voltage increased until a well defined muscle twitch was obtained (~40-50V). The current was then decreased until a barely perceptible twitch was obtained only in one area of the muscle. This was assumed to be the motor point. The cathode was moved around over the surface of the long fibres of the muscle to make sure that a twitch was only obtained at that point. With the voltage held at that level, the button electrode was moved downwards towards V.M.O. In a certain area over V.M.O. a twitch was again perceptible. This indicated the presence of another motor point. The same method was used over V.L.L. and V.L.O. The cathode was also moved around over the surface of R.F. This experiment was performed on two subjects.

Results

Two motor points were found over vastus lateralis, and two over medialis, but only one over R.F., in the positions denoted in the diagram.
Fig. 2.1. Positions of the motor points of V.M., V.L. and R.F. (R. thigh).

Discussion

The results supported the hypothesis that both vastus medialis and vastus lateralis do indeed consist of two parts (V.M.L. and V.M.O., and V.L.L. and V.L.O.). However, the same result might not have been obtained with every subject because Lieb and Perry (1968) have shown in dissections that the nerve supply to V.M.O. is variable, but it was decided not to pursue this matter with different subjects, because the main reason for the experiment was to find optimum positions for the placement of electrodes.

2.2.2. Description and Use of the EMG Equipment

The electrodes which were used to record the EMG were manufactured by Motion Control Inc. and supplied by Oxford Metrics. Each pair of electrodes were encased in plastic cases which were rectangular in shape. Thus the interelectrode distance
was always the same (3.5cm). Between the active electrodes was a reference electrode. Each electrode unit also contained a pre-amplifier with a gain of about 380. The active electrodes were made of stainless steel and were a.c. coupled. These stainless steel discs, which were 1.2 cm in diameter, and protruded below the casing so that they indented the skin to provide a good contact electrical contact. They were designed to be used without gel, but firm external pressure was necessary for a noise free signal to be obtained, by cutting down the interface resistance between the skin and electrode as much as possible. They had a high input impedance (the amplifier also had a high input impedance of 1MΩ, as recommended by Basmajian (1974)).

Each patient and normal subject was first asked to sit on a couch with their legs extended. The skin over the front of the thigh where the electrodes were applied was prepared by rubbing fairly vigorously with alcohol pre-injection swabs (Sterets*), so that a slight erythema was produced and the alcohol had removed the surface grease on the skin. They were then asked to contract statically their quadriceps muscles, so that the individual portions could be delineated. Each pair of electrodes was placed over the individual portions, in the positions found in the study of motor points, and in the direction of the muscle fibres in each case. The electrode pair over V.M.O. was placed at an angle 55° to the line of the femur, so that the distal edge was 1.5 cm from the medial edge of the base of the patella. The location for V.L.O. placement was found by taking a line from the iliotibial band to the

---

1 * Manufactured by Seton Prebbles Ltd

67
lateral edge of the base of the patella, as far down the muscle belly as possible. At an angle of $40^\circ$ to the shaft of the femur this electrode pair was not as acutely angled as that over V.M.O., because that is the angle at which the fibres approach their patellar insertion (Javadpour et al. 1991). The fibres of V.M.O. are more horizontal (the lowest ones are angled at $80^\circ$ to the vertical axis of the femur). This was demonstrated in the dissections made by Bose et al. (1980). The position for R.F. was slightly medial and about midway along the length of the thigh. Since R.F. is a bipennate muscle, the electrodes were angled at $15^\circ$. This is, however, an approximation because the muscle architecture is more complicated in this muscle, with the deep fibres running ventrally. The electrode pairs over V.M.L., and V.L.L. were placed near the motor points (as previously identified), angled to lie along the length of the muscle fibres. The electrodes over these parts of the muscle were not angled as acutely as over either V.M.O. or V.L.O. The electrodes were fixed in position with strips of $^2$Micropore*, and then the appropriate width of $^3$Tubigrip* was rolled onto the leg to apply the required pressure, and keep the electrodes in position during any movement of the thigh which might occur.

When the electrodes were in position the leads, which were well screened against electrical interference, were connected to an amplifier, which had a gain trim to ensure that there was no interelectrode variability. There were different gain settings which could be used (the amplification was from 1000-10000). The raw signal could

---

$^2$ *Manufactured by 3M
$^3$ *Manufactured by Seton Healthcare Group
be collected, or it could be rectified, and smoothed by means of a "leaky" integrator, and the time setting constant of this could be changed (50, 100 or 200 ms) to vary the degree of smoothing. The amplifier ran at 6 V supplied by a separate a.c. adapter, thereby isolating the subject from the mains supply. The three channel EMG then passed via a junction box to an analogue to digital converter (A/D) connected to a computer via a serial port. The digital signal could then be displayed on screen, and stored on disc, using the programme "Microscope". With this software the duration of sample and the number of sampling points could be adjusted, as well as many other functions. For the isometric experiments the duration of sampling was 4 s and the number of sampling points was 1000. However, for the isokinetic experiment the duration was increased to 8 s, because a greater time was needed to enable the exercise to be completed. Since the trace had been rectified and smoothed, in most cases 1000 sampling points were sufficient. However, for any experiment using the raw data, including recording action potentials, the number of sampling points was increased to 4000 so that the maximum amount of data could be saved (see Fig. 5.3). The data was further analysed (via ASCII files) using various programmes.

2.2.3. Calibration of Amplitude of EMG Signal

To avoid interelectrode variability, because the gain of each pre-amplifier was slightly different, the gain was adjusted at the amplifier. This was calibrated before use and checked at frequent intervals during the period of the experiments. The

\(^4\) Written by Michael Wilson
electrodes were placed in perfect apposition to three stainless steel discs on the surface of a battery powered box, which when switched on, supplied 1mv square pulse. Although the interface was metal upon metal, a small amount of electrode gel was used to ensure a good electrical contact. The gain setting of the amplifier was 1000. Each electrode in turn was tested with the output from the raw signal channel. The output was connected to a portable Nicolet oscilloscope, so that 2V could be accommodated on the screen above the base line. The screen was marked with a grid in position, to enable each electrode to be calibrated at an output of 1V. Had it proved necessary the trim could have been adjusted, but after the initial calibration this was not necessary.

2.2.4. Calibration of EMG Amplifier Integrator

The amplifier integrator was set on the 100 ms time constant for the experiments, because it was found that the signal on this time setting was smooth enough for the estimation of amplitude, but quick enough for a change in the amplitude to be apparent. However to enable a user to be confident that the time settings were accurate, the time constants were calibrated.

Method

The integrators in all three populated channels were calibrated for all available time-constants, using the following procedure:-

The EMG amplifier was driven directly from a function generator, avoiding the use of the active electrodes. The function generator was adjusted to give a 200Hz square wave signal, of amplitude approximately 100mV, gated on and off at
approximately 1Hz. The integrated output of EMG amplifier and the gate signal from the function generator were displayed on a digitising oscilloscope. The oscilloscope time base, trigger and y-sensitivity controls were adjusted to display the rise in the integrator output following the gate signal’s “on” transition. The integrator output displayed an exponential rise from zero to an equilibrium value during the gate “on” time.

\[
\text{integrator output} = V_m(1-\exp(u/T))
\]

where \(V_m\) is the equilibrium voltage, and \(T\) the integrator time constant, which is defined as the time taken to reach \((1-1/e)\) times the equilibrium voltage.

The time constant was measured by measuring the equilibrium voltage using the oscilloscope’s marker system. This value was multiplied by \((1-1/e)\) (approx 0.63), and a voltage marker set to the resulting value. The oscilloscope time markers were then used to measure the time between the start of the gate signal’s rise and the point where the voltage marker intersected the integrator output signal.

The actual time constant setting are shown in Table 2.2.

<table>
<thead>
<tr>
<th>Channel</th>
<th>50</th>
<th>100</th>
<th>200</th>
</tr>
</thead>
<tbody>
<tr>
<td>Channel 1</td>
<td>50.0</td>
<td>101</td>
<td>196</td>
</tr>
<tr>
<td>Channel 2</td>
<td>50.4</td>
<td>102</td>
<td>201</td>
</tr>
<tr>
<td>Channel 3</td>
<td>50.4</td>
<td>101</td>
<td>200</td>
</tr>
</tbody>
</table>

This showed that there was minimal difference between the time setting constant markings and the actual values.
2.2.5. Measurement of Skin Resistance Using the Same Preparation as the EMG Recording.

Method

A sinusoidal current (1kHz) was generated from a power function generator. The output was passed in series through a 22000Ω resistor, and two stainless steel electrodes. The voltage output and the voltage across the electrodes were separately monitored by two channels of a Nicolet 3091 oscilloscope (1MΩ input impedance). The grid on the screen was put into operation, and the oscilloscope signal was adjusted until a 5 V signal was registered on the screen. Without the electrodes in contact, and therefore with an infinite resistance between them, the two signals were in phase and of the same magnitude. They were then placed on the skin round the knee the same distance apart as were the discs of the EMG apparatus. Because they might not have quite such a smooth surface as the stainless steel discs incorporated into the EMG recording apparatus, they were lightly gelled. The difference in voltage of the two signals was then measured.

The surface of the skin was then prepared by rubbing with alcohol, to remove the surface grease on the skin (using Sterets preinjection swabs), which was the same method of preparation as that used during the EMG experiments. The experiment was repeated to see whether the skin preparation had any effect on the impedance.
This same method was used on each of 4 subjects. The results obtained were then analysed in the following way:-

Where \( V_2 \) is the voltage produced by the oscillator, and \( V_1 \) is the voltage across the electrodes, the value of the skin resistance (\( R_i \)) can be found from the equation:-

\[
R_i = \frac{V_1 R_2}{V_2 - V_1}
\]

where \( R_2 \) is the known resistance (22,000 \( \Omega \)).

**Results**

The mean skin resistance for the four subjects before preparation was 30.3k\( \Omega \), with a standard deviation of 13.1k\( \Omega \). After rubbing the skin with alcohol it was 25.6k\( \Omega \) with a standard deviation of 8.7k\( \Omega \). The skin resistance of the subjects therefore would be of the same order, and since the EMG equipment had a high input impedance (1M\( \Omega \)), was well within the recommended levels. It is generally considered that the input impedance should be at least ten times that of the skin impedance.

**2.2.6. Cross Correlation of EMG Signals**

It is known that cross-talk (signals from one muscle being picked up at a second recording site) is a problem when recording from surface electrodes, so it was therefore decided to investigate the percentage of cross-talk from the individual recordings. Different motor units have individual firing frequencies; if, for example, some of the signal from VLO is being detected by the electrodes over VMO they will fire at almost the same time as the source signal, which will have the effect of making
both signals large at that time. This is the way in which the cross talk was detected.

The results obtained were analysed in two ways.

Method

In the experiments the quadriceps were either contracted isometrically at different angles of flexion, or isokinetically. Therefore, one of the conditions of muscle activation used during the experiments was reproduced in this experiment to estimate the percentage of contamination of the signals from one recording site to the other. The recording electrodes were positioned over V.M.O. and V.L.O. in the usual way, and the knee was bent to 20° of flexion. The subject was asked to make a maximal voluntary extensor effort. The raw signals were collected from both sites when the maximal effort was being sustained, with a sampling rate of 1000 Hz. The length of the sample was the same in both cases (576 sampling points), and was recorded in an ASCII file to analyse as described below. Deliberate contamination was produced to test whether this was detected by the method of analysis in the following manner:-

Analysis 1.

1. ) The signal from V.M.O. was correlated with the signal from V.L.O., obtained from the same recording.

2) A cross correlation was made of the signal from V.M.O., with the addition of 5% of the signal from V.L.O. (that is 0.95 of the V.M.O. signal plus 0.05 of V.L.O.)

3) The signal from V.M.O. with 10% of the signal from V.L.O.

4) The signal from V.M.O. with 15% of the signal from V.L.O.

5) The signal from V.M.O. with 20% of the signal from V.L.O.
6) The signal from V.M.O. with 25% of the signal from V.L.O.
7) The signal from V.M.O. with 30% of the signal from V.L.O.
8) The signal from V.M.O. with 35% of the signal from V.L.O.
9) The signal from V.M.O. with 40% of the signal from V.L.O.

Before the analysis, the mean value was first subtracted from each signal used (V.M.O. and V.L.O). The signal was then divided by its standard deviation. With this pre-treatment the central point of the cross correlation function (CCF) is numerically equal to the correlation coefficient (r). The correlation function was then calculated for shifts between -25 and +25 points (each point is equivalent to 1 millisecond). This is likely to be more sensitive an analysis than Analysis 2, because account is taken of the time which is taken by the propagation of the signal, so that it will show up contamination even if there is a delay in the common signal reaching one channel. The results are shown in Fig 2.2.a). Fig 2.2. b) is the same as a) on a larger scale. Examination of Fig. 2.2. b) suggests that there is a peak at a shift of -6 ms. The peak is equal to 0.124 ie 12% contamination.

Fig.2.2. a) and b). To show the correlation of the EMG recordings from V.M.O. and V.L.O. (in each case the vertical scale is the correlation coefficient and the horizontal scale is the shift).
The CCF for the addition and subtraction of the signal due to the deliberate contamination is shown in Fig. 2.2 c) to j).

Fig. 2.2 c) to j: To show the cross correlation for the addition and subtraction of the signal due to the deliberate contamination.
The range of the CCF for each contamination is plotted in Fig. 2.3. The additions to j) are to indicate what "range" means, viz. the difference between the maximum and minimum.

Fig. 2.3. The range of the cross correlation for each contamination

Armed with the information from Fig. 2.2.b), the analysis was repeated with the V.M.O. signal shifted 6 ms with respect to V.L.O. The results are shown in Fig. 2.4 and Fig 2.5.
Fig. 2.4. The same analysis with the V.M.O. signal shifted 6 ms to the left.
It can be seen that Fig. 2.4.d) gives a flatter signal than any of the others, suggesting that the contamination due to cross talk has been removed.

Fig. 2.5. The result of signals obtained from deliberate contamination, with those from V.M.O. shifted 6 ms with respect to V.L.O.

Conclusions

a) If there is any contamination its probable magnitude is approximately 10% and it is 6 ms out of phase

b) There is no statistically compelling evidence of any contamination. This is shown by the fact that the 6 ms peak in Fig. 2.2.b) is no higher than the other peaks.

Analysis 2.

Data was taken from 576 sampling points as before. From this the correlation coefficient and Student's t was found.
The graph of V.M.O. against V.L.O. had a slope of -0.031. This showed that one signal is not increasing with the other.

The correlation of V.M.O. with V.L.O. (r) = -9.678*10^{-3}

\[ t = \sqrt{r^2 * (\frac{576}{2})} \]

\[ t = 0.232 \text{ (which was not significant)} \]

To test whether this method could detect cross talk, the signals were deliberately mixed as before with 200 increments.

Combine \((f) = (f * \text{VMO}) + (1 - (f)) * \text{VLO}\)

\(\text{CC} (f) = \text{Corr} (\text{combination} (f) * \text{VMO})\)

The result of this is shown by the graph below.

---

**Fig. 2.6.** To show the effect of adding and subtracting increasing amounts of the other signal. It can be seen that around 0 (i.e. one signal correlated with the other with very small additions) that the slope is very slight. \((j=0\ldots200, \text{mix}_j = -1 + j/100, \text{Res}_j = \text{CC} (\text{mix}_j))\)
Discussion

Using the first method, the estimation of cross talk was 12%, use of the second produced an estimate of 3%. The reason why the estimate may be higher in Analysis 1. than 2. has already been suggested, i.e. there may be a time delay for the signal from one channel to reach the other. At most the percentage of cross talk was low, which would have a minimal effect on the signals at the two sites. In most of the experiments information of the differences between the individual portions of the quadriceps was sought. These are likely to be a little greater than demonstrated. In a few cases the similarity of the action of the portions was being demonstrated, in which case this would be slightly less than the results showed.

In the case of recording from other parts of the quadriceps the % of cross talk would not be any greater than has been shown, because the positions of the recording electrodes were not any closer.

2.2.7. Estimation of the Depth of Subcutaneous Adipose Tissue at the Sites of EMG Recording

Introduction

Although the paper relating the skin fold thickness over different muscles to the median firing frequency (Bilodeau et al. 1992) had not been published at the start of this project, many papers had been published comparing surface electrodes with intramuscular electrodes (e.g. Milner-Brown and Stein 1975, Perry and Bekey
1981). Several authors had sought to explain the finding that some muscles had linear EMG/Force relationships and some non-linear (e.g. Woods and Bigland 1983). Perry and Bekey (1981) suggested that the tissue between the muscle and the surface electrode can act as a low pass filter. It was therefore thought to be prudent before commencing muscle recording, to make an estimation of the depth of subcutaneous fat over V.M.O. and V.L.O. to see whether there was any difference between the two.

Permission was asked to use equipment (Futrex 5000) which estimates the depth of subcutaneous adipose tissue at another University. This was granted. The method utilised was near infrared interreactance.

Several studies have been made on the validity of the use of the Futrex 5000. This depends on the light absorption and reflection properties of different body tissues. Conway et al (1984) compared the results obtained with use of the Futrex 5000 with a) deuterium oxide b) skin fold and c) ultrasound. They found a very good correlation ($r=0.94$, $r=0.90$, and $r=0.89$ respectively). McLean and Skinner (1992) determined body composition by using Futrex 5000. They found that it was accurate except for extremes of obesity and leanness; with moderate amounts of obesity it was comparable with skin fold measurements.

When electromagnetic radiation strikes a material, the energy is reflected, absorbed or transmitted depending on the composition of the sample. Fat will absorb the
radiation differently from muscle. In estimating body fat the manufacturers found
that the best signal was obtained at 940nm and 950nm, and the instrument gives a
read out at these two wavelengths. The figures obtained are a ratio of light absorbed
and reflected , compared with the standard obtained when the probe is placed in a
Teflon block.

Method

9 subjects (4 males and 5 females) gave their consent to take part in this experiment.
None of them were extremely thin or extremely obese, and this was also true of the
patient group and the normal subjects in the main experiments. All the subjects were
seated in the same position. The position for the probe was found by asking them to
brace the thigh muscles, so that the muscle outlines were delineated. The same
position as used for the placement of the electrodes for the EMG experiments was
found over V.M.O. and V.L.O. This was marked with a skin pencil. They were then
asked to relax, with the lower limb extended. The probe was surrounded by a piece
of black foam, which also had a collar which was applied to the thigh. A reading
was taken at both 940nm and 950nm, but the reading at 940nm was the one which
was used. The instrument was standardised between each reading. The absorption of
fat is highest between 930 and 940 nm, so that this apparatus is very good at
distinguishing between different thicknesses of fat at different sites. The %
difference between the readings at the two sites was found for all 9 subjects (that is
over V.M.O. and V.L.O.), the average of this and the sem +/- was then calculated.
Results

There was in all subjects (except for one male) very slightly more fat over V.L.O., than over V.M.O., and as expected the females had more subcutaneous adipose tissue than the males at both sites. The differences between the two sites are set out in the following table:-

Table 2.4. Percentage difference in the amount of subcutaneous adipose tissue over the two sites

| % difference in the amount of fat over VMO and VLO | 2.33 |
| Sem+/− | 0.966 |
| Student’s t test | 2.411 |

Although the Student’s t test showed that there was significant difference between the two sites, this is unimportant because it is so small, and likely to have little effect on the EMG recordings.

It had been hoped to obtain readings for R.F., and the experiment was repeated to this effect. In the interim, however, the machine had been reset and the readings obtained were not as reliable. Therefore, it was decided to rely on the measurement of skin fold thickness. These results are shown together with the results for V.M.L. and V.L.L. in Chapter VI.
2.3. CYBEX II PLUS DYNAMOMETER.

The Cybex dynamometer is manufactured by Nomeq for isolated joint testing. This apparatus was used for all the experiments involving isometric and isokinetic testing together with monitoring of the muscles by EMG, but it was not used in the experiment described in Chapter III.

2.3.1. Description of the Cybex

The subjects were seated in the chair, with either none, one, or two back supports inserted according to the length of the thigh of the subject, so that the thigh was supported but the bend of the knee was free. The seat of the Cybex was marked with tape so that the thigh was aligned in the same way with each subject. Care was taken that the hip was in neutral rotation and neither abducted or adducted. Each subject was then stabilized by use of the chest and lap straps. The fulcrum of the force arm was placed over the knee jointline. 90° was found and verified with a goniometer (Akron) to take account of the slope of the thigh when the subject was seated. The goniometer was aligned along lines taken from the greater trochanter, through the jointline and terminating with the external malleolus of the fibula. Other angles could then be set by the Cybex.

In the experiments described in Chapter IV and Chapter VI, the Cybex was used in the isometric mode. In the experiment described in Chapter V the Cybex was used in the isokinetic mode.
Fig. 2.7. Patient seated in the Cybex chair with the EMG electrodes in position

2.3.2. Calibration of the Cybex

For the first few experiments the force arm of the Cybex was used for recording, and this was calibrated in the following way:-
The foot piece was removed and it was then locked into a horizontal position. Weights were then loaded sequentially on to the bar, and the voltage produced by each weight was recorded. The duration of the recording was set at 64s so that each weight and combination of weights could be loaded and unloaded on the bar in one recording. The weights which were used for the calibration were 5, 15, 40 and 65 pounds. The force that each of these weights represented was then calculated and expressed in Newtons (N). The calibration graph is shown below.

Fig. 2.8. Cybex calibration

After a limited period of use, it was felt that the Cybex force arm was not entirely satisfactory, because of a certain amount of play on it. Because of this a custom
built arm, which could be very firmly tightened, was instituted. It also had a better frequency response, which enabled the changes in torque to be more accurately assessed. Incorporated into this force arm were strain guages. It was extensible to accommodate different lengths of lower leg. At the lower end of the force arm was a bar, to which was attached a pad and strap with velcro fastening. The pad was fitted round the subject's lower leg as low down on the leg as possible but clear of the ankle. Thus the moment arm could be measured. This arm was connected to its own amplifier, and via the same junction box as the EMG signals (the junction box had 4 channels, 3 for collection of EMG signals and 1 for the force), to the computer, where the recording could be seen on the screen and stored on a floppy disc for future analysis.

This arm was also calibrated in the same way as the Cybex arm, i.e. it was locked into a horizontal position with the time setting at 64 s, and weights were loaded onto and unloaded from the bar. The voltage produced was recorded. However, on this occasion it was felt that the voltage which corresponded to more force should also be recorded (the force exerted by the quadriceps of men can be more than 700N). Therefore, two men were weighed, and first one and then the other stood on the horizontal bar at the end of the lever or force arm (the moment had to be the same for every weight), and the voltage was again recorded. Balance was not easy on this bar so speed was essential. Also the individual weights were weighed to see that they were accurate. The weights used were 1) 2.2 kg, 2) 4.6 kg, 3) 11.2 kg.
4) 11.2 Kg. The sequence of loading was 1, 1+2, 1+2+3, 1+2+3+4. The weights were expressed in Newtons (N), from this the moment or torque was found and expressed in Nm. The voltages produced by unloading were the same as the loading. The calibration graph is again shown below.

![Lever arm calibration 18.5.93 (G=500)](image)

Fig. 2.9. Calibration graph of the custom built force arm

Table 2.5. Values obtained from the calibration graph

<table>
<thead>
<tr>
<th>Newtons</th>
<th>Voltage</th>
<th>Slope</th>
<th>Intercept</th>
<th>Correlation</th>
<th>Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0</td>
<td>0.006675 V/N</td>
<td>0.005119 V</td>
<td>0.999994</td>
<td></td>
</tr>
<tr>
<td>21.574</td>
<td>0.1450</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>66.688</td>
<td>0.445</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>176.526</td>
<td>1.196</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>770.83</td>
<td>5.14</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>880.669</td>
<td>5.89</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The calibration was repeated at intervals throughout the length of the experiments involving this recording.
The results of these were:-

<table>
<thead>
<tr>
<th>In Newtons</th>
<th>In Nm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 volt = 149.82N</td>
<td>1 volt = 47.94 Nm</td>
</tr>
<tr>
<td>1 volt = 154.71N</td>
<td>1 volt = 50.28 Nm</td>
</tr>
<tr>
<td>1 volt = 154.86N</td>
<td>1 volt = 50.32 Nm</td>
</tr>
<tr>
<td>1 volt = 153 N</td>
<td>1 volt = 49.73 Nm</td>
</tr>
<tr>
<td>1 volt = 152.2N</td>
<td>1 volt = 49.47 Nm</td>
</tr>
<tr>
<td>1 volt = 150.8N</td>
<td>1 volt = 49.01 Nm</td>
</tr>
<tr>
<td>1 volt = 153.43N</td>
<td>1 volt = 49.83 Nm</td>
</tr>
</tbody>
</table>

The mean of these results expressed as N was 152.69N per volt with a standard error of 0.7152, when expressed as Nm the mean was 49.51 Nm

2.4. STATISTICAL METHODS

Parametric statistical methods have been used throughout this thesis. Variation between individual results has been expressed by the standard deviation (s.d.) calculated as

$$\sqrt{\frac{\sum (y_i - \bar{y})^2}{n-1}}$$

(where \(n\) is the number of observations and \(y\) are the results)

Variations in the measured parameters have been expressed as +/- the standard error of the mean (s.e.m. or SEM) where \(s.d./\sqrt{n}\).

Groups were compared using Student’s t test, \(t = \frac{\text{difference between means}}{\sqrt{s.e.m.}_1^2 + s.e.m.}_2^2}\)

In all the cases except in Chapter III and Chapter VI the test for unmatched samples was used. In Chapter III where the predicted quadriceps muscle strength was compared with the strength measurement, and in Chapter VI where the change in
ratios were found, Student’s t test for matched (or paired) samples was used. The formula used for finding this was:-

\[ t = \frac{\sum \text{differences}}{\sqrt{n \sum d^2 - (\sum d^2)^2}} } \]

\( \text{where } n = \text{number of subjects} \)

Values of the probability of the null hypothesis (p) were obtained from Statistical Tables of the “two tailed” test. Critical significance level was set at p<0.05. In all cases the degrees of freedom were taken by subtracting one from the number of subjects (n-1).

Correlation coefficients (r) were calculated using the Pearson product-moment formula, t values were obtained from this using the formula

\[ t = \sqrt{r^2 \times (n-2)} \]

\( 1-r^2 \)

F values have been used to compare two estimates of variance in Chapter III (pg 80). The larger variance (the less affected limb of the men patients) was divided by the smaller variance (the less affected limb of the women patients). When the result was greater than the value listed in Tables for the 2 degrees of freedom (DF for numerator \( v_1 \) and for the denominator \( v_2 \)), the difference in variance (for p = 0.05) was significant; a smaller value was not significant.
When error bars have been shown they have been taken from the standard error of
the mean (s.e.m.)

All the statistical formulae used in the calculations were those of Snedicor and
Cochran (1976).

2.5. OTHER TESTS

The methods used in other tests and measurements (including the method used for
the CT) are described in the chapter concerned.
CHAPTER III

EXPERIMENT TO TEST THE MAXIMUM VOLUNTARY ISOMETRIC STRENGTH IN A GROUP OF PATIENTS WITH CHRONIC PATELLOFEMORAL DYSFUNCTION

Introduction

It is well known that many kinds of knee pathology cause wasting of the whole of the quadriceps, for example arthritis, knee trauma and operation, whereas there has not been much evidence of the corresponding wasting of the hamstrings. Stokes and Young (1984) called this quadriceps wasting arthrogenous muscle weakness, and showed that it exists after a meniscectomy and persists for some weeks. The mechanism of this weakness is not fully understood but was not found to be associated with pain. They demonstrated this dissociation in two ways. They found that 3-4 days after the operation when the median pain was minimal, the inhibition was near maximum. They also injected the knee pre-operatively (10ml of 5% bupivacaine), which although temporally preventing most of the pain, did not change the severity of the inhibition. The inhibition was lessened but not abolished by aspiration of the knee effusion.

Other papers have demonstrated the presence of inhibition of the quadriceps in the absence of either pain or effusion but where there is a chronic mechanical derangement of the knee (e.g. Newham et al. 1989). Dvir et al. (1989)
demonstrated that this weakness was not accompanied by any loss of hamstring strength, in a isokinetic study on anterior cruciate ligament (ACL) deficient knees. How this inhibition could lead to muscle atrophy has been postulated by Stokes and Young.

Fig. 3.1. “Vicious circles” in arthrogenous muscle wasting (from Stokes and Young)

Loss of strength thought to be caused by this mechanism is a very important factor in all knee pathology, leading to further trauma and abnormality, and so it was considered important to find out whether this group of patients with patellofemoral problems did exhibit loss of strength. In the other experiments control groups were used because it was not possible to use the contralateral limb as a control, since this was frequently symptomatic and where it was asymptomatic, there were muscle abnormalities. In this experiment no control group of normal subjects was thought to be necessary, because there has been much previous work done testing the isometric strength of the quadriceps of normal subjects.
The prediction of quadriceps strength is not easy, and it is a complicated procedure to obtain the cross-sectional area of the muscles, in order to obtain the absolute strength. It has been shown that there is a clear correlation between the height of an individual and his/her muscle strength. Rutherford and Jones (1992) found that strength in women had a significant correlation with height \( r=0.17; \ p=0.018 \) but not with weight \( r=0.11; \ p=0.14 \). Parker et al. (1990) found that in girls from 5 to 11.9 yrs, strength was related to height \( r=0.752 \); in boys from 5 to 12.9 the correlation was 0.808. With adolescents the correlation was less. The method for predicting the strength of the quadriceps in this experiment was based on the calculation which Parker et al. derived from this regression. However the extrapolation from the height of growing children to adults who have finished growing is not ideal, but it was the best that was available. Other factors which would have an effect on the overall strength are the hormonal status, genetic considerations and the level of fitness.

The difficulty in measuring the force exerted by any muscle lies in deciding whether the maximum voluntary contraction is indeed truly maximal. The measurement of patients is especially difficult. A patient will avoid producing a maximum voluntary contraction for a variety of reasons, including fear of pain, actual pain, or even to impress how severely incapacitated they are, as well as the other causes of inhibition alluded to above. The measurement of pain would have been a
possibility, but this would only have taken account of one factor, and all the other unconscious factors would have been excluded. However, the experiment by Merton (1954) showed that when adductor pollicis was voluntarily fully activated, and then an electrical shock was interpolated during this contraction, there was no twitch visible on the tension record. Since then this method has been used as a method of deciding whether the voluntary contraction obtained is maximal (McComas et al. 1983, Rutherford et al. 1986). It is commonly referred to as “twitch interpolation” (Bulow et al. 1993). The percentage of inhibition can be estimated by the formula used by Bigland-Ritchie et al. (1986), which made a comparison of the height of the twitch obtained when the muscle was resting, with that when the subject was fully activating his/her muscles.

The test on the patients was made when the knee was bent to 90° of flexion because this is the position where the previous experiments were performed, and comparisons could be made with the predicted values. However, before the main experiment was performed, a small experiment was carried out to see at what length the quadriceps exerted their maximum force. The length-tension relationship of a whole muscle reflects the mechanical behaviour of the muscle fibre, which is dependent upon the overlap (Gordon et al. 1966). At the length where there is a certain degree of overlap a muscle will develop maximum tension. Quadriceps are frequently functionally used strongly at around 90°, e.g. getting out of a chair without arms (Seedhom et al. 1976). It was wished to see how well these three
factors agreed. It was considered acceptable if the quadriceps strength at 90° was within 10% of the maximum voluntary contraction.

3.1. Experiment to Compare the Force Exerted by the Quadriceps at Three Different Lengths

Method

Four normal subjects (2 women and 2 men) were each seated in a Cybex II Plus dynamometer with chest and lap straps firmly secured to stabilize the trunk and pelvis. The right knee was bent to 90° and the right thigh stabilized. The maximum force exerted by the quadriceps was recorded during a maximal voluntary isometric extensor effort lasting 2 seconds; this was repeated twice. The resultant signals were digitised and recorded on disc for further analysis. The highest force reading was taken as the maximum voluntary contraction (MVC). The knees were each straightened by 15° increments, after rests between each new position, and the readings compared.

The force was calculated and normalised against the maximal, so that the percentage of this obtained at each angle could be found.
Results

Normalised results of 4 subjects

Table 3.1. The ratios of the maximum force exerted by the quadriceps at different knee angles

<table>
<thead>
<tr>
<th>knee angle in degrees</th>
<th>90</th>
<th>75</th>
<th>60</th>
<th>45</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td>normalised</td>
<td>force</td>
<td></td>
</tr>
<tr>
<td>subject 1 (female)</td>
<td></td>
<td>0.9091</td>
<td>0.9632</td>
<td>1.00</td>
</tr>
<tr>
<td>subject 2 (female)</td>
<td></td>
<td>0.9022</td>
<td>0.913</td>
<td>1.00</td>
</tr>
<tr>
<td>subject 3 (male)</td>
<td></td>
<td>0.8786</td>
<td>1.00</td>
<td>0.789</td>
</tr>
<tr>
<td>subject 4 (male)</td>
<td></td>
<td>0.9966</td>
<td>1.00</td>
<td>0.9656</td>
</tr>
<tr>
<td>mean</td>
<td></td>
<td>0.9216</td>
<td>0.9691</td>
<td>0.9386</td>
</tr>
</tbody>
</table>

Fig. 3.2. The effect of length of quadriceps on force.

The 2 men were found to develop maximum force at 75° of flexion, and the 2 women at 60° of flexion, but the differences were within 10% of the maximum, so
it was felt that the difference would not materially affect the outcome of the experiment, and 90° of flexion was chosen as the angle at which the muscle strength was recorded.

3.2. Experiment to Test the MVC of Patients with Chronic Patellofemoral Problems.

Method

Each of the patients was seated in a metal chair surrounded by a metal frame which was quite stable and would not tip or move in any way during the testing. A lap strap was used to stabilise the pelvis, and the patients were instructed to keep their arms folded in their laps, so that other muscles were not brought into play. At the back of the metal frame was a horizontal metal bar with a internal strain gauge, which was attached via a metal cable to a canvas sling, which was placed over the front of the ankle with comfortable padding. The strain gauge signal was recorded on a UV recorder.
Fig 3.3. The chair for testing quadriceps strength. (A is the adjustable back support, B is the adjustable bar containing the internal strain gauges, C is the padded strap above the ankle)

The padded strap was always placed just above the bend of the ankle, so that the length of the lever arm varied with the length of the patients' tibia. The height of the transducer was at a right angle to the cable which was connected to the padded strap. If the subject was very tall the height of this was changed. The apparatus was calibrated before use and at frequent intervals during the time that patients were being tested, by turning the metal frame over, so that a bag containing weights could be hung free on the strain gauge. Weights were chosen (10.5kg) to be nearly
equivalent to 100N. The excursion of the UV recorder at gain=20 was 1cm, and at 50 it was 2.5cm; this was seen not to vary throughout the length of the experiment.

Percutaneous electrical stimulation was given to each patient via flexible pads of conductile rubber (7.5cm by 9 cm), coated in gel, which were strapped to the anterior surface of the thigh, proximally and distally along the fibres of vastus lateralis (V.L.), with a separation of about 6 cm. It was not possible to stimulate all the different portions of the quadriceps together, and V.L. is a large fleshy muscle which makes a big contribution to the overall strength, and therefore this was the muscle which was chosen for the twitch interpolation test. A stimulator and pulse generator supplied the current of a square wave pulse of a width of 50 μs, (the pulse width denotes the duration of each stimulus) at a frequency of 1Hz. The current voltage was gradually increased until the muscle twitch was clearly visible on the photographic paper (a height of 1cm was aimed at, which was at least 10% of the total force). Most of the patients tolerated this well, and only two refused permission to proceed, so in these two cases the force exerted by the quadriceps had to be recorded without the twitch interpolation.

Each patient (n=72) was instructed to push outwards against the cuff as hard as possible, and to try to move the beam of light which recorded the force on the photographic paper of the UV recorder, as far to the left as they could, thus providing visual encouragement. Verbal encouragement was also given. After
maintaining the extensor effort for a second, they then relaxed and had 2 minutes rest. This recording was repeated twice. The intensity of the stimulating current was then appropriately adjusted and turned off, and the patient made ready for another MVC. The speed of delivery of the photographic paper was increased. The current was turned on; three twitches were recorded before the MVC which was held for 3 seconds, and two twitches were recorded after the cessation of the effort. This was repeated with the other lower limb. Care was taken that one side was not always recorded first. The height of each patient was recorded by a stadiometer.

The % inhibition was worked out by measuring the size of the twitches which were made before the extensor effort and comparing them with those made during the quadriceps contraction, using the formula (Bigland-Ritchie et al. 1986)

\[
\text{% activation} = (1 - \frac{\text{height of superimposed twitch}}{\text{height of resting twitch}}) \times 100
\]

\[
\text{% Inhibition} = 100 - \text{% activation}
\]
Fig. 2.5 Twitch interpolation in a patient showing inhibition

The true strength of the muscle was then estimated by the addition of the figure that the % inhibition represents on to the measured result.

The predicted strength of the quadriceps was estimated as follows:

**Men**

Pred. strength in N = H^{3.01} * 98 \ (H = \text{height in m})

(over 16 and below 25 * 1.12)

**Women**

Pred. strength in N = H^{2.78} * 91.2

(Calculation from Parker et al 1990).

The predicted strength for the men aged 18-25 was multiplied by 1.12, because over a long period of practical application, the predicted strength was found to
underestimate the actual strength of men of that age group (Jones verbal communication).

The results of the men and the women were analysed in two separate groups, because in the results of the hamstring length experiment showed differences due to sex: and since the men were so much stronger than the women to treat them as one group would cause a very large standard deviation. The % loss of strength (compared with the predicted values) was found for both the men and the women, on the most affected limb, and the least affected or unaffected limb.

A paired t test was performed for all groups
A confidence limit was set at 95%, for the percentage difference of measured quadriceps strength compared with the predicted strength. The critical level for assessment of the probability of the null hypothesis was 0.05.

Results

Table 3.2 Results of the strength test

<table>
<thead>
<tr>
<th>Number</th>
<th>Predicted F in N</th>
<th>Measured F in N</th>
<th>% difference (less than the predicted)</th>
<th>Measured + inhib</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MEN</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n=27</td>
<td>Worse</td>
<td>599 ± 85</td>
<td>461 ± 167</td>
<td>4.1 ± 5.3</td>
</tr>
<tr>
<td>n=16</td>
<td>Less affected</td>
<td>623 ± 80</td>
<td>557 ± 174</td>
<td>1.3 ± 2.6</td>
</tr>
<tr>
<td>n=11</td>
<td>Unaffected</td>
<td>563 ± 79</td>
<td>508 ± 104</td>
<td>1.3 ± 1.3</td>
</tr>
<tr>
<td>WOMEN</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n=42</td>
<td>Worse</td>
<td>382.2 ± 42.2</td>
<td>290 ± 104</td>
<td>5.3 ± 5.3</td>
</tr>
<tr>
<td>n=35</td>
<td>Less affected</td>
<td>379.4 ± 41</td>
<td>348 ± 83</td>
<td>2.6 ± 8.30</td>
</tr>
<tr>
<td>n=7</td>
<td>Unaffected</td>
<td>407 ± 42</td>
<td>403 ± 87</td>
<td>0.1 ± 0.95</td>
</tr>
</tbody>
</table>
The total number of patients tested was 69 (27 men and 42 women) so that 61% of the patients were women.

The results of the men showed that there was a loss of quadriceps strength on the more affected side, compared with the predicted muscle strength. This was highly significant (p<0.001, confidence 99.9%). On the less affected leg (n=16) the % weakness was 9.605% SEM +/-7.256%, which was not significant, however the percentage of the weakness was altered by two patients who exceeded the predicted force by more than 25%. With the group who had unilateral symptoms only (n=11), the mean measured force was 8.297% less than the predicted force, SEM +/-6.607%, which was again not significant. Five of the men showed inhibition (that is that the superimposed twitch was still visible on the maximal application of extensor force), the mean value of which was 37.5%. When this inhibition was accounted for in the patients who exhibited this, the mean strength of the more affected leg for the whole group was 475.76N.

The results of the women also showed a loss of quadriceps strength on the more affected side compared with the predicted force; the % weakness on this side was again highly significant (p<0.001, confidence 99.9%). There was also weakness on the less affected side. This weakness, although less than the more severely affected leg was significant (p<0.02). In cases where the symptoms were unilateral (n= 7)
the measured mean force was 402.9N on the unaffected side, compared with the predicted force of 406.7N. In only one case was the predicted force considerably underestimated. This patient was a professional ballet dancer. The number of women patients who showed inhibition was eleven, (but two did not tolerate the stimulation) and when this added force was accounted for in these eleven patients, the mean actual force for all the women was 309N. The mean inhibition was 25.25%.

A comparison of the results of the two groups of patients (men and women) showed a very similar % loss of strength on the more affected side, (approximately 22% and 23%) showing the effect of the pathological process to be similar. The variances of the men and women were also similar, the F value of the comparison of the two groups was 1.04, which was not significant. On the less affected side where the loss of strength was significant in the group of women but not in the group of the men, a comparison of the two groups gave an F value of 2.35 which showed a significant difference between the variance of the two groups (men and women). However, when the results of the two men patients which were atypical, in that they exceeded the predicted force by more than 25%, were removed, a comparison of the two groups gave an F value of 1.93 which was not significant, indicating that there was no proven difference in variance between the two groups. Also the mean result for the men, after leaving out the two outlyers was 15.29%, which is also fairly close to the result for the women.
Discussion

All the results suggest that the low forces which were recorded were due to arthrogenous muscle wasting, leading to weakness. Young et al. (1982), in an earlier paper, found that the muscle wasting due to knee trauma was due to a reduction in the size of the muscle fibres, and that there was no other abnormality. This is different from the wasting due to age where there is also a reduction in the number of fibres (Larsson et al. 1979). The conclusion that there was arthrogenous muscle wasting could be drawn from the fact that the most affected side was also the weaker, so that where the pathological changes were more severe the wasting was more pronounced. This same conclusion could also be drawn from the results in patients with unilateral symptoms. On the unaffected side there was very little loss of strength. If the loss of strength had been bilateral with a marked difference between the measured values and the predicted values, it might have been forced changes in lifestyle causing the deficit, because general immobility or disuse can cause muscle wasting; but this was not so.

Inhibition did not seem to account for a major part of this weakness, although when it was present it was frequently of considerable magnitude. When the contribution due to inhibition was accounted for, in none of the patients was the force brought up to normal values. It may be that the inhibition was underestimated, because the test was undertaken at 90° where the inhibition has been shown to be less (e.g. Shakespeare et al. 1984, Jones et al. 1987). It has been demonstrated that

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inhibition is greatest near full flexion and full extension of the knee (Jensen and Graf 1993). It must also be borne in mind that the duration of symptoms of the patients in this study was considerable (4 months-20 years), which means that the full effect of these “vicious circles” could be felt (see diagram in introduction). While the majority of the patients did not experience pain performing this experiment, all of them had found other activities painful over a long period.

Which of the reflex mechanisms is responsible for the recorded changes, is open to question. Newham et al. (1989) reported a lowered quadriceps strength and inhibition on the affected limbs of patients with ligamentous injuries in which the common factor was ACL rupture. In that condition there are changes in the activation of the hamstrings (Solomonov 1987) thought to be a mechanism to compensate for the loss of stability, so the inhibition seen in that condition in the quadriceps may have been a reflex to prevent the tibia subluxating forwards on the femur. In the same way in patellofemoral dysfunction, there may well be a diminution of overall quadriceps activation to compensate for the changes in the pattern of activation which has demonstrated in the oblique fibres of the vasti (see Chapters IV and V). The purpose of this might be to prevent too great a disparity between the activation of different portions of the quadriceps, to ensure that differences in the pull on the patella are minimised, and stability maintained as much as possible. Newham et al. (1989) also reported inhibition in the unaffected leg.
which was not a factor in this experiment. The reflex mechanisms behind muscle
balance must be very complex and are not in all cases understood.

In cases where there was marked inhibition the impression was gained that it was
largely due to pain since the patients complained of this, but this was not true in the
majority of patients. The pain mechanism is not totally understood in retropatellar
pain. While cartilage is aneural there is evidence that there are changes in the
underlying bone (eg Bjorkstrom et al 1980), and there are likely to be free nerve
endings (Type IV receptors) in the bone (Rothwell 1994). Stener (1969) found
that in a patient with a tumour of the lateral epicondyle there was inhibition in V.M.
and V.L. but not R.F. If, then, the site of patellofemoral pain is in the subchondral
bone, this may cause a reflex pathway to be activated. Fulkerson et al. (1985), due
to their findings of the evidence of retinacular nerve injury, thought that it is likely
that the retinaculum itself is painful in many patients.

There are Golgi and Ruffini endings in the capsule which are receptive to
deformation of the capsule, at knee angles where there is stress on the capsule. In
an artificially induced effusion Jensen and Graf (1993) found that quadriceps
inhibition was greatest at these angles. While these are mechanoreceptors it is not
always easy to differentiate between these and nociceptors (Torebjork 1985), and
Spencer et al. (1984) found that an artificially induced effusion which caused reflex
inhibition was abolished on injection of lidocaine. However, changes in motor
reflexes reflect the consequences of action and does not pinpoint the exact steps in processing the information (Torebjork 1985). Hunt and Rossi (1985) reported that 50% of the primary afferents in nociception were peptide containing and 50% were not.

Marked effusion is very rare in patients with patellofemoral pain; in some of these patients there was a slight “puffiness” but the majority of them had no effusion at all, so the inhibition cannot all have been capsular in origin. In this experiment 60-70% of the patients experienced no pain at all in performing the MVC, so pain experienced at the time cannot have been a factor in these patients. In conclusion arthrogenic inhibition leading to wasting must be multifactorial.
CHAPTER IV
A COMPARISON OF THE ACTIVATION OF MUSCLES MOVING THE
PATELLA IN NORMAL SUBJECTS AND IN PATIENTS WITH
CHRONIC PATELLOFEMORAL PROBLEMS

4.1. Experiment to Test the Activation of the Oblique Fibres of Vastus Medialis
and Vastus Lateralis at 20° of Knee Flexion

Introduction

The quadriceps muscle has as its main function the extension of the knee, via its action on the patella and the patellar ligament. The anatomy and action has already been discussed in the main introduction, including the division of the whole muscle bulk into four heads, that is:- vastus intermedius (V.I.), rectus femoris (R.F.), Vastus medialis (V.M.), and vastus lateralis (V.L.). Each of them act on the patella from somewhat different angles, and therefore the possibility should be considered that an additional function of the quadriceps is to control the position of the patella with respect to the trochlear surface of the femur (Bose et al. 1980, Lieb and Perry 1968). The most oblique parts of V.M. (V.M.O.) and of V.L. (V.L.O.) as well as R.F. would be the sections most suited anatomically to exert this control. V.M.O. has been seen, in many cases to be separated from the long fibres by a fascial plane, and most of the fibres of V.M.O. arise from the tendon of adductor magnus (Bose et al. 1980). The innervation appears to be variable, and in some cases a separate motor nerve to the belly of V.M.O. has been observed (Terry 1986, Lieb and Perry 1968). V.L. has also been shown to be divided into two parts; the proximal fibres are
straighter, originate from the femur and are inserted into the middle layer of the quadriceps tendon, whereas the distal, or postero-lateral fibres originate from the iliotibial band, are more oblique in their direction and are inserted into the base and lateral border of the patella (Javadpour et al. 1991). They, therefore, suggested that they have a controlling effect on the patella acting in opposition to that of V.M.O., however no investigation of this has been undertaken. R.F. in contrast exerts a relatively straight pull (i.e. little medio- lateral component) on the patella.

An example of the need for control of patellar position is when the knee is nearly fully extended. At small angles of flexion there is a lateral component in the force which all the quadriceps except V.M.O. exert on the patella, which can only be dynamically counterbalanced by the action of V.M.O. (Hungerford and Barry, 1979).

Static or passive constraint is also provided by the medial retinaculum. As the knee is flexed, the patella moves downwards and medially over the femur (Kujala et al. 1989), entering the trochlear groove by about 20° of flexion. The trochlea is deeper distally, so that the congruity of the bony parts gives the joint more stability of the patella at increased angles of flexion. The timing of activation of V.M.O., of V.L.O. and of R.F. has therefore been investigated during isometric contractions with the knee flexed at 20°. If these parts of the muscle are effectively to control the position of the patella it would be expected that they would be rapidly activated, at the same time as, or even in advance of the larger parts of the muscle which generate the majority of the isometric force. The timing of their activation has therefore been compared with that of the the onset of extensor torque. The method of motor
control of the patella is not known, but there may be an initial ballistic movement. This "learnt" control of movement has been described by Rothwell (1994). "This pattern of EMG activity....represents a package of nervous commands which can be 'preprogrammed' in advance by the central nervous system...this does not mean that commands cannot be modulated under normal conditions by peripheral feedback mechanisms." Because this type of motor control is stereotyped, the first 80% of the force rise was monitored, in the expectation that this might exhibit a relatively simple behaviour.

Method

49 patients with chronic patellofemoral pain were studied. Criteria for their choice was as has already been set out in Chapter II. 20 normal subjects were also recruited to be a control group, chosen in the way which has been stated in the same chapter. Both lower limbs of the patients were studied and only the right lower limb of the controls. In previous studies no differences in activation of the quadriceps in normal subjects due to dominance had been seen. In this part of the report only the affected side (or most affected side) of the patients is presented.

The subject was positioned on a couch with his/her lower limbs extended and was instructed to brace his/her quadriceps so that the individual muscles of the group could be delineated and electrode placings selected. The electrode placings were as has already been described, that is one pair over V.M.O., one pair over V.L.O. and one over R.F., all of them orientated along the line of the fibres. The subjects were then seated in a Cybex II Plus Isokinetic Dynamometer, with the thigh carefully aligned so that the hip was in neutral rotation and neither abducted or adducted. The
force arm of the Cybex was replaced by a custom built force arm which was used for all the experiments in the Cybex. The fulcrum of this force arm was placed over the joint line of the knee. 90° of knee flexion was found and verified with a goniometer \textit{(Akron)}, to take account of the slope of the thigh when the subject was seated. The goniometer was aligned along lines taken from the greater trochanter, through the jointline and terminating with the external malleolus of the fibula. The knee was extended in the Cybex to 20° of flexion and fixed. The knee angle was again verified by the use of the goniometer. With change in angle of the knee the muscle fibres shorten and lengthen but no gross movement of the skin was seen to occur, so that the fact that the electrodes were positioned, and then the knee angle changed, was not seen to have any effect on the EMG signal obtained.

The subjects were instructed to apply maximum force as fast as possible and hold it for 2 seconds. The exhortations to produce the force quickly were repeated over a few trials when patients, or subjects, appeared to produce the force rather slowly. The EMG equipment and data storage were as have already been described. The EMG and force data were then analysed.

**Results**

A typical result from a normal subject is shown in Fig.4.1A. Each record has been expressed as a proportion of the maximum during this contraction. Force, and therefore the activation of the bulk of the muscle rises to 80 % of its maximum within less than 200 msec. Within this time all the EMG signals rise rapidly to a peak, and all the parts of the muscle are activated more or less together, with some tendency for VMO and VLO to be activated more quickly than the bulk of the
muscle. A result from a patient is shown in Fig.4.1B. In this example, which is
typical, the force develops rather more slowly than in the normal subject and the
EMG of those parts from which recordings were taken, show a delay in activation
relative to the force development.

Fig.4.1. Recording of force and rectified, smoothed EMG from two subjects during
maximum contraction of quadriceps. A is a normal subject; B is a patient with
patellofemoral problems. All the recordings are expressed relative to the maximum
value observed during the period shown.

In Fig.4.2 V.M.O. EMG records are plotted against the force record for the first
80% of the tension rise, to see whether the activation of these muscles keeps pace
with the development of force, which indicates the timing of the activation of the
other parts of quadriceps. With the EMG recordings of the normal subjects the resultant line falls above, or close to, the line of identity \((y = x)\). A close adherence to the line of identity would indicate that all parts of the muscle are activated together. Thus the oblique fibres kept pace with, or occurred before the activation of the long fibres producing the force. In the patient’s recordings, in contrast, the line falls well below the line of identity, so that the oblique fibres did not keep pace with the development of force by the long fibres.

Fig. 4.2. A comparison of the time course of activation of V.M.O. with the long fibres and other parts of the quadriceps. Plots of recorded EMG from V.M.O. against force developed (predominantly by the long fibres and other parts) during the first 80% of force rise for the contractions shown in Fig. 4.1. The thin line shows the line of identity which the data would follow if V.M.O. was activated with the same time course as the bulk of quadriceps.

The proximity to the line of identity has been quantified by measuring the area between the line of identity and the EMG data. This area, which is referred to as the
lag factor will be zero when the EMG data falls about the line of identity, positive when it falls below the line and negative when it falls above the line. The scale of the lag factor is such that a value of +1 would indicate no activation at all, and -1 would indicate instantaneous full activation. For the patient shown the value is +0.321, whereas for the normal subject shown in these figures the value is -0.046.

After the lag factors had been found for both the patient and the normal subject group, a Student's t test was applied, and the probability of a null hypothesis (that is that there is no difference between the two groups) set at p<0.05. A table of all the results is shown below in Table 4.1.

Table 4.1. A comparison of the lag factors for the 20 normal subjects and the 49 patients.

<table>
<thead>
<tr>
<th></th>
<th>Normal Subjects Mean ± SEM</th>
<th>Patients Mean ± SEM</th>
<th>Significance of difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time for 80 % tension development (msec)</td>
<td>185.8 ± 13.3</td>
<td>385.8 ± 31.4</td>
<td>p &lt;0.001</td>
</tr>
<tr>
<td>Force developed (N)</td>
<td>224.5 ± 12.3</td>
<td>174.0 ± 8.3</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>Lag factors</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VMO</td>
<td>-0.158 ± 0.047</td>
<td>0.161 ± 0.032</td>
<td>p &lt;0.001</td>
</tr>
<tr>
<td>VLO</td>
<td>-0.204 ± 0.040</td>
<td>0.083 ± 0.032</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>RF</td>
<td>-0.019 ± 0.057</td>
<td>0.180 ± 0.028</td>
<td>p &lt; 0.002</td>
</tr>
<tr>
<td>Number (number of males)</td>
<td>20 (7)</td>
<td>49 (16)</td>
<td></td>
</tr>
<tr>
<td>Age Range</td>
<td>20 - 33</td>
<td>20 - 37</td>
<td></td>
</tr>
</tbody>
</table>

The force exerted by the patients is slightly, but significantly, less than for the control subjects and its onset is on average more than twice as slow. There is also,
for each of the regions from which recordings were made, a large and significant difference between the patient group and the normal subjects in the timing of activation of V.M.O., V.L.O. and R.F., relative to the activation of the main bulk of the muscle. The difference seen for the oblique fibres (V.M.O. and V.L.O.) is greater than for R.F. Although these differences are large there is still some overlap between the control and patient groups for each of these measurements. This is illustrated by the histogram of the lag factors for V.M.O. shown in Fig. 4.3.

![Histogram of lag factors for VMO](image)

**Fig. 4.3.** A histogram showing the distribution of the lag factors (see text) for normal subjects (dark bars) and patients (light bars)

Considering all the observations (patients and control subjects) as a group, there are correlations between the three different lag factors. This correlation is strongest between V.M.O. and V.L.O. ($r=0.743$) and least between V.L.O. and R.F. ($r=0.432$). There is also (an inverse) correlation between the time for 80% tension
development and the V.M.O. lag factor. This might suggest that the increased lag factor was somehow due to the slower rise of tension. This was tested by selecting those of the patients (n = 21) whose rise times were within the normal range. Eleven of these patients had a positive lag factor. Comparing the mean V.M.O. lag factor for this group (-0.002) with that for the controls (-0.158), shows the former to be still significantly (p<0.05) greater. Thus the slowness of tension development cannot be the only reason for the change in the lag factor.

Discussion

As was expected from the hypothetical function of V.M.O. and V.L.O. each of these sections of the quadriceps in the normal subject is activated somewhat ahead of the main bulk of the quadriceps, as is shown by the significant negative lag factors in Table 1. R.F. by contrast, in the normal subjects is activated along with the bulk of the muscle. Grabiner et al. (1991) have previously reported that activation of V.M.O. leads that of vastus lateralis in normal subjects; he studied isokinetic extension of the knee, but did not find any statistically significant effect during isometric contraction. For V.L.O., which has not been investigated physiologically before, a similar mean negative lag factor as for V.M.O. was found and a strong correlation between the lag factors for these two parts of quadriceps. Thus they seem to share a common activation pattern as suggested by the anatomical finding of Javadpour et al. (1991). It is likely that the need for this common activation will be great with the knee angle that has been used, where the congruity of bony parts does not give much stability. Because of the insertion of V.M.O. onto the medial border of the patella an unapposed pull of V.M.O. would not only produce the necessary
slight medial patellar shift, but also an unwanted medial tilt in a horizontal plane due
to the fact that it would pull the patella dorsally.

The patient group showed large and significant differences from the control group in
the lag factors of the muscles tested. Quadriceps activation in patellofemoral
dysfunction has also been studied by Moller et al. (1986) and by Grabiner et al.
(1992). The former study is of the comparative amplitude of V.M.O. and V.L.
EMGs and found no significant differences between the groups; however they used
as a control group the contralateral \( \text{limb} \) of the patients. Such data has not been used
here as controls because it was noticed at an early stage of this study that it was not
common for the contralateral \( \text{limb} \) to be entirely asymptomatic, and where it was,
there was a tendency for the asymptomatic \( \text{limb} \) to show some abnormality. Grabiner
et al. (1992) studied isometric contractions with fast and slow activation and
reported that the patients showed decreased activation of both V.M.O. and V.L. in
the fast but not the slow contractions. This is in agreement with these findings that
the tension rise in isometric contractions made as fast as possible is slower in the
patients than in the control group. They, however, did not record from the oblique
portion of V.L.

The abnormality in activation of these oblique portions is unlikely to be simply a
response to pain because the majority of patients did not experience pain on being
asked to undertake the isometric contraction that was studied (at 20° of flexion).
Frequently patients did find an isometric contraction at 30° of flexion
uncomfortable. There was also a small group who did not experience any difficulty
in performing an isometric contraction at either 20 or 30°, but did have difficulty at 60 or 90°. These findings resemble those of other workers cited by (Grabiner et al. 1994) and short arc exercises are very frequently given for rehabilitation because that arc of movement is considered the most comfortable.

In the study by Lieb and Perry (1968) on cadavers, loads were applied to the muscles to produce a knee extension movement from 0-90 degrees. When the muscles were loaded singly, vastus intermedius was seen to be the most effective extensor. All the other long heads needed a mean 12% increase in force to produce the same movement. V.M.O. could not, by itself, effect a knee extension movement, no matter how great the loading, but V.L., by itself, laterally subluxated the patella without a countering force from V.M.O. It is likely that V.M.O. has a similar function in vivo, and that its action is necessary as the patella moves medially to enter the trochlea at about 20° of flexion (Kujala et al, 1989). If the activation of V.M.O. is sluggish and inadequate to the purpose, then the patella would not enter the trochlea at the correct angle, when the knee is flexed and the quadriceps are working eccentrically. This would mean that by 30°, in which position the patella is somewhat deeper in the groove, it would not articulate correctly with the femur, as is necessary for the area of articulation to be a large enough band as shown by Goodfellow and Hungerford(1976), for the patella contact force to be applied in the usual manner. There would then be areas of hyperpressure, which is thought to cause pathological changes (Insall et al. 1976, Outerbridge and Dunlop 1975), and also hypopressure, which is thought to cause disturbances of nutrition (James 1979). The
direction of the force applied would also not follow the way the trabeculae are laid down in the subchondral bone as found by Townsend et al. (1979).

A longitudinal study would be necessary to answer the question whether these abnormalities of muscle action are a cause or effect of chronic patellofemoral pain, but large and significant differences in the activation timing of V.M.O. and V.L.O. have been demonstrated in the patient group, compared with that in the normal subjects.

4.2. Activation of V.M.O. and V.L.O. in the Group of Patients with Unilateral Symptoms.

Introduction

The unanswered question which was left by the results of this experiment was whether the muscle abnormality shown was a result of the symptoms or a cause of them. While the majority of the patients concerned did not appear to experience pain while performing the maximal isometric extensor effort at 20° of knee flexion, it could be argued that the abnormality of the muscle activation which has been demonstrated could be inhibition to avoid the pain. Spencer et al. (1989) have shown that there is muscle inhibition, particularly noticeable in V.M.O., in subjects with an artificially produced effusion. It is also likely that the dip in amplitude of the EMG, again particularly noticeable in VMO in the isokinetic experiment, (see Chapter V) was also due to inhibition. It has also been shown that inhibition can occur in the whole of the quadriceps during a maximal isometric extensor effort (see Chapter III) in this group of patients. This abnormality of muscle action then could be due

*(an artificially produced effusion is easy to demonstrate experimentally whereas other pathological changes would be almost impossible to reproduce)*
to fear of pain, or to any of the pathological processes which habitually give rise to pain in these patients.

In most of the patient group studied, the symptoms were bilateral, even if usually they were more severe in one knee than the other. However, in 17 patients they were unilateral. In this group the patients were quite sure that one knee was asymptomatic. Therefore, it was considered important to see whether the abnormality in muscle action was present in the asymptomatic leg, in order to illuminate further its origin, and to this end V.M.O. action was analyzed in the same way in this leg, in all of the 17 patients.

Results

Sixteen of these patients had a positive lag factor on the symptomatic limb. The mean for the group as a whole was +0.2419 with a standard deviation of 0.2179. The results for the asymptomatic side gave a lag factor of +0.1618 with a standard deviation of 0.1868. In the one patient who did not have any slowing of the activation of V.M.O., and a lag factor of -0.382 on the symptomatic limb; on the asymptomatic side the lag factor was -0.372, which was remarkably similar. The results of the whole group show that there was a slowness of V.M.O. activation on the asymptomatic leg but not as marked.

Discussion

Had the lag factors been exactly the same on both sides one could have argued that the positive lag factor had nothing to do with the pathological process and was therefore of no significance. However, the results show that the significance lies in the magnitude of the lag factor. It is possible that these abnormalities are
developmental and give a predisposition to patellofemoral problems. Once this occurs there could be a downward spiral of all the factors which together give rise to patellofemoral problems, in which the bony and soft tissue adaptation further impair the effectiveness of the muscles, particularly V.M.O. Another factor leading to the difference between the asymptomatic and the symptomatic side in the same individual could be explained by reflex inhibition leading to muscle wasting. Muscle wasting is unlikely to be a factor in the asymptomatic knee because there was no evidence of loss of strength on the asymptomatic side in Experiment 3. It is possible that there might be reflex inhibition from the symptomatic knee. Newham et al. (1989) demonstrated this effect in the case of unilateral ACL rupture and other ligamentous deficit, but neurophysiologists have demonstrated crossed extensor reflexes. However, this is unlikely in this case because no such inhibition was discernable in Experiment 3, and both sides were tested for inhibition. Also reflex inhibition as demonstrated by Stokes and Young (1984) leads to muscle weakness, and as already stated this was not seen.
CHAPTER V

EXPERIMENT TO TEST PATIENTS WITH PATELLOFEMORAL PROBLEMS ISOKINETICALLY FROM 0 TO 90 DEGREES OF KNEE FLEXION

Introduction

There have been many studies of isokinetic testing which have been performed using the various makes of apparatus which have been industrially manufactured for the purpose (eg Cybex, Lido, Kincom). These devices control the velocity of the limb to be moved, at an angular velocity which can be set, from 30°/s to 360°/s. During this controlled velocity the torque can be recorded. It is usually considered that the slower speeds are more useful for studying the strength of the muscles concerned, and the higher speeds for studying endurance (Kannus 1994). In a research context they enable muscle action to be studied in a dynamic situation, albeit a somewhat artificial one, since in isolated movements account cannot be taken of muscle synergists. Some of the studies have been directed at establishing the relationship between isokinetic and isometric torques (eg Grabiner et al. 1990, Kaufman et al. 1991, Bobbert and Harlaar 1993). Some have been directed at evaluation of the apparatus and the techniques which are in use (Winter 1981, Rothstein 1987, Kannus 1994). Peak torque has been the most frequent parameter which has been evaluated. However, Reid et al. (1989), in an isokinetic study of shoulder strength, considered that the arc of maximum strength might be a more functional measurement than peak torque. The angle of peak torque has also been studied; Kannus and Beynon (1993) found that for
knee extension this had a mean of 54° of flexion. However, Herzog et al. (1991) demonstrated that the angle of peak torque of R.F. is dependent on the hip angle, and for it to be comparable with the rest of the quadriceps the hip angle has to be at 90°.

These studies have been performed on normal subjects, and frequently EMG monitoring of the active muscles has also been employed. Afzali et al. (1992) studied the shape of the normal torque curve with a view to discovering differences in shape in pathological conditions. There have also been other studies of the shape of the curve. Bobbert and Harlaar (1993) performed the test from a low threshold moment and a high threshold moment (i.e. the isometric tension at the start of the movement which was at 85° of knee flexion). When starting from a high threshold moment the first part of the curve was nearly horizontal, but from a low threshold moment, time was taken for the torque to be built up. They postulated that in the former condition the muscles are operating in a more favourable region of their force/velocity relationship, which enabled them to generate a larger force. They also studied the smoothed, rectified EMG for rectus femoris, vastus lateralis, and vastus medialis. The EMG signals were normalised to those obtained during the maximum phase of an isometric contraction at 105° (75° in the system of notation of knee angle used in this project) to enable comparison between subjects. The normalised, smoothed EMG signals from the isokinetic movement were found to have a plateau between 85° and 40° of knee flexion, which was the limit of the results presented.
There have also been isokinetic studies of groups of patients with different knee conditions. Newham et al. (1989), Dvir and Halperin (1989) and Itoh et al. (1992) performed both isometric and isokinetic testings on patients with cruciate ligament deficiencies. Newham et al. found that at low velocities the ratio of the forces generated between the affected and unaffected leg was the same in both the isometric and the isokinetic test.

Dvir et al. (1990) measured both the peak torque and the average torque in patients with patellofemoral pain syndrome at different velocities and found between 30% and 40% reduction in strength in the patients. Hsieh et al. (1992) also performed isokinetic testing on female patients with patellofemoral pain at different velocities. They found that the peak torque in the patients was most abnormal at 60°/s. The lowest values were on the affected side of the unilateral group, followed by both sides of the bilateral group, and then the unaffected side of the patients. All of these were lower than the torques of the control group. 17% of the patients showed abnormal torque curves. Dvir and Halperin (1992) related quadriceps strength deficit, and torque curve irregularities to the pain provoked according to a Borg pain scale. The patients also had a bone scan, CT and X-Ray imaging. In addition to the deficit in the quadriceps moment there was also a dip in the eccentric curve which they termed a “break”. In none of these studies were the extensor muscles monitored by means of EMG.

A normal torque curve resembles a Poisson distribution curve (see diagram)
Fig. 5.1. A normal torque curve

From the angle of peak torque the moment gradually declines towards full extension, because of the decline in mechanical advantage of the muscles. The muscles also have to support the weight of the lower leg, and also the weight of the force arm below the strain gauges. This will approach maximum as the knee extends. At $90^\circ$ gravity has no effect. The weight of the lower leg can be expressed by the following equation:

$$w \cdot c \cdot \cos \alpha$$

Fig. 5.2 Diagram to show how the weight of the lower leg is distributed (w is the weight of the lower leg and c is the centre of mass, and $\alpha$ is the angle of knee flexion.)

The weight, w has been worked out by Winter (1990) to be equal to 0.061 of the total body weight, and the centre of mass to be 0.433 of the shank length. So in an individual weighing 60 kg, and a shank length of 0.39m, at $60^\circ$ of knee flexion
(approximate angle of peak torque) the moment, therefore, which needs to be produced to just lift the leg is 0.169*35.9*0.5 = 3.03Nm, which is very little.

The weight of the force arm can be worked out in the same way. The total weight of the force arm was 2.16kg, and the weight below the strain gauges was 1.08kg. The length of the force arm was 0.325m, and the length below the strain gauges was 0.16m, so the centre of mass of this is 0.08m.

Moment necessary to just lift the force arm = 0.08*10.59*0.5 = 0.42Nm. These factors are therefore negligible and can be disregarded, and even towards full extension would not be much more. This shows that the shape of the torque curve seen is very similar to actual torque values.

The aim of this experiment was to compare the EMG signals from the oblique fibres of the vasti (V.M.O. and V.L.O.) in this group of patients with those of a group of normal subjects; (a) to see whether they were inhibited at specific angles in the movement, in which case the smoothed and rectified signals would exhibit a dip at a certain point in the curve; (b) to see whether rectus femoris (R.F.) behaved in the same way; and (c) to find out whether any inhibition was reflected by a corresponding dip in the torque. Bobbert and Harlaar have demonstrated that when subjects start with a maximum isometric contraction, and then proceed to an isokinetic movement the EMG signals increase to 150% of the isometric signal. They considered that this finding confirmed that of Perrine and Egerton (1978) that subjects are unable to activate fully their muscles in isometric testing. However, when moving, a muscle needs a higher frequency for full activation (Joyce et al 1969), so that the EMG signal for full activation will not be the same.
in both situations. Hence there is a need to study the muscle action by EMG in both situations.

Although the shape of the torque curve was of importance in the experiment described here, together with the EMG’s, it was not considered of great importance to obtain values for the peak torque, because it has been shown by other workers that the values obtained are similar for isometric and low velocity isokinetic exercise, and there are other chapters in this thesis where the force exerted by the quadriceps muscles are presented (Chapter III, and Chapter IV). What was of more interest was the quadriceps/ hamstring ratio.

In this experiment a Cybex II Plus dynometer was used, with the constant velocity set at 60°/s, because endurance was not of so much interest. Also, as pointed out by Perry and Bekey (1981) the simple relationships which exist between EMG and tension in isometric testing, do not exist in dynamic situations, and muscle excitation has been shown to increase with speed (Eloranta and Komi 1980). Therefore, one is more confident that at a constant velocity at a moderate speed the same level of muscle excitation for a maximal voluntary effort is achieved. Like Hsieh et al. (1992), Conway et al. (1992) found the lowered force output in patients with patellofemoral problems greatest at slower velocities. This, they considered, was due to the fact that at higher velocities the joint is exposed for a shorter time to external resistance, leading to lower load on the patellofemoral joint, and hence reduced potential inhibition. The reflex arc causing inhibition may also be too slow to react at high velocities, and since the
inhibition of the individual portions of the quadriceps was being monitored by
EMG in this experiment, this factor was of particular importance. A specially
adapted force arm was used to improve accuracy and frequency response. Thus
any dips or breaks in the torque curve would be more easily seen. In recording
from the long fibres of the quadriceps, there is a direct relationship between the
muscle signals and the torque which is obtained, since they contribute to the
extensor force. However, in the case of V.M.O. and V.L.O., although previous
studies have shown that there is an indirect relationship with the extensor force
which is built up, we do not know what the direct relationship is, because from
the study by Lieb and Perry (1968) V.M.O. is unlikely to contribute greatly to the
extensor force. The contribution to extensor force of V.L.O. is unknown. Also,
when the knee is at different angles, the extensor muscles acting on the knee will
be at different lengths. This might alter the area of the muscle under the recording
electrodes. Before the main experiment commenced, it was therefore necessary
to find out whether this alone had any effect on the EMG signal, and led to the
following experiment.

5.1. Experiment to Investigate the Effect of Knee Angle on the Size of the
Superimposed Action Potential Obtained from the Three Recording Sites.

Introduction
The effect of knee angle on quadriceps muscle fibre conduction velocity has been
studied by Arendt-Neilson et al. (1992), who found that the muscle fibre
conduction velocity increased with decreasing muscle length. Garland et al.
studied the effect of knee angle on both the M wave and the H reflex (1994).
They found an increase in amplitude in both the M wave and the H reflex as the
knee is straightened, but the effect was much less marked in the amplitude of the M wave than the H reflex. Hashimoto et al. (1994) studied the effect in muscles which move the finger. They found that the amplitude of the compound motor action potential increased on shortening the muscle with a concomitant reduction in the duration.

Motor units in normally contracting muscle fire at different frequencies, but in this experiment motor units were synchronously activated by stimulation of the nerve supplying the quadriceps i.e. the femoral nerve. The pulse width of each stimulation was 100\,\mu s. The three sites (over V.M.O., V.L.O. and R.F.) for the recording of the action potentials each cover a fairly large area (the separation of the active electrodes was 3.5 cm), which means that there is massive superimposition of motor unit potentials. In this experiment the area under the curve of the sum of the superimposed action potentials, produced by supramaximal stimulation of the femoral nerve, which was recorded under each pair of EMG electrodes, was measured. Therefore, account was taken of both the amplitude and the duration of the action potentials obtained. The aim of the experiment was to see whether this varied at different knee angles, because if this is a major effect it will affect the EMG recording used in isokinetic testing.

**Method**

The subjects (3 men and 1 woman) who volunteered for this experiment did not have any knee symptoms. The three men (Subjects 2, 3 and 4) were from the same age range as the patients. The woman subject (Subject 1) was considerably older than the other subjects, but apart from a possible slowing of the nerve
conduction velocity, it was not anticipated that age differences would have any effect on this experiment. The electrode pairs were placed over V.M.O., V.L.O., and R.F., in the usual manner. The skin preparation, the method of placement, and the apparatus used was exactly the same as that described hitherto. The leads from the EMG amplifier were connected to the raw data output (the EMG signals were not rectified or smoothed). Each subject was seated in the chair of the Cybex in the usual way, with the knee flexed to 90°. The outputs from the three EMG channels, and one force channel, were connected to the computer via the A/D and was visible on the screen. The duration was set at 4 s, and the number of sampling points to 4000, so that as much data as possible could be recorded.

One of the flexible pads made of conductile rubber (which were used to provide electrical stimulation to the patients) was used as an indifferent electrode, gelled and strapped to the outer aspect of the hip. The cathode was a small button electrode, with the metal head covered with gauze. The button electrode was gelled and placed over the femoral nerve as it descended in the femoral triangle. The position for the electrode was located just lateral to psoas major which could be palpated. A firm pressure was applied to the electrode in a slightly upwards direction, because the hip was flexed to 90°. The width of the pulse to stimulate the nerve was 100μs and the pulse generator delivered a single shot which was repeated as necessary. The voltage was increased until the electrical stimulation resulted in a twitch of the quadriceps, which produced a recordable extensor force visible on the computer screen. Minor adjustments to the position of the
electrode to produce the biggest force were then made. The voltage of stimulation was again increased until there was no increase in the force exerted by the twitch. When this was achieved the voltage was still turned up (to ~ 70 volts) to make quite sure that the stimulation was indeed supramaximal. At supramaximal stimulation a pure M wave is obtained since the stimulus intensity necessary to evoke a Hoffmann (H) reflex is much lower and it is extinguished at higher intensities (Gottlieb and Agrawal, 1976). The superimposed action potentials which were generated at each of the three sites were saved for future analysis.

The same procedure was followed at 60° of flexion and then again at 30° and 20°. The saved data was then analyzed by a programme which estimated the area of the action potentials, so that a comparison could be made of the area at each angle. The estimation was obtained by defining the limits of the action potential, obtaining a base line and digitally integrating the rectified curve of the action potential, so that the negative part of the signal was added to the positive. The units of area obtained were then normalised to those obtained at 90° (see results for Subject 1, for example the ratio of the area of the action potential for V.M.O. at 20° was 1.042, compared to the area at 90° which was taken as 1).

**Results**

There was very little variation in the area of each successive summated action potential at the same angle. In general there was a slight increase in area with progressive extension, with a more marked effect in the rectus femoris. A good
clean signal was obtained, with the action potential biphasic in V.M.O. and V.L.O., and that from R.F. of a more complex shape.

![Graphs of V.M.O., V.L.O., and R.F. action potentials]

Fig. 5.3. The action potential which was obtained from each recording site (the first narrow spike in each case was the stimulation artifact A, the remainder is the M wave). The duration of the signal was measured from B to C.
The fact that it was not popular with the subjects, because the stimulation had to be truly supramaximal, made this a difficult experiment to perform. It was found in practice that it was better to ask the subjects to hold the active electrode, rather than have it applied. The pressure had to be firm and even and exactly in the right place to stimulate the femoral nerve, even when the lower leg jerked into extension. It would have been somewhat easier to get consistent results, if the back support of the Cybex could have been reclined, so that the hip angle was not so acute, and a slightly larger button electrode could have been placed in the groin.

The results from the female subject (Subject 1) were consistent and the units of area obtained from three repeated tests showed the mean ratio of the area of action potential at 20°, compared with that at 90°, during three successive tests to be 1.042 for V.M.O. with a SEM+/- of 0.04553; for V.L.O. the mean ratio was 1.09 with a SEM+/- of 0.01473. The mean ratio for R.F. was slightly greater (1.202) with a SEM+/- 0.03568.
Fig. 5.4. Graphs of action potential areas (normalised to 90°) against knee angle for Subject 1.

The results from the three male subjects (Subjects 2, 3 and 4) are also presented even though they are not as consistent.
Figure 5.2 (a) Graphs of action potential areas (normalised to 90°) against knee angle for Subjects 2.

Subject 2
Subject 3

Fig 5.5. (b) Graphs of action potential areas (normalised to 90°) against knee angle for Subject 3
The reason why more reliance is placed on the results for Subject 1 rather than for example Subject 4, is the force obtained from what was supposedly a supramaximal stimulation. If the stimulation is supramaximal, the same stimulation is being given at all the angles, but because of the mechanical disadvantage of the extensor muscles approaching extension, and because the lower leg weighs more (since gravity has a greater effect), the force that is produced by the stimulation is less. The force produced by Subject 4 at 30° was nearly as much as that produced at 90°, whereas the forces produced at different
angles by Subject 1 is what is normally found. It was therefore assumed that
Subject 4 did not maintain the even pressure on the femoral nerve, and that this
was the reason for the greater variation in the results for Subjects 2, 3, and 4.
Because it was not a popular experiment, and therefore not easy to recruit
subjects, it was decided not to try and obtain further results, especially as the
repeatability in the case of Subject 1 was good.

![Graph showing force production at different angles](image)

**Fig. 5.6. The force produced by supramaximal stimulation at different angles**

The timing of the action potential was found for one of the subjects and it was
seen that there is indeed a slight increase in the velocity as the knee becomes
straighter.

**Table 5.1. The time taken for the superimposed action potentials at different
angles for one subject (measured as shown in Fig 5.3).**

<table>
<thead>
<tr>
<th>Knee angle in degrees</th>
<th>Duration of action potential in ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>90</td>
<td>31.5</td>
</tr>
<tr>
<td>60</td>
<td>29</td>
</tr>
<tr>
<td>30</td>
<td>26</td>
</tr>
<tr>
<td>20</td>
<td>25</td>
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</table>
Discussion

It might be that the angle had a more pronounced effect on the size of action potential from R.F. than the oblique portions of the vasti because, although it is a bipennate muscle, the fibres of this muscle are straighter compared with the vertical and the overall shortening might be greater, because R.F. undergoes a greater excursion. The more complex shape might be due to the complexity of the muscle architecture and the spread of the signal.

Overall it could be concluded that the change in shape of the muscles due to different knee positions does not have a great effect on the EMG recordings in isokinetic movements.

5.2. Experiment to Test whether there is Inhibition of V.M.O. and V.L.O. during Isokinetic Testing at 60°/s in Patients with Patellofemoral Problems

Method

Forty nine patients, who were from the selected group, were asked to undergo isokinetic testing. There was also a group of ten normal subjects, who had not had any knee symptoms. They were aged 20-33, and had a similar range of accustomed activity as the patient group.

Each subject had the same skin preparation as for the previous experiments, and the three recording electrodes were placed in the same sites as previously (i.e. over V.M.O., V.L.O. and R.F.), and fixed as before. The EMG equipment used was also as before. The subject was then seated in the Cybex chair as before, with chest and lap straps tightened to stabilise the trunk and pelvis. The test
speed was set at 60°/sec. The subject was then instructed to move the leg through the range of movement until it was fully extended. The stop was then placed in position at this angle. This was to ensure that some of the patients who showed hyperextension of the knee would not strain the knee at this angle. Equally they would not be afraid of hyperextending the knee and avoid applying maximum torque in the last 30° of the movement. Each subject was instructed to push against the resistance of the machine as hard as possible, throughout the whole range of movement, applying the torque somewhat faster than the machine, so that he/she would be slowed down to a uniform speed. When each subject reached the stop he/she was then instructed to pull the knee back as hard as possible, so that the torque produced by the hamstrings was also tested, until the knee was back at 90° of flexion. Each was given the chance to practise this several times until the technique had been perfected. This was done because it is not as easy to perform this manoeuvre as a maximal isometric contraction. Both legs of the patient group were tested, but only the right legs of the normal subjects. However, only the results of the most severely affected leg, or in the case of the patients with unilateral symptoms, the affected leg, are presented here.

The duration of test was then set at 8 s so that each subject was able to complete three extensions and two of the three flexions. Three of the patients had by this time started to complain of the pain induced, and it was felt that it was unethical to provoke too much pain, and so they were asked to complete just one movement. Considerable patellofemoral contact forces are built up in this experiment.
EMG data was collected from 3 of the channels and the torque data from the fourth. This was saved on disc for further analysis.

**Mathematical analysis**

Each subject completed three extension movements. The cycle where the torque produced was the maximum, was chosen for further analysis. Because the forces built up are at a maximum, it was anticipated that any inhibition of the muscles would be most pronounced. This was usually the second cycle. Each of the three EMG traces was normalised to maximum, and plotted against the angle (from 0-90°). The torque was normalised in the same way to the peak torque, and plotted against the angle.

In order to quantify any evidence of inhibition it was necessary first to smooth the EMG signals. This was done as follows:-

A probability curve was fitted to the four sets of data to simulate the main response, and another inverted probability curve to simulate any dip in the data where there was a diminution of the signal as the subject passed over the part of the curve corresponding to specific angles. The parameters of the two curves were manipulated until the best fit was obtained between the plotted data and the two curves (see Fig.5.8.). The minimum correlation between the fitted and the observed data which was aimed at was $r^2 = 0.99$, and was obtained in most cases (a minimum of 0.98 had to be accepted in some cases). It was then felt that the curves adequately described the data.
Dvir et al. (1990) in their study of isokinetic testing of patients with patellofemoral pain used the term “break” when discussing the dip in the torque curve. However, in this study the term “dip” is used for both the torque curves, and those of the EMG data. While the term “break” might be appropriate for the torque curve, in the case of the EMG data where there is a diminution rather than a cessation of the data the term “dip” is thought to be better.

The mathematical equation of the curves could thus be described as follows, where $x$ denotes the angle ($=50 - 200^\circ$), and $A =$ amplitude, and $w =$ width. The mathematical equation for a probability curve is $A \cdot e^{-\left(\frac{x - \text{midpoint}}{\text{width}}\right)^2}$.

This describes both the main curve and the dip.

The mathematical analysis of a hypothetical result will be shown in full as follows:

A main = 1 (normalised)

$w_{\text{main}} = 30^\circ$

$\text{Main}(x) := A_{\text{main}} \cdot e^{-\left(\frac{x - \text{Midmain}}{\text{Widthmain}}\right)^2}$

A dip = 0.4

$w_{\text{dip}} = 8^\circ$

$\text{Dip}(x) := A_{\text{dip}} \cdot e^{-\left(\frac{x - \text{Dipmid}}{\text{Widthdip}}\right)^2}$

Both $(x) = \text{Main}(x) - \text{Dip}(x)$
Fig. 5.7. Graph which would be obtained from hypothetical data

It can be seen from this that “both max” which is shown in Table 5.2. denotes the maximum amplitude of the EMG activity, compared to the activity it would have had without the dip.
Fig 5.8. The EMG data obtained from VMO in a normal subject.

Fig 5.9. The EMG data obtained from VMO in a patient with patellofemoral problems, showing the centre of the dip in mid range of movement.
Results

Table 5.2. The dips in the curves for isokinetic torque and EMGs for both normal subjects and patients

<table>
<thead>
<tr>
<th>Force</th>
<th>Patients</th>
<th>Normal subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>both max dip max dip pos width</td>
<td>both max dip max dip pos width</td>
</tr>
<tr>
<td>Mean</td>
<td>0.9902 0.0337 148.85° 13.83</td>
<td>0.9993 0.0414 148.61° 16.83</td>
</tr>
<tr>
<td>n</td>
<td>49 49 49 49</td>
<td>10 10 10 10</td>
</tr>
<tr>
<td>SD</td>
<td>0.0770 0.0430 21.61 8.574</td>
<td>0.0119 0.0428 12.28 7.268</td>
</tr>
<tr>
<td>SEM</td>
<td>0.0110 0.0061 3.087 1.225</td>
<td>0.0038 0.0135 3.885 2.298</td>
</tr>
</tbody>
</table>

mean difference (patients:normals) -0.0077
SEM 0.0148
\( t = -0.5192 \) (NS)

<table>
<thead>
<tr>
<th>VMO</th>
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<tr>
<td>Patients</td>
</tr>
<tr>
<td>--------</td>
</tr>
<tr>
<td>both max dip max dip pos width</td>
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<tr>
<td>Mean</td>
</tr>
<tr>
<td>n</td>
</tr>
<tr>
<td>SD</td>
</tr>
<tr>
<td>SEM</td>
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mean difference (patients:normals) -0.09002
SEM 0.01947
\( t = 4.625 \) p<0.001 Confidence 99.9%

<table>
<thead>
<tr>
<th>VLO</th>
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<tr>
<td>Patients</td>
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<td>--------</td>
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<tr>
<td>both max dip max dip pos width</td>
</tr>
<tr>
<td>Mean</td>
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<tr>
<td>n</td>
</tr>
<tr>
<td>SD</td>
</tr>
<tr>
<td>SEM</td>
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</table>

mean difference (patients:normals) 0.05307
SEM 0.02619
\( t = 2.0623 \) p< 0.05 Confidence 95%

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<thead>
<tr>
<th>RF</th>
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<tr>
<td>Patients</td>
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<tr>
<td>both max dip max dip pos width</td>
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<tr>
<td>Mean</td>
</tr>
<tr>
<td>n</td>
</tr>
<tr>
<td>SD</td>
</tr>
<tr>
<td>SEM</td>
</tr>
</tbody>
</table>

mean difference(patients:normals) 0.0515
SEM 0.0247
\( t = 2.083 \) p<0.05 Confidence 95%

The results showed that there was very little dip in the torque curve of the patients or in that of the normal subjects, and the difference between the two groups was not significant. There was only an abnormal shaped curve in 8
patients. There may have been a greater proportion in curves obtained from the eccentric mode, as was found by Dvir and Halperin (1992).

The results of the EMG data were:-

V.M.O.

The dip in the patients' data was the largest of the three sets of EMG, and the difference between the patient and the normal group was the greatest. A comparison of the two groups yielded a mean difference of 0.09002, with a SEM of 0.01947. The t value was 4.625, which was highly significant (p<0.001), so that the confidence was 99.9%, that it was not due to chance. This means that the amplitude of the EMG from V.M.O. was markedly reduced at a particular part of the curve, as the isokinetic movement was between certain angles. There was not a corresponding dip in the torque curve.

V.L.O.

The dip in the patients data was less than the dip in V.M.O. The mean difference between the two groups was 0.05307 with an SEM of 0.02619, which gave a t value of 2.02627, which was also significant (p<0.025), or 97.5% confidence.

R.F.

The mean for the maximum dip for the patients was 0.105 with SEM of 0.0124; for the controls it was 0.05353 with a SEM of 0.02139. The mean difference was 0.0515 with a SEM of 0.02472, which yielded a t value of 2.083. (p<0.05)

A comparison of the differences of the magnitude of the dips between patients and normal subjects could be summarised thus:-
Table 5.3. Differences in the dips in the EMGs for normal subjects and patients

<table>
<thead>
<tr>
<th></th>
<th>normals</th>
<th>patients</th>
<th>difference</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>VMO</td>
<td>0.06435</td>
<td>0.1544</td>
<td>-0.09002</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>VLO</td>
<td>0.0827</td>
<td>0.1358</td>
<td>-0.05307</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>RF</td>
<td>0.05353</td>
<td>0.1050</td>
<td>-0.0515</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>FORCE</td>
<td>0.0414</td>
<td>0.0337</td>
<td>-0.0077</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

Fig. 5.10. A histogram to show the differences in the size of the dips between the patient and the normal group.

A frequently observed occurrence in the patient group which was not seen in the normal group was quadriceps activity during flexion. This is presented in 5.4.

**Discussion**

The results showed that the dip in the EMG at specific angles in the range of isokinetic movement which the patients undertook, was most marked over V.M.O. The dip was considerably smaller in both V.L.O. and R.F. The fact that
it was only apparent at certain angles, points to the mechanism involved being that due to inhibition. As has already been stated in the chapter on overall quadriceps inhibition, it has not been elucidated where the receptors are, nor the precise nature of the mechanism of the inhibition. What is interesting in this experiment is the fact that it is most marked in V.M.O. action, which may contribute to an alteration in the mechanics of the joint. While it is unlikely to be the root cause of the condition, it might contribute to a progressive spiral of poor function.

Spencer et al. (1984) studied the effect of effusion on the H reflexes obtained from vastus medialis, vastus lateralis, and rectus femoris. The study of H reflexes (Hoffmann reflexes) gives information of the excitability of the motoneuron pool under study. They injected saline into the knee joint space of ten subjects with normal knees to produce a simulated effusion. The reflex inhibitory response in vastus medialis was greater than that in the other two muscles, and it was also noticeable that the threshold for reflex inhibition was less in this muscle. When lidocaine was injected afterwards into one of the knees there was no reduction in the H reflex, so they concluded that quadriceps inhibition is mediated through afferent activity from intracapsular receptors. Patellofemoral problems can be secondary to other pathological conditions affecting the knee joint, which might cause effusion. If, therefore, there is a greater degree of inhibition of V.M.O. than other parts of the quadriceps, they postulated that this might cause altered mechanics in other knee conditions and therefore secondary patellofemoral problems.
5.3 Quadriceps/Hamstring Ratio

From the extensor and flexor torques obtained, the quadriceps/hamstring ratio was estimated. Hamstring/quadriceps balance is thought to be clinically important in knee conditions, and so it was decided that it should be considered in this group of patients. Another consideration was that there was a statistically significant hamstring tightness in the male, but not the female group (see Chapter VI). There have been various hypotheses as to why tight hamstrings might contribute to patellofemoral problems, for example that of Jacobson and Flandry (1989) who thought that tight hamstrings cause the quadriceps to work harder and so increase the patellofemoral joint reaction force (P.F.J.R.F.); or that the attachment from the hamstrings to the patella could alter patellar position (Terry 1984); or that differences in the tightness of the medial and lateral hamstrings alters alignment. While all these factors could contribute to pathology is some cases, and certainly P.F.J.R.F. is largely dependent on quadriceps force, they cannot be a major cause of patellofemoral pain because, in this group of patients, the ratio of female to male patients was about 3:2 and there was not significant tightness in the female patients. The mechanism behind this tightness is not known at all. It could be that the generalised quadriceps weakness which has been noted in these patients could cause an abnormal quadriceps/hamstring ratio, and this might be a cause of hamstring tightness.

Hamstring strength has been studied, and in some studies there have been estimations of the quadriceps/hamstring ratio. Kroll et al. (1988) studied a group of patients with polymyositis by isometric testing. The quadriceps were
tested at 60° of knee flexion, and the hamstrings at 30°. Their results showed the control group of normal subjects to have a quadriceps/hamstring ratio of 1.93; their patient group had a ratio of 1.60. Dvir and Halperin (1989) studied eccentric and concentric ratios in ACL deficient knees. One of the parameters which they presented was Hc/Qc, and the ratio for the sound knee was 0.52, compared with the ratio for the deficient knee of 0.53. However they did find a significant difference between the He/Qc of the deficient knee and the sound knee. The study by Dvir and Halperin was performed at 30°/s, whereas in this study the velocity was 60°/s. Using the same notation as the study by Kroll et al. (1988) viz Qc/Hc their ratio for the sound knee was 1.92, which was very similar.

It was not possible to test the He/Qc ratios in this experiment because it is beyond the scope of a Cybex Plus II testing device.

Results

Table 5.4. The quadriceps/hamstring ratio for both normal subjects and patients

<table>
<thead>
<tr>
<th>SEX</th>
<th>GROUP</th>
<th>Number</th>
<th>Mean ratio</th>
<th>SD</th>
<th>SEM +/-</th>
<th>t of diff</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MEN</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Patients</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>16</td>
<td>1.976</td>
<td>0.534</td>
<td>0.138</td>
<td>0.479</td>
</tr>
<tr>
<td></td>
<td>Controls</td>
<td></td>
<td>4</td>
<td>1.875</td>
<td>0.423</td>
<td>0.211</td>
<td>1.111</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>8</td>
<td>1.896</td>
<td>0.294</td>
<td>0.104</td>
<td>1.111</td>
</tr>
</tbody>
</table>

The results were divided by sex because the results of the hamstring tightness was different in men and women. In the normal subjects the ratio of the quadriceps torque to the hamstring torque of both sexes were very similar (1.875 for the men, with a standard deviation of 0.4227, as against 1.896 with a standard
deviation of 0.2941 for the women). These results were very similar to the results of the control group in the study by Kroll et al. (1988) and Dvir and Halperin (1989). The mean ratio for the men patients was slightly higher than the control group (1.976 with a standard deviation of 0.534), however this change was not significant (p>0.05). The mean ratio for the women patients was 1.731, with a standard deviation of 0.6009, which was slightly lower than that of the controls. A Student's t test of the difference between women patients and controls gave a t of 1.111 which was again not significant, p>0.05.

Discussion

The results showed no significant change in the patients' quadriceps/hamstring ratio compared with those of the normal subjects, although there was a significant loss of quadriceps strength isometrically. The results here reported were similar to those of Dvir and Halperin (1989) who did not find any change in the Hc/Qc of ACL deficient knees. Had it been possible to test the He/Qc a change in the ratio might have been apparent.

Imbalance of an agonist/antagonist group, which is in this case quadriceps/hamstrings, might cause shortness of the dominant group. However, these results do not support this. The men who did have shortness of the hamstrings had no significant change in the ratio; the women who did not have shortness also had no significant change in the ratio. This means that the hypothesis that it is the dominance of the hamstrings which causes the shortness has not been proved. A more likely explanation is that the hamstring tightness is
a compensatory mechanism. Solomonov et al (1987) demonstrated the presence of a secondary reflex arc, either from mechanoreceptors in the hamstring muscles or joint capsule, which provides hamstring activation upon knee instability. Perhaps hamstring shortness is due to a resetting of the stretch reflex which governs agonist/antagonist activity.

5.4. Quadriceps Activity during Flexion of the Knee

This is an example of co-contraction of the antagonists during agonist activity which is effecting joint movement. The agonists in this case are the hamstrings bringing about knee flexion. The purpose of co-contraction is thought to be to stabilize the joint, and a certain amount of co-contraction is entirely normal (Rothwell 1994).

This was investigated to see whether there was any difference in the duration of the co-contraction of the quadriceps in the patient group compared with the normal subjects, during an activity of the same velocity (that is 60°/s). Therefore, the EMG recordings from 3 parts of quadriceps (V.M.O., V.L.O. and R.F.) were analysed to see the length of time that they were switched on during the flexor phase, starting from the time of application of flexor force. Since the isokinetic machine was set at 60°/s, the subjects took 1.5s to produce 90° of movement. This was verified by measuring the actual time taken. The number of degrees of flexion during which the 3 parts of quadriceps were activated could thus be calculated. The recordings of 46 patients was analysed in this way.
Results

Table 5.5. Duration of co-contraction of quadriceps with hamstrings during flexion of the knee: patients compared with normal subjects.

<table>
<thead>
<tr>
<th></th>
<th>Patients</th>
<th>Normal subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>46</td>
<td>10</td>
</tr>
<tr>
<td>Range of joint angle with co-cont</td>
<td>17°</td>
<td>7°</td>
</tr>
<tr>
<td>sd</td>
<td>8.10</td>
<td>3.92</td>
</tr>
<tr>
<td>sem+/−</td>
<td>1.19</td>
<td>1.24</td>
</tr>
<tr>
<td>Student’s t pt/norm</td>
<td>5.988</td>
<td></td>
</tr>
<tr>
<td>p</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Number with active V.M.O. longer than R.F.</td>
<td>20</td>
<td>1</td>
</tr>
</tbody>
</table>

The angle of peak flexor torque was seen to be ~30° of knee flexion. The mean angle at which the quadriceps ceased to be active in the group of normal subjects was 7°, and in only one subject was V.M.O. active longer than V.L.O. and R.F. (10%). This was in contrast to the group of patients where the mean angle of flexion at which the quadriceps ceased to be active was 17°. This was at an angle where the peak flexor torque was approaching maximum (in some patients the quadriceps remained active beyond the angle of peak flexor torque). There was, therefore, a highly significant difference between the duration of co-contraction in the patients compared with the normal subjects (p<0.001). In 20 patients the duration of activation of either V.M.O. alone or with V.L.O., was longer than that of R.F. (43%). In 11 patients the longer duration of activation was in V.M.O. alone and not seen in the other two.
Fig. 5.11. To show the extent of co-contraction with hamstring action in a patient

**Discussion**

The extra co-contraction of quadriceps with flexion of the knee, could cause overload of these muscles which might lead to slowing of these muscles. Pette (1984), when giving isometric twitch contractions to tibialis anterior muscle, where one side had been chronically stimulated to become slow, found that the half relaxation time was longer in the slow muscle. He suggested that this was due in part, to differences in the myosin structure and to Ca$^{++}$ outflow. Kennedy et al. (1988) demonstrated a transformation of overloaded chicken muscle; one of the myosin chains was replaced by an embryonic form. Pette and Vrbova (1985) quoted studies in human muscle which show that when muscles are overloaded, fast (Type II) muscle fibres are transformed into slow fibres (Type I). Overload, they defined, not as an increased force but the length of time that they were
active. The transformation of the muscle fibres is an energy saving device, since the energy cost of activation of slow muscle is lower than fast muscle.

This has implications for the muscles in this situation, and could well account for the high proportion of Type II C fibres (evidence of muscle transformation) found by Floyd et al. (1987) in the V.L. of patients with recurrent dislocation of the patella. If, as in the patients in this experiment, the muscles are activated not only when the quadriceps are effecting extension, but during flexor activity when in the normal subject they are silent, then they are likely to suffer from overload. This has particularly important implications for V.M.O. It has been shown in Chapter VI that the way in which it is activated points to a predominantly phasic muscle with many fast fibres. If, then in many of these patients it is transformed into a tonic muscle due to overload, it cannot then react to the changes in activation necessary in movements undertaken at high velocity. It was interesting that, when talking to the only normal subject who showed a prolonged activation of V.M.O. compared with the recordings from the other two sites (he also had the longest co-contraction time), it was discovered that he was an oarsman. He said that he made that choice of sport because he did not think that he would be any good at sports involving high velocity movements.
CHAPTER VI

CHANGES IN VMO ACTIVATION AT DIFFERENT RATES OF APPLICATION OF FORCE AND DIFFERENCES IN KNEE POSITION

Introduction

Much of the work towards an increased understanding of how biomechanical changes affect the functioning of the patellofemoral joint, has either been done using geometrical models, or in vitro with cadavers. The oblique portions of the vasti (V.M.O. and V.L.O.) must adapt to changes in position of the patellofemoral joint caused by rotatory movements of the knee but this mechanism has rarely been studied. Although in vivo muscle studies have been made (e.g. Moller et al. 1986, Grabiner et al. 1992) using EMG recording from V.M.O., they have been carried out only in neutral rotation. The only study of vastus medialis and vastus lateralis action to consider changes in rotation which has come to light after a thorough search is one by Schmitt and Mittelmeier (1978). They tested a subject in standing on one leg, with the knee bent to 30° and the tibia rotated first externally and then internally. They showed that when the tibia was externally rotated, the action of V.M. was greater than V.L.; internal rotation caused the opposite to occur. They made no distinction between the long and the oblique fibres. Gray’s Anatomy 36th Edition (p 598) states that electromyographical studies indicate that the vasti do not act synchronously or equally in external rotation of the thigh, but the editors commented that confirmed details were not available. They did not say to what study they were referring.
Although abnormal Q angles have been noted by many authors as a predisposing factor for patellofemoral pain, it is not understood how in the normal subject the muscles compensate for normal changes in Q angle. In the seated position, the Q angle, which is the angle that the quadriceps make with respect to the patella tendon, is at a maximum when the knee is extended because of the terminal external rotation of the tibia, and approaching zero at 90° of knee flexion. When there are changes in rotation at 90° of knee flexion, there are functional changes in Q angle, because the angle of pull of the tendon has changed. The extent of external rotation which is possible is 40°, and that of internal rotation is 30° (Kapandji 1971). The force vectors are then thought to be changed in the way as is shown in the diagram.

a) Neutral rotation

\[ F_R = \text{resultant quad force} \]
\[ F_{T1}, F_{T2} = \text{forces at tibia = st line} \]
\[ \text{Lat force = zero} \]

b) External rotation

\[ F_R = \text{resultant quad force} \]
\[ F_{T2} \text{ not straight line} \]
\[ \text{Lat force generated R_L} \]

Fig. 6.1. Diagram of how vectors change with change in rotation of the knee

160
Muller (1983) thought that it was a pity that our understanding of rotational physiology of the knee is so poor, since in so many sports much of the muscle work is undertaken in either external or internal rotation. The examples which he gives are footballers, hurdlers, and weight lifters. He considered that footballers and hurdlers would need greater vastus medialis oblique activity than weight lifters because their sport involves external rotation of the knee. Many more examples of activities which involve rotation of both the hip and the knee could be found; all team sports or racquet sports involve cutting where there is considerable rotation. Swimming is often advised when there are knee problems because it is non weight-bearing, but in breast stroke swimming the knee is rotated.

The hypothesis put forward by Muller (1983) is that in internal rotation of the tibia on the femur, the medial facet of the patella is pushed against the femur, and that V.M.O. then works less to compensate for this. He thought that there would, at the same time be an increase in activation in V.L. to pull the medial border away. In external rotation he thought that the opposite should happen. Van Kampen and Huiskes (1990) in their study with cadavers to find out what movements of the patella occurred during movements of the knee, showed that the patella can move medially and laterally (patella shift); can rotate around a vertical axis (patella tilt); and can also rotate around a transverse axis. They considered that tilt and shift were coupled and occurred mostly at small angles of flexion. At 90° of flexion, if there is tibial rotation, they said that the patella movement which mostly occurs, is rotation about a transverse axis (frontal
plane), so that external tibial rotation causes lateral patellar rotation, due to the laterality of the patellar ligament. If Van Kampen and Huiskes (1990) are right then VLO and VMO would not necessarily have to work antagonistically to one another. One of the reasons why the following experiments were undertaken, was to test Muller’s hypothesis, or alternatively, to see whether the patella movements detected by Van Kampen and Huiskes, were supported by different changes in the muscle activation. The muscle control of these small movements of the patella is not known.

Fig. 6.2. Muller’s hypothesis of the effects of external and internal rotation of the tibia on patellar position, and the necessary compensation in V.M.O. and V.L. action (from Muller 1983).
There also seemed to be an important clinical reason for increasing understanding of this question, as it quickly became apparent in this study of patients with patellofemoral problems, that there was a prevalence of biomechanical changes in the whole lower limb, due to altered rotation. These in their turn led to changes in the gait pattern; some of these were structural, but many patients also showed a marked disinclination to risk any external rotation of the hip. The knee was firmly held in. Therefore, it was wished to see what effects, in the normal subject, might be caused by changes in rotation of the hip. The patients had been forced, in many cases, to give up the sports which they enjoyed. Many patients said that they could swim as long as they did not attempt breast stroke, which involves much rotation of the knee. This led the author to try and find out the reason for these observations. It was not considered ethical to subject patients to changes in position which might increase the pain, or even cause damage, so these series of experiments were done on normal subjects.
In another chapter (Chapter IV) evidence is presented that the activation of V.M.O. and V.L.O. is different from that of the rest of the quadriceps, however, there is a body of opinion which questions whether there is selective activation of V.M.O. For example Grabiner et al. (1994) considers that the evidence in favour of its occurrence is not compelling. A further aim of these experiments, therefore, was to understand more fully the activation, not only of the oblique fibres, but of the long fibres as well. It is generally considered that there is an orderly recruitment of motor units, with the slow twitch fibres being recruited first, and at greater force levels the additional recruitment of fast fibres (Henneman and Mendell 1981). Bilodeau et al. (1991) stated that the optimum condition to observe this is during a slow steady increase of force (this is a ramped contraction). On the other hand there have been shown to be differences of activation of a muscle, according to the type of contraction investigated. The author, therefore, wished to compare the differences in activation of the quadriceps muscles concerned, at slow speeds with fast speeds. Grabiner et al. (1992) found that, compared with normal subjects there was a decreased activation of V.M.O. and vastus lateralis in patients with anterior knee pain, during isometric contractions where there was a rapid application of force, but not when it was applied slowly. It is for this reason that in the following experiments, where muscle contraction adapting to changes in knee position was being studied (Experiment 6.2 and 6.3.), each subject did a slow ramped application of force from rest to maximum, and also force applied rapidly in
increasing steps. However when the recruitment strategy of the muscles was being investigated (Experiment 6.1.), then a slow ramped contraction was done.

It has been shown that different muscles give different shaped curves when their activation is plotted against force (Solomonov et al. 1986). While Henneman's size principle remains a controversial issue, in that the mechanism by which the small slow muscle units are recruited first is not clear, within specified limits it still holds good (Enoka and Stuart, 1985). At small forces, as already stated, the slow fibres are recruited and with increasing force the faster fibres are recruited (Bigland and Lippold 1954), so that the median firing frequency is increased. This factor would cause a more rapid increase in EMG amplitude. Thus a muscle with many slow (Type I) fibres will have a more linear Force vs EMG graph, than a muscle which has a large proportion of fast (Type II) fibres. The aim in this part of the experiment was to see whether there are differences of Force/EMG relationship between different portions of the quadriceps. These factors give us clues about the control strategy of the individual muscle.

However, it has been shown that where the method of recording EMG is by surface electrodes, the subcutaneous adipose tissue which attenuates the signal before it is collected at the skin - electrode interface can have the effect of acting as a low pass filter, so that the fast fibres (Type II) with a higher frequency are preferentially blocked, causing a non-linear relationship to appear more linear (Lindstrom and Peterson, 1983). This means that differences of linearity could be due to different distribution of fat over the recording sites.
When force is applied rapidly it has been shown that the fast fibres are recruited at a lower force than in a slower contraction. Gillies et al. (1972) found that when force is applied rapidly, the frequency of the firing rate was 70 Hz at 40% of the maximum force, but when the force was applied slowly the firing rate was only 20-30Hz. Citterio and Agostini (1984) found differences in quadriceps activation at different cycling rates. In the experiment which is being described here, the Force/EMG relationship is studied with the two different ways of applying the force (Experiments 6.2. and 6.3.). The first was where the isometric force was gradually and smoothly increased to maximum, taking 8 s to complete (slow ramped contraction) and secondly where the force was applied as rapidly as possible. The second regime was tried with several different magnitudes of force.

Lastly, changes in rotation might bring about changes in the activation of V.M.O. and V.L.O., but not in the long fibres, so it was decided to investigate this. In this way further evidence was sought that the control strategy of V.M.O. is different from V.M.L. This evidence would help in understanding the differences in quadriceps action which have noticed in the patients compared with the normal subjects.

These series of experiments have been split into several sections:-
6.1. Experiment to See whether there is a Difference of Activation of the Oblique Fibres of the Vasti Compared with the Long Fibres, at Slow Activation Rates.

6.2. Experiment to Investigate any Differences in V.M.O. and V.L.O. Action in Changes of Rotation of the Hip with the Knee Flexed to 20°: and with Changes of Rotation of the Knee, Flexed to 90°.

6.3. Experiment to Investigate whether Changes in the Activation of V.M.O. in External Rotation of the Hip, at 20° of Flexion of the Knee are Parallelled by Changes in V.M.L.

Experiment 6.1.

Experiment to See whether there is a Difference of Activation of the Oblique Fibres of the Vasti Compared with the Long Fibres, at Slow Activation Rates.

Introduction

Chaffin et al. (1980) demonstrated that the Force/EMG amplitude curve could be well fitted by two straight lines. In the first 40-60% of the curve where the recruitment was of mainly slow fibres, the gradient was slight, and later when the recruitment was mainly of the fast fibres the gradient was much steeper. If, therefore, the control strategy of one muscle is different from another, and its proportion of fast fibres is different, the ratio of its two slopes would be different.

It was decided to use this hypothesis to test differences between V.M.O. and V.M.L., and between V.L.O. and V.L.L., as evidence of differences of control.
strategy, and therefore function, between the oblique and long fibres of the two vasti.

**Method**

4 subjects (2 men and 2 women) were asked to participate in this experiment, the criteria for their selection was as in all the experiments. Before the main experiment skinfold thickness was measured to test that variation over the sites did not occur and cause the amplitude/force curve from the long fibres to appear more linear than the activation of the fibres would cause it to be, due to the preferential blocking of the signal from the fast fibres due to this effect (as reported by Bilodeau et al. 1992). They tested the median frequency/force curve in anconeus and found it to be markedly different from triceps brachii. In anconeus, where there was a very low skinfold thickness, the increase of the median frequency with an increase in force was much greater. This suggests that the amplitude/force curve would be similarly affected. Moreover, there was a significant gender effect in both the median frequency/force curve and the skin fold thickness. When comparing triceps brachii and anconeus, the skinfold thickness was 4.1 times greater in triceps brachii than anconeus in the group of men, and 5.2 in the group of women.

**6.1.1. Measurement of skinfold thickness**

**Method**

Before each of the participants was tested, they each had their skinfold thickness measured over the four sites, that is the skin over V.M.O., V.M.L., V.L.O. and V.L.L.; the skin over R.F. was also tested at the same time to ensure that none of the results in the other experiments was biased by this effect. The subject was
comfortably seated on a couch with his/her legs stretched out and supported in slight flexion over a pillow so that the quadriceps muscles were as relaxed as possible. Skinfold was picked up between the operator's index finger and thumb and the calipers applied. Each measurement was done three times to test repeatability. Two of the subjects were expected to have higher skin-fold thicknesses from their weight; the result of one has been presented, the other could not be tested because she had much subcutaneous adipose tissue over the thighs, allied to strong musculature, and it was impossible to pick up the skinfold.

Table 6.1. Results of skinfold thickness test

<table>
<thead>
<tr>
<th>Subject 1 (male) Skin over,</th>
<th>VMO</th>
<th>VML</th>
<th>VLO</th>
<th>VLL</th>
<th>RF</th>
</tr>
</thead>
<tbody>
<tr>
<td>thickness in mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.2</td>
<td>10.2</td>
<td>5.8</td>
<td>6.6</td>
<td>9.2</td>
<td></td>
</tr>
<tr>
<td>7.8</td>
<td>10</td>
<td>6</td>
<td>6.8</td>
<td>8.8</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>10</td>
<td>5.8</td>
<td>6.6</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>mean in mm</td>
<td>8</td>
<td>10.1</td>
<td>5.9</td>
<td>6.7</td>
<td>9</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Subject 2 (mean)</th>
<th>VMO</th>
<th>VML</th>
<th>VLO</th>
<th>VLL</th>
<th>RF</th>
</tr>
</thead>
<tbody>
<tr>
<td>15.3</td>
<td>18</td>
<td>16</td>
<td>16.2</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>Subject 3 (mean)</td>
<td>11.9</td>
<td>12.1</td>
<td>11.1</td>
<td>16.8</td>
<td>16.1</td>
</tr>
<tr>
<td>mean of group</td>
<td>11.7</td>
<td>13.4</td>
<td>11</td>
<td>13.2</td>
<td>13.7</td>
</tr>
</tbody>
</table>

The mean ratio of the skinfold thickness over V.M.L. compared with V.M.O. was 1.15. That over V.L.L compared with V.L.O was 1.22. This result was very much less than the ratio of skinfold thickness over triceps brachii compared with anconeus (4.1), reported by Bilodeau et al. (1992). Also in one of subjects (Subject 3) there was very little difference between the skinfold thickness over V.M.L compared with V.M.O (1.02). No differences were ascertainable in the results of the main experiment in this case, compared with the other subjects. One would also expect effects of this type on the resultant EMG signal to be
greater with a larger interelectrode distance than that used here. Therefore, it can be concluded that any difference in attenuation of the signal over the long fibres must be minimal.

6.1.2. Method

Each of the 4 subjects were seated in the Cybex chair with the specially fitted force arm, with an integral strain gauge connected to an amplifier. The same inclusion criteria as used previously, governed the choice of each subject i.e. that their age range was the same as the patients, their accustomed activity was similar, and they were free from any knee symptoms. A pair of electrodes was placed over the fibres of V.M.O. in the direction of the line of the fibres, after skin preparation. Another pair of electrodes was placed over the fibres of V.L.O. also in the direction of the fibres. A third pair of electrodes was placed over V.M.L. (the direction of the fibres has been discussed in Chapter II). Care had to be taken with this placement, to avoid both sartorius and rectus femoris. Sartorius is a thin strap-like muscle which crosses the thigh from the lateral side, crossing the knee medially to be inserted on the medial aspect of the tibia. The placement of the electrode pair had to be distal to this muscle, but medial to rectus femoris. To ensure that this placement was correct the subject was asked to flex the hip, since both these muscles are hip flexors, with sartorius acting particularly in external rotation. If there was no signal when this was done, the contribution from cross talk could not be great. In this experiment there were 4 channels (3 to collect the EMG signals and 1 to record the force). The raw signal from the muscles was recorded first during a sub-maximal contraction of the quadriceps to ensure that a noise free signal was obtained from each channel.
The subjects were asked to make a maximum voluntary contraction (MVC), with the knee flexed to 90°, and the amplitude of the force obtained was marked on the screen. They were then asked to make one slow linear contraction in 8 seconds from zero to maximum, trying to reach this mark. This type of contraction is commonly called a “ramped” contraction (Bilodeau et al. 1991). The same EMG equipment was used as in all the muscle experiments, and the amplified, rectified, and smoothed signal stored for future analysis.

When this was completed, the third pair of electrodes was then removed from its placement over VML and placed over the long fibres of vastus lateralis (VLL); those over VMO and VLO were left in situ and the test was repeated.

**Analysis of the results**

A programme called *Viewdac* was utilised to analyse the results. A specific programme was written to fit the data to 2 lines. From this the slope of the first line was obtained, and the slope and also the intercept of the second line.
Fig. 6.4. To show how the data fitted the two slopes.

The data fitted the two slopes excellently with three of the results obtained, but only moderately in one case, where the subject did not make a smooth and steady increase in force. For each of the 4 subjects, Slope 1 was then subtracted from Slope 2 for each set of data for the two muscles, and the result obtained for V.M.L. was subtracted from the result for V.M.O., and from this the mean of the difference was found. The same process was repeated to compare V.L.O. with V.L.L. A Student's t test was applied to the mean difference in slopes obtained from the values of V.M.O. and V.M.L., and V.L.O. and V.L.L.; p<0.05 was set as a level of significance.

Results

\[(\text{Slope}_2 - \text{Slope}_1)_{\text{VMO}} - (\text{Slope}_2 - \text{Slope}_1)_{\text{VML}} = 0.2283 \text{ (mean) SEM} +/-.0646.\]

\[n_1 = 4 \quad n_2 = 4 \quad n_3 = 4 \quad n_4 = 4\]
A paired t test was performed between the two samples (VMO/VML) each of which contained 4 measurements. A $t$ of 3.532 was obtained ($p<0.05$).

The same test applied to the comparison of V.L.O. with V.L.L. gave 0.106 which was not significantly different from zero.

**Discussion**

There were differences in linearity of the Force/EMG relation between V.M.O. and V.M.L., which could not be demonstrated between V.L.O. and V.L.L. V.M.O. activation is more non-linear which is indicative of more Type II fibres.

It also adds to the circumstantial evidence that there is selective activation of these fibres, and therefore a different function. The case for V.L.O. cannot be argued as convincingly.

**Experiment 6.2.**

a) **Rotation of the Hip with the Knee Flexed to 20°**

**Method**

Seven subjects gave their consent to be subjects in the experiment. 4 of them were women (57%). Their age range was 20 - 33. Absence of any knee pathology was a prerequisite for inclusion. The same pairs of electrodes as used in all the EMG experiments were placed over V.L.O., V.M.O. and R.F., after skin preparation. Each subject was asked to sit on a couch with his/her lower limb extended. Each was requested to contract statically his/her quadriceps to facilitate correct placement over the belly of the muscle. The pair over V.M.O. was placed in the same position as in all the experiments, as were the ones over V.L.O. and R.F., and all of them along the line of the fibres.
Each subject was seated in the Cybex II Plus dynamometer, with the specially fitted force arm with an integral strain gauge connected to an amplifier. This force arm had a padded bar strapped round the lower end of the tibia just above the ankle. An extension was attached to the side strut of the force arm to which was attached a footplate on a swivel joint. The heel was supported in a heel piece, and the front of the foot by a series of straps. On the side of the footplate was a marker which rotated around a semicircular piece of metal marked out in degrees, so that the foot could be fixed in various positions. Thus zero was in the middle, with increasing degrees in either direction to measure both internal and external rotation. The subject was stabilised in the chair by a chest and hip restraining strap. The knee strap was put in position below the patella so that it did not interfere with the EMG electrodes.

The subject’s foot was strapped into the footpiece and the straps over the instep and the forefoot done up tightly and secured with velcro. The knee was flexed to 90° and then extended to 20°. The angle was verified by a goniometer and re-adjusted to account for changes in the slope of the thigh due to different thigh girth. The upper arm of the goniometer was aligned with the femur so that it was over the greater trochanter, and the lower arm along the fibula.
The same EMG equipment was used as in all the muscle experiments, and the signal was amplified, smoothed and rectified, and then digitised and stored on computer for future analysis. When the subject was in position, the electrodes were connected to the amplifier. There were thus 4 channels, one for each for the muscles and one to record the force. The raw signal from the muscles was recorded first during a sub-maximal contraction of the quadriceps, to ensure that a noise free signal was obtained from each channel.

The subject’s hip was put into a neutral position and the thigh aligned to the marks in the Cybex chair. The footpiece was fixed with the marker reading zero,
and the angle of dorsiflexion at the ankle at 90°. The subject was asked to make a maximum isometric contraction, exerting an extensor force with the lower leg only. The EMG signal was rectified and smoothed (100msec time setting) and the total gain set at 5000. This recording was saved on disc, and the amplitude of the force marked on the screen. The timing of the screen read-out was set at 8 s, and, and as before the subject was requested to make a smooth, slow, and steady continuous increase in force starting at the baseline and finishing at the mark for maximum 8 seconds later.

Some subjects were good at activating their muscles in this manner, and some were less good and needed several practices before a satisfactory recording was obtained. Rests were given to ensure that fatigue was not a factor. These recordings, both from the muscles and the force, were again saved, and using the same mark on the screen for maximum force, estimations were made for 25%, 50%, 75% of the maximum. These were again marked on the screen and the subject was instructed to apply the force rapidly and then release it rapidly, reaching each mark in turn. Again the recordings were saved on disc. These different types of activation were termed “ramped contractions” and “stepped contractions” (following the same terminology as Bilodeau et al. (1991), and other workers in the same field).

The subject was then aligned so that the hip was externally rotated 20°, and the footplate fixed at the 20° mark. Care was taken that the rotation did occur at the hip and the thigh strap placed firmly over the patella to hold the knee in its new position. The same test was repeated in this position, and the recordings saved on disc. The test was then repeated with the hip in internal rotation, and fixed in
the same way with the footplate fixed at 20° (in a medial direction) and the knee firmly strapped.

The saved recordings were then analysed in the following manner:

**Mathematical Analysis**

ASCII files were made and again read by a programme called *Viewdac*. Each set of EMG data (VMO and VLO) was normalised, thus $E_{\text{max}}$ and $F_{\text{max}}$ was found (where $E_{\text{max}}$ is the maximum EMG amplitude and $F_{\text{max}}$ is the maximum force).

$$E' = E/E_{\text{max}}, \text{ and } F' = F/F_{\text{max}}$$

$E$ is the EMG data, $F$ is the force recorded.

Because the EMG/force curve was not linear, each curve was fitted to the equation:-

$$E' = I + L \cdot F' + Q \cdot F'^2$$

(where $I$ is the intercept, $L$ is the linear function, and $Q$ is the quadratic function)

The values of $I$, $L$ and $Q$ could then be found.

The following information of the EMG data was obtained for each subject:

<table>
<thead>
<tr>
<th>VMO</th>
<th>Neutral linear (L)</th>
<th>quad (Q)</th>
<th>EMG max ampl in mV</th>
<th>max force in mV</th>
<th>EMG ampl at test force in mV</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.029</td>
<td>0.744</td>
<td>373</td>
<td>645</td>
<td>284</td>
</tr>
</tbody>
</table>

where "test force" is the lowest of 3 values for maximum force.

From this the EMG amplitude at test force in each of the three positions (that is with the hip in a neutral position, with the hip internally rotated by 20°, and with the hip externally rotated by 20°) was found. The EMG amplitude had to be found at the same force each time, because even though the force was maximal,
exactly the same force cannot be produced in each of the three positions. The EMG amplitude at these positions could then be compared.

The hypothesis which is being tested is that when there are changes in the position of the hip there are also changes in the activation of V.M.O. and V.L.O., because these portions of quadriceps are those which are thought to influence the position of the patella. R.F. with its relatively straight pull is unlikely to have much effect on this. Therefore in 5 subjects, recordings were also taken from R.F. The ratio of the activation of these muscles on external and internal rotation of the hip was compared with that when the thigh was in a neutral position, to see whether there is an increase or decrease in the amplitude of the EMG signals.

An example of the graph obtained when the normalised EMG was fitted to the EMG/Force curve is shown below.

Fig. 6.6. Graph of EMG data fitted to EMG/force curve.
When the ratio of the activation of the muscles at the different angles had been found, a Student's t test was applied to the ratio, and the level of probability of the null hypothesis that the ratio=1, was set at p<0.05.

Results

Table 6.3. The results of the ratios of the EMG amplitude with the knee flexed to 20°, for different hip rotations

<table>
<thead>
<tr>
<th>Ramps</th>
<th>VMO</th>
<th>External/Neutral</th>
<th>Internal/Neutral</th>
<th>External/Internal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>n=7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VMO</td>
<td>External/Neutral</td>
<td>0.810</td>
<td>0.966</td>
<td>0.869</td>
</tr>
<tr>
<td></td>
<td>Internal/Neutral</td>
<td>0.091</td>
<td>0.106</td>
<td>0.101</td>
</tr>
<tr>
<td>Student’s t test</td>
<td></td>
<td>2.082</td>
<td>0.321</td>
<td>1.2914</td>
</tr>
<tr>
<td>p&gt;0.05</td>
<td>p&gt;0.05</td>
<td>p&gt;0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VLO</td>
<td>External/Neutral</td>
<td>1.082</td>
<td>0.782</td>
<td>1.535</td>
</tr>
<tr>
<td></td>
<td>Internal/Neutral</td>
<td>0.072</td>
<td>0.098</td>
<td>0.228</td>
</tr>
<tr>
<td>Student’s t test</td>
<td></td>
<td>1.053</td>
<td>2.221</td>
<td>-2.347</td>
</tr>
<tr>
<td>p&gt;0.05</td>
<td>p&gt;0.05</td>
<td>p&gt;0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Steps</td>
<td>VMO</td>
<td>External/Neutral</td>
<td>Internal/Neutral</td>
<td>External/Internal</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VMO</td>
<td>External/Neutral</td>
<td>0.514</td>
<td>0.828</td>
<td>0.622</td>
</tr>
<tr>
<td></td>
<td>Internal/Neutral</td>
<td>0.053</td>
<td>0.058</td>
<td>0.053</td>
</tr>
<tr>
<td>Student’s t test</td>
<td></td>
<td>9.221</td>
<td>2.976</td>
<td>7.059</td>
</tr>
<tr>
<td>p&lt;0.001</td>
<td>p&lt;0.05</td>
<td>p&lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VLO</td>
<td>External/Neutral</td>
<td>0.908</td>
<td>0.821</td>
<td>1.045</td>
</tr>
<tr>
<td></td>
<td>Internal/Neutral</td>
<td>0.103</td>
<td>0.086</td>
<td>0.169</td>
</tr>
<tr>
<td>Student’s t test</td>
<td></td>
<td>-0.893</td>
<td>2.081</td>
<td>0.266</td>
</tr>
<tr>
<td>p&gt;0.05</td>
<td>p&gt;0.05</td>
<td>p&gt;0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Steps</td>
<td>RF</td>
<td>External/neutral</td>
<td>Internal/ neutral</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RF</td>
<td>External/neutral</td>
<td>1.016</td>
<td>0.812</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Internal/neutral</td>
<td>0.121</td>
<td>0.065</td>
<td></td>
</tr>
<tr>
<td>Student’s t test</td>
<td></td>
<td>0.134</td>
<td>2.89</td>
<td></td>
</tr>
<tr>
<td>p&gt;0.05</td>
<td>p&lt;0.05</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
With the knee flexed to 20°, and the quadriceps contraction performed rapidly in increasing steps, there was a highly significant lowering of the EMG amplitude of V.M.O. when the hip was outwardly rotated, compared with that in the neutral position. There was a less marked but still significant decrease in amplitude when the hip is internally rotated. The corresponding results when the contraction was done slowly (ramped contraction) was not significant (p>0.05), but there was one subject whose results were atypical, and when 3 other results from another experiment which involved the same ramped contraction was added, the result was significant. This larger set of results (n=10), had a mean ratio of 0.851 with a standard deviation of 0.06. When a Student's t test was applied the t value was 2.492, which gave p<0.05. The atypical result could have been due to compensation for biomechanical changes. Any change in the activation of V.L.O. was not consistent.

There was no change in the ratio of R.F. (mean=1.016) during the stepped contractions when there was such a strongly significant change in V.M.O., but there was a change in the ratio on internal rotation. Thus this experiment shows clearly that V.M.O. activity decreases by about 50% when the thigh is external rotated. At the same time there is no change in R.F.

It was noticed that in this and also in the other experiments involving the two sorts of isometric contractions that the data from the steps which were done rapidly was much more linear than the ramps which were performed slowly. This showed that at moderate forces for the steps the EMG amplitude was higher.
Fig 6.7. A comparison of the data obtained for steps and ramps showing greater degree of linearity with steps

Discussion

Using the primary meaning of Q angle (quadriceps angle), the only change in the angle of applied force of the quadriceps with external rotation of the hip, is a slight increase in the component force medially of rectus femoris, which is the only part of the quadriceps to have its origin on the pelvis. Although there might be effects to the fascial stocking, it is hard to find a clear cut biomechanical reason for the results. The origin of V.M.O. arises mostly from the tendon of adductor magnus (Bose et al. 1980). Adductor magnus is a composite muscle, but the long fibres which end in the tendon which is attached to the adductor tubercle on the medial condyle, are almost vertical, arising from the ischial ramus. Their action would be slightly to internally rotate the thigh (Grays Anatomy 36th edition quotes de Sousa and Vitti (1966) as suggesting that all the adductors slightly inwardly rotate the thigh but that this has not been confirmed). In external rotation this tendon would be pulled back stretching V.M.O., which might then be at a poor physiological length for maximum contraction.
The lowering of the activation in VMO which was so highly significant in external rotation of the thigh, during the application of force in rapid steps, was less apparent in the slow, ramped contraction. It may be, because of the need of a fast reaction time for V.M.O., that it is a learnt response, occurring in the cerebellum (this has been discussed in Chapter IV). These responses which frequently occur in voluntary movements in the body, enable feedforward programming to occur. The pre-programming which is necessary for these responses, may be in this instance specific for V.M.O. at a certain length i.e. for neutral rotation. It has been noticed that these responses are in general length specific. During the ramped contractions, there is more time for adjustment by reflexes, which are probably myotatic in origin, to increase the EMG amplitude. Such a hypothesis would fit the disinclination of the patients to externally rotate, for it would be advantageous for a muscle which is compromised, to be in the most favourable position for its action. The normal subject, who showed the least change in this experiment, might have “learned” a better strategy to cope with external rotation. The above hypothesis for the muscle control would account for the patients having lower activity in the rapid application of force as found by Grabiner et al. (1992). It would also fit in with the difficulty experienced by the patients in participating in sporting activities which might involve rapid activation of these muscles round the knee. It has been shown by Bose et al. (1980) who studied the origin of V.M. in many species of primates that none of them had fibres arising from the adductor magnus tendon, but from the femur only, in other words they had no V.M.O. It may be that V.M.O. action, which is a late adaptation in evolutionary terms and which can be thought
to be a source of stability in the upright gait, may be an imperfect adaptation, whose weakness can be detected in external rotation. Or it may be that there is a medial shift of the patella in lateral rotation, and V.M.O. does not need to work as hard in this position.

The change in activation of V.M.O. on lateral rotation of the hip was not accompanied by any change in the activation of R.F., however there was on internal rotation. There is a considerable change in the position of the underlying muscle (R.F.) with respect to the skin on rotation of the hip, and it is likely that on internal rotation the pair of electrodes was no longer fully over the muscle. On the other hand it would be unlikely for there to be such a large corresponding change in electrode position which could account for the difference in the results for the oblique fibres, because the oblique orientation is not being changed although it is likely that the length is; also if the effect was due to a change in electrode position there would be no difference between the steps and ramps. There may be other reasons for the findings for the oblique fibres which have not been detected. When applying force in this position, the lowering of V.M.O. activity might be a response to changes in tension in the knee which has been altered with different forces being transmitted and differences in the stretching of the soft tissues in the knee, perhaps in the retinacula. It is not known where receptors are located, whereby an inhibitory reflex might be initiated. Further research would be a great advantage in answering the unsolved questions. Local anaesthetic applied to different structures in the knee would be an informative experiment. Had access to better gait analysing equipment been possible, the
effects of different rotation in weight bearing would have been studied, again using EMG but also studying differences in moment with the CODA or similar system, and the transference of weight by means of a force plate.

The greater degree of linearity in muscle activation plotted against force, when the subjects made a rapid application (rather than slow) and relaxation of different degrees of force (stepped contractions), were probably similar to the findings of Gillies (1966). Citterio and Agostini (1984) postulated that differences in activation in fast and slow bicycling rates were due to the preferential activation of Type 2 fibres at smaller force. This caused an increased amplitude of the EMG signal at fast rates, and therefore a corresponding increased linearity for smaller forces. It is generally considered that a marked increase in amplitude of the signal obtained at higher forces is due to Type II fibres (Chaffin et al. 1980), as seen in the slow ramped application of force.

In trying to understand the slow activation of V.M.O. and V.L.O. in the experiment where the patients were asked to make a rapid application of force at 20° of flexion which was termed the lag factor (Chapter IV), a superficial assessment might lead one to suppose that the non-linearity was due to the relative slowness of the force application which the patients exhibited. In this experiment it could be argued, either that the slowness in the rate of application of force in the patient group might have been due to fear or other factors causing the difference in muscle activation compared with normal subjects; or that their
inability to activate these portions of the quadriceps in the normal manner led to the slow application of force.

When a closer look was taken at the slow ramped muscle activation in normal subjects in this experiment, it was seen that the shape was altogether different from the patients' muscle activation in Experiment 4. 80% of the maximum force was covered by the steepest part of the curve (see Fig. 6.6.), when it is postulated the Type II fibres have mostly been recruited. It has been shown that although activation varies in individual muscles, this is true of most muscles (Lenman 1979). This was not the case in the previous experiment; in many cases when the patients had reached 80% of the maximum force, the amplitude of the EMG for V.M.O. and V.L.O. was only 40-50% of the maximum. Muscle biopsies of V.L. have demonstrated that patients with recurrent dislocation of the patella had a low percentage of Type IIB fibres and a high percentage of embryological Type IIC (Floyd et al. 1987). Type II B fibres are normally activated at high forces. Pette (1984) has demonstrated that a high proportion of Type IIC denotes a fast
to slow transition in the fibre content of a muscle. If, therefore, there is a loss of fast fibres in the oblique sections of the patients' muscles, the shape of the curve when the activity of the muscle is plotted against the force (Fig 6.7.) could well be changed and account for the slow activation rates of the oblique muscles.

b) Rotation of the Knee at 90° of Flexion

Introduction

The situation with the knee flexed to 90° is quite different from the foregoing experiment. Here in actual change in rotation of the knee, there is a change in the orientation of the patella to the femur, and also a change in the force vectors.

Method

6 normal subjects, selected in the same way as those in the previous part of the experiment took part; 4 of them were women (67%); 3 of them took part in Expt. 1a) and 3 were new subjects, this was because it was considered inadvisable to complete the whole experiment at one sitting, not only because they did not have the time available, but because fatigue in the muscles could be a factor in lowered activity.

Each was seated in a Cybex chair, with the straps in position, and the force arm with the extension and foot plate in place. The knee was bent to 90° and the extent of flexion verified with the use of the goniometer. The ankle was fixed at a right angle and the lower leg in neutral, with the marker on the foot piece reading zero. The electrodes were in place over V.M.O. and V.L.O. Noise free raw signals from the muscles was obtained as before, and then the smoothed and rectified recordings from the two portions of the muscles together with the force
during a maximal isometric extensor effort, were saved on disc. The extent of
this was marked on the screen and each subject did a slow ramped contraction
lasting 8 seconds, which was followed by rapid application of force to 25%, 50%,
and 75% of the maximum (these forces were also indicated by markers on the
screen). This was repeated with the lower leg inwardly rotated by 20 degrees
and externally rotated by 20 degrees.

Results

Table 6.3. Results of the ratios of the EMG amplitude with the knee bent to 90°,
for different tibial rotations

Ramps

<table>
<thead>
<tr>
<th></th>
<th>External/Neutral</th>
<th>Internal/Neutral</th>
<th>External/Internal</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>VMO</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n=6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>1.021</td>
<td>1.046</td>
<td>0.969</td>
</tr>
<tr>
<td>sem +/-</td>
<td>0.105</td>
<td>0.059</td>
<td>0.034</td>
</tr>
<tr>
<td>Student's t test</td>
<td>0.2</td>
<td>0.78</td>
<td>0.912</td>
</tr>
<tr>
<td>p&gt;0.05</td>
<td>p&gt;0.05</td>
<td>p&gt;0.05</td>
<td></td>
</tr>
<tr>
<td><strong>VLO</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n=6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>1.038</td>
<td>1.023</td>
<td>1.009</td>
</tr>
<tr>
<td>sem +/-</td>
<td>0.088</td>
<td>0.06</td>
<td>0.041</td>
</tr>
<tr>
<td>Student's t test</td>
<td>0.432</td>
<td>0.383</td>
<td>0.22</td>
</tr>
<tr>
<td>p&gt;0.05</td>
<td>p&gt;0.05</td>
<td>p&gt;0.05</td>
<td></td>
</tr>
</tbody>
</table>

Steps

<table>
<thead>
<tr>
<th></th>
<th>External/neutral</th>
<th>Internal/neutral</th>
<th>External/internal</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>VMO</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n=6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>0.998</td>
<td>0.859</td>
<td>1.175</td>
</tr>
<tr>
<td>sem +/-</td>
<td>0.038</td>
<td>0.055</td>
<td>0.048</td>
</tr>
<tr>
<td>Student's t test</td>
<td>0.053</td>
<td>2.564</td>
<td>3.662</td>
</tr>
<tr>
<td>p&gt;0.05</td>
<td>(p&gt;0.05)</td>
<td>p&lt;0.02</td>
<td></td>
</tr>
<tr>
<td><strong>VLO</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n=6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>0.941</td>
<td>0.808</td>
<td>1.212</td>
</tr>
<tr>
<td>sem +/-</td>
<td>0.088</td>
<td>0.079</td>
<td>0.063</td>
</tr>
<tr>
<td>Student's t test</td>
<td>0.67</td>
<td>2.424</td>
<td>-3.372</td>
</tr>
<tr>
<td>p&gt;0.05</td>
<td>p&gt;0.05</td>
<td>p&lt;0.02</td>
<td></td>
</tr>
</tbody>
</table>
There was a lowered EMG signal from both V.M.O. and V.L.O. in the stepped contractions, in internal rotation compared with the neutral position. This was not significant (p>0.05) in the case of V.M.O., and not significant for V.L.O., nevertheless the results were very similar, and the ratio for external rotation compared with internal rotation was also very similar (the change for both was significant and p<0.02). This would indicate that the muscles work more in synchrony than at 20°, and suggest that there may be a reflex loop between the two. There was not a significant change in the ramped contractions. The maximum force at 90° was more than double what it was at 20°, which was as expected because of the considerable mechanical disadvantage when the knee is nearly straight.

Changes in external rotation were not significantly different from the ratio=1.

Discussion

The results of the muscle action in internal rotation showed that there was a marked correlation between the activity in V.M.O. and V.L.O., so that a decrease in activity in the one was followed by a decrease in activity in the other. Therefore, it seems as if the method of their control is for them to work synergistically, and not antagonistically as they would have to do if Muller was right in his hypothesis. This correlation was much greater at 90° than at 20°.

However, in the experiment described in Chapter IV of a rapid application of force at 20° there was a correlation, which was greater in the patients than in the normal subjects. Van Kampen and Huiskes (1990) in their study found that in internal rotation at 90° of knee flexion there was a medial rotation of the distal
pole of the patella, which since it rotates round a transverse axis would cause a lateral rotation of the proximal pole. V.M.O., which is inserted into the proximal half of the medial border of the patella, would be stretched. Compared with its activity in neutral rotation, V.M.O. might not be at the best length for maximum activation, and V.L.O. would follow suit. The reason for greater correlation between V.M.O. and V.L.O. at 90° than at 20° during internal tibial rotation, could well be due to the relative absence of tilt at that angle. However, the extent of tilt at 20° would be more variable due to differences in geometry between individual knees.

Hehne (1990) has postulated that the retinacula provide stability at flexed angles of the knee because they are pulled tight at these angles. Hungerford and Barry (1979) consider that it is when the knee is nearly straight that there is necessity for effective V.M.O. action to counteract the lateral pull, and that when the patella has entered the trochlea bony congruity will provide sufficient stability. However, at 90° the articulation of the patella with the trochlea is at a position where the trochlea is much more shallow, and after 90° when it lies between the condyles it is not stable at all. The oblique fibres of the vasti were certainly working strongly at 90°, and it has been shown by this experiment that they do show a change in activity to accommodate for change in rotation. It is likely that the movement of the patella which occurs is mostly passive, but that V.M.O. and V.L.O. do accommodate for the very small movements which occur and provide added stability.
It is postulated by the author that the reason why there was no significant change in activity in external tibial rotation is that there was a mistake in the design of the experiment. In internal rotation, 20° is 2/3 of the limit of range whereas 20° of external rotation is only half of the permitted range. When the experiment was designed it was thought that what was being tested was an appreciable change in the vectors. When the results of Experiment 6.3. became known, and showed that the long fibres are capable of adaptive change, then the hypothesis could be put forward that oblique fibres “fine tune” changes in patella position, and the long fibres adapt to change in the force vectors. It is the long fibres which produce a major part of the extensor force, whereas it has been shown that V.M.O. contributes very little to that force. Future research should certainly include further studies of the action of the long fibres as well as the oblique fibres in changes of rotation in this position, as well as studying the changes in activity at half as well as at the full range of movement possible.

**Experiment 6.3.**

**Experiment to Investigate whether Changes in the Activation of V.M.O. in External Rotation of the Hip at 20° of Knee Flexion are Paralleled by Changes in V.M.L.**

**Method**

5 subjects volunteered to take part in this experiment. 3 of these were women (60%) and 2 were men (only 2 were the same subjects as in Expt. 1). The same inclusion criteria governed their choice, and the same methods were followed. Electrodes were placed over V.M.O. and V.L.O. in the same way as in the previous experiments. The only difference was that in this experiment a third
electrode was placed over V.M.L. As in Experiment 1, care had to be taken over
this placement, to avoid both sartorius and rectus femoris. Sartorius is a thin
strap-like muscle which crosses the thigh from the lateral side crossing the knee
medially to be inserted on the medial aspect of the tibia. The placement had to
be distal to this, but medial to rectus femoris. To ensure that this placement was
correct the subject was asked to flex the hip, since both these muscles are
accessory hip flexors, with sartorius acting particularly in external rotation. If
there was no signal when this was done, the contribution from cross talk could
not be great. The subjects applied the force both in slow ramps, and in separate
trials in rapid increasing steps.

The ratio of the EMG amplitude at the test force in external rotation compared
with neutral rotation was found for V.M.O. and V.M.L. The ratio of the change
in activation for V.M.O. was then compared with the ratio of change for V.M.L.
for each individual subject, and from this the mean of the 5 ratios
(V.M.O./V.M.L.) was found. A Student’s t test was then applied in the normal
way.
Results

<table>
<thead>
<tr>
<th></th>
<th>VMO</th>
<th>VML</th>
</tr>
</thead>
<tbody>
<tr>
<td>RAMPS</td>
<td>External/Neutral</td>
<td></td>
</tr>
<tr>
<td>n=5</td>
<td>VMO</td>
<td>VML</td>
</tr>
<tr>
<td>mean</td>
<td>0.835</td>
<td>1.444</td>
</tr>
<tr>
<td>sem +/-</td>
<td>0.071</td>
<td>0.192</td>
</tr>
<tr>
<td>STEPS</td>
<td>VMO</td>
<td>VML</td>
</tr>
<tr>
<td>n=5</td>
<td>mean</td>
<td>0.491</td>
</tr>
<tr>
<td></td>
<td>sem +/-</td>
<td>0.105</td>
</tr>
<tr>
<td>RAMPS</td>
<td>VMO/VML</td>
<td></td>
</tr>
<tr>
<td>n=5</td>
<td>mean</td>
<td>0.597</td>
</tr>
<tr>
<td></td>
<td>sem +/-</td>
<td>0.136</td>
</tr>
<tr>
<td>Student’s t test</td>
<td>probability of null hypothesis (ratio=1)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>p&lt;0.05</td>
<td></td>
</tr>
<tr>
<td>STEPS</td>
<td>VMO/VML</td>
<td></td>
</tr>
<tr>
<td>n=5</td>
<td>mean</td>
<td>0.385</td>
</tr>
<tr>
<td></td>
<td>sem +/-</td>
<td>0.105</td>
</tr>
<tr>
<td>Student’s t test</td>
<td>probability of null hypothesis (ratio=1)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>p&lt;0.01</td>
<td></td>
</tr>
</tbody>
</table>

The EMG amplitude at the test force was again much reduced in outward rotation in the steps. The mean ratio was 0.491, which was again a highly significant reduction (p<0.05). The EMG amplitude in the ramps was not significant (a t test gave a t factor of 2.324), and the mean ratio was again higher than the steps (0.835). The ratio of the change of EMG amplitude on external rotation of V.M.O. to the change of that of V.M.L. was however, highly significant for both the ramped contractions and the stepped contractions. The results, therefore, showed that the decrease in the amplitude of EMG for V.M.O. in outward rotation are not matched by a corresponding lowering in amplitude for V.M.L., in fact in most cases the amplitude of V.M.L. was greater in external rotation than it was in neutral rotation.
Discussion

The experiment showed that there were differences in the activation of the oblique and long fibres of V.M. in changes of rotation of the hip, which adds to the evidence that selective activation of V.M.O. is indeed possible. It would appear that the function of the two portions of the muscle are different, which need different strategies for the recruitment of motor units. The precise function of these different portions needs to be elucidated by future research. The increased activation of the long fibres might compensate for the lowered activity of the oblique ones.
CHAPTER VII

EXPERIMENT TO TEST THE LENGTH OF THE HAMSTRINGS AND THE PLANTARFLEXORS IN PATIENTS WITH PATELLOFEMORAL DISORDERS COMPARED WITH NORMAL SUBJECTS

Introduction

Although tight hamstrings have been implicated as a possible cause of patellofemoral problems, no study has been seen by the author which compares hamstring length in patients with patellofemoral dysfunction, with a group of normal subjects. It has been noted that the hamstrings in males are shorter than in females (Wang et al. 1993). Although it has been suggested that the effect of short hamstrings is to cause the quadriceps to work harder and therefore to increase patellofemoral contact force, and that this is a cause of symptoms (Jacobson and Fandry 1989), there is very little direct evidence that this is so. It has also been suggested that hamstring tightness might cause symptoms because the lateral hamstrings (biceps femoris) is connected to the iliotibial band. Terry et al. (1986) during their dissections found that the short head of biceps inserts into the layer of the iliotibial band which they termed the osseo-capsular layer all the way along its course. The iliopatellar part of the iliotibial band is attached to the patella, so that it is possible that biceps femoris has an effect on the patella, and could exert a lateral pull. The iliotibial band also has an effect on the patella through the retinaculum. LaBriar and O’Neill (1993) found evidence of tight hamstrings in patellofemoral stress syndrome. They used this term to describe the condition where there is no retropatellar pain, or evidence of malalignment,
but lateral peripatellar pain and tight iliotibial band and lateral retinaculum. They advocated hamstring stretching for treatment of the condition.

Tight hamstrings are seen in other conditions. Hamstring length is often assessed in anterior cruciate ligament (ACL) injuries (for example Harner et al. 1994). Fisk et al. (1984) noted that in Sheuermanns disease there was a marked correlation between the radiological evidence of the disease in males, and tight hamstrings. However, they could find no evidence of a correlation between dynamic activity and the condition, and concluded that prolonged sitting might be the cause of both effects. It has been noted that the preferred activity has an effect on hamstring length, Wang et al. (1993) found that runners had shorter hamstrings than non-runners, and that long distance runners had the shortest hamstrings of all the groups. The shortest hamstrings that have been seen by the author, were two male cricketers whose preferred position was wicket keeping. However, it is likely that postural factors such as prolonged sitting, as cited by Fisk et al. (1984) has a marked effect. It has been noted that children’s hamstrings are shorter after the age of 5 years, that is when they start school and sit for fairly long periods.

Various methods have been used to measure hamstring length. Gajdosik et al. (1993) made a comparison of four clinical tests for assessing hamstring muscle length. These were 1) straight leg raising with the pelvis strapped 2) straight leg raising with a flat pelvis, and supported by a pillow if necessary 3) active lower leg straightening with the thigh flexed to 90° of hip flexion 4) passive movement
in the same position. There was very little difference between the results of the first two, but the measurement of the popliteal angle in tests 3) and 4), gave a lower angle in the active test. The results they obtained for the passive test was 31° +/- 7.5°. Method 4) has been chosen for the measurement in this study. The method was based on that used by Clendaniel et al. (unpublished) on normal subjects. However in this experiment, the patients and normal subjects had the contralateral leg fully extended, in contrast to the previous method, in order to fix the pelvis more effectively. The notation in this study is different from the results given by Gajdosik et al. (1993). The angle that is measured in this study is the limit of the angle achieved, starting from the horizontal position, so that 31° in their notation would be 59° in this notation.

There have been many studies undertaken to find how much increase in length can be achieved by stretching regimes (for example Starring et al. 1988, Bandy and Irion, 1994). However, Halbertsma and Goeken (1994) cast doubt on the effectiveness of stretching regimes, and concluded from their study that stretching does not make the short hamstrings any longer or less stiff, but only increases the tolerance to stretch.

The length of the plantarflexors was also investigated since it is thought that tight plantarflexors contribute to the incidence of increased sub-talar pronation in the stance phase of gait (Carson et al. 1984, Root 1977).
7.1 Experiment to Investigate the Length of the Hamstrings

Method

The same group of patients were asked to undergo this test. A group of normal subjects was also tested, belonging to the same age group as the patients, and the ratio of women to men was the same as the patient group. None of them had had any knee symptoms.

Each subject lay supine on a couch with their legs extended. The measuring device, which was attached to an upright stand, was placed on the side of the couch of the leg being measured. The upright stand had an extendable flange to accommodate different lengths of femur, and on the end of this was a goniometer. The marker for the goniometer was a long piece of plastic which was marked along its length at the centre. It was aligned to the length of the fibula. The upright stand was attached to the subject’s thigh by means of a strap, tightly secured by velcro. The length of the upright piece was extended until the centre of the goniometer was at the level of the knee joint line. With one hand supporting the stand and the thigh at 90°, the operator pushed the lower leg upwards towards the ceiling, together with the goniometer marker. When it became hard to achieve more movement due to resistance from the hamstrings, the angle at which this occurred was read from the goniometer. The subjects did not protest or complain of pain, and any remark that the muscles felt tight coincided with the operator’s assessment of the end-point.
Fig. 7.1. Diagram of hamstring measurement ($\theta$ is the angle being measured)

Fig. 7.2. A subject whose hamstrings are being measured
Results

Table 7.1. Hamstring length of patients compared with normal subjects

<table>
<thead>
<tr>
<th></th>
<th>patients</th>
<th>patients</th>
<th>normal</th>
<th>normal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>men</td>
<td>women</td>
<td>men</td>
<td>women</td>
</tr>
<tr>
<td></td>
<td>most aff</td>
<td>least or unaffected</td>
<td>side</td>
<td>side</td>
</tr>
<tr>
<td>number</td>
<td>27</td>
<td>27</td>
<td>43</td>
<td>43</td>
</tr>
<tr>
<td>mean in°</td>
<td>55.2</td>
<td>59.4</td>
<td>63.6</td>
<td>66.8</td>
</tr>
<tr>
<td>sd</td>
<td>10.9</td>
<td>9</td>
<td>13.7</td>
<td>12.5</td>
</tr>
<tr>
<td>sem+/−</td>
<td>2.1</td>
<td>1.7</td>
<td>2.1</td>
<td>1.9</td>
</tr>
</tbody>
</table>

When a Student's t test was applied there was a significant difference (mean=6.1°) between the hamstring length on the more affected side for the men patients compared with the group of male normal subjects (t was 2.33, p<0.05).

In women patients difference was much less (2.6°), which was not significant (t=1.22). When comparing the less or unaffected leg of the men patients with the normal group there was no significant difference.

Discussion

This result was similar to the finding of Wang et al. (1993) that men have shorter hamstrings than women. The results indicate that there is an incidence of hamstring tightness in men patients with patellofemoral problems, but it is not likely to have a great pathological significance because the group of women patients did not show the same effect. It is not possible to see from these results which of the hypotheses put forward (for example Jacobson and Flandry, 1989, Terry et al. 1989) is likely to be correct. The possible mechanism behind tight hamstrings was discussed in Chapter V during the discussion of quadriceps/hamstring ratio (part 5.2.)
7.2. Experiment to Investigate the Length of the Plantarflexors

Method

The plantarflexors were measured in prone lying with the foot being measured over the bottom of the couch. In this position the operator was able to see whether it is only the extent of dorsiflexion which is being measured. In supine lying when the operator pushes up the foot it is possible to evert the foot, and so increase the angle of dorsiflexion, thus masking the tight plantarflexion.

Each subject was in prone lying with the lower limb straight. A specially made goniometer made by Langer instruments, which was the right size and shape for a foot, was used. One arm of this instrument was aligned with the fibula and the other arm aligned with the head of the 5th metatarsal. The foot was pushed into dorsiflexion, taking care neither to evert or invert the foot until the endpoint was reached. The angle at which there was resistance to further movement was read from the goniometer.

Fig 7.3. Instrument for measuring the extent of dorsiflexion
Each foot of 44 normal subjects was measured, and since there was no mean difference between the right and left feet, the mean of both feet of the whole group was found (88 measurements). This was compared with the measurements taken from the most affected side of 68 patients, and then the least affected or unaffected side. A Student’s t test was applied.

Results

The angle which is being presented is the angle of dorsiflexion beyond the right angle.

Table 7.2. Extent of dorsiflexion in normal subjects

<table>
<thead>
<tr>
<th>Subjects</th>
<th>right</th>
<th>left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>44</td>
<td>44</td>
</tr>
<tr>
<td>Mean angle of dorsiflexion</td>
<td>11.61°</td>
<td>11.45°</td>
</tr>
<tr>
<td>sd</td>
<td>4.1°</td>
<td>4.26°</td>
</tr>
<tr>
<td>sem+/-</td>
<td>0.604°</td>
<td>0.629°</td>
</tr>
</tbody>
</table>

Table 7.3. A comparison between the normal subjects (88 measurements) and the patients.

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Patients (most affected limb)</th>
<th>Patients (least aff. or non aff. limb)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of measure</td>
<td>88</td>
<td>68</td>
</tr>
<tr>
<td>mean angle of dors</td>
<td>11.53°</td>
<td>11.46°</td>
</tr>
<tr>
<td>sem+/-</td>
<td>0.4438</td>
<td>0.4438</td>
</tr>
<tr>
<td>Student’s t test</td>
<td>0.1249</td>
<td>1.5491</td>
</tr>
<tr>
<td>Subjects/patients</td>
<td>p&gt;0.05</td>
<td>p&gt;0.05</td>
</tr>
</tbody>
</table>

When considering the patient group, in 7 patients the angle of dorsiflexion beyond right angle was less than 10° on the most affected side, whereas it was 10° or more on the least affected side. However, since the number of patients was 68, the incidence of this characteristic is very low. In 5 patients the angle...
was less than 10° bilaterally, even though the symptoms were greater in one knee than the other.

When considering the normal subject group, in 9 of these subjects the angle was less than 10° bilaterally, and in 4 unilaterally. On comparing the measurements of the patients and the normal subjects, there is no evidence of any difference between the two.

Discussion

While it has been demonstrated in other gait studies that tight plantarflexors do have an effect on gait, in that there is either a premature heel raise in the stance phase, or a tendency for increased sub-talar pronation to occur, this alteration probably occurs in the normal subjects who did not have any knee symptoms. Other studies giving normative data have not been seen by the author, but this group of patients compared with a large number of normal subjects, show no significant differences in the extent of dorsiflexion. However, tight plantarflexors is only one of several causes of sub-talar pronation, and foot abnormalities were noted in 57 patients (84%), which is a very high incidence and likely to be higher than the general population, although no normative data is available. Of these 52 showed sub-talar pronation and 5 uncompensated rearfoot varus.
CHAPTER VIII

BIOMECHANICAL MEASUREMENTS

8.1. External and Internal Rotation of the Hip

Introduction

Patella malalignment may not only be due to maltracking of the patella, but may be due to an underlying torsion of the femur (Carson et al. 1984, Ekhoff et al. 1994). This thinking has prevailed clinically for several decades, and it was thought that femoral anteversion altered the patellofemoral articulation. However, the evidence of Ekhoff et al. (1994) (to be explained below) did not support this view. On the other hand Lee et al (1994), in a study on cadavers, demonstrated that there was a change in patellofemoral contact pressures in femoral rotation, and also on tibial rotation.

A certain degree of torsion of the long bones is a part of normal development, and is thought to be the result of a bipedal gait (le Damany 1903, translated 1994). The epiphyses rotate round the diaphyses, and the diaphyses remain unaltered, so that in the case of the femur there is a change in angle between the shaft and the head (Grays' Anatomy 36th Edition p 392, and Eckhoff et al. 1994), causing a medial or lateral rotation of the shaft. In utero there is rotation of the femur, so that there is 40° of anteversion in the neonate. This means that with development a derotation has to occur, and by the time skeletal maturity has been reached there is only an average of 10° anteversion (Crelin 1981, Staheli et al. 1977).
The femoral torsion which has been most frequently implicated clinically in the pathogenesis of patellofemoral dysfunction is excessive femoral anteversion. However, femoral retroversion can also occur. There are various imaging techniques which could be used to discover the position of the head of the femur with respect to the shaft, e.g. X-Ray, CT, MRI, ultrasound, Laser imaging.

Upadhyay et al. (1990) did a study on normal children using ultrasound and found that there was a highly significant negative correlation between femoral anteversion and age. The most recent study (Ekhoff et al. 1994) was undertaken to discover the difference in femoral anteversion between normal subjects, and patients with patellofemoral pain which was resistant to conservative measures of alleviation. They used CT scanning and took three “cuts”; one was taken at the level of the mid femoral head; the second at the level of the lesser trochanter; and the third proximal to the intercondylar notch. Their hypothesis was that if there was a femoral anteversion, there would be a compensatory change in the patellofemoral articulation, causing both a change in the congruence angle and lateral patellofemoral angle, that is patella shift and tilt.

Fig. 8.1. To show femoral anteversion (on the right) (after Ekhoff et al. 1994)
What they found, however is that although there was indeed an increased femoral anteversion in the patients compared with the asymptomatic group, there was no change in the patellar articulation. Therefore, any changes that the excess anteversion might cause are likely to be functional rather than structural.

It was decided to try to quantify the incidence and magnitude of femoral anteversion in the sample of patients in this study, and to see whether there was a correlation between this structural abnormality, and changes in quadriceps muscle action. Lee et al. (1994) thought that the rhythm of the quadriceps mechanism would be disturbed by changes in rotation. The method chosen was to compare the extent of internal and external rotation of the hip. This is a useful marker for femoral anteversion as shown by Staheli et al. (1968) and Svenningen et al. (1989 and 1990). Staheli et al. (1968) showed that there was a high degree of correlation between the extent of internal rotation of the hip and femoral anteversion in children (the results were significant and p<0.01). In a study in children by Svenningen et al. (1990), a decrease in internal rotation mirrored a decrease in anteversion, during improvement of an intoeing gait. However, Svenningen et al. (1989) did point out that femoral anteversion was not the only cause of increased internal rotation, which was also caused by the shape of the acetabulum, and capsular and muscle tightness.

The various imaging techniques, which could have been used, were discarded partly on account of cost, but also radiation and other considerations, so that this marker (that is the extent of internal rotation in excess of external rotation) was
used to give an indication of femoral anteversion, information which would otherwise be extremely difficult to obtain. Carson et al. (1984) also regarded it as a useful marker.

Hip rotation can be measured in supine with the legs extended; in supine with the knees bent; or in prone with the knees bent. The first of these has been used in this experiment, as suggested by Root (1977), who thought that in supine with the knees extended, the hip structure determines the extent of the movement, whereas in supine with the knees bent, ligamentous tightness is brought into play. However, he may have had no objective justification for this assertion. The two factors which are most likely to influence the result are capsular tightness and ligamentous tightness. Gray's Anatomy (36th Edition p 477) examines the structure of the capsule. It is stated that the fibrous capsule consists of circular fibres which form a collar around the neck of femur. They are blended with the pubofemoral and ischiofemoral ligaments. The longitudinal fibres are greatest in number at the upper and front part of the capsule, where they are reinforced by the iliofemoral ligaments. The pubofemoral and the ischiofemoral ligaments also strengthen the articular capsule. It is clear from this that the ligaments have a big effect on the capsule. However, Kapandji (1970) says that when the hip is straight the ligaments are moderately relaxed, but when the hip is flexed the total range of lateral rotation is greater because the hip flexion relaxes the iliofemoral and pubofemoral ligaments which play a vital part in limiting lateral rotation. Therefore, it does seem reasonable to choose a method where the hip is straight.
The position most often employed to measure rotation in the hip joint is prone with the knee bent to 90°, but care has to be taken not to rotate the knee as well. One reason why the supine position was chosen is because it is easier for one person to perform the passive movement and to take the measurement, whereas in prone this is very difficult. The extent of flexion or extension of the hip is the same in both the prone and the supine position, and therefore the relationship of internal rotation to external rotation should not be changed. This is the important relationship in this experiment. It could be that the relationship would be changed with the hips flexed, in which case there would be a larger number of patients and normal subjects with a greater external external rotation. However, Svenningen et al. (1989), who showed the correlation between femoral anteversion and the extent of internal and external rotation, performed the movement in prone with two examiners, one performing the movement and one taking the measurement. Staheli et al. (1968) also employed the same position. Also in prone or supine (that is midway between flexion and extension) there is greater similarity with the erect position.

Method

The subject was asked to lie flat, in supine position, on a couch. Each subject was asked to hold his/her pelvis, with the fingers over the anterior superior iliac spine and the thumbs over posterior part of the pelvis, they were asked to report immediately if any movement of the pelvis took place during the measurement of hip rotation. A pressure monitor could have been used, but this way of stabilising the pelvis enabled one person passively to move the hips and take the measurement. The medial and lateral epicondyle were palpated at the knee and
marked with a skin marking pencil. A gravity goniometer (Martin goniometer) which is manufactured by Langer Instruments, was used with Micropore tape wound over the points to make it more comfortable and to ensure that the metal did not slip on the skin. This was placed in position with the leg in a neutral position. The pointer then was in a vertical position. With the other leg slightly abducted, the hip was manually rotated inwards with the hold over the lower leg until the end point was felt, when the number of degrees of rotation at which this occurred was read of the goniometer dial. The hip was rotated into external rotation in the same way and the reading taken.

The repeatability of this measurement was studied on 4 subjects before being used on the patients. They were measured, and a week later were re-measured without the previous results being looked at. The difference between the extent of internal rotation compared with the extent of external rotation was calculated (a negative number indicates the the extent of external rotation was greater than internal rotation). The standard deviation of the two observations was calculated. One standard deviation was unacceptably large so the observation was repeated once more. The standard deviation of these 3 measurements was then found.

**Result**

Table 8.1. Repeatability of hip rotation measurement

<table>
<thead>
<tr>
<th>Subject</th>
<th>Right hip mean diff</th>
<th>sd</th>
<th>Left hip mean diff</th>
<th>sd</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject 1</td>
<td>-10°</td>
<td>0</td>
<td>-9°</td>
<td>1.4</td>
</tr>
<tr>
<td>Subject 2</td>
<td>-7.5°</td>
<td>3.5°</td>
<td>-12.5°</td>
<td>3.5°</td>
</tr>
<tr>
<td>Subject 3</td>
<td>-12°</td>
<td>2.8°</td>
<td>-1.5°</td>
<td>2.1°</td>
</tr>
<tr>
<td>Subject 4</td>
<td>-15°</td>
<td>5.7°</td>
<td>-14.5°</td>
<td>5.2°</td>
</tr>
</tbody>
</table>
The mean s.d. of all the 18 observations was $3^\circ$. This is adequate for studying the quantity of measurements in this experiment where there is a variation between individuals of almost $30^\circ$.

The results of the most symptomatic leg are presented, and each leg of the normal group. The extent of internal hip rotation compared with the extent of external rotation has been calculated.

**Summary of Results**

Table 8.2. Hip rotation in patients compared with normal subjects

<table>
<thead>
<tr>
<th></th>
<th>Patients</th>
<th>Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>number</td>
<td>58</td>
<td>32</td>
</tr>
<tr>
<td>mean int rot-ext rot</td>
<td>-4.05°</td>
<td>-8.42°</td>
</tr>
<tr>
<td>sd</td>
<td>10.06°</td>
<td>4.72°</td>
</tr>
<tr>
<td>sem +/-</td>
<td>1.32°</td>
<td>0.92°</td>
</tr>
</tbody>
</table>

When a Student’s t test was applied, the difference between the groups gave a value of 2.71 which was significant ($p<0.01$). There were 8 patients whose extent of internal rotation was greater than the extent of external rotation by $10^\circ$ or more (14%), whereas in the normal group there was only one normal subject whose extent of internal rotation of one hip was greater than the external rotation (by $1^\circ$). There was also a small group of patients who exhibited an increased range of external rotation of the hip, but a lowered range of internal rotation. It is not possible to be sure whether this is comparable with a true femoral retroversion, because no work has been done comparing the extent of external rotation with retroversion.
Discussion

The group of patients did exhibit a significant increase of internal hip rotation, which was shown by Svenningen et al. (1989 and 1990) to have a high degree of correlation with femoral anteversion. Eckhoff et al. (1994) did not find any differences in the articulation of the patellofemoral joint, but since his measurement were taken with the knee straight, it is not known how this abnormality would alter the articulation at different angles of knee flexion. However, whether or not there are structural effects, there are likely to be functional effects. Changes in these which are likely, are alterations in patellofemoral contact pressures, and alteration in the action of the muscles.

8.2. External Tibial Torsion

Introduction

The high incidence of external tibial torsion in patients with patellofemoral problems has long been noted (e.g., Carson et al. 1984, Insall et al. 1976), as has the clinical observation that it is frequently combined with proximal tibia vara. The latter has been termed the “bayonet sign” because the upper end of the tibia resembles a bayonet. The effect that both these abnormalities are thought to have on the functioning of the joint, is to alter the functional Q angle by the laterality of the pull of the patella tendon (see Fig. 6.1) and thus to cause a lateral vector which needs to be compensated for by muscular action (Hehne 1990). Therefore, it was considered important to record the incidence of this abnormality in this sample of patients, and to attempt to quantify it, and correlate it with other abnormalities. Quantification has been previously undertaken by imaging techniques, for example by CT (Jacob et al. 1980), or X-Ray (Wangarmez and
There have also been measurements made in cadavers (eg Dupuis 1951, quoted by Turner and Smillie 1981), or in vivo in one of two ways. The first in vivo method was with calipers with flexion of the knee to 90°; one report gave a mean reading of 14° of external tibial torsion and in the other study this was 16°. Other studies used the same method of fixing the leg in extension in a frame and measuring the angle of the malleoli, and gave remarkably consistent results (Turner and Smillie 1979, Jaffres 1970). In one study the mean was 19° and the other two 20°. Turner and Smillie took the tibial tuberosity as their reference point, and used a pointer to fix it.

The advisability of fixing the tibial tuberosity was difficult to ascertain. As already stated in the section on femoral torsion, it is the epiphysis which rotates round the diaphysis. In this case the epiphysial line falls in the middle of the tibial tuberosity. The upper part, which forms the attachment of the ligamentum patellae, is thus twisted in this abnormality, whereas the lower part (which is the subcutaneous area) is not. This means that the accuracy of positioning is paramount.

Turner studied the association between tibial torsion and knee joint pathology (Turner and Smillie 1981, Turner 1994). He found that the average external torsion in patellofemoral instability was 24.5°, and in chondromalacia it was 24°. In other instances of anterior knee pathology which were not included in this study, it was also increased. He states that it is still not known whether it accentuates or compensates for femoral anteversion. Fabry et al. (1994) found
that 40.3% of children with femoral anteversion also had external tibial torsion. Le Damany (1901, translated 1994) suggests that it is to gain greater stability in weight bearing that increasing external tibial torsion normally takes place during childhood, and is due to muscular forces. In the neonate external tibial torsion is zero, or the tibia is even internally rotated (Turner 1994) and by the age of 5 the adult level has been attained. What causes external tibial rotation greater than normal is not known.

Methods

Each patient subject was asked to lie supine and flat on the couch with the whole body aligned. The same calipers (Martin Goniometer) were used for this measurement as for the previous measurement. The foot was dorsiflexed to a right angle and the margins of the lateral malleolus were palpated. A line was drawn on the skin vertically at the centre of the extent of the width. The distal tip was also palpated and a horizontal line drawn at the centre of the raised portion of the bone. The intersection of these two lines was taken for the positioning of the point of the caliper. The same procedure was repeated to find the centre of the medial malleolus. The leg was manually orientated so that the distal pole of the patella was facing upwards, and the subject asked not to move the leg in any way. The ankle was again dorsiflexed, the points of the calipers of the gravity goniometer were placed on the marks, checking that the knee had not moved, and the number of degrees of rotation that the malleoli made from the vertical read on the dial.
The apex of the patella was taken as the reference point, and the lower leg was not fixed, but I am confident that the lower leg did not rotate on measurement. It is true that due to the geometry of the knee joint there is a terminal external rotation of the tibia. In full extension the patella also lies more laterally on the femur than it does at 20°, causing a lateral shift in a frontal plane. Any changes due to the geometry of the tibio-femoral joint in this position of the knee would be the same in both the patient and the normal group. Turner and Smillie (1981) stated that the reason for a mean external tibial torsion of 19° in their study, whereas in the 2 studies where the angle had been measured with the knee flexed to 90° was 5° and 4° less, was probably due to the difference in tibial rotation due to positioning at the two angles of knee flexion. The same also applies to this study. Therefore, it is the difference between the two groups which is a measure of the extent of changes in the patients.

Results
The results of the patients' most symptomatic limb is presented here, and both limbs of the normal subjects.

Summary of Results
Table 8.3. External tibial torsion in patients compared with normal subjects

<table>
<thead>
<tr>
<th></th>
<th>Patients (worst leg)</th>
<th>Normal subjects (both legs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>number</td>
<td>58</td>
<td>29</td>
</tr>
<tr>
<td>mean ext tibial torsion</td>
<td>22.3°</td>
<td>17.9°</td>
</tr>
<tr>
<td>sd</td>
<td>5.5°</td>
<td>4.3°</td>
</tr>
<tr>
<td>sem+/-</td>
<td>0.64°</td>
<td>0.79°</td>
</tr>
</tbody>
</table>

The extent of external tibial rotation was greater in the patient group than in the normal subjects. When a Student's t test was applied, the value for t was 4.27
(p<0.001). Although the results for the less or asymptomatic limb are not presented here, in many cases the worse limb showed greater external tibial torsion. The percentage of patients with external tibial torsion equal to or greater than 20° was 71% (41 patients)

Discussion

Alteration of the position of the patella due to positioning of the limb would make the same difference in the patient and the normal subject group, and there are clear differences between the two groups. The tilt of the patella which causes lateral hyperpressure, often seen in patients with patellofemoral problems might cause the whole lower limb to be positioned to a very small degree in medial rotation, which would tend to minimise slightly the measurement taken, and therefore the difference between the two groups.

Turner and Smillie (1981) did not make any suggestion as to the possible effect of the high incidence of external tibial torsion in patients with patellofemoral problems. The percentage of patients with external tibial torsion above 20° was 71% in this sample of patients, whereas the percentage of the patients with internal hip rotation more than 10° greater than external rotation was only 14% (8 patients), so that external tibial torsion cannot have occurred purely to compensate for the femoral anteversion as suggested by Le Damany. However, a correlation between the two factors performed in Chapter XI, while not significant did suggest that there might be a link. An intoeing gait is both unstable and biomechanically inefficient. In normal gait the propulsive phase of stance takes place in supination; whereas in the intoeing gait there is prolonged
pronation. The number of the patients who had both femoral anteversion and external tibial torsion was 5 (that is 63% of those who had femoral anteversion also had external tibial torsion). It may have been that choosing 10° internal rotation in excess of external rotation was too stringent a yardstick, since the normal group did not exhibit that characteristic, but Fabry et al. (1994) regarded a 10° excess of internal rotation over external rotation as normal. This view was also shared by Carson et al. (1984) and Svenningen et al. (1989).

Fox (1975) suggested that the high incidence of external tibial torsion was a sequel to abnormalities in the quadriceps mechanism. Although he did not instance muscle studies to support his hypothesis, he thought that if there was an imbalance in the muscle activation in favour of vastus lateralis, then there would be a lateral pull on the patellar tendon. Furthermore, he thought that activation of tensor fasciae latae, could pull on the iliotibial band and via its insertion into the proximal shaft of the tibia, exert a lateral pull on the tibia.

In 22 of the patients who exhibited an external tibial torsion greater than 20°, this was more marked on the more symptomatic side (54%) which suggests, in these cases, difference in mechanical usage. It, therefore, may be due to abnormalities of the quadriceps mechanism which are greater on the more symptomatic side, but this is unlikely to be the only factor because of the absence of a positive direct correlation between the two groups (see Chapter XI). 69% of 44 patients had a tight iliotibial band (ITB), and as has been shown by Huggler and Jacob (1983), it is tensioned by tensor fasciae latae. Pare et al (1981) found that the posterior
fibres externally rotate the thigh, and the anterior fibres internally rotate the thigh. Gluteus maximus also inserts into the iliotibial band and can be an external rotator. If, therefore, one takes the view of these two authors that muscle action affects the tension of the ITB, any change in the force vectors of the rotator muscles of the hip will have an effect on the ITB. The effect of a tight iliotibial band on the tibia and the lateral retinaculum, can be seen from this photograph made during the dissection.
interdigitates with retinaculum

insertion onto tibia

Fig. 8.2. To show the insertion of the iliotibial band

The effect that external tibial torsion has on the position of the patella, or the effect of a combination of external tibial torsion and femoral anteversion, is
apparent at first glance in many of the patients: it causes the patella to "squint" (that is instead of appearing vertical the two patellae face towards one another). This has been noted by very many authors, including Carson et al. (1984). Lee et al. (1994) has shown in cadavers that torsional changes of both the femur and the tibia change the distribution of patellofemoral contact force causing an increase in pressure in certain areas. If the areas of contact are reduced, this is accompanied by an increased pressure per unit area, and whether or not there cartilage changes, could cause the pain threshold of the underlying bone to be exceeded. This factor might have been a contributory cause of the inhibition noted in the isokinetic experiment.

The effect that external tibial torsion has on the gait pattern, (if it is not allied to femoral anteversion which appeared to be the case in the majority of this group of patients), would be to cause "out toeing", which would cause increased and prolonged internal rotation of the tibia during the stance phase. However, in some of the female patients there was an objection to "out toeing" on cosmetic grounds, so they adopted a gait pattern to compensate for this. The effect of this was to increase internal rotation of the hip, and since the structural position put the forefoot into a supinated position, increased forefoot pronation was necessary to compensate for this.

It would be instructive to consider the two hypotheses which have been put forward as to the cause of increased external tibial torsion (Fox et al 1975, or Le Damany, reported in 1994). If Fox is right, then one would expect to see a
correlation between an abnormality in quadriceps muscle activation, and external tibial torsion which will be discussed in Chapter XI.
CHAPTER IX

ASSESSMENT OF PATELLA POSITION BY COMPUTERISED TOMOGRAPHY

Introduction

There have been a considerable number of studies reported in which the position of the patella has been visualised, using a variety of different techniques. Probably the best method is that used by Schutzer et al. (1986) using computerised tomography. They tried to match the customary standing posture when the patient was lying in the CT machine, and took cross sectional views at 5°, 15°, 25°, and 30° of knee flexion. They found that between these angles, any abnormality in the congruence tended to decrease, but that the tilt tended to increase. In this study, it was felt that at 18° both abnormalities would be apparent and this was the angle which was chosen. When this project was planned, there had not to the knowledge of the author been any work done which correlated patellar position with any of the other abnormalities seen with this type of patient. However, in 1994 Eckhoff et al. did study femoral anteversion with its effect on patella position, by means of computerised tomography (CT). This work has already been alluded to in other chapters (in the Introduction and in Chapter VII). It was felt at the beginning of this project, that with the additional knowledge gained by this method of visualisation, the correlation of several causes of malalignment and patella position could be found. Permission was given to use the University College Hospital scanner in the lunch hour, and permission was granted by the ethical committee. This was carried out on some ten
patients, but by this time the drawbacks had become all too apparent, and it was discontinued.

The main drawback was not of the technique, but of available time. All the patients were young and most working, and although they were keen to participate, it meant giving up their lunch hour to attend. The scanner could only be used if the morning session had finished on time and there were no emergencies, which the staff did not know in advance. Added to this was the difficulty of frequent breakdowns as the equipment was becoming obsolescent. This meant that all too often the patient came to the hospital to be sent away again, which it was felt was not fair to them. They were already giving up much time to do the other tests.

There were other difficulties inherent in the technique. Shellock et al (1989) in their MRI study said that the greatest difficulty was that of the frequency of patella alta, and in any cross sectional visualisation, the level may be correct for the patella but too high for the condyles. They also discussed other difficulties of positioning the patient to allow for anatomical differences, which may or may not be pathological.

The work done by Brattstrom (1964) in measuring the sulcus angle (the angle made by the two condyles in cross section), and Merchant et al. (1974) in measuring the congruence angle (the angle made by the line of bisection of the sulcus angle and a line drawn from the deepest part of the sulcus through the longitudinal ridge on the
underside of the patella, also in cross section) has already been discussed in the Introduction on page 27. Ficat and Hungerford (1977) considered that angles in excess of 145° indicated a shallow sulcus which was a pathological finding. Merchant et al. (1974) considered that if the line from the deepest part of the sulcus through the ridge of the patella was lateral to the line of bisection of the sulcus angle, then the congruence angle was positive, which was pathological. This indicated that the patella was shifted laterally and was not properly aligned. The Laurin lateral patellofemoral angle (Laurin et al. 1978) was measured by assessing the angle made by the intersection of a line drawn across the condyles at their greatest height and another line drawn along the lateral facet of the posterior surface of the patella. This is a measure of patella tilt. These were the angles which have been measured in this study from the CT cross sections of the small number of patients and some normal subjects, but equally well the patella tilt angle used by Schutzer et al. (1986) could have been used instead of the lateral patellofemoral angle.

Wiberg in his extensive studies of the patellofemoral joint (1941) found evidence of different patella shapes. He classified these according to the width of the medial facet compared with the lateral facet:

a) Type I patella - the medial and the lateral facets have equal width.

b) Type II patella - the medial facet is smaller than the lateral facet.

c) Type III patella - the medial facet is very much smaller than the lateral.

He considered that the Type III predisposes a patient to patellofemoral problems.
Hehne (1990) also studied patellar shape, and considered that it did not have any great pathological significance, and was because the patella has to accommodate to different condylar shapes.

Despite the fact that it by no means represents the patient group as a whole this report has, however, been presented and the result shown for a small number of patients, to demonstrate that in this group of patients there were indeed abnormalities of patellar position. Any inference as to the incidence, or correlation with other results cannot be drawn. More results could have been shown, but unfortunately the CT scans were sent to the referring doctors, who were very interested but did not return them as requested. In the interim the machine became obsolete and the discs could not be read.

**Method**

A polystyrene wedge was made, backed by a thin piece of wood. The height of the wedge was adjusted so that when tested on a number of subjects, it enabled the knee to be fixed in $18^\circ$ of flexion, at which point the patella is beginning to articulate with the femoral groove or trochlea. This was verified by a goniometer. This wedge was placed in a cotton covering, incorporated into which was a strap with a velcro tab which came up and over both the wedge and the patient’s thigh. The top of the far end of the wedge was rounded so that it did not dig into the back of the patient’s knee.
The subject was supine in the scanner, covered by a lead apron which came halfway down the thigh so that the gonads were carefully protected. The wedge was positioned so that the high end came just to the bend in the knee. The strap was positioned so that the thigh did not fall into external rotation. Another strap was placed round the ankles to ensure further that the leg was in neutral rotation. The patella was palpated so that the lateral and medial side of the base was felt between the index finger and thumb. The hand was moved distally half way down from the base to apex of the patella. In the male subject a notch can be felt here, in the female subject it is not as easy. The scanner was positioned so that a beam of light fell at this point. When the scanner was switched on, cross sectional CT was taken at this point.

CT scans were taken of 10 patients and 4 normal subjects, but only the scans of 3 normal subjects could be measured.

**Geometric analysis**

The CT scan was placed over a viewing box, with tracing paper on top, so that the margins of the patella and condyles could be drawn. The resultant tracings are reproduced in the results. The sulcus angle of the femur could then be found (BAC); this was then bisected,(AO) and another line drawn from the apex of the sulcus through the ridge of the patella (DA). Thus the congruence angle was found; it was negative if DA was medial to AO, and positive if it was lateral. A positive congruence angle indicates lateral patellar shift. A line was also drawn along the lateral border.
of the patella (B’ B’), and another from the height of one condyle to the other (A’ A’).

A normal result is when these two lines intersect making an angle laterally (lateral patellofemoral angle). If they do not intersect, or make a medial angle there is patellar tilt.

Results

None of the normal subjects had abnormal congruence angles or lateral patellofemoral angles. A summary of the results which was obtained from the individual tracings of the scans is shown in Fig. 9.

Table 9.1. Sulcus and congruence angles for normal subjects

<table>
<thead>
<tr>
<th>Number of measurements</th>
<th>Sulcus angle</th>
<th>Congruence angle (-ve)</th>
</tr>
</thead>
<tbody>
<tr>
<td>mean in degrees</td>
<td>134</td>
<td>6.2</td>
</tr>
<tr>
<td>s.d.</td>
<td>2.9</td>
<td>1.5</td>
</tr>
<tr>
<td>number with Type II patella</td>
<td>6</td>
<td></td>
</tr>
</tbody>
</table>

The patients' results are shown after Fig. 9.
Normal Subjects

Subject 1

Right

Wiberg Type II patella
Sulcus angle = 137°
negative congruence angle (8°)

Lat

Med

Left

Wiberg Type II patella
Sulcus angle = 132°
negative congruence angle (7°)
Subject 2

Right

Wiberg Type II patella
Sulcus angle = 132°
negative congruence angle = 7°

Left

Wiberg Type II patella
Sulcus angle = 131°
negative congruence angle = 5°
Subject 3

Right

Wiberg Type II patella
Sulcus angle = 138°
negative congruence angle = 6°

Left

Wiberg Type II patella
Sulcus angle = 134°
negative congruence angle = 4°
b) PATIENTS

1. T.C. Right knee is the more symptomatic

   RIGHT

   Patella alta, because a cross section of the patella is too high to show a cross section of the condyles. Although the patella is not making contact with the trochlea at this angle and therefore one cannot assert with certainty that there will be a positive congruence angle and a tilted patella when it does, nevertheless that appears likely.

   Wiberg Type II patella

2. P.D. Right knee is the more symptomatic

   RIGHT

   Sulcus angle is 155° showing evidence of a shallow lateral condyle. The congruence angle is negative (3°)

   Wiberg Type I patella
3. C.W. Right knee is the more symptomatic

Sulcus angle is 131°
There was a negative congruence angle (26°)
The Laurin lateral patellofemoral angle shows evidence of slight patellar tilt
Wiberg Type I Patella

4. C.M
Both knees are symptomatic but the right knee is more symptomatic

Bilateral patella alta
Wiberg Type I patella
5. J.W.
Both knees symptomatic, the right slightly more than the left
The CT from the right could not be analysed because of poor positioning due to genu varum

RIGHT
LAT MED

LEFT
MED LAT

Sulcus angle $133^\circ$
The patella is markedly shifted outwards giving a strongly positive congruence angle $(40^\circ)$
Wiberg Type II patella
Patients results:-

2 patients showed patella alta

1 patient had a sulcus angle greater than 145° (155°), the others who could be measured had a mean of 132°.

1 patient had a tilted patella shown by the Laurin lateral patellofemoral angle.

1 patient had a strongly positive congruence angle (40°), showing a marked lateral shift of the patella. The other patients (those who could be measured) had a negative congruence angle.

1 patient had a Wiberg Type II patella

The two patients with patella alta illustrate the inherent difficulty of the technique which was mentioned in the introduction viz. the cross section of the patella does not coincide with a cross section of the condyles with the trochlea between. Patella alta has been demonstrated but there are easier ways of showing this with a lateral X-Ray (Blackburne and Peel 1977 or Insall et al. 1976).

The other difficulty is illustrated by the CT of patient J.W. of her right knee viz. the difficulty of positioning when there is femoral and tibial malalignment. This certainly was more marked on the right side. There may also have been a slight leg length abnormality which is common.
Advice was sought from a radiologist as to whether any information could be gained about the cartilage from the CT scans. His view was that any conclusion would be unreliable without the presence of contrast media.

However, despite the far from complete results, the few patient results which are available do show evidence of abnormalities of patella position which were not shown by the normal subjects.
CHAPTER X
THE CASE HISTORIES OF FOUR PATIENTS

Subject 1

Male

Right knee only

Age 35

Duration of symptoms -3 yrs

Site of pain- Retropatellar on the lateral side, also around the patella on the lateral side.

Intensity of pain Scale 0-10 (/10) (Squatting 2, climbing stairs 1, descending stairs 3, cycling 3, running 10, sitting with bent knees, standing, and walking minimal.

Total score 1.9.

Disability (restriction of activity by pain) Running, hill walking, squash, football, swimming, all activities severely limited.

Clinical Assessment

Retropatellar tenderness was tested by Clarke's sign, which combines downward patellar pressure with a voluntary static quadriceps contraction. Pain was elicited showing that the test was positive. There was no sign of patella alta clinically.

There was marked appearance of patellar tilt, and there was appearance of wasting of V.M.O. on the affected side.

The tightness of the iliotibial tract or band was tested by asking the patient to lie on his side with the right side uppermost and the left knee bent. He was then asked to
tilt the pelvis towards the left by pressing into the couch with the waist, while the examiner took the weight of the leg, in this way the iliotibial tract was put on the stretch. The patient kept pressing the waist into the couch while the examiner passively lowered the leg, making sure that the hip was neither in flexion or extension. There is no information available on normative values, but it is considered clinically that normal limits of tightness enable the leg to be lowered nearly to the couch. In this case the iliotibial tract appeared extremely tight and became stretched when the leg was about 5 ins from the couch.

**Biomechanical Assessment**

Although the length of the hamstrings was within normal limits, they were shorter on the affected side (on the left, hamstring length was 63°, but on the right it was 55°). External tibial torsion was within normal limits (20°).

With the patient prone and the lower leg bisected and the calcaneum bisected, there was a rear foot varus.

There was poor mobility of the sub-talar joint, which meant that in standing the heel was in supination (uncompensated rear foot varus). The gastrocnemius muscle was short on the affected leg, so that in prone lying only 7° of dorsiflexion could be obtained. On the other foot the extent of dorsiflexion was 10°.

No gait video could be taken but on visual inspection there was internal tibial rotation in mid to late stance. The hip was held internally rotated.
Fig 10  Subject 1 standing

← hip held in internal rotation
Muscle tests

There was a small drop in the force exerted by the quadriceps at 90° on the isometric strength test, which was 520N on the right compared with predicted values of 565N. However the predicted values were on the low side since the quadriceps force exerted on the unaffected side was 690N. Before the onset of symptoms he had been very athletic. There was a marked slowness of activation of V.M.O. at 20° of knee flexion during a rapid application of extensor torque. the lag factor was 0.55. The dip in the EMG amplitude during the isokinetic movement was 0.179.

Treatment

The patients' knee was strapped by the method advised by McConnell (1986) to correct the tilted patella. Strapping made by Biersdorff was used. This consisted of a very adhesive inner layer which covered the patella and a surrounding area of skin. Over this, very strong zinc oxide plaster was applied firmly with a certain amount of traction, from the vertical midline of the patella extending over the medial side of the knee. He was advised to wear it all the time, re-applying it as necessary in the way shown, and covering it with a plastic bag on showering.

Another aspect of the treatment regime aimed at quadriceps training, and included short arc quadriceps exercises, and static quadriceps contractions concentrating on VMO. He was also advised to try and stretch the tight structures, which were hamstrings, gastrocnemius, and the iliotibial tract. For the latter the test position was adopted, and the help of his wife enlisted to try to increase the passive range.
Re-assessment after 8 weeks

Intensity of pain  He had minimal pain on descending stairs fast, and sitting with bent knee for long periods, also sprinting.

Restriction of activity was now minimal; he could now run for 5 miles, and only occasionally easing off hard running because of the pain. He could now swim and again only felt the knee occasionally on hard kicking.

Biomechanical re-assessment

The hamstring length on the left was 66°, and on the right 68°. The extent of dorsiflexion had increased also (right=12°, left = 14°) . Whether the iliotibial tract can be stretched is open to question, because of the toughness of this structure, but the range of passive movement appeared to be greater. It has been doubted whether it is possible to stretch muscle either (Halbertsma 1989). They assert that it is the tolerance of the individual to tightness which is being re-educated.

All the structural changes noted were of course unchanged. The patient had been offered orthoses for the feet but these had been declined.

The only change in the gait pattern which was observed was the fact that he no longer held his knee in internal rotation.

Muscle tests

The lag factor was considerably decreased and was now only 0.076. The dip in the isokinetic movement was normal (0.003)

The quadriceps strength on the right had been considerably improved and the force exerted by both quadriceps was now 680N.
Result of conservative treatment

Good

Subject 2
Female
Age 28
R>L
Duration 7 yrs, fell on knee, then onset gradual. Although it is likely that there was initial trauma it was still regarded as of insidious onset. It cannot be proved that in the insidious onset of a condition that initial trauma does not act as a trigger even if it is not noted at the time.

Site of pain Retropatellar, and around the margins of the patella.

Intensity of pain. Scale 0-10 (/10) 1.6 (squatting 4, climbing stairs 2, descending stairs 1, sitting with bent knees 5, standing 3, walking 1)

Disability
She was stopped going up stairs because of the pain, occasionally, and any activity involving deep bending.
X-Rays were not available, but she had recently had an arthroscopy which showed slight cartilage changes on the posterior surface on both facets.

Clinical examination
Genu recurvatum both knees, genu valgum right.
Patellar mobility seemed normal, but the right patella was tilted and the left patella less so.

The iliotibial tract did not seem tight.

Since there were no X-rays available, the Insall index could not be measured, so patient was asked to sit on the side of the couch, and clinical signs of patella alta were looked for. If the patella is high riding the patella is more horizontal than it should be, when viewed from the side, and the infrapatellar fat pad is uncovered. There was evidence of patella alta on the right side but not the left.

Some effusion inferiorally.

There was some crepitus on movement, but no crackling or popping.

Retropatellar tenderness

She stood with outwardly rotated hips, and the feet appeared pronated.

**Biomechanical assessment**

The range of external rotation at the hip was 20° more than internal rotation.

The hamstrings were tighter on the right than the left, but not excessively (60° on left, 52° on right. However biceps femoris was tighter than semitendinosis and semimembranosis.

External tibial torsion was 20° which was not much greater than the norm.

When the patient was standing, on bisecting the calcaneum, and testing the angle with a protractor, there was 6° of pronation. There was also evidence of subtalar pronation from the position of the head of the talus.
Muscle tests

There was no loss of strength on the isometric strength test at 90°. The predicted strength for her height was 427N, on the right the force exerted by the quadriceps was 415N and on the left 427N.

There was no lag factor on either side, the lag factor for V.M.O. on the right was -0.247 (on rapid application of extensor torque at 20° of knee flexion). The size of the dip for VMO on isokinetic testing was 0.123. Pain was experienced at 60° of knee flexion but not at any other angle.

Treatment

The patella was strapped, as the previous patient, in the way recommended by McConnell to control the tilt. There was also strapping firmly applied across the base of the patella, because it was felt that there was some fat pad entrapment. She was advised to wear the strapping all the time.

Advice was given to do short arc quadriceps exercises, even though there was no loss of strength, because of the ligamentous laxity.

She was also advised to seek advice over the foot problem with a view to obtaining orthoses.

Stretching of the hamstrings was also given because of the inequality between the medial and lateral hamstrings.

The patient's progress was monitored once a week for 6 weeks and then she was retested after 8 weeks.
Re-assessment after 8 weeks

She rated herself 80% better, however on filling in the questionnaire again she still rated the pain total at 1.3.

She no longer avoided stairs and was able to do all the activities she wished.

No effusion

Muscle tests

The quadriceps were stronger, R=500N, L= 470N

The dip in the EMG for V.M.O. was not changed.

The hamstring measurement on both sides was 62°

Response to treatment

Fair to good

Subject 3

Male

Age 37

R>L

Duration of symptoms 17 years

Site of pain Retropatellar on the lateral side, peripatellar on lateral side and superior

Intensity of pain Scale 0-10 (/10) total score 4.3 (squatting 7, climbing stairs 5, descending stairs 4, cycling 6, running 8, sitting with bent knee 3, standing 3, walking 3)

Disability (limitation of activity by pain) any sport severely, cycling occasionally
Clinical examination

Retropatellar tenderness, marked tilting both patellae, tight iliotibial band R>L

Both quadriceps appeared very wasted, particularly over V.M.O.

Both patellae showed marked “squinting” (that is instead of appearing vertical patellae faced inwards towards one another).

Pain in the right knee was experienced on testing (in the Cybex) at 60° of knee flexion (he did not complain during the test in the chair at 90° although there was inhibition), and 90° and 60° in the left knee (in the Cybex) and 90° in the chair.

Biomechanical assessment

Internal hip rotation - external hip rotation = 11°R, 10°L, external tibial torsion 24°R, 22° L

Length of hamstrings R = 45°, L =55°,

Extent of dorsiflexion beyond 90° = 15° R and L

Pronation in standing = 5° R and L

Muscle tests

On testing the force exerted by the quadriceps isometrically at 90° of knee flexion, R = 252 N (with 20-30° inhibition), L 236N. The predicted values for a man of his height was 701N so that the percentage loss of strength was 68%. When the estimated force deficit which could be attributed to the inhibition was added to this, the true force estimation was 315N, which leaves a % difference of 55% between the true force estimation and what was predicted, so that there must have been much wasting of the quadriceps.
On the right the lag factor for VMO was 0.255.

The dip in EMG from VMO during the isokinetic test was 0.1911

**Treatment**

The patella was strapped in the way recommended by McConnell, to control the tilt. Also, other strapping as advised by her, was applied to encourage anti-rotation in a frontal plane. Advice given to the patient was the same as with the previous two patients.

Static quadriceps exercises were also given to be done with persistence concentrating on V.M.O., together with short arc exercises.

Hamstring stretching and iliotibial band stretching were advised. It was suggested that in-shoe orthoses to correct pronation might improve symptoms

**Re-assessment after 8 weeks**

Iliotibial band appeared the same, also tilting of the patella.

Hamstring length 65° L, and 55° R

Intensity of pain 4.4, unable to any sports as previously

**Muscle tests**

There was no improvement in the strength of the quadriceps; on repeating the isometric strength test, the force exerted by the right and left quadriceps was 236N. There was no apparent improvement of muscle bulk, indicating that there was still much wasting, and the percentage deficit compared with the predicted strength remained the same.

The lag factor in the EMG from V.M.O. on restesting the right limb was 0.129
The dip in the EMG for V.M.O. on isokinetic testing was less than before 0.065.

**Response to treatment**

No improvement

**Comments**

The combination of a very marked excess internal rotation of the hip (marker for femoral anteversion), and external tibial rotation, caused a very poorly aligned patella on both lower limbs, and therefore presumably an abnormal P.F.J.R.F. All the muscle tests showed poorly functioning quadriceps. The strapping could not control the abnormality and therefore on the whole the inhibitory influences on muscle function were not ameliorated, so that function did not improve, although there was some improvement in the dip in the EMG from V.M.O.

**Subject 4**

Female

**Age** 30 years

**Site of pain** Retropatellar, peripatellar, medial round to lateral side.

L very much more than R. Same distribution of pain on L but less severe.

**Intensity of pain** Scale 0-10 (/10) 5.4.(squatting 8, climbing stairs 8, descending stairs 8, sitting with bent knees 4, standing 4, walking 4, cycling 8, running 8

Disability (limitation of sports severely, and also general exercise).

**History**

**Duration of symptoms** 16 years
She dislocated her left patella three times when she was aged 14, and the right once. This could have been due to trochlear dysplasia. Since then she has not redislocated. However she stopped playing hockey and netball and it was hard for her to find sports that she could do. 3 or 4 years ago she started tennis, squash and skiing, which caused a gradual build up of pain. She was advised to build up the quadriceps; some exercises aggravated it especially lifting weights from bent knee. She gave up sports except for skiing.

Recent arthroscopy (L) showed marked malacic changes on medial facet. Pain much more severe after arthroscopy.

Clinical examination

Marked wasting of L quadriceps particularly V.M.O.
Marked retropatellar tenderness. Slightly tilted patella L and R.
Slight effusion L
Proximal tibia vara L, very tight iliotibial band L

Biomechanical assessment

Hip internal rotation- external rotation L = 3°, R = -10°

Hamstring length both 40°

External tibial torsion 27° L, 25° R

Dorsiflexion beyond 90° = 10° R and L.
Subtalar pronation in standing = 5°
**Muscle tests**

Isometric test of quadriceps strength at $90^\circ = 500\text{N}$, $L = 188\text{N}$ (10-15% inhibition).

The predicted values for a woman of her height was $478\text{N}$, which was close to the force which was exerted by the right quadriceps. This means that the loss of strength of the left quadriceps was 61%. Even when the force loss due to 10-15% inhibition was added, the estimated true force only amounted to $212\text{N}$, so that in real terms the percentage deficit was still 56%.

No lag factor from the EMG of the left V.M.O.

Isometric Cybex testing caused pain at $60^\circ$ of knee flexion which was greater than $30^\circ$ knee flexion; on the right there was the same pattern. There was no pain at $20^\circ$ of flexion.

Dip in EMG from the left V.M.O. in isokineti$^\text{c testing} = 0.3169$

**Treatment**

McConnell strapping R to correct tilt, L to correct tilt, and also antirotational strapping.

Stretches for iliotibial band, and hamstring stretches. Concentrated exercise for quadriceps both statically and short arc exercises.

Saw podiatrist and given in shoe orthoses.

**Reassessment after 8 weeks**

Orthoses made symptoms much worse so stopped wearing them.

Initially pain was 80% improved with strapping, but that improvement was not maintained, now 30-40% better than before. The subjective assessment was that the
right knee felt functionally better and stronger, but the left knee did not feel any stronger.

**Muscle Retests**

There was some improvement in the strength of the left quadriceps when tested isometrically at 90° of knee flexion. The force exerted by the left quadriceps was 250N.

She declined to undergo the isokinetic test, because she was afraid of exacerbating the pain.

Hamstring length improved on both sides by 10°, and the iliotibial band appeared to be less tight.

**Response to treatment**

Slight improvement

**Discussion of the treatment for all four patients**

All the patients appeared to improve the length of the hamstrings, even when there was no improvement in symptoms, so one would doubt whether this characteristic has very much effect on this type of patellofemoral problems, in fact as suggested in Chapter V, to stretch the hamstrings may have an adverse effect on stability.

The strapping as advised by McConnell appeared to be more efficacious when used to influence soft tissue, and not so effective on marked structural changes. Where there was improvement in the symptoms the patient could make progress in improving the quadriceps function.
CHAPTER XI

CORRELATION AMONG DIFFERENT RESULTS

In the previous chapters clear differences, both in the muscle function and also biomechanically, have been shown between the group of patients and the normal subjects. In this chapter the author is going to attempt to correlate the sets of results with one another. There has, as far as she is aware, been no quantitative work of this nature attempted, which is a pity because, although only a longitudinal study could conclusively demonstrate the pathogenesis of patellofemoral dysfunction, nevertheless the causes are likely to be multifactorial, and study of this sort would give indications as to what factors are primary and what are secondary. It would also give information to enable grouping of patients, so that their treatment could be made more selective.

There were many exclusion factors with the sample of patients studied, and therefore it is not claimed that the results obtained are representative of all patients with patellofemoral problems. Not every patient who qualified for inclusion agreed to make the considerable outlay of time which was necessary. The fact that so many of them did shows the strength of their motivation.

The prevalence of malalignments indicates the likelihood that there are failures in the correct development of the musculo-skeletal system of the patients studied. While there are few subjects whose development would be so perfect as to have optimum
biomechanical effects, perhaps the defects are one of degree and more marked
defects predispose a patient to accumulative minor trauma. If the whole population
had limbs where the biomechanical effects were optimum, we should all be elite
athletes.

Many authors have postulated (Frost 1964-90, Pauwels 1965) that it is the
mechanical usage which determines the growth of bone, not only the length but the
direction in which it grows. And while bone retains a certain amount of plasticity
throughout life; it is during childhood until skeletal maturity, that it is most plastic.
It is likely, therefore, that long bone malalignment occurs during bony development,
but the question as to whether the malalignment alters the direction of pull of the
muscles, or whether there is muscle imbalance which alters the direction of bony
development, is unanswered. There is no direct evidence to support either
hypothesis. Le Damany (1901, translation 1994) postulates that external tibial
torsion occurs because it is more stable to out-toe in walking than in-toe. However,
although external tibial torsion was so prevalent amongst the sample of patients
tested, many of the patients had a marked disinclination to out-toe, especially the
women, some of them because they felt it was cosmetically unattractive. However,
this may have been an adult adaptation. Fox’s hypothesis, (1975) is that if the pull of
vastus medialis is inadequate, and he considers that there is a big genetic component
in this, then there is a unequalised lateral pull on the patellar tendon which causes
external tibial torsion. Frost (1989), shows how the direction of pull of the muscles
causes uneven compression and traction so that growth occurs in uneven spurts both medially and laterally. Bose et al., (1980) in their dissection of many types of primates demonstrated the absence of any oblique fibres of V.M. They considered from this that activation of V.M.O. was a late adaptation, in evolutionary terms, by man to accommodate to an upright gait. Hehne, after an exhaustive analysis of the biomechanics of the patellofemoral joint, concluded that patellofemoral dysfunction cannot be explained by biomechanics alone, but by failure of adaptation to the upright gait.

80-90% of the patients dated the real onset of their problems to adolescence. At this time demands on the muscles have become greater, due to growth and the desire to perform well in sports activities. However, at this time the fusion of epiphysis and diaphysis means that the plasticity of the bone has markedly decreased. Any imperfect musculoskeletal development then becomes apparent. An important effect of this might be instability of the patellofemoral joint. One important cause of instability, is trochlear dysplasia. Accurate patellar tracking is likely to be an important prerequisite for adequate trochlear depth. If the patella is not properly centred, the lateral condyle might not develop adequate height. Another cause of instability is thought to be patella alta. The incidence of patella alta in patients with patellofemoral problems has been estimated in various studies to be between 45-50%, and while in this study the impression was gained that it was not as high as that, it was nevertheless considerable. Any instability of a joint has to be
compensated for, and it is suggested that many of the soft tissue adaptations seen e.g. a tight lateral retinaculum, and/or a tight iliotibial band, might be to compensate for this instability. The co-contraction of the quadriceps during knee flexion described in Chapter V might be to improve the stability of an otherwise unstable joint. Unfortunately compensatory mechanisms frequently have disadvantages. This often makes rehabilitation complicated.

One of the constraints of studying muscle action by EMG is that the experimenter cannot compare one subject with another, so that it cannot be said that the EMG showed that magnitude of the V.M.O. signal was either adequate or inadequate. Nevertheless, when compared with the maximum i.e. normalised, it does give much information not obtained by measurements of the muscle force developed by the muscles. In the experiment described in Chapter IV the slow activation of V.M.O., in particular, in the patient group is indicative of faulty timing of the activation of the muscle. This is not likely to be due to the same mechanism as the inhibition described in Chapter V, since these two results were not well correlated, i.e. the patients with the largest lag factor were not necessarily the ones with the greatest inhibition.

Results

1. There was a poor correlation between the occurrence of femoral anteversion and demonstration of slowing of the activation of V.M.O. at 20°of flexion (r = 0.119, \( r^2 = 0.0142, n = 48, t = 0.813, p = 0.42 \)).
2. The patients with recurrent dislocation of the patella were not a large group n=3, but none of them had external tibial torsion, although two of them showed slowing of the activation of VMO. They were treated as a separate group.

3. One patient was atypical in that he did not have recurrent dislocation or retropatellar pain, although he had pain over the insertion of V.M.O. All the rest had retropatellar pain; this means that the percentage of the patients with retropatellar pain was 96% (Bentley and Dowd gave 98% as the estimate in their paper).

4. If the reason for the retropatellar pain is, as Insall et al. (1979) asserts, due to the pain threshold in the subchondral bone being exceeded, because of a sideways movement, then the slowness of activation of V.M.O. might mean that the patellar is not in the right position when it enters the trochlea. Moreover this abnormality might be the cause of an increased external tibial torsion during development (due to a lateral pull of the quadriceps). If this is so, one would expect to find a correlation between the lag factor and external tibial torsion in patients with retropatellar pain.

For this reason the correlation between the extent of external tibial torsion and the extent of slowness of activation of VMO (i.e. the lag factor) was investigated in the patients with retropatellar pain, who did not have recurrent dislocation. \( r \) was equal to 0.179, \( n = 42, t = 1.154 \). This gave \( p=0.25 \).

5. There was poor correlation between the occurrence of the dip in the activation of V.M.O. greater than other parts of the quadriceps, and external tibial torsion. \( r \) was = 0.072, \( n = 49, t = 0.495 \).
6. When the lag factor was correlated with the inhibition of V.M.O. seen in the isokinetic experiment, there was poor correlation. \( r = 0.0548, n = 49, t = 0.3762 \)

7. When the measurements of external tibial torsion, were correlated with the extent of internal hip rotation compared with external rotation, \( r^2 = 0.0579, r, \) therefore was 0.3498, which yielded a t factor of 1.97, and \( p = 0.055. \)

6. The extent of patellar tilt could not be quantified, however in 26 patients it was clinically graded from 0-2, and correlated with the lag factor. \( r^2 = 0.1272, \) and \( r = 0.355, \) which yielded a t factor of 1.87, and \( p = 0.06. \)

The last two correlations gave the lowest \( p \) values.

**Discussion**

The figures show that while there no direct correlation between the different sets of results there is likely to be considerable overlap in these different findings. However, since there was poor correlation in the dip in the activation of the muscles (which might be due to inhibition) in the isokinetic experiment, and the slow activation of the muscles at 20° of knee flexion, these two sets of results are unlikely to be due to the same mechanism. As already stated in Chapter III, previous work by many authors has shown that muscle inhibition can have many causes, rather than a direct consequence of one pathological change.

The correlation between the patellar tilt and the lag factor, while not significant at the 95% confidence limit, does suggest that there may be a link between tight lateral
structures and the ability to activate VMO with sufficient velocity and that further investigation would be worthwhile.

If Le Damany's (1903, translation 1994) hypothesis was extrapolated to explain how excessive external tibial torsion occurs, it is to be expected that where there is insufficient derotation of the femur there would be greater external tibial torsion to compensate. The correlation between these two sets of measurements while again not significant, suggests a trend towards this compensation. The group of patients who experienced recurrent subluxation was very small which makes correlation among the sets of results difficult. This small number did not exhibit external tibial torsion, but this may not have held good with a greater number. Measurements of sulcus angle or estimation of sulcus depth which might have been abnormal were not quantified.
The absence of correlation between external tibial torsion and the abnormality of activation of V.M.O., does not preclude the possibility that it was initially a contributory factor, although it does not prove that it did happen. Patellofemoral problems are likely to be multifactorial, as already stated, which makes correlating the various factors very complicated. There may be several contributory factors to inadequate activation of V.M.O., both past and present.
CONCLUSIONS

While there is no direct evidence that the mechanism of activation of V.M.O. to help to control the position of the patella, is that which has been suggested in Chapter IV, (that is that it is a learnt mechanism similar to a ballistic movement), and that failure of this mechanism is a primary cause of problems, the burden of circumstantial evidence built up in this thesis, suggests that it is a factor in the pathogenesis of this condition: but other pathological changes are also likely to affect the function.

The findings here presented do have a practical application in the rehabilitation of these patients. Several salient facts emerge. One is that if no effort is made to improve the malalignment or increase the stability of the joint, incorrect tracking will cause inhibition particularly to V.M.O., leading to wasting in this portion of the quadriceps. Therefore, efforts towards improving its function will be of no avail.

The strapping introduced by McConnell (1986) was relatively successful in controlling malalignment due to soft tissue tightness. However, although it would be hard to draw conclusions on the basis of one patient, it did not appear to be successful in this case in correcting marked bony torsions, for which she also recommends strapping.

If transformation of the fast muscle fibres of V.M.O. and V.L.O. contributes to the lag factor in Chapter IV, and is caused in part by the co-contraction in Chapter V, then after the strapping the rehabilitation should include work done at increasing
The results of the experiments which were carried out in Chapter VI could also be applied to muscle re-education. If one is seeking to improve the function of V.M.O., then performing straight leg raising with the thigh externally rotated, in which position that part of the quadriceps is activated less efficiently, is not the best method. At present this is a frequently used exercise.
velocity. Very high velocities are built up in normal knee function and it does not appear logical to rely on static muscle work in rehabilitation. The muscle re-education at increasing velocities can be undertaken in a pain free arc of movement.

**FUTURE RESEARCH**

Longitudinal studies are infrequently done because of time, difficulty, and expense, but it would be a valuable exercise with these patients. There is also much more which needs elucidating as to how both the long and oblique fibres of the vasti adapt to changes in position of the limb. Further understanding of the control of the patella in dynamic situations would also be valuable, including the effect of increasing velocity.


Conway, J.M., Harris, K.H., and Bodwell, C.E., 1984, A new approach for the
1130.


Darracott, J. and Vernon- Roberts, B., 1971, The bony changes in “chondromalacia

Dehaven, K.E., Dolan, W.A. Mayer, P.J., 1979, Chondromalacia in athletes: clinical

Dehaven, K.E. and Lintner, D.M. 1986, Athletic injuries: comparison by age, sport,

Dejour, H., Walch, G., Nove-Josserand, L. and Guier, Ch., 1994, Factors of patellar

de Sousa, O.M. and Vitti, M., 1966, Estudio electromiographica de los musculos


Hungerford, D.S. and Barry, M., 1979, Biomechanics of the patello-femoral joint, 

Hunt, S.F. and Rossi, J., 1985, Peptide and non-peptide containing unmyelinated 
primary afferents: parallel processing of nociceptive information, In *Nociception and 


Insall, J., Bullock, P.G. and Burstein, A.H., 1979, Proximal “tube” realignment of the 

Itoh, H., Ichihashi, N., Maruyama,T., Kurosaka,M. and Hirohata, K., 1992, 
Weakness of thigh muscles in individuals sustaining anterior cruciate ligament injury, 


Grants GM26395 and GM23732 Rancho Los Amigos Hospital Downey California.


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APPENDIX I

Glossary of Abbreviations and Terms Used in this Thesis

Anatomical Abbreviations
ACL  anterior cruciate ligament
ITB  iliotibial tract or band
R.F.  rectus femoris
V.I.  vastus intermedius
V.L.L.  the long fibres of vastus lateralis
V.L.O.  the oblique fibres of vastus lateralis
V.M.L.  the long fibres of vastus medialis
V.M.O.  the oblique fibres of vastus medialis

Biomechanical Abbreviations
P.F.J.R.F.  patellofemoral joint reaction force

Electrical Terms
EMG  electromyography
pulse width  the duration of each individual electrical impulse

Imaging
CT  computer aided tomography
MRI  magnetic resonance imaging

Muscle
MVC  maximum voluntary contraction
twitch interpolation  the technique of superimposition of an electrical stimulus on a
MVC to test for muscle inhibition.
A COMPARISON OF THE ACTIVATION OF MUSCLES MOVING THE PATELLA IN NORMAL SUBJECTS AND IN PATIENTS WITH CHRONIC PATELLOFEMORAL PROBLEMS

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ABSTRACT. The activation of the oblique fibres of vastus medialis, the posterolateral fibres of vastus lateralis, and rectus femoris was studied by surface electromyography, during the force development of a maximal isometric contraction, performed near full extension (20° of flexion), by 49 patients with chronic patellofemoral problems and 20 normal subjects. In the normal subjects activation of oblique portions of the vasti was in advance of force rise, during the time for 80% tension development. In the patient group, however, the activation of these lagged behind force rise. Force rise was slower in the patients even though the contraction was generally pain free. In all groups the activation of these two sections of the muscle remained approximately synchronous, suggesting that they have a reciprocal action in controlling patellar position, disruption of which might contribute to patellofemoral problems.

Key words: activation, electromyography, force, muscle, patella.

INTRODUCTION

The quadriceps muscle has as one of its main functions the extension of the knee, via its action on the patella and the patellar ligament. The muscle is very large and is conventionally regarded as having these four sections which are anatomically distinct: vastus lateralis (VL), vastus intermedius (VI), vastus medialis (VM) and rectus femoris (RF). Different sections of the muscle pull on the patella from somewhat different angles, and therefore the possibility should be considered that an additional function of the quadriceps is to control the position of the patella with respect to the trochlear surface of the femur (e.g. 1, 12). The most oblique parts of VM (VMO) and of VL (VLO) as well as RF would be the sections most suited anatomically to exert this control. VMO has been seen, in many cases to be separated from the long fibres by a fascial plane, and most of the fibres of VMO arise from the tendon of adductor magnus (1). The innervation appears to be variable, and in some cases a separate motor nerve to the belly of VMO has been observed (12, 17). VL has also been shown to be divided into two parts; the proximal fibres are straighter, originate from the femur and are inserted into the middle layer of the quadriceps tendon, whereas the distal, or posterolateral fibres originate from the iliotibial band, are more oblique in their direction and are inserted into the base and lateral border of the patella (10). It has therefore been suggested that they have a controlling effect on the patella, acting in opposition to that of VMO. Rectus femoris in contrast exerts a relatively straight pull on the patella.

An example of the need for control of patella position is that when the knee is nearly straight all the quadriceps, except VMO, have a lateral pull on the patella, which can only be dynamically counterbalanced by the action of VMO (7). Stability is also provided by the retinacula, but the transverse fibres can scarcely be dynamized by muscular action (6). As the knee is bent, the patella moves downwards and medially (11), entering the trochlea by about 20° of flexion. The trochlea is deeper distally, so that the congruity of the bony parts gives the joint more stability at increased angles of flexion. We have therefore investigated the timing of activation of VMO, of VLO and of RF during isometric contractions with the knee flexed at 20°. If these parts of the muscle are actively to control the position of the patella we should expect them to be rapidly activated, at the same time as, or even in advance of, the larger parts of the muscle which generate the majority of the

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isometric force. We have therefore compared the timing of their activation with that of the onset of extensor torque. The method of motor control of the patella is not known, but there may be an initial ballistic movement. This learnt control of movement has been described by Rothwell (16). "This pattern of EMG activity...represents a package of nervous commands which can be 'preprogrammed' in advance by the central nervous system... this does not mean that commands cannot be modulated under normal conditions by peripheral feedback mechanisms."

Because this type of motor control is stereotyped, we monitored the first 80% of the force rise, in the expectation that this might exhibit a relatively simple behaviour.

**MATERIALS AND METHOD**

Forty-nine patients with chronic patellofemoral pain were studied. This pain was exacerbated by climbing or descending stairs, or undertaking sports and other activities involving running and/or deep flexion of the knee. In some cases the retropatellar pain was exacerbated by prolonged sitting. Each had been diagnosed by an orthopaedic surgeon, and each was also re-examined for suitability for inclusion. Criteria for their choice were, not only the presence of the above symptoms but also retropatellar tenderness, and that the onset of their symptoms was insidious rather than a response to trauma, and had occurred between the ages of 12 and 30. In 34 patients the symptoms were bilateral, but usually the symptoms were more severe in one knee than the other. The duration of their symptoms was 6 months to 10 years. Exclusion criteria were: bipartite patella, femoral trochlea fracture, osteochondritis dissecans, muscle tear, meniscal or ligamentous pathology, reflex sympathetic dystrophy, Osgood Schlatter disease, Sinding-Larson-Johannson disease, patellar tendinitis, prepatellar bursitis, scarred or inflamed plicae, gross effusion, or recent knee operation, not including arthroscopy.

The age range of the patients was 20–37 with a median age of 26, and 67% were female.

A group of 20 normal subjects were recruited, of age range 20–33 with a median age of 25 and 65% were female. Although they were mostly university staff and students, care was taken that not only athletic subjects were chosen, so that the range of accustomed physical activities was similar in patient and control groups. The controls were carefully questioned to ensure that they did not suffer, and had not previously suffered, from any knee symptoms.

Both legs of the patients were studied and only the right leg of the controls. In this report only the most affected leg (or the only affected leg) of the patients is presented. The subject was positioned on a couch with legs stretched out and was instructed to brace his/her quadriceps so that the individual muscles of the group could be delineated and electrode placings selected. Each pair of electrodes was mounted in a fixed unit (Motion Lab Systems Inc.). The electrodes, which were AC coupled, were raised stainless steel discs 3.5 cm apart, with a diameter of 1.2 cm with the indifferent electrode situated between the active electrodes. They were used without gel but provided a very good contact, which was accentuated by being fixed, first with micropore, and then firm, fitted tubigrip. This ensured that the good contact was maintained during any movement which might occur. The electrodes over VMO were placed at an angle of 5° to the line of the femur, that is along the line of the fibres; so that the distal edge was 1.5 cm from the medial edge of the base of the patella. The location for the electrodes over VLO was found by taking a line from the iliotibial band to the lateral edge of the base of the patella, as far down the muscle belly as possible. The position for the electrode on the muscle belly of RF was located over the motor point which had been previously found, in a small sample of subjects, to be slightly medial and about midway along the length of the thigh. Since RF is a bipennate muscle, the electrodes were angled at 15°. The skin was rubbed with alcohol, before the application of the electrodes.

The subjects were then seated in a Cybex II Plus Isokinetic Dynamometer, with the thigh carefully aligned so that the hip was in neutral rotation and neither abducted or adducted. The force arm of the Cybex was replaced, for greater accuracy, by a custom-built force arm which contained strain gauges. This arm could be very firmly tightened and had a better frequency response. It was frequently calibrated and was found not to vary over the experimental period. The fulcrum of this force arm was placed over the jointline of the knee. Ninety-degree flexion was found and verified with a goniometer (Akron), to take account of the slope of the thigh when the subject was seated. The goniometer was aligned along lines taken from the greater trochanter, through the jointline and terminating with the external malleolus of the fibula. The knee was extended in the Cybex to 20° of flexion and fixed. The knee angle was again verified by the use of the goniometer. The subjects were instructed to apply maximum force as fast as possible and hold it for 2 seconds. The amount of extension caused by a maximal extensor effort was about 4°. The exhortations to produce the force quickly were repeated over a few trials when patients, or subjects, appeared to produce the force rather slowly.

The electrodes contained preamplifiers with a gain of about 400. The leads from these preamplifiers were well screened. Each time it was used care was taken that a noise-free preamplifier output was obtained. These signals were further amplified with a gain trim so that there was no inter-electrode variability. Also the signals were rectified and smoothed with a leaky integrator (100-millisecond time constant). After digitization they were displayed and stored on a computer, together with the record of force exerted. This apparatus was regularly calibrated.

**Statistical methods**

Parametric statistical methods were used throughout. Variation in the measured parameters are expressed as ± the standard error of the mean (s.e.m.). Groups were compared using Student's t-test (unmatched samples) and the values of the probability of the null hypothesis (p) were obtained from Tables of the "two-tailed" t-test. Correlation coefficients R were calculated using the Pearson product–moment formula. All the statistical formulae used in these calculations are those of Snedecor and Cochran (17).

**RESULTS**

A typical result from a normal subject is shown in
Fig. 1A. Each record has been expressed as a proportion of the maximum during this contraction. The force rises to 80% of its maximum within less than 200 milliseconds. Within this time all the electromyographic (EMG) signals rise rapidly to a peak. In Fig. 2 the EMG record from VMO is plotted against the force record for the first 80% of the tension rise, to see whether the activation of this muscle keeps pace with the development of force, which indicates the timing of the activation of the other parts of quadriceps. The resultant line falls above, or close to, the line of identity ($y = x$) indicating that all parts of the muscle are activated more or less together, with some tendency for VMO and VLO to be activated more quickly than the bulk of the muscle.

A result from a patient is shown in Fig. 1B. In this example, which is typical, the force develops rather more slowly than in the normal subject and the EMG records show a delay in activation of those parts from which we recorded, relative to the force development, i.e. relative to the activation of the bulk of the muscle. This is seen most clearly by the plot in Fig. 2, in which the line falls well below the line of identity, instead of along the line of identity as it did in the normal subject, when oblique fibres kept pace with the activation of the long fibres producing the force. This has been quantified by measuring the area between the line of identity and the EMG data. This area, which we refer to as the lag factor will be zero when the EMG data falls about the line of identity, positive when it falls below the line and negative when it falls above the line. The scale of the lag factor is such that a value of +1 would indicate no activation at all, and −1 would indicate instantaneous full activation. For the patient shown the value is 0.321, whereas for the normal subject shown in these figures the value is −0.046.

A comparison of these factors for the 20 normal subjects and the 49 patients is shown in Table I. The force exerted by the patients is slightly, but significantly, less than for the control subjects and its onset is on average more than twice as slow. There is also, for each of the regions from which recordings were made, a large and significant difference between the patient group and the normal subjects in the timing of activation relative to the activation of the main bulk of the muscle. The difference seen for the oblique fibres (VMO and VLO) is greater than for RF.
Although these differences are large there is still some overlap between the control and patient groups for each of these measurements. This is illustrated by the histogram of the lag factors for VMO shown in Fig. 3.

Considering all the observations (patients and control subjects) as a group, there are correlations between the three different lag factors. This correlation is strongest between VMO and VLO ($r = 0.743$) and least between VLO and RF ($r = 0.432$). There is also (an inverse) correlation between the time for 80% tension development and the VMO lag factor. This might suggest that the increased lag factor was somehow due to the slower rise of tension. This was tested by selecting those of the patients ($n = 21$) whose rise times were within the normal range. Eleven of these patients had a positive lag factor. Comparing the mean VMO lag factor for this group ($-0.002$) with that for the controls ($-0.158$), shows the former to be significantly ($p < 0.05$) greater. Thus this cannot be the only reason for the change in the lag factor.

**DISCUSSION**

As we expected from the hypothetical function of VMO and VLO each of these sections of the quadriceps in the normal subject is activated somewhat ahead of the main bulk of the quadriceps, as is shown by the significant negative lag factors in Table I. RF by contrast, in our normal subjects is activated along with the bulk of the muscle. Grabiner et al. (3) have previously reported that activation of VMO leads that of vastus lateralis in normal subjects; he studied isokinetic extension of the knee, but did not find any statistically significant effect during isometric contraction. For VLO, which we are the first to investigate physiologically, we find a similar mean negative lag factor as for VMO and a strong correlation between the lag factors for these two parts of quadriceps. Thus they seem to share a common activation pattern, as suggested by the anatomical finding of Javadpour et al. (10). We feel that the need for this common activation will be particularly great with the knee at the angle we have used because otherwise an unapposed pull of VMO would not only produce the necessary slight medial patellar shift, but also an unwanted medial tilt because of the insertion of VMO on the medial border of the patella.

The patient group showed large and significant differences from the control group in the lag factors of the muscles tested. These oblique portions were activated more slowly in the patients relative to the main bulk of quadriceps. Quadriceps activation in patellofemoral dysfunction has also been studied by Moller et al. (13) and by Grabiner et al. (4). The former study is of the comparative amplitude of VMO and VL EMG records and found no significant differences between the groups; however they used as a control group the contralateral leg of the patients. We have not used such data as controls because we noticed at an early stage of our study that the contralateral leg was often not entirely asymptomatic. Also abnormalities of muscle function have been found on the asymptomatic leg of other knee conditions (14). Grabiner studied isometric contractions with fast and slow activation and reported that the patients showed decreased excitation of both VMO and VL in the fast but not the slow contractions. He, however, did not record from the oblique section of VL.

The abnormality in activation of these oblique portions is unlikely to be simply a response to pain because the majority of patients did not experience pain on being asked to undertake the isometric contraction that we studied. Frequently patients did find an isometric contraction at 30° of flexion uncomfortable. There was also a small group who did not experience any difficulty in performing an isometric contraction at either of these angles, but did so at 60°
Quadriceps activation in chronic patellofemoral dysfunction

Table 1. A comparison of the results for the 20 normal subjects and for the 49 patients

<table>
<thead>
<tr>
<th></th>
<th>Normal Subjects</th>
<th>Patients</th>
<th>Significance of difference</th>
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<tbody>
<tr>
<td></td>
<td>n = 20</td>
<td>n = 49</td>
<td></td>
</tr>
<tr>
<td></td>
<td>male/female: 7/13</td>
<td>male/female: 16/33</td>
<td></td>
</tr>
<tr>
<td></td>
<td>age: 20–30 years</td>
<td>age: 20–37</td>
<td></td>
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<tr>
<td>Time for 80% tension development</td>
<td>185.8 ± 13.3</td>
<td>385.8 ± 31.4</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Force developed (N)</td>
<td>224.5 ± 12.3</td>
<td>174.0 ± 8.3</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Lag factors</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VMO</td>
<td>-0.158 ± 0.047</td>
<td>0.161 ± 0.032</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>VLO</td>
<td>-0.204 ± 0.040</td>
<td>0.083 ± 0.032</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>RF</td>
<td>-0.019 ± 0.057</td>
<td>0.180 ± 0.028</td>
<td>p &lt; 0.002</td>
</tr>
</tbody>
</table>

or 90°. Our findings resemble those of other workers and short arc exercises are very frequently given for rehabilitation because that arc of movement is considered the most comfortable (5).

In a study on cadavers, loads were applied to the muscles to produce a knee extension movement from 0° to 90° (12). When the muscles were loaded singly, vastus intermedius was seen to be the most effective extensor. All the other long heads needed a mean 12% increase in force to produce the same movement. VMO could not, by itself, effect a knee extension movement, no matter how great the loading, but VL, by itself, subluxated the patella laterally without a countering force from VMO. It is likely that VMO has a similar function in vivo, and that its action is necessary as the patella moves medially to enter the trochlea at about 20° of flexion (10). If the activation of VMO is sluggish and inadequate to the purpose, then the patella would not go down at the correct angle. This would mean that by 30°, in which position the patella is somewhat deeper in the groove, it would not articulate correctly with the femur, as is necessary for the area of articulation to be a large enough band as shown by Goodfellow et al. (2), for the patellar contact force to be applied in the usual manner. There would then be areas of hyperpressure, which is thought to cause pathological changes (8, 15) and also hypopressure, which is thought to cause disturbances of nutrition (9).

This cross-sectional study cannot answer the question as to whether these abnormalities of muscle action are a cause or effect of chronic patellofemoral pain, but we have demonstrated large and significant differences in the activation of VMO and VLO in the patient group, compared with that in the normal subjects.

ACKNOWLEDGMENTS

This study was supported by a grant from the Sir Jules Thorn Charitable Trust. We also thank Professor David Jones of the University of Birmingham for his help in the initiation of the project. Permission was granted by the ethics committee of the University College London Hospitals.

REFERENCES


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