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REFLEX INHIBITION OF THE HUMAN QUADRICEPS IN THE PRESENCE OF KNEE JOINT DAMAGE

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CONTENTS

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ABBREVIATIONS

AHC	Anterior Horn Cell
CSA	Cross-sectional Area of Quadriceps
CAT	Computerized Axial Tomography
EMG	Electromyography
FRA	Flexor Reflex Afferents
Inj	Injured
MFA	Mean Fibre Area
ml	Millilitres
ms	Milliseconds
MVA	Maximal Voluntary Activation
MVC	Maximal Voluntary Contraction
NSAID	Non-steroidal Anti-inflammatory Drug
PAN	Posterior Articular Nerve
RF	Rectus Femoris
s	Seconds
SD	Standard Deviation
SLR	Straight Leg Raise
St	Semitendinosus
TNS	Transcutaneous Nerve Stimulation
Uninj	Uninjured
V	Volts
μV	Microvolts

VARIABILITY OF MEASUREMENTS

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Maximal Voluntary Activation of Quadriceps	CV 6%	41
Maximal Isometric Quadriceps Strength	CV 8 %	34
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ABSTRACT

REFLEX INHIBITION OF THE HUMAN QUADRICEPS IN THE PRESENCE OF KNEE JOINT DAMAGE

by Maria Stokes

Quadriceps weakness can occur by atrophy and by reflex inhibition due to stimuli from a damaged knee joint. The mechanisms by which quadriceps weakness occurs are not understood enough to allow weakness to be prevented.

The nature of reflex inhibition has been studied in patients undergoing arthrotomy and meniscectomy in order to find ways of preventing inhibition. The maximal voluntary activation (MVA) of quadriceps was recorded, using surface integrated electromyography, during straight leg isometric contractions before and after surgery. Post-operative inhibition was expressed as the percentage reduction from the pre-operative MVA. Knee pain experienced during each contraction was recorded on a linear analogue scale.

Post-meniscectomy reflex inhibition of quadriceps is severe (70-80% during the first 3 days), prolonged (35-40% at 2 weeks) and is not related to pain after 24 hours. Inhibition can be temporarily prevented by per-operative infiltration of the knee with a local anaesthetic (Chapter 3). Prolonged voluntary tourniquet ischaemia in normal subjects did not alter subsequent quadriceps function, indicating that the reduced MVA observed in the meniscectomy patients was not due to ischaemia (Chapter 4). Isometric quadriceps contractions are inhibited less with the knee flexed than extended (Chapter 5). Transcutaneous nerve stimulation (TNS) had only a small effect on inhibition, and pain relief was similar in both the treatment and the control groups (Chapter 6). In patients who developed knee joint effusions post-operatively, aspiration always reduced inhibition but did not abolish it (Chapter 7).

The effect of knee joint afferent activity on quadriceps activation was studied in normal subjects with knee joint infusions. Intra-articular pressure/volume relationships at rest were similar to results reported by other authors, and during contraction the pressures were higher at each volume (Chapter 8). Inhibition of reflex activation of quadriceps was examined by measuring quadriceps H-reflex (Chapters 9,10 & 11). The central neural pathway of the joint afferent stimuli was investigated by testing for spatial facilitation between joint stimulation (by infusion) and known pathways of quadriceps H-reflex inhibition (by stimulating various nerves). The results suggest that the joint afferents have connections with Ib and cutaneous flexor reflex afferent (FRA) inhibitory pathways, but results for reciprocal inhibition were equivocal.

The present investigations have quantified the severity and duration of post-meniscectomy quadriceps inhibition, and have confirmed that the inhibition occurs by a reflex mechanism originating from the knee joint and that it is possible to block the inhibitory stimuli. Investigation of the pathway of the afferent stimuli suggested convergence of joint afferents with Ib pathways and with cutaneous FRA pathways.

CHAPTER 1

INTRODUCTION TO THE INVESTIGATION OF REFLEX INHIBITION OF QUADRICEPS IN THE PRESENCE OF KNEE JOINT DAMAGE

DISABILITY DUE TO QUADRICEPS WEAKNESS

MECHANISMS OF QUADRICEPS WEAKNESS

HISTORY OF STUDIES OF REFLEX INHIBITION

The Existence of a Reflex Mechanism
Suggested Causes of Reflex Inhibition
  Pain
  Joint Effusion
Quadriceps Weakness Due to Ischaemia
Pathway of Inhibitory Joint Afferent Stimuli

AIMS OF THE PRESENT INVESTIGATIONS OF REFLEX INHIBITION OF QUADRICEPS

INTRODUCTION TO THE INVESTIGATION OF REFLEX INHIBITION OF QUADRICEPS IN THE PRESENCE OF KNEE JOINT DAMAGE

DISABILITY CAUSED BY QUADRICEPS WEAKNESS

Injury to the knee joint, whether it be due to trauma, surgery or disease, results in weakness and atrophy of the quadriceps femoris muscle group. The action of the quadriceps is to extend the knee and its functional importance is realized when the muscle becomes weak. Severe quadriceps weakness may prevent patients from, e.g.,rising from a squatting position, climbing the stairs, rising from the toilet or a low chair. These problems are often seen to trouble elderly patients who might have 'arthritis', or may not have recovered after a fall. Many of the patients seen by physiotherapists are young and have short term problems of quadriceps weakness which may be due to knee trauma or surgery. While it is necessary to improve rehabilitation techniques in order to speed the recovery of these patients, it is essential that improvements are made for patients with joint disease whose disabilities are often more severe and long term.

The cost-effectiveness of physiotherapy is increasingly scrutinized. A study by Forster & Frost (1982) found that "physiotherapy" made no difference to the recovery of function following knee surgery. It is necessary to increase our knowledge and understanding of quadriceps weakness before the effectiveness of physiotherapy can be improved and disability can be prevented. If the choice of treatment is made without knowing why the weakness has occurred, the treatment may be inappropriate if the cause is still present.

MECHANISMS OF QUADRICEPS WEAKNESS

The mechanisms which cause quadriceps weakness are poorly understood. Quadriceps weakness can occur by atrophy due to immobilization (either deliberate i.e.'therapeutic',or due to unwillingness to move because of pain). The weakness causes the mechanics of the knee joint to be altered during movement. This produces abnormal loading in the knee which can cause further damage to the knee joint. Abnormal tracking (or malalignment) of the patella may also occur, leading to patello-femoral joint irritation (Paulos et al.,1980; Williams,1980; Kettelkamp,1981; Reider et al.,1981). Further wasting and weakness then occur after joint damage and a vicious cycle of events is set up (Fig 1.1). Basmajian (1970) suggested that this vicious cycle could only be interrupted by regaining quadriceps strength. This approach is often taken and can be sucessful, but the story is not always that straightforward and in some patients the quadriceps does not respond to strengthening exercises.

A common observation after knee surgery is that patients cannot contract their quadriceps no matter how hard they try, even though the attempt may not be painful. These patients often say that they have "no control" over the quadriceps of their operated leg. Basmajian (1970) suggested that voluntary muscle contraction may be inhibited due to either voluntary inhibition (unwillingness to contract due to pain) or involuntary reflex inhibition. Reflex inhibition of quadriceps is the inability to contract the muscle due to afferent stimuli from the knee joint receptors which prevent activation of quadriceps' aplha motor neurones in the anterior horn of the spinal cord. The afferent pathway of the stimuli from the joint is not known. The phenomenon of reflex inhibition, which causes weakness and can contribute to atrophy, can be added to the cycle of events already described (Fig 1.2.). Although the existence of reflex inhibition is recognised (e.g. Blockey,1954;

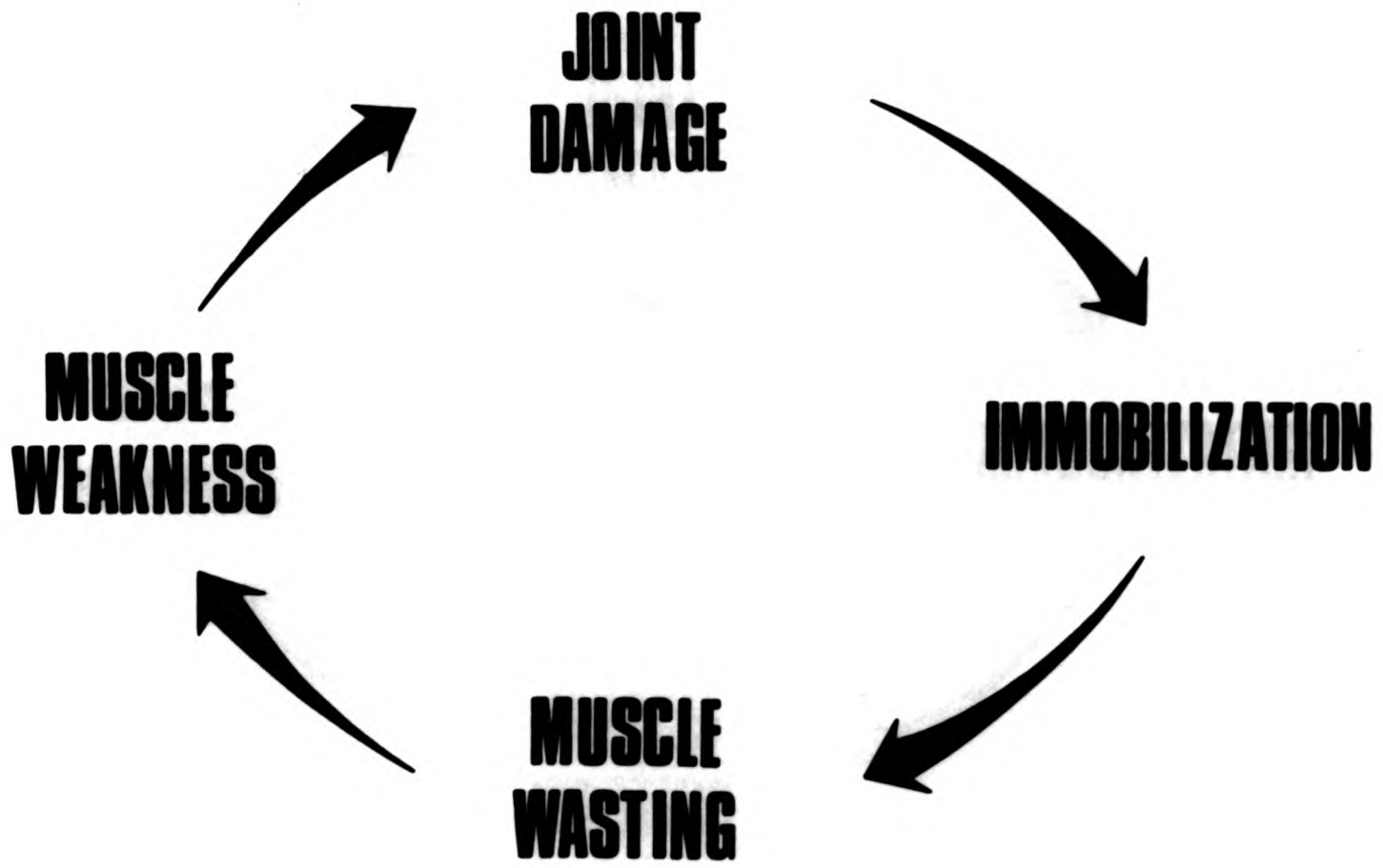

Figure 1.1. **The cycle of events initiated by joint damage leading to muscle atrophy and weakness (after Basmajian, 1970).**

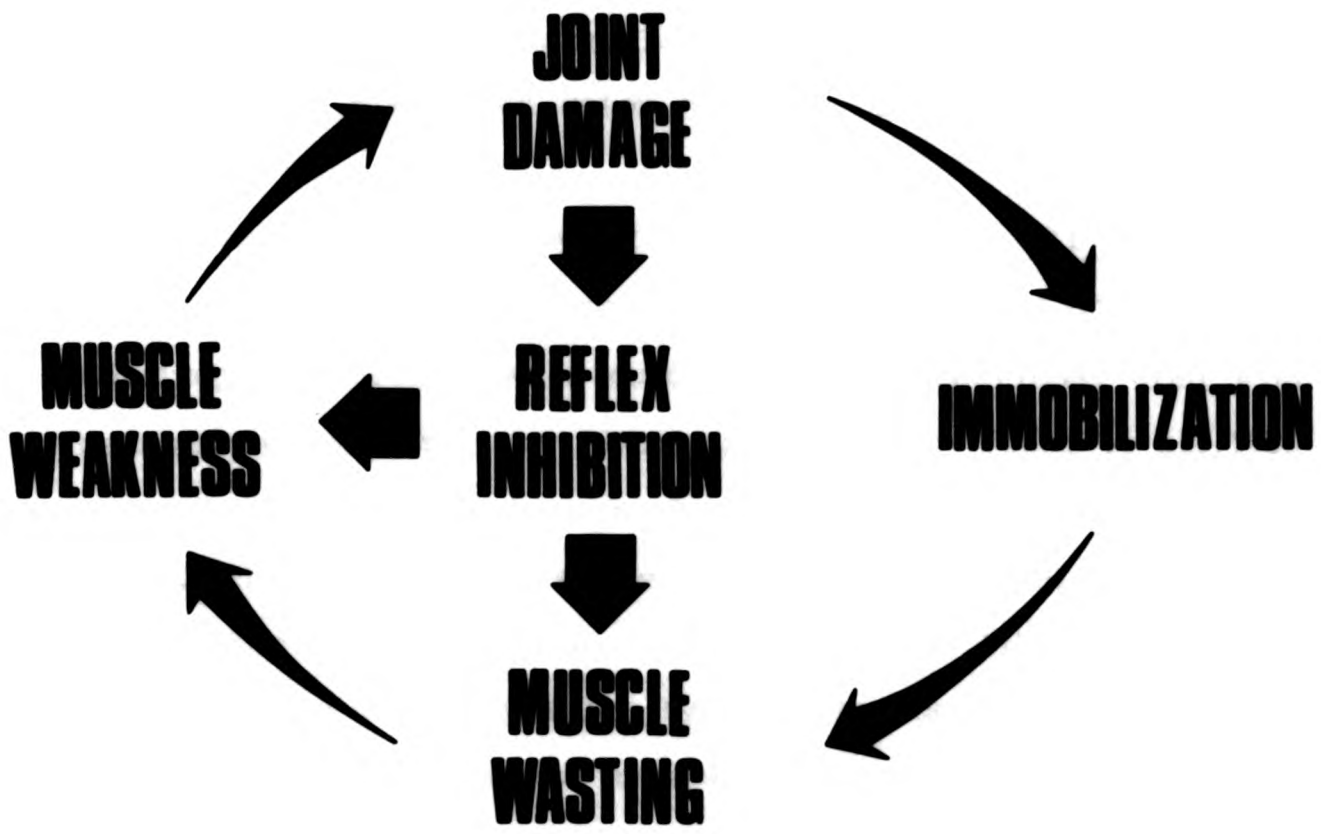

Fig 1.2. Reflex inhibition of voluntary quadriceps activation contributes to muscle atrophy and weakness in the vicious cycle of events initiated by joint damage.

Nichols,1980) few appreciate its potency. Sherrington (In: Denny-Brown,1979a) spoke of reflex inhibition in terms of reciprocal inhibition by contraction of antagonists but he also discussed inhibition of spinal reflexes resulting from peripheral afferent stimuli Sherrington (In: Denny-Brown,1979b). Throughout the present studies, the term 'quadriceps inhibition' refers to reflex inhibition of activation of quadriceps motor neurones due to afferent stimuli from the knee joint.

Methods of measuring quadriceps atrophy (both macroscopically and microscopically) have improved our understanding of quadriceps weakness. Whole quadriceps cross-sectional area (CSA) can be measured accurately by ultrasound scanning (Young et al.,1980) or by computerized axial tomography i.e. CAT scanning (Häggmark et al.,1978). The needle biopsy technique (Young,1979; Edwards et al.,1980) and microscopy allow individual muscle fibre areas to be measured, and the different fibre types to be identified. Young has discussed the implications for rehabilitation of studies which used such techniques and he also acknowledged the important contribution of reflex inhibition to quadriceps weakness (Young,1982). Some studies of quadriceps wasting have demonstrated selective wasting of the different muscle fibre types (Young et al.,1982; Ingemann-Hansen & Halkjaer-Kristensen,1983). Specific types of exercise for increasing the size of each fibre type would be beneficial in the absence of reflex inhibition, but if the selectiveness of the atrophy is dependent on a selective reflex mechanism, the specific exercises will be ineffective if the inhibitory reflex still operates. It is therefore necessary to find the cause of the inhibition first and to find ways of preventing it.

HISTORY OF STUDIES OF REFLEX INHIBITION

The Existence of a Reflex Mechanism

In the 19th century many workers (particularly in France) became interested in the cause of muscle weakness and atrophy in the

presence of joint damage. Theories in the early 19th century were based on clinical observations and it was currently accepted that disuse could not entirely explain muscular atrophy and that a reflex mechanism involving the joint was operating.

In a series of lectures delivered in the 1860's, Hilton described how he had traced the distribution of nerves and found that the same nerve trunk supplied branches to the interior of a joint, the muscles acting over that joint and the skin over the muscles and the joint (Hilton 1907). He explained how swelling of the shoulder joint produced spasm in the surrounding muscles and made the following statement:

> " When the interior of the joint is in a state of inflammation or of irritation, the influence of this condition is carried to the spinal marrow, and thence reflected to the various muscles of the joint, through the medium of the associated motor nerves, the muscles being supplied by the same nerves that supply the interior of the joint."

This statement, which became known as Hilton's Law, was based on anatomical studies and was not supported by physiological evidence. Hilton's Law outlines the same pathway which was later described in the theory of reflex inhibition.

Vulpian (1875) remarked that atrophy which was of rapid onset and development could not be caused by disuse as this would cause the atrophy to occur slowly. Charcot (1889) described the case of a young man demonstrating severe and persistent quadriceps weakness after a minor knee injury. Due to the history of injury and the persistence of mild symptoms, Charcot believed that this weakness was not due to disuse and that it was due to a reflex effect originating in the knee joint and he termed the weakness "atrophic articular paralysis". He also stated that:

> " There is no relation necessary between the intensity of the joint affection and that of the paralytic and atrophic phenomena."

The existence of a reflex mechanism involving joint afferents was supported by physiological evidence by Raymond (1890) when he demonstrated the phenomenon in dogs with artificially induced arthritis. The posterior nerve roots of the lower 3 lumbar, and upper 3 sacral nerve roots were cut on 1 side. The side on which the reflex arc was intact showed marked muscle wasting, while there was no wasting on the side of the cut nerves (i.e. the deafferented side). Harding (1929) also demonstrated the existence of a reflex mechanism when she demonstrated the difference between the quadriceps atrophy resulting from immobilization and that from joint pathology. Her experiments, performed using cats and rabbits, showed that deafferentation of a limb, by dorsal root section, prevented quadriceps atrophy in the presence of arthritis of the knee, but did not prevent atrophy during joint immobilization.

Suggested Causes of Reflex Inhibition

The early animal studies of quadriceps inhibition (e.g. Raymond,1890; Harding,1929) were designed to confirm that weakness in the presence of knee joint injury was caused by a reflex mechanism. More recently, studies have set out to identify the inhibitory stimuli which cause reflex inhibition.

Pain. Pain has been suggested as a cause of reflex inhibition (Eriksson ,1981; Smith et al.,1983) but even several of the authors of the early studies observed that reflex inhibition often occurred in the absence of pain (Vulpian,1875; Charcot,1889; Raymond,1890; Harding,1929). Studies which suggested that pain caused reflex inhibition did not produce any evidence that the inhibitory stimuli were those carried by the small pain fibres. Stener & Petersén (1963) showed that pressure and tension (applied by passive abduction) in the injured medial collateral ligament of the knee during a quadriceps contraction, caused reflex inhibition. The fact that both manoeuvres were accompanied by pain is not enough evidence to suggest that the inhibitory stimuli were from the pain receptors

and not the mechanoreceptors (i.e. pressure and stretch receptors). Eriksson (1981) also suggested that the reflex inhibition of quadriceps which occurs after knee surgery was caused by pain but this is open to similar criticism (Chapter 3, page 43). Pinching the knee joint capsule in the cat has been shown to inhibit quadriceps, again with no evidence that the pain receptors were responsible (Ekholm et al.,1960). Other studies have demonstrated reflex inhibition in the absence of pain (Blockey,1954; deAndrade et al.,1965; Jayson & Dixon,1970).

Effusion. Increased intra-articular pressure produced by joint infusion caused inhibition of the monosynaptic reflex from quadriceps in both decerebrated and spinalized cats (Ekholm et al.,1960). The clinical observation that a knee joint effusion was often associated with quadriceps weakness and atrophy prompted deAndrade et al. (1965) to study quadriceps inhibition under controlled conditions in man. They demonstrated that distension of the knee joint (with human plasma) caused quadriceps weakness and that pain was not always present. deAndrade et al. (1965) concluded:

> "It is futile to attempt to strengthen the quadriceps in the presence of a distended knee."

Jayson & Dixon (1970) also found that infusion of fluid (dextrose and sodium chloride) into the knee joint produced quadriceps weakness in the absence of pain.

Very recently, reflex (i.e.involuntary) activation of quadriceps was shown to be reduced by knee joint infusion in man (Spencer et al.,1984) using a similar approach to that described in Chapter 11.

Quadriceps inhibition often occurs in the absence of both pain and effusion (personal clinical observation) so other factors must also be involved. Other possible mechanisms are discussed in Chapter 3 (page 44) and Chapter 12 (page 125).

Quadriceps Weakness due to Ischaemia

It has been suggested that the period of tourniquet ischaemia during knee surgery may contribute to post-operative quadriceps weakness (Saunders et al.,1979; Dobner & Nitz,1982). If this were true, the quadriceps weakness observed after knee surgery would not involve the same mechanism as that causing the weakness in patients with joint disease. Therefore, the results of studies of inhibition performed on surgical patients (e.g. Chapters 3,5,6 & 7) would not be entirely relevant to patients with joint disease and no history of surgery.

Pathway of Joint Afferent Stimuli

The reflex pathway from joint receptors to the quadriceps' anterior horn cells (AHC) is unknown. It has been suggested that a common pathway is shared by joint, cutaneous, and Group II and III muscle afferents (Eccles & Lundberg,1959). The afferents contributing to this common pathway have been termed the flexor reflex afferents (FRA) and it is thought that they may also have other, separate, reflex connections or 'private' pathways. One suggested alternative pathway for joint afferents is convergence with Ib afferents (from tendon receptors) on to common interneurones (Lundberg et al.,1978; Baldissera et al.,1981; Brink et al.,1984).

Baldissera et al. (1981) summarized the state of knowledge about the pathway of human joint afferents in the following statements:

> " Further investigation of the effect of joint afferents on spinal interneurones is highly desirable, particularly with regard to the inhibitory actions they may exert on interneuronal paths activated from primary afferents and / or descending fibers. At present there is only scanty indirect evidence for such inhibition ".

Experiments to identify the pathway of quadriceps inhibition are described in Chapter 11.

AIMS OF THE PRESENT INVESTIGATIONS OF REFLEX INHIBITION OF QUADRICEPS

1. To examine reflex inhibition of voluntary quadriceps activation after arthrotomy and meniscectomy and to find ways of reducing and preventing it. (Chapters 3,5,6 & 7)

2. To demonstrate reflex inhibition of both voluntary and reflex activation of quadriceps by knee joint infusion. (Chapters 8, 10 & 11)

3. To determine the minimal amount of knee joint infusion necessary to cause inhibition of voluntary and reflex activation of quadriceps. (Chapters 8 & 11)

4. To determine the central nerual pathway of the afferent inhibitory stimuli from a damaged knee joint. (Chapter 11)

CHAPTER 2

METHODS FOR INVESTIGATING REFLEX INHIBITION OF QUADRICEPS

CHOICE OF EXPERIMENTAL MODELS

Patients Undergoing Arthrotomy and Meniscectomy
Normal Subjects with Knee Joint Infusions

CHOICE OF METHODS OF MEASUREMENT

Maximal Voluntary Activation of Quadriceps
Measurement of Knee Pain
Reflex Activation of Quadriceps
Quadriceps Size and Strength

SUBJECTS

Patients
  Surgical details
  Routine post-operative treatment
Normal Subjects

ETHICS

ELECTROMYOGRAPHIC TESTS

Maximal Voluntary Activation of Quadriceps
  Surface electromyography
  Needle electromyography
  Knee joint position
Hamstring Activation
Soleus H-reflex
Quadriceps H-reflex

PAIN

MAXIMAL ISOMETRIC QUADRICEPS STRENGTH

KNEE JOINT INFUSION IN NORMAL SUBJECTS

Intra-artucular pressure
Infusion of saline into the knee joint

STATISTICS

METHODS FOR INVESTIGATING REFLEX INHIBITION OF QUADRICEPS

CHOICE OF EXPERIMENTAL MODELS

Patients Undergoing Arthrotomy and Meniscectomy

This surgical procedure provides a suitable model of controlled knee trauma in which to study quadriceps inhibition. This experimental model allows comparison of post-operative with pre-operative measurements. It also allows patients who have marked quadriceps inhibition before the joint is subjected to surgery, to be excluded from the study.

Normal Subjects with Knee Joint Infusions

Painless knee joint distension with saline (deAndrade *et al*.,1965; Jayson & Dixon,1970) allows quadriceps inhibition to be studied under more controlled conditions. Inhibition can be increased or decreased by altering the volume of infusion, and the intra-articular pressure can be monitored throughout the procedure.

CHOICE OF METHODS OF MEASUREMENT

Maximal Voluntary Activation (MVA) of Quadriceps

Surface and needle electromyography (EMG) were used to measure quadriceps MVA in the present studies. The integrated electrical output of a muscle during a maximal voluntary contraction (MVC) is dependant on 2 factors *viz*. 1) the number of motor units recruited, and 2) the rate of firing of the motor units. Although changes in either factor will alter the strength of contraction, MVA is not a measure of muscle strength. In a patient with unilateral quadriceps atrophy, MVA should be almost the same for both quadriceps although their strengths would be very different. Unequal recording conditions may cause a slight difference in MVA but a large difference may be considered to indicate inhibition. Repeated recordings using surface electrodes are reliable provided that the

electrode is placed at the same site over the muscle and that there is no change in muscle length (Ralston, 1961).

Measurement of Knee Pain

Linear analogue scales were used to assess the severity of knee pain experienced during quadriceps contractions. This type of scale has been found to be reliable (Downie et al.,1978). Linear analogue scales were used in preference to descriptive scales (which require more concentration to use) because patients were required to record pain scores in the early post-operative period while they were still drowsy.

Reflex Activation of Quadriceps

The H-reflex was used to measure reflex (involuntary) quadriceps activation. The H-reflex, which is elicited by low intensity stimulation of Ia spindle afferents of the femoral nerve resulting in a small quadriceps contraction, may be influenced by sensory stimuli in the spinal cord (Ekholm et al.,1960). Surface electrodes were used to record the muscle response over rectus femoris.

Quadriceps Size and Strength

Quadriceps' mid-thigh CSA can be measured by ultrasound scanning (Young et al.,1980). The maximal voluntary isometric strength of quadriceps can be measured by dynamometry with the knee at 90^{o} (Edwards et al.,1977). The methods for measuring quadriceps size and strength are described in the Appendix and Chapter 2 respectively. One possible method of measuring quadriceps inhibition in females might be to use the linear relationship between quadriceps size and strength which has been shown to exist in young and old normal women (Young et al.,1984a). This relationship was used to study 2 young female patients with unilateral quadriceps weakness and wasting secondary to knee injury (Fig 2.1.). Their uninjured limb's quadriceps fell within the normal range of size and strength

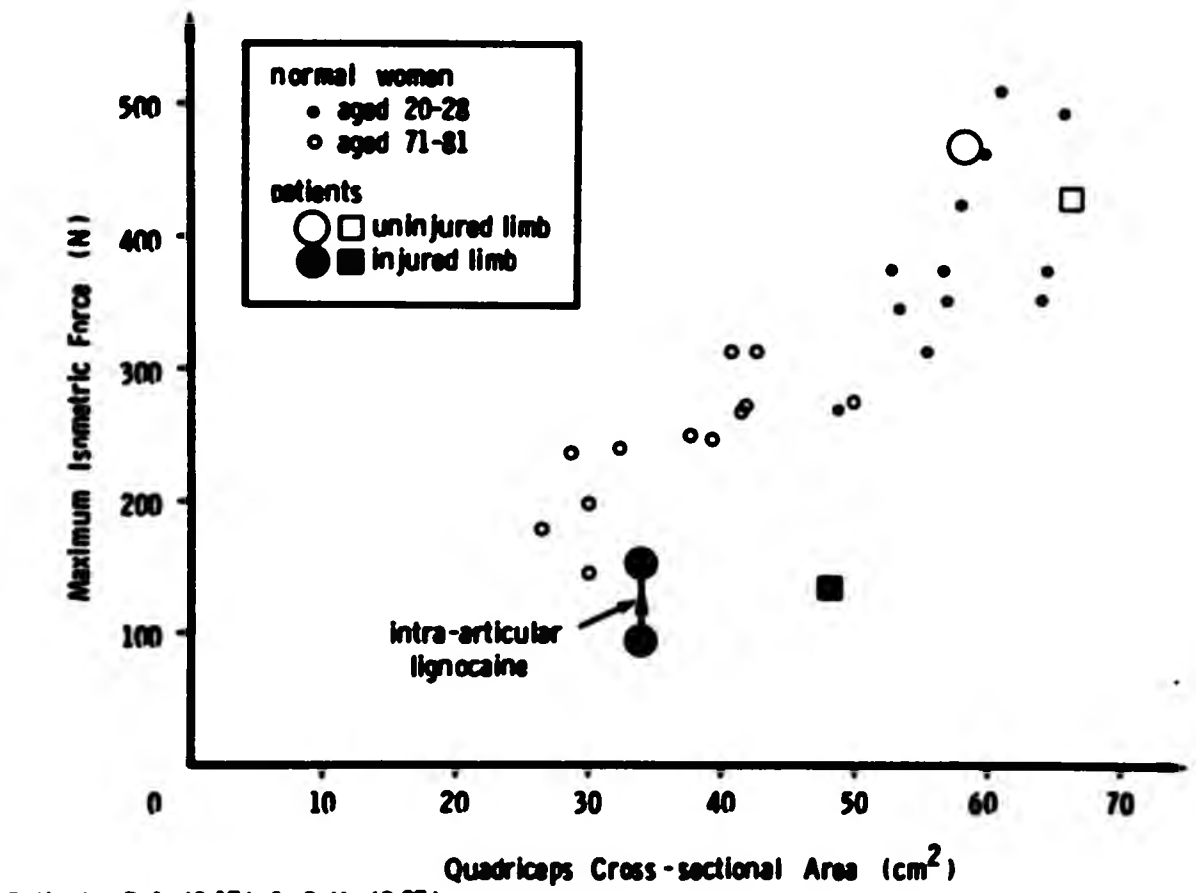

Figure 2.1. The relationship between quadriceps size and strength in normal females was used to examine the wasted quadriceps of 2 young female patients with unilateral knee damage. Their normal quadriceps were within the normal range and their injured limbs' quadriceps showed 'excess' weakness. In one patient, intra-articular injection of local anaesthetic enabled her to exert more force.

for young females. The quadriceps of the injured limbs showed severe atrophy and weakness. In both cases the weakness was more severe than would have been expected for the size of muscle. This suggested that the injured limbs' quadriceps might be inhibited. In 1 patient, intra-articular injection of a local anaesthetic enabled her to exert more force. These 2 patients experienced knee pain which may have caused voluntary inhibition, accounting for the 'excess' weakness. It was thought that, in patients without pain, excess weakness could be a potential measure of reflex inhibition. Nevertheless, this method was considered unsuitable as a measure of inhibition for the purposes of the present investigations because: 1) only female patients could be studied as the relationship between quadriceps size and strength in males is not so straightforward (some young men are stronger than would be expected from the size of their quadriceps; Young et al.,1984b) and 2) strength measurements could not be made if knee flexion was limited as it is in the early post-operative period.

It was later found that quadriceps contractions are inhibited less with the knee flexed (Chapter 5), so the use of the size / strength relationship would also have been unsuitable for this reason as the true severity of the reflex inhibition would not have been detected.

Measurements of quadriceps size and strength were used to monitor the progress of a patient with severe quadriceps weakness during a programme of quadriceps strengthening exercises (see Appendix). Although the patients' quadriceps were inhibited, size and strength were not used to measure inhibition for the reasons already given.

SUBJECTS

Patients

Males undergoing medial (1 lateral) arthrotomy and meniscectomy were studied. Patients were recruited from the operating lists of the orthopaedic consultants at the Nuffield Orthopaedic Centre, Oxford. None of the patients had undergone any previous knee surgery, had any history suggestive of neuromuscular or musculoskeletal disease, or had injured either leg so severely as to require immobilization of a joint for more than 1 week within the previous 2 years.

Surgical details. Ischaemia during surgery was achieved in the routine way for patients undergoing meniscectomy at this hospital, the patient being positioned supine with both knees flexed over the end of the operating table. The injured leg was elevated, exanguinated with a rubber Esmarch bandage, and a pneumatic cuff (9.3cm wide) was placed around the thigh as far proximally as possible. The cuff was inflated to 101b/sq in (517 mmHg) as usual. The rubber bandage was then removed and the leg was returned to its original position. Surgery was performed with the patient in this position (Fig 2.2.).

Medial meniscectomy was performed through a medial para-patellar arthrotomy. In some patients a bucket-handle fragment was removed (partial meniscectomy) and in others the whole meniscus was removed (total meniscectomy).

The capsular incision was sutured with 'dexon' (polyglycolic acid, which is absorbed by 60-90 days) and the skin incision was closed with nylon sutures (non-absorbable). The skin sutures were removed on the tenth post-operative day and the capsular sutures were left to be absorbed. The wound was dressed with 'melolin', cotton wool and crepe bandaging, and the leg was placed (with the knee extended) in a removable plaster of Paris backslab for 2-3 days.

Figure 2.2. **Position of the patient for meniscectomy showing the location of the pneumatic tourniquet. The cuff was placed as far proximally as possible.**

Figure 2.2. Position of the patient for meniscectomy showing the location of the pneumatic tourniquet. The cuff was placed as far proximally as possible.

Routine post-operative treatment for meniscectomy patients. Pre-medication was papaveretum 15mg and hyoscine hydrobromide Ø.3mg ('Omnopon - scopolamine'). Anaesthesia was by barbiturate induction then halothane and nitrous oxide maintenance. Post-operative analgesia was papaveretum 1Ø-2Ømg ('Omnopon') for the first 24 hours and then a variety of painkillers was available on request during the remainder of the hospital stay. The drugs prescribed depended on which consultant was responsible for the patient. They included: nitrazepam (5-1Ømg), 'Distalgesic' (dextropropoxyphene hydrochloride) (32.5mg), paracetamol (325mg), temazepam (1Ø-2Ømg), ibuprofen (4ØØmg), paracetamol (5ØØmg), and DF118 (dihydrocodeine tartrate 3Ømg).

The standard post-operative physiotherapy regime at this hospital was followed. Hourly straight leg maximal quadriceps contractions were commenced on the day of operation. Straight leg raising was practised and when this was achieved (usually 2-3 days post-operatively) the patient was permitted to walk partially weight-bearing with elbow crutches. Gentle knee flexion (passive and then active) was commenced on the second or third day. All decisions regarding progression of rehabilitation were made by the surgeon, and progression was supervised by the physiotherapist attached to the particular orthopaedic 'firm'. All patients were discharged by 5 days, and all were fully weight-bearing by 1Ø days.

Normal Subjects

Males and females within the age range of the patients were recruited from the Nuffield Orthopaedic Centre and departments of the University of Oxford. Some subjects were co-authors of different studies in the present series. Subjects were studied if they satisfied the same exclusion criteria as the patients.

ETHICS

The present investigations were conducted with the approval of the Nuffield Sector Ethics Committee, Oxford. The subjects gave their written informed consent (Figs 2.3. & 2.4).

ELECTROMYOGRAPHIC TESTS

Maximal Voluntary Activation of Quadriceps

Surface electromyography (EMG). Quadriceps MVA was measured during maximal voluntary isometric contractions using surface integrated EMG. Prior to recording MVA, skin resistance was reduced by shaving (if necessary), abrasion with fine sandpaper and cleaning with alcohol. Surface electrodes with saline-soaked felt pads (Medelec, Type EL210M) were placed (1cm apart) over rectus femoris at approximately mid-thigh (Fig 2.5.). The electrode sites were marked on the skin with a pen and were recorded, together with permanent skin blemishes,on a transparent sheet to allow accurate relocation for subsequent tests (Dons et al.,1979). The raw signal was transmitted through a pre-amplifier (Medelec PA62) and amplifier (Medelec AA6) and was then rectified and integrated. (Integration does not differentiate between changes in frequency and amplitude of action potentials). The electronic integrator (Medelec I6) was set at the auto-reset mode which produced a continuously integrated record of muscle activity (Fig 2.6.). The signal was emitted from a fibre optic recorder (Medelec MS6) onto ultra-violet light sensitive paper (Kodak Linagraph, Type 1895) and the paper speed was 10cm/s . The frequency of the waveform provided a measure of the electrical activity over a given time period (0.9s) and was measured by counting the number of times the signal reset in 5 lines of signal. The total voltage output per 0.9 s was calculated as follows:

$$\text{IEMG units} = \frac{\text{Number of resets per 0.9s}}{\text{Auto reset setting}} * \text{Amplifier gain}$$

N.B. * = multiplied by

CONSENT FORM

TITLE OF STUDY: The Treatment and Prevention of Quadriceps Muscle Weakness after Meniscectomy.

PURPOSE OF STUDY: To find the cause of the weakness which occurs in the quadriceps (the muscle on the front of the thigh) after knee surgery and to attempt to prevent the weakness. It is hoped that the results of this study can be used to help patients with joint disease.

NATURE OF PROCEDURES: Measurement of muscle activity produced while tightening the quadriceps muscle. Recording electrodes are placed on the thigh over the muscle.

Treatment of the weakness i.* injection of a local anaesthetic into the knee at the end of the operation. ii.* stimulation of nerves in the skin 2 days after operation.

CONSENT OF THE SUBJECT

I,.....of.....

.....

give my consent for.....to study my legs. He/she has explained the procedures to me. I understand that the study is being carried out for research purposes and is not essential for the management of my condition. I understand that I can withdraw from the study at any stage.

Signed.....Date.....

I confirm that I have explained to.....the nature and purpose of the study.

Signed.....

Witness.....

* Delete as necessary

Figure 2.3. Consent form used to obtain written informed consent of the meniscectomy patients before taking part in the studies.

CONSENT FORM

TITLE OF STUDY: Investigations of Quadriceps Muscle Weakness

PURPOSE OF STUDY: To find out how a swollen knee joint prevents maximum use of the quadriceps (the muscle on the front of the thigh), which causes the muscle to become weak and shrink. This knowledge would help us understand and treat the muscle weakness in patients with joint disease.

NATURE OF PROCEDURES: Measurement of reflexes of the quadriceps produced by electrical stimulation. This is not painful and causes a feeling of "pins and needles" which disappears immediately afterwards. Electrical stimulation will be applied to nerves in the groin and behind the knee and ankle.

Measurement of the changes in pressure inside the knee as fluid is inserted and removed. At the end of the experiment, the fluid will have been removed.

CONSENT OF SUBJECT

I.....of.....

.....

Give my consent for.....to study my knee and thigh muscles as indicated above. He/she has explained the nature, purpose, and possible consequences of the procedure. I understand that the study is being carried out for research purposes and that I can withdraw from the study at any stage.

Signed.....Date.....

I confirm that I have explained to.....the nature, purpose, and possible consequences of the proposed study.

Signed.....

Witness.....

Figure 2.4. Consent form used to obtain written informed consent of normal subjects before taking part in the study.

Figure 2.5. Measuring quadriceps MVA. Surface IEMG was recorded over rectus femoris during a straight leg maximal voluntary isometric quadriceps contraction.

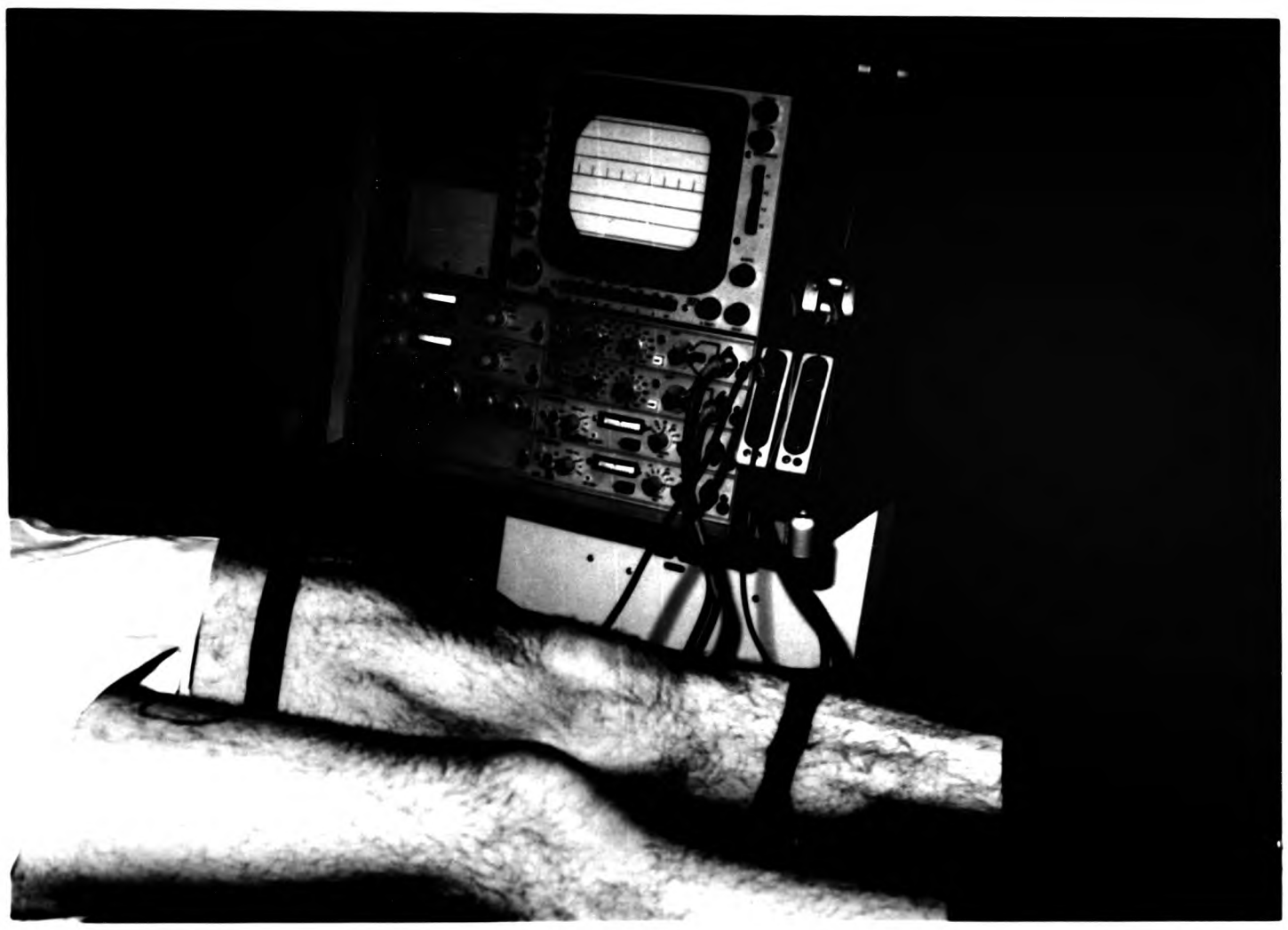

Figure 2.5. Measuring quadriceps MVA. Surface IEMG was recorded over rectus femoris during a straight leg maximal voluntary isometric quadriceps contraction.

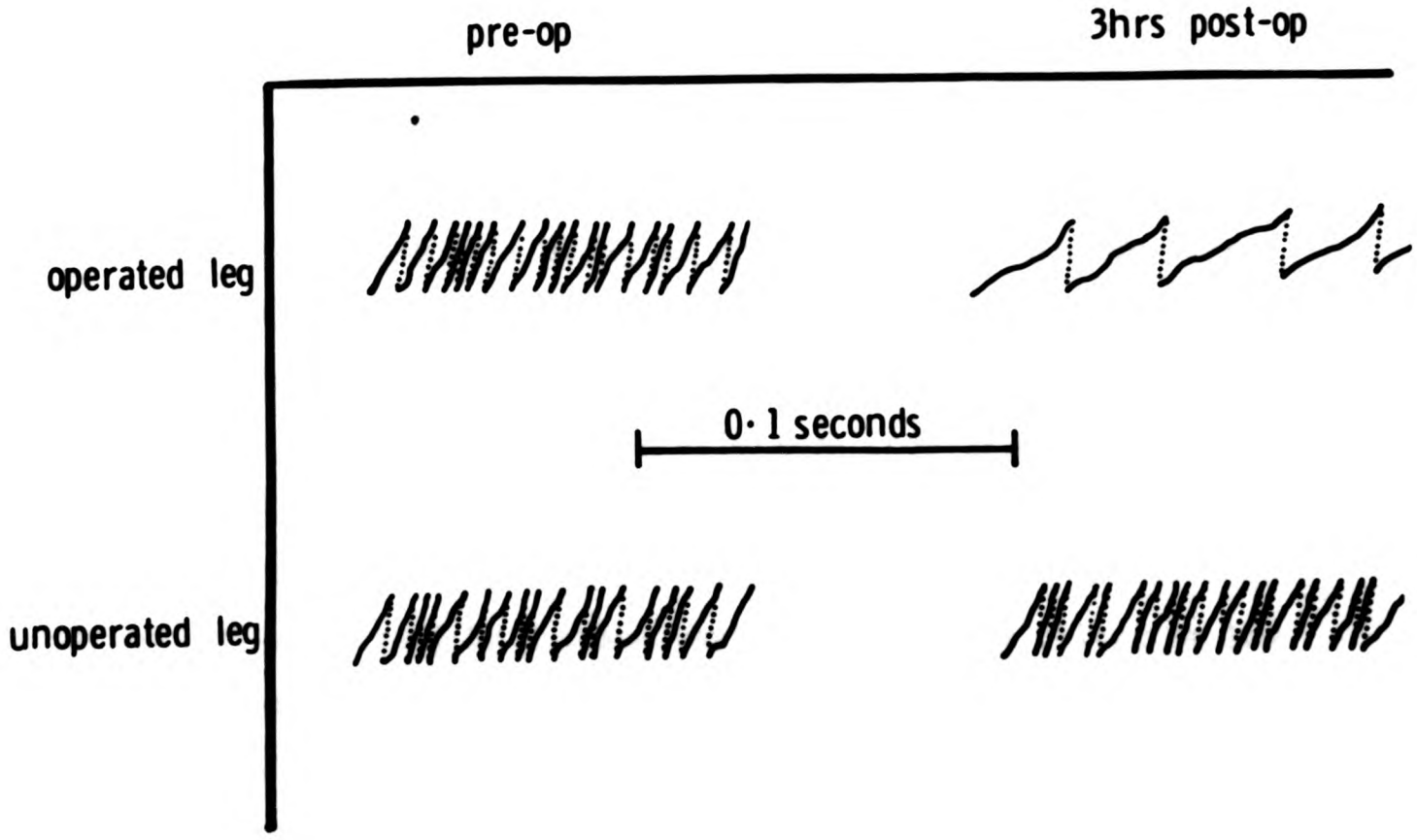

Figure 2.6. These sample recordings of the IEMG activity were recorded over rectus femoris before and after meniscectomy. There was a reduction in activity in the operated limb after surgery. Machine settings were: auto reset = 20 resets / s / division, amplifier gain = 5 μV / division, paper speed = 10cm / s.

The MVA was measured over the Ø.9 s period with the greatest number of resets during a maximal isometric quadriceps contraction lasting about 4 s. The reliability of repeated tests is discussed in Chapter 3. The between-days coefficient of variation (CV) in normal limbs was 6%.

Needle electromyography. A single concentric needle electrode, 12mm long and with a lead attached (Medelec, Type CF12 53513) was used to record quadriceps activation. The contraction was performed exactly as when recording with surface electrodes. The electrode was placed in the rectus femoris muscle belly at approximately mid-thigh, after cleaning the skin with alcohol. The electrode led to a pre-amplifier (Battery run, Type AlØl) which was set with a low frequency (LF) cut of 2ØØ Hz (due to problems with interference from 5Ø Hz) and a high frequency (HF) cut of 5KHz. The signal was displayed on an oscilloscope screen (Tektronix dual beam, Type 5Ø2A) and was then rectified and integrated by a state variable averaging filter (a low pass filter with a rapid dynamic response) with a time constant of 2ØØms (Garland et al.,1972). The signal was then amplified and displayed by a hot-wire chart recorder (Devices). The maximum activity over a 3 s period was measured.

Knee joint position. Unless otherwise stated, tests of quadriceps MVA were performed with the subject supine and the knee extended (Fig 2.5.). The subject was instructed to dorsiflex his foot, push his knee back towards the bed and contract his quadriceps as hard as possible. The best of 2 or more contractions was used in the analysis.

In the meniscectomy patients, recordings were made on both legs at each test session. A pre-operative value for MVA in the injured limb of greater than 75% of the value for the normal limb was required for inclusion in the studies (i.e. there was no evidence of major inhibition pre-operatively). Post-operative MVA was expressed as a percentage of the pre-operative MVA, and this figure was

subtracted from 100 to give a measure of the severity of inhibition.

In some experiments, tests of MVA were performed at different angles of knee flexion. The knee was flexed over a block or a hinged board (Fig 2.7.) with the hip also flexed. As the subject tried to straighten his knee, the lower leg was prevented from moving by a downward force applied at the ankle (either manual or a strap) so that the contraction was still isometric. Knee joint angle was measured with a goniometer.

Knee angles were also obtained with the hip in neutral by flexing the knee over the end of the bed and using a hinged board which could be set at different angles.

Throughout the present investigations the angles of the knee joint refer to the degree of flexion from the fully extended position which is 0^{o}. This system is used in clinical practice and is found in the medical research literature. Physiological research literature, however, uses a system in which knee extension is designated the angle of 180^{o}. So, for example, in the present studies 30^{o} of flexion would be a knee angle of 150^{o} according to the physiological system.

Hamstring Activation

Recordings of hamstring activation (made simultaneously with quadriceps recordings when possible) were made in some experiments. The subject lay supine on a plinth with a gap in it which allowed placement of the hamstring electrode. Surface electrodes were placed over the bellies of RF and semitendinosus and recordings were made during maximal voluntary isometric quadriceps contractions.

Maximal hamstring activation (HMVA) was recorded during a maximal isometric hamstring contraction ($10-15^{o}$ flexion) so that hamstring activation during quadriceps contractions could be expressed as a percentage of HMVA.

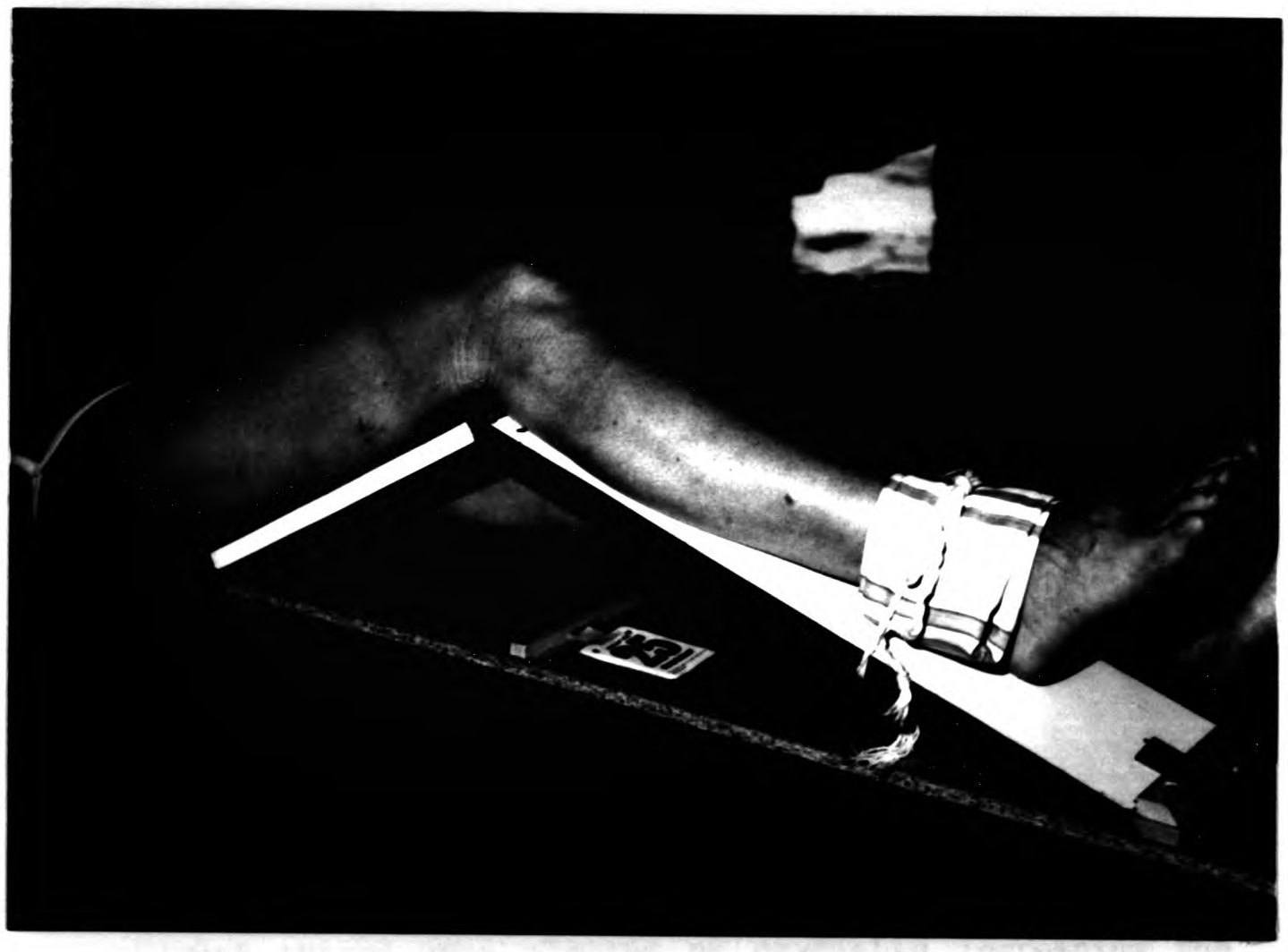

Figure 2.7. Subject performing an isometric quadriceps contraction with the knee and hip flexed.

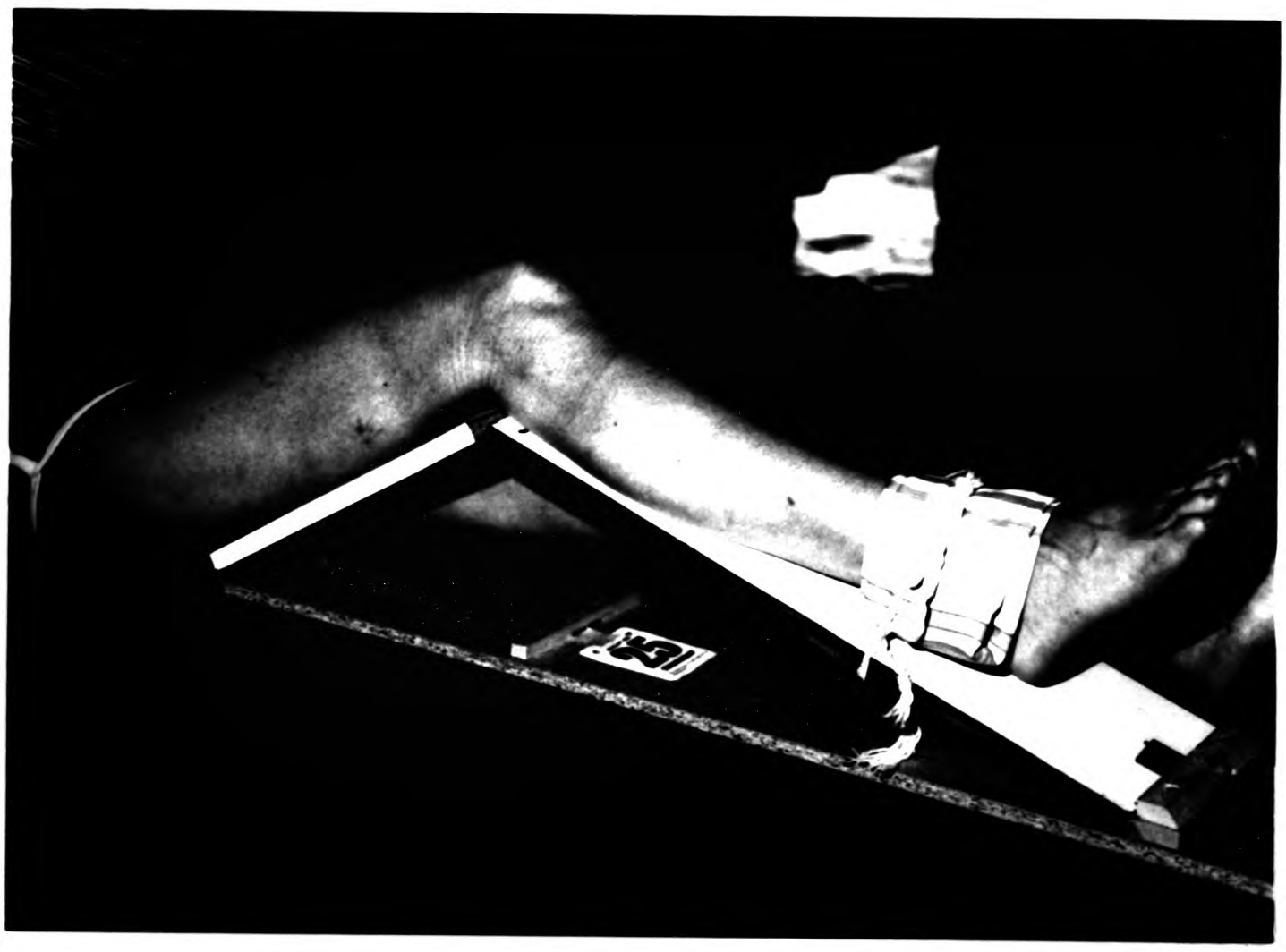

Figure 2.7. Subject performing an isometric quadriceps contraction with the knee and hip flexed.

Soleus H-reflex

The subject was seated with the knee in 60° of flexion and the ankle in 10° of plantarflexion with the foot resting on a platform.

Electrode placement. The tibial nerve was stimulated in the popliteal fossa with a Simon (1962) electrode (metal rod) and the anode (a silver strip) was placed on the medial side of the knee and held in place with rubber tubing. Two electrocardiographic (ECG) electrodes, containing electrode jelly, were placed over soleus after skin resistance had been reduced by abrasion and wiping with alcohol. An earth electrode was placed close to these.

The common peroneal nerve (CPN) was stimulated with 2 silver strip electrodes which were held in place with rubber tubing. The cathode was placed at the neck of the fibula and the anode was placed on the antero-medial aspect of the knee, just inferior to the patella.

Stimulation of soleus H-reflex. The tibial nerve was stimulated at a frequency of 0.2 Hz with long duration stimuli (0.5 ms) using an isolated stimulator (Digitimer 3072). The H-reflex and M response recruitment curves were plotted by gradually increasing the stimulation intensity from zero until the M response was maximal. Throughout the experiments, when changes in H-reflex amplitude were being observed, the intensity was such that a small M response was also produced to monitor the stability of the stimulation conditions i.e. H-reflex changes were only valid if the M responses were similar as different sized M responses would be accompanied by different sized H-reflexes anyway (see Chapter 9 re: H/M recruitment curves).

Controlled voluntary contraction of soleus. Soleus H-reflex was recorded during a small, controlled, voluntary contraction of the plantarflexors. The foot was strapped to a platform which could be rotated about a coronal axis. Tension springs attached to an arm on a motor shaft provided calibrated torque resisting plantarflexion.

Visual feedback of position from a meter enabled constant torque and position to be maintained.

Conditioning stimulation. 1) Prolonged vibration of tibialis anterior (TA) - a physiotherapy vibrator (Pifco) was screened with aluminium foil and earthed to avoid 50 Hz interference with the recording equipment. The vibrator operated at 100 Hz, peak to peak amplitude 0.5-1mm under load (depending on pressure against muscle, as described by Cussons et al.,1980), and was applied to the belly of TA. Test (i.e. control) reflexes were recorded before conditioned reflexes to avoid any influence from after effects of vibration (i.e. tonic vibration reflex - TVR) (Chapter 9,page 93). Vibration was applied continuously whilst conditioning reflexes were recorded, and recording began after 3s of vibration as the TVR may develop slowly (Burke,1981). 2) CPN (Common peroneal nerve) stimulation - an isolated stimulator (Digitimer 3072) was used to apply a shorter duration stimulus (0.2 ms) at a frequency of 0.2 Hz. CPN stimulation (either single or double shock) was applied at selected time intervals before the H-reflex. The timing of tibial and CPN stimulation was controlled by a gated programmer (Digitimer D4030). If double shocks were used, the time interval was taken from the second shock. An inhibitory time curve was plotted by recording H-reflex inhibition over a range of conditioning stimulus latencies from 0 - 19ms (Chapter 10, page 101). The intensity of stimulation was approximately alpha threshold.

Recording of soleus H-reflex. The recording electrodes were attached to a battery-run pre-amplifier (Type A101) with the LF cut set at 2Hz and the HF cut at 5KHz. Electromyographic recordings were amplified and displayed on an oscilloscope screen (Dual Beam, Type 502A, Tektronix, Guernsey Ltd.). Because of fluctuations of the response latencies between trials, records were full wave rectified before 16 responses were averaged by a computer (Biomac 1000 averaging computer, Data Laboratories Ltd.). When a conditioning stimulus was used, 16 control and 16 conditioned reflexes were

averaged. When CPN stimulation was used, the control and conditioned reflexes were alternated and accumulated separately in the computer. Timing was controlled by a modified Digitimer D4030 programmer. H-reflexes were elicited throughout the experiment (whether being recorded or not) to avoid the period of depression of amplitude found at the beginning of a train of reflexes (Iles & Roberts, unpublished). If the baseline of the H-reflex was too irregular, the amplitude of the averaged reflex was measured (Fig 2.8.a). Otherwise, the area above the baseline of the averaged reflex was measured using an integration facility (Fig 2.8.b). Either averaged or averaged and integrated recordings were plotted and measured for analysis. The inhibitory effect of the conditioning stimulus on the test reflex was expressed as the percentage reduction from the test reflex and was calculated as follows:

$$100 - \frac{\text{cond}}{\text{test}} * 100 = \% \text{ inhibition}$$

N.B. * = multiplied by

Quadriceps H-reflex

The subject lay supine with the knees extended.

Electrode placement. The cathode (a button electrode) was placed over the femoral nerve (just lateral to the femoral pulse and superior to the inguinal ligament) and the anode (an aluminium pad electrode(3cm by 3cm) was placed over the greater trochanter (Aiello et al.,1982). The electrodes were covered with lint which had been soaked in saline and were held in place with an adjustable frame (Fig.2.9.).

Electrodes for recording the H-reflex were 2 saline-soaked felt pads set 1cm apart in a plastic unit (Medelec sensory electrodes SE20 53056) (Fig 2.9.). The electrode unit was placed over RF at approximately mid-thigh.

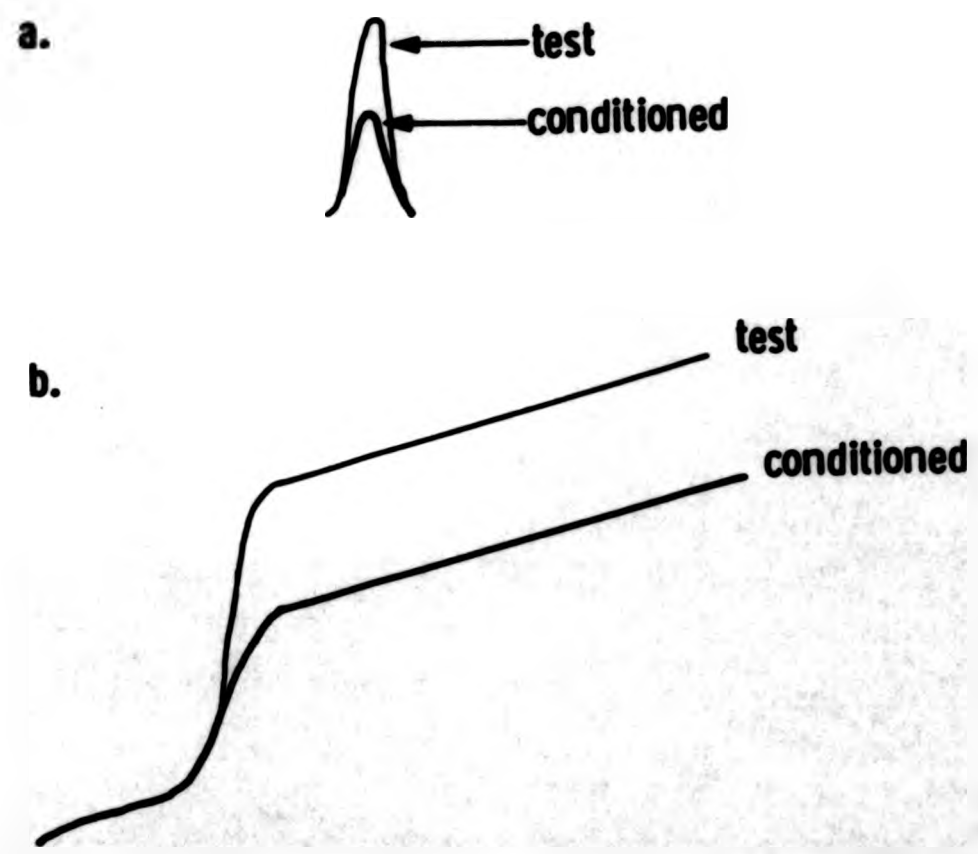

Figure 2.8. Recordings of a) averaged, and b) averaged and integrated H-reflexes showing test (control) and conditioned (inhibited) reflexes.

Figure 2.9. Positioning of electrodes for stimulating and recording quadriceps H-reflex.

Figure 2.9. Positioning of electrodes for stimulating and recording quadriceps H-reflex.

Biofeedback recording electrodes (2 discs), filled with electrode jelly, were placed over RF, proximal to the H-reflex recording electrodes. An earth electrode was placed lateral to these. Prior to fixing the recording electrodes in place, skin resistance was lowered by shaving (when necessary), abrasion and cleaning with alcohol.

The electrodes used for conditioning stimulation were: 1) sciatic nerve stimulation - the cathode was placed just below the ischial tuberosity at the inferior edge of gluteus maximus,and the anode was distal to this. The electrodes were fixed in a unit about 3cm apart. 2) tibial nerve stimulation - the same arrangement was used as when stimulating soleus H-reflex. 3) Sural nerve stimulation- the stimulating electrodes were placed inferior to the lateral malleolus (cathode) and on the dorso-lateral aspect of the foot (anode).

Stimulation parameters for eliciting the H-reflex. The femoral nerve was stimulated using the same equipment and parameters (except frequency which was 0.2 - 0.25 Hz) as for the tibial nerve when eliciting soleus H-reflex (see page 29). M response and H-reflex recruitment curves were also plotted in the same way.

Controlled voluntary quadriceps contraction. The H-reflex was elicited at rest (when possible) and also during a small isometric quadriceps contraction (Chapter 10) which was controlled by EMG biofeedback (Autogen 1700) in the 'normal' limb. A target of approximately 15-20% MVA was set on the meter and auditory feedback was also given to help the subject maintain a constant contraction. The aim was to make equal contractions of both quadriceps throughout the experiment to ensure the same amount of effort (rather than activation) as inhibition of the quadriceps in the test limb occurred (Chapter 11).

Conditioning stimulation. 1) Prolonged vibration of quadriceps, semitendinosus, TA and gastrocnemius / soleus. Vibration was applied

to the muscle bellies in the same way as for vibration during soleus H-reflex recording. 2) Sciatic nerve stimulation. The same parameters were used as when stimulating the CPN (see page 30). Only single shocks were used for the sciatic nerve. An inhibitory time curve was plotted for quadriceps H-reflex. 3) Tibial nerve stimulation. The tibial nerve was stimulated in the popliteal fossa using the same stimulation parameters as used to stimulate the CPN (see page 30), and an inhibitory time curve was plotted. 4) For sural nerve stimulation see Chapter 11, Experiment III.

The effects of the different types of conditioning stimuli on quadriceps H-reflex are described and discussed in Chapter 10.

Recording of quadriceps H-reflex. The response was recorded exactly as for soleus H-reflex. The test and conditioned reflexes (whether electrical or mechanical) were alternated, and the results calculated in the same way to give values for percentage inhibition of the H-reflex.

PAIN

The severity of knee pain experienced by the patient while performing the test for quadriceps MVA was recorded using a linear analogue scale. The scale was a 10 cm horizontal line drawn on paper, and was without word cues. The patient was reminded at each test that the left hand end of the line represented no pain, and the right hand end represented the worst pain he could imagine. The scale was marked immediately after each quadriceps contraction. The patient was instructed to mark the scale (with a pen) according to the severity of his pain while making that particular contraction. Previously recorded scores were not visible.

MAXIMAL ISOMETRIC QUADRICEPS STRENGTH

The force of a maximal voluntary contraction (MVC) of quadriceps was measured at the ankle by isometric dynamometry (Fig 2.10). The subject was seated in an adjustable straight backed chair with the lower leg dependant and the knee at 90^{o} (Edwards *et al.*,1977; Fig 2.10). The force exerted was recorded by a strain gauge (Gould Universal Transducing Cell, UC3), amplified (Medelec Strain Gauge Amplifier, AD6) and displayed on an oscilloscope screen (Medelec MS6). The signal was recorded on light sensitive paper (Kodak Linagraph, Type 1895). The maximum height of the signal over a 1 second period was measured and converted into Newtons. This method of strength measurement does rely on the motivation of the subject but the repeatability of the test has been shown by the author to be reliable in normal subjects *viz.* the coefficient of variation was 8% in young adults, and also 8% in elderly subjects (Young *et al.*,1984).

KNEE JOINT INFUSION IN NORMAL SUBJECTS

Knee joint infusion was performed by Dr A Young.

Intra-articular Pressure

The subject lay supine with the knee extended. An infusion system was set up as shown in Fig 2.11. The pressure transducer could not be sterilized so a length of tubing was placed between a 3-way tap and the transducer to keep it apart from the rest of the tubing system. The transducer and syringe were secured into clips on a board. Saline was flushed through the tubing (but not through the cannula) before the cannula was inserted into the knee.

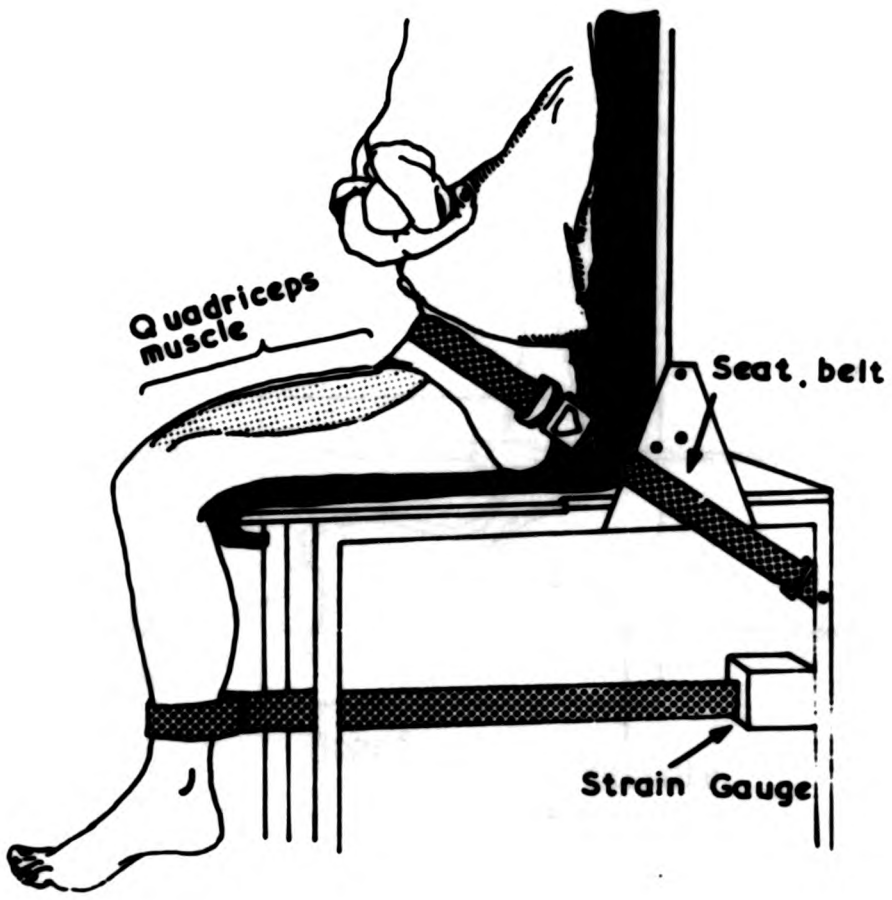

Figure 2.1Ø. Measurement of maximal voluntary isometric quadriceps strength. The force of a MVC was recorded as the subject tried to straighten his knee against the inextensible strap placed around the ankle.

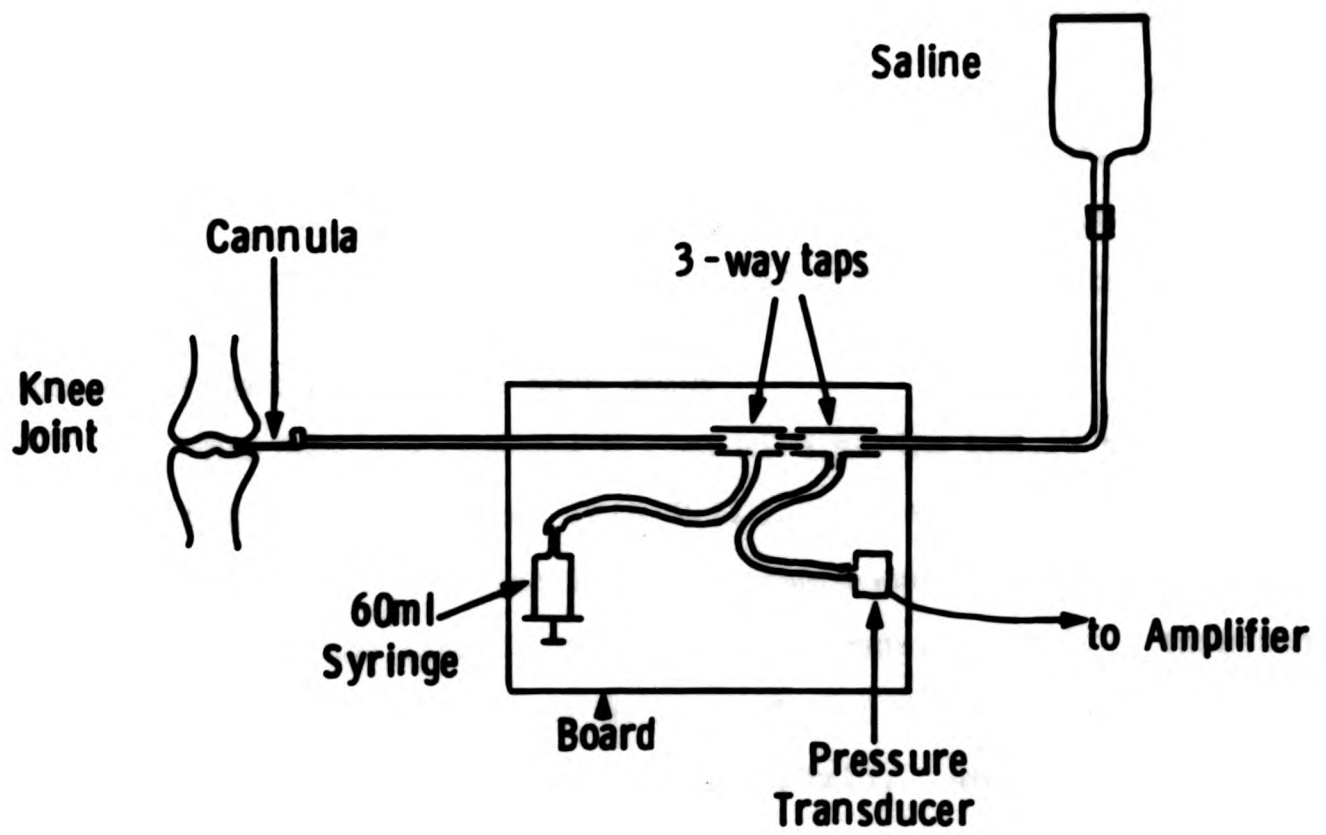

Figure 2.11. System for infusion of the knee joint with saline and recording of intra-articular pressure.

The skin was cleaned with Chlorhexidine in Spirit (0.5%) and, observing sterile technique, a 16 gauge cannula ('Venflon' Leur-lock) was inserted through unanaesthetized skin into the lateral aspect of the knee, posterior to the patella.

An electrically isolated pressure transducer (National Gauge Pressure Transducer, National Semiconductors, LX1 701GB) was used to record intra-articular pressure. The transducer was connected to an amplifier which relayed signals to a hot-wire chart recorder (Devices). The transducer was calibrated using a mercury manometer at the end of each experiment. The maximum pressure recorded over a chosen period was measured and converted to mmHg.

Infusion of Saline into the Knee Joint

The 60ml syringe was filled with saline before the cannula was inserted into the knee (Fig 2.11.). After insertion of the cannula, the initial intra-articular pressure was recorded and saline was infused into the knee in increments of usually 10ml. Infusion was ceased when sufficient inhibition of the H-reflex was achieved (Chapter 11), so large volumes (> 100ml), which might cause discomfort were not reached.

STATISTICS

Parametric tests were used for testing the repeatability of methods (vide infra, 1,3 & 4). Non-parametric statistics were used for comparisons between groups as the numbers were small, normal distributions were not observed, the variances were sometimes large and the pain scales were not necessarily linear (vide infra, 2, 5, 6 & 7). The medians and ranges of results were therefore used throughout the studies. All test formulas were obtained from the same statistics book (Sokal & Rohlf,1969). Tests used were:

1. Wilcoxon's 2 sample test for differences between different groups of subjects (Chapter 3).

2. Single classification analysis of variance (anova) with equal sample sizes - to test for variation within the same subjects on more than 2 occasions (Chapter 3).

3. Coefficient of variation - to test for variation within the same subjects on 2 or more occasions (Chapter 3).

4. Fisher's exact probability test - to see if results seen in a small number of subjects could have been due to chance (Chapter 4).

5. Wilcoxon's paired signed ranks test - for changes in values for the same individuals after treatment (Chapters 5,6 & 7).

6. Spearman's rank correlation - to test for correlation between 2 variables which are not directly comparable (Chapter 7).

7. F max test - for difference between 2 coefficients of variation (Appendix).

8. Regression analysis - the slope of the regression line was used to describe the relationship between 2 variables (Chapter 11).

CHAPTER 3

EFFECTS OF INTRA-ARTICULAR BUPIVACAINE ON POST-MENISCECTOMY PAIN AND QUADRICEPS INHIBITION

INTRODUCTION

PATIENTS

METHODS

- Experimental Groups
- Surgical Details
- Measurements

RESULTS

- Control Group
- B1Ø Group
- B15 Group
- Surgery and Other Medication
- Effusion
- Repeatability of Measuring Quadriceps Activation

DISCUSSION

- Severity and Duration of Post-meniscectomy Quadriceps Inhibition
- Dissociation of Inhibition and Pain
- Effects of Bupivacaine on Inhibition and Pain
  - Site of infiltration
  - Dose-related effect on inhibition
  - Possible clinical role for bupivacaine
- Possible Causes of Post-meniscectomy Quadriceps Inhibition
  - 'Learned' response
  - Pain
  - Effusion
  - Ischaemia
  - Peri-articular stimuli

CONCLUSIONS

EFFECT OF INTRA-ARTICULAR BUPIVACAINE ON POST-MENISCECTOMY PAIN AND QUADRICEPS INHIBITION

INTRODUCTION

Weakness of the quadriceps occurs within hours after knee surgery. It is not known what causes this inhibition, how severe it is or how long it lasts. Some believe that pain is the cause (e.g. Eriksson,1981; Smith et al.,1983). Reflex inhibition of quadriceps occurs in response to an artificially induced knee infusion and can be prevented by the previous intra-articular injection of local a anaesthetic (deAndrade et al.,1965; Jayson & Dixon,1970a).

The present study was undertaken to quantify the severity and duration of post-meniscectomy quadriceps inhibition, to determine the relationship of inhibition to pain, and to observe the effect of local anaesthesia on inhibition and on the relationship between pain and inhibition. A reduction in pain might indicate a role for local anaesthesia in post-operative analgesia, with the thought that even a few hours' relief of pain might prevent a 'learned' inhibition.

In a pilot study, 10ml of either 0.5% bupivacaine hydrochloride ('Marcaine Plain') or saline was injected into the lateral compartment of the knee joint 1 hour after meniscectomy. Neither pain nor inhibition was reduced in either the 'treatment' patients (n=2) or the 'control' patients (n=2). It was decided that, in the trial proper, the bupivacaine would be infiltrated extensively into the knee joint and around the incision before suturing, and that a higher dose would also be used.

Preliminary reports of aspects of this work have been published as abstracts (Sherman et al.,1983; Young et al.,1983) and have been referred to in a review (Stokes & Young,1984). A full report of these studies has been accepted for publication in 'Clinical Physiology' (Shakespeare et al., 1985).

PATIENTS

Fourteen males (aged 18-46 years) who were undergoing medial arthrotomy and meniscectomy were studied (Table 3.1.).

METHODS

Experimental Groups

Subjects were randomly allocated to 1 of 2 treatment groups, each of 4 patients, or a control group of 6 patients (Table 3.1.). At the completion of meniscectomy the treatment groups (B10 and B15) received 10ml or 15 ml respectively, of 0.5% bupivacaine into the meniscal bed and tissues surrounding the capsular and skin incisions. The control group received no injection. The surgeon gave the injection, and neither the patients nor those making subsequent measurements knew to which group the patients belonged.

Surgical Details

Periods of tourniquet ischaemia ranged from 25-65 minutes (median = 37.5) and are shown for each group separately in Table 3.1. The type of meniscectomy (i.e. partial or total) performed on the patients in each group is also shown in Table 3.1.

Measurements

Quadriceps MVA and pain scores were recorded (as described in Chapter 2) for each leg, within 5 days pre-operatively, and at 1-2 hours, 4-5 hours, 7-9 hours, 1 day, 3-4 days and 10-15 days post-operatively.

RESULTS

There was no significant change in activation in the contralateral normal limb at any stage. The between days variation in the unoperated legs over 4 days (pre-operatively, 1 day 3-4 days and 10-15 days post-operatively) was not significant (P>0.1, 2-tailed, single classification anova with equal sample sizes). The between days coefficient of variation was 6%.

Group	No. of Patients	Age Range (yrs)	Medial Meniscectomy		Tourniquet Times (mins)	
			Partial	Total	Median	Range
Control	6	22-36	4	2	37.5	30-65
B10	4	24-46	2	2	39.0	30-60
B15	4	18-38	1	3	35.0	25-50

Table 3.1. Details of the patients studied. The B10 and B15 groups received 10ml and 15ml, respectively, of 0.5% bupivacaine.

Control Group

The controls had severe inhibition and pain during maximal contractions made during the first 24 hours after surgery (Fig 3.1.; Table 3.2.). Inhibition was still severe at 3-4 days (median = 73.5%) and persisted at 10-15 days (median = 37%). Pain was reduced by 3-4 days and was mild or absent at 10-15 days (Fig 3.1.; Table 3.3.).

B10 Group

Compared with the control group at 1-2 hours the B10 group had similar inhibition but less pain ($P<0.005$, Wilcoxon 2-sample, 1-tailed test) (Fig 3.2.). A 1-tailed test was used because the hypothesis stated that bupivacaine would specifically reduce both pain and inhibition.

B15 Group

This group had both less inhibition ($P<0.05$) and less pain ($P<0.01$) than the controls (Fig 3.2.). These effects had already been lost by 4-5 hours after surgery (Table 3.2.). Throughout the remainder of the 10-15 day period there were no differences in inhibition or pain among the 3 groups (Fig 3.3.; Table 3.3.).

Surgery and Other Medication

The type of operation performed did not influence the severity of inhibition or pain. The experimental groups were comparable in respect of the duration of tourniquet ischaemia during surgery (Table 3.1.). During the first 24 hours after surgery all patients requested papaveretum except for 3 in the B15 group and 1 in the control group. From the second day until discharge from hospital there were no differences in the consumption of analgesics between the 3 groups.

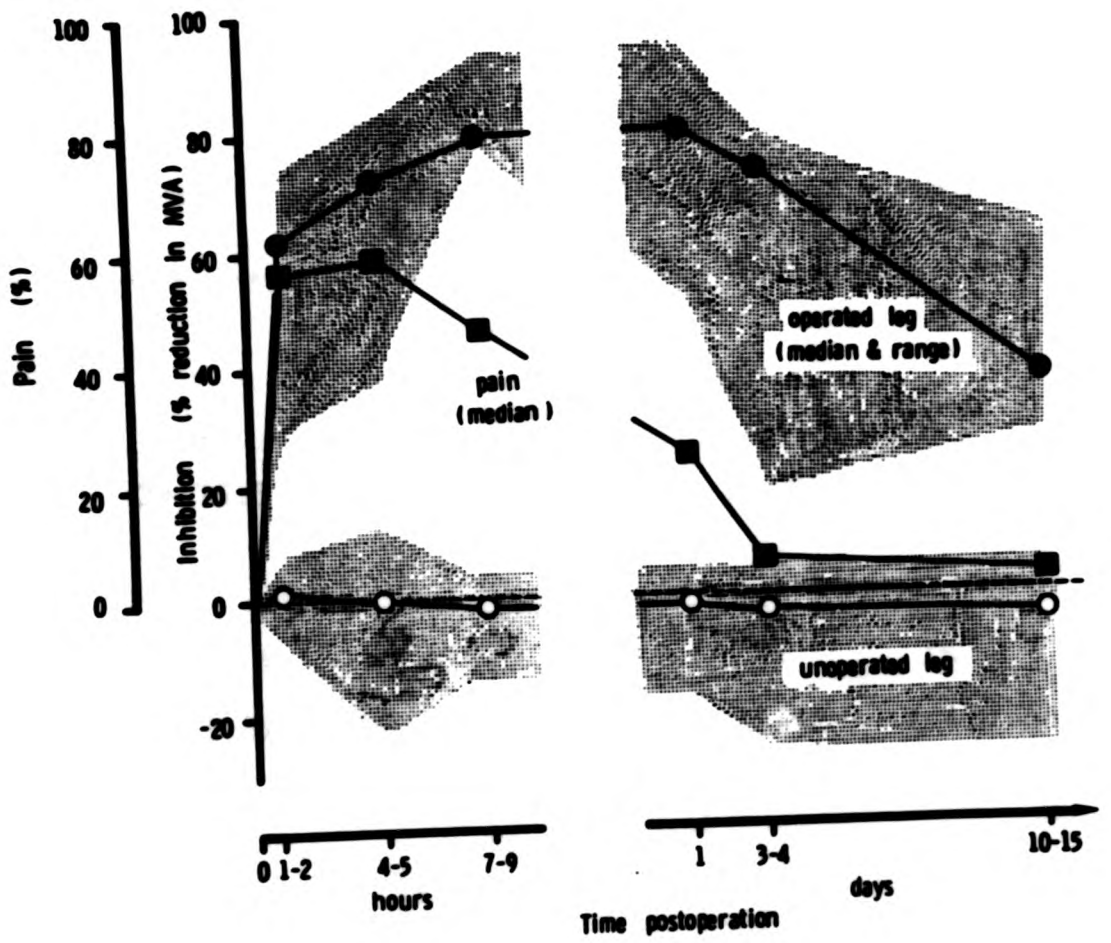

Figure 3.1. Quadriceps inhibition and knee pain during contraction in the control group. Medians ● ○ inhibition ■ pain. ▒ Range for inhibition.

	TIME AFTER OPERATION					
	Hours					
	1 - 2		4 - 5		7 - 9	
	Pain %	Inhib %	Pain %	Inhib %	Pain %	Inhib %
CONTROLS						
Median	57	62	58.5	72	46.5	79
Range	29–78.5	29–75.5	22–66	37–83	22–67	77–94
B1Ø						
Median	13.5	57.5	23	76	15	86.5
Range	2–26	28–85	2–55	71–82	4–41	79–94
B15						
Median	8	23	22	7Ø	51	96
Range	Ø–3Ø	1Ø.5–54	Ø–69	8–93.5	16–87	25–1ØØ

Table 3.2. Quadriceps inhibition and knee pain during contractions: early post-operative period.

	TIME AFTER OPERATION					
	1 Day		3–4 Days		1Ø–15 Days	
	Pain %	Inhib %	Pain %	Inhib %	Pain %	Inhib %
CONTROLS						
Median	24.5	8Ø	6	73.5	3	37
Range	11–45	52–96	1–63	17–8Ø	Ø–1Ø	27–64
B1Ø						
Median	14.5	79.5	Ø	66	Ø	46
Range	5–57	61–87	Ø–4	62–78	Ø	11–81
B15						
Median	33	91	6	76	4.5	23.5
Range	Ø–67	35–96	Ø–29	39–87	Ø–21	-2–46

Table 3.3. Quadriceps inhibition and knee pain during contractions: late post-operative period.

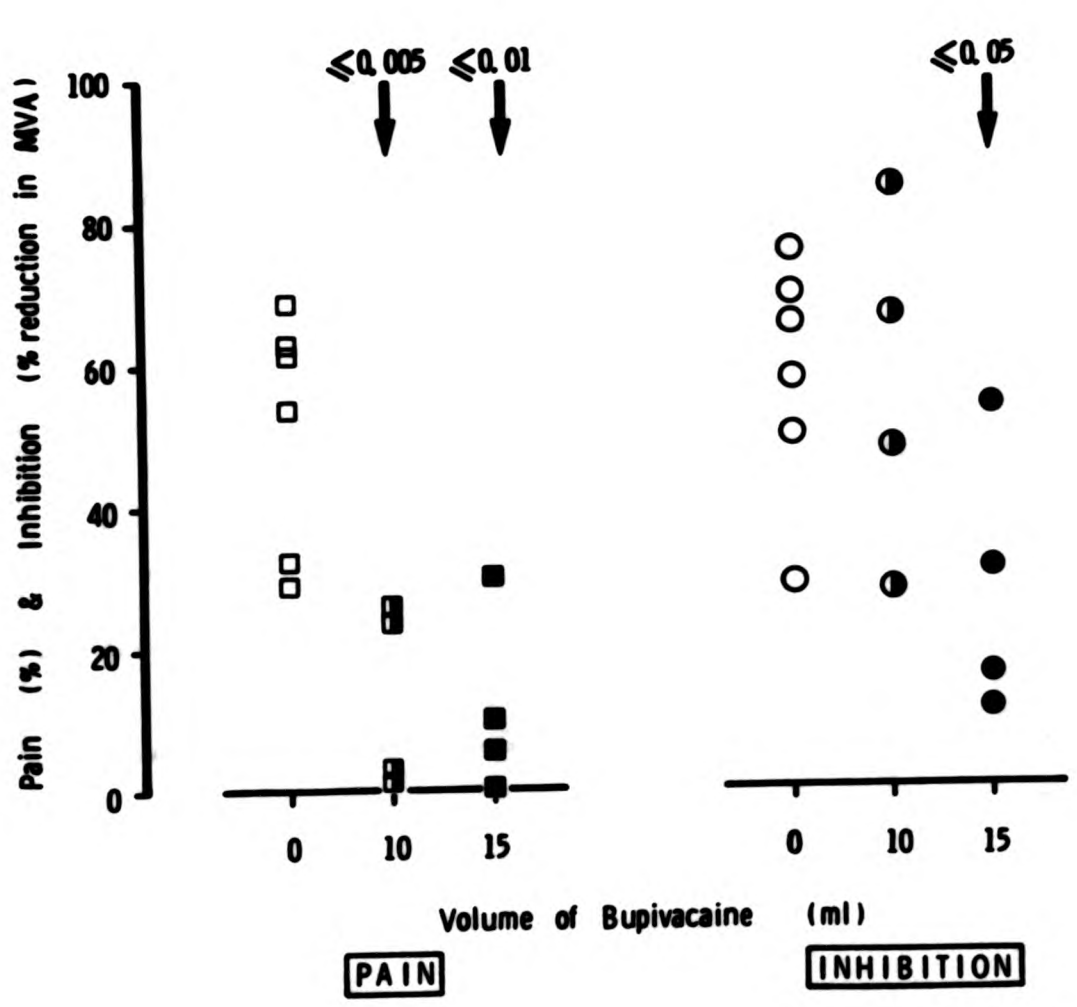

Figure 3.2. Severity of knee pain and quadriceps inhibition 1-2 hours after per-operative infiltration of the knee with bupivacaine.

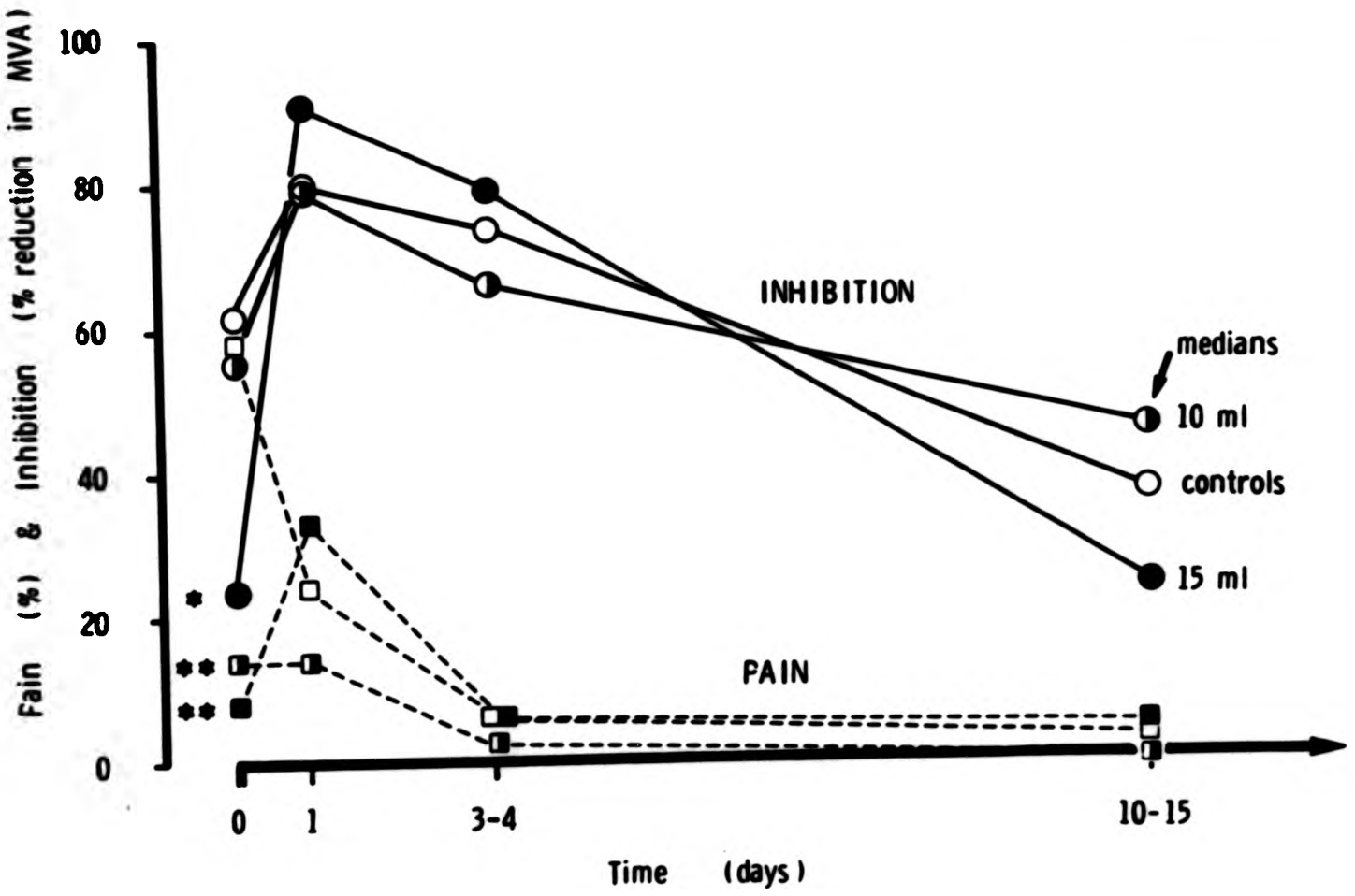

Figure 3.3. Group medians for quadriceps inhibition and knee pain during contractions made in the first 2 post-operative weeks.

* $p < 0.5$ ** $p < 0.01$

Effusion

One patient in the B15 group developed a knee effusion on the 3rd post-operative day and this was aspirated prior to testing quadriceps MVA for the present study.

Repeatability of Measuring Quadriceps Activation

Between days variation. The coefficient of variation for repeated measurements of MVA made in the unoperated limbs was 6%. The use of surface IEMG is therfore a reliable method for monitoring post-meniscectomy quadriceps activation.

Between attempts variation. The variation between the 2 attempts made on each occasion was very small for both legs. Pre-operatively, the coefficient of variation (CV) was 2.6% for the unoperated legs and 2.8% for the operated legs. The first post-operative efforts with the unoperated legs had a CV of 2.4%. The CV for the first post-operative efforts with the operated legs was not calculated because the absolute values for IEMG were so small that it would have given a misleadingly high CV. Instead, the absolute differences between efforts made with the operated legs were compared with those from the unoperated legs. The histogram (Fig 3.4.) shows the absolute differences for both legs in the 6 patients who did not receive per-operative bupivacaine.

The results for the unoperated limbs of all other patients who had been studied at this stage of the investigations and who demonstrated severe quadriceps inhibition (median = 70%) were included in the calculations. These patients did not take part in the main study and were otherwise unselected for the purposes of the repeatability study. These were: 2 medial meniscectomy patients who received 10ml bupivacaine 1 hour post-operatively (these results were obtained before injection), 2 medial arthrotomy patients, and 2 lateral meniscectomy patients.

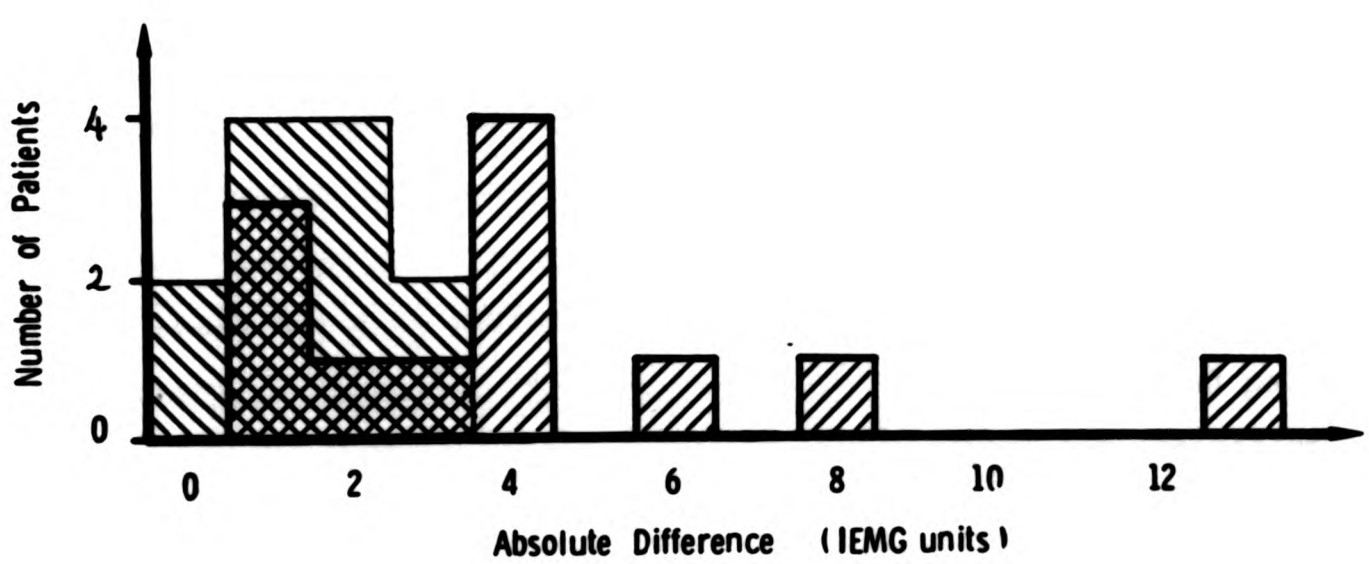

Figure 3.4. **The absolute differences between the first and second efforts of the earliest post-operative tests for MVA in both legs.**

DISCUSSION

Post-meniscectomy quadriceps weakness is due to reflex inhibition of voluntary activation.

Severity and Duration of Post-meniscectomy Quadriceps Inhibition

The present study has shown that 70-80% quadriceps inhibition is typical for the first few days following medial arthrotomy and meniscectomy and that 30-50% inhibition is commonly present even 2 weeks after surgery.

Dissociation of Inhibition and Pain

During the early post-operative period severe inhibition is accompanied by severe pain. After 24 hours the inhibition is still severe but pain subsides and by 10-15 days inhibition persists with little or no pain. This dissociation of inhibition and pain indicates that a stimulus other than pain is causing the inhibition after 24 hours.

Effects of Bupivacaine on Inhibition and Pain

This study has confirmed, by the effect of an afferent block, that the inhibition of quadriceps is caused by stimuli originating from the knee joint. The fact that the temporary blocking of early pain had no effect on the subsequent severity of inhibition, also rules out the possibility that inhibition is a 'learned' response to early pain.

Site of infiltration. In the pilot study the intra-articular injection given post-operatively would not be expected to reach capsular endings (as opposed to synovial endings). It has been shown that peri-articular stimuli can cause quadriceps inhibition (Stener,1969; see Chapters 12 & Appendix). The tissues surrounding the incision were infiltrated with bupivacaine in the present study and not in the pilot study. Therefore, if the inhibitory stimuli originated from the incision they would not have been influenced by

the post-operative injection. The fact that 10ml of bupivacaine reduced pain with the per-operative infiltration but not the post-operative injection, suggests that the post-operative injection did not reach the area of origin of the painful stimuli.

Dose-related effect on inhibition. The smaller dose of bupivacaine reduced the severity of pain without having any effect on the severity of inhibition. The larger dose was sufficient to block both pain and inhibition. This was not due to pain being controlled more effectively by the larger dose (Fig 3.2.). The effects of the different doses of bupivacaine on inhibition show that inhibition and pain are not closely linked even in the early period when they are both severe. Bupivacaine is said to have a preferential analgesic effect before it acts as an anasethetic i.e. the small pain fibres (A delta and C) are blocked before the larger fibres carrying sensations of cold, heat, touch and deep pressure (Atkinson et al.,1982; Cousins & Bridenbaugh,1980). It is possible, therefore, that only the larger dose was sufficient to block the afferent stimuli in the larger fibres (i.e. from receptors other than pain receptors) and that it is these stimuli which result in inhibition, rather than those carried by the small fibres. The possibility that some of the inhibition might be caused by pain impulses which do not reach conciousness cannot be tested in man. It can be said, however, that neither the late nor the early inhibition seen in the patients in the present study can be entirely attributed to perceived pain.

In a study by I.Arvidsson, E.Eriksson & E.Knutsson (unpublished work, referred to by Eriksson,1981) epidural injection of a dilute local anaesthetic increased the MVA of quadriceps after knee surgery. Because pain was blocked and motor fibres were spared, they assumed that the inhibition was pain-induced but their anaesthetic may not have blocked only the smaller fibres; other afferent fibres may also have been blocked.

Possible clinical role for bupivacaine. The effect of local bupivacaine on inhibition might well permit the effective performance of quadriceps exercises but it is much too brief to be clinically useful. Opiate requirement was less in the B15 group but the numbers are too small to allow any conclusion other than that it might be worthwhile carrying out a clinical trial to evaluate per-operative bupivacaine as a post-operative analgesic (see Chapter 12- 'Topics for Further Study'). The effect would only last for a few hours, but the period of severe pain is also short.

Possible Causes of Post-meniscectomy Inhibition

'Learned' response. The inhibition was shown not to be a 'learned' response to disuse of the injured limb prior to surgery because pre-operative values of MVA were similar to those of the uninjured limb and were severely reduced immediately after surgery.

Pain. The results of the present study exclude perceived pain as the cause of quadriceps inhibition in the patients studied. This was demonstrated by the dissociation of inhibition and pain after 24 hours, and by the dose-related effect of bupivacaine on early inhibition and pain.

Effusion. Experimental joint distension has been shown to cause reflex inhibition of voluntary quadriceps contractions in the absence of pain (deAndrade et al.,1965; Jayson & Dixon,1970a). Aspiration of effusion which developed in some meniscectomy patients (including 1 from the present study) reduced, but did not abolish, the inhibition (Stokes & Young,1984; Chapter 7). While joint effusion is clearly a potential cause of inhibition it could not be implicated as a cause in the present series of meniscectomy patients as none of them had a clinically apparent effusion when the reported data were obtained.

Ischaemia. It has been suggested that the use of a tourniquet during surgery causes post-operative impairment of quadriceps function (Saunders et al.,1979; Dobner & Nitz,1982). The reduced MVA observed

in the present meniscectomy patients would not have been prevented by bupivacaine if it had been due to tissue damage produced by ischaemia. Voluntary ischaemia in normal subjects (for periods similar to those necessary for meniscectomy) failed to produce any abnormalities in quadriceps function (Chapter 4). Ischaemia was therefore ruled out as the main cause of the inhibition observed in the present meniscectomy patients.

Peri-articular stimuli. Preliminary studies suggest that the severity of inhibition may be influenced by the type of incision (i.e. arthrotomy or arthroscopy). This topic will be discussed later (Chapter 12).

CONCLUSIONS

1. Following arthrotomy and meniscectomy, inhibition of voluntary quadriceps activation is severe for at least 3 days (typically 70-80%) and persists for at least 2 weeks (typically 35-40%).

2. Post-meniscectomy quadriceps inhibition persists in the absence of pain and of obvious knee joint effusion.

3. Extensive intra- and peri-articular infiltration with an adequate dose of bupivacaine reduces post-meniscectomy quadriceps inhibition confirming that the mechanism causing the inhibition is a reflex involving stimuli from the knee.

4. The dose-related dissociation of the effects of bupivacaine on inhibition and pain suggests that the inhibitory impulses were not transmitted along the small pain fibres.

5. The effect of bupivacaine on reflex inhibition is too brief to permit a useful period of effective post-operative quadriceps exercises.

CHAPTER 4

QUADRICEPS FUNCTION AFTER VOLUNTARY ISCHAEMIA IN NORMAL SUBJECTS

INTRODUCTION

SUBJECTS

METHODS

- Period of Ischaemia
- Muscle Function Tests
  - Needle electromyography
  - Maximal voluntary activation of quadriceps
  - Isometric quadriceps strength
  - Impulse of one-legged leaps
  - Height of one-legged leaps
- Timing of Tests

RESULTS

DISCUSSION

- Aspects of Quadriceps Function Studied
- Possible Explanations for the Present Negative Findings
  - Tourniquet position
  - re-establishment of Neural Transmission

CONCLUSION

QUADRICEPS FUNCTION AFTER VOLUNTARY ISCHAEMIA IN NORMAL SUBJECTS

INTRODUCTION

It has been suggested that the period of tourniquet ischaemia during knee surgery may contribute to post-operative weakness (Saunders et al.,1979; Dobner & Nitz,1982). If this were true, the findings with the meniscectomy 'model' would be less relevant to patients with, for example, rheumatoid arthritis (i.e. joint pathology without a history of surgery & tourniquet ischaemia).

The present study was therefore carried out to see if per-operative tourniquet ischaemia could have contributed to the reduced MVA of quadriceps observed in meniscectomy patients. The effects of unilateral lower limb ischaemia were studied in 4 normal subjects. This study is to be published as a chapter in a book on biomechanical measurement (see 'PUBLICATIONS').

SUBJECTS

Three males and 1 female, aged 25-36 were studied. They were all involved in conducting the present series of studies.

METHODS

Period of Ischaemia

A pneumatic tourniquet was applied to 1 thigh exactly as for a patient at this hospital (Chapter 2). Duration of ischaemia was for as long as each subject could tolerate viz. 37, 38, 47 and 50 minutes.

Muscle Function Tests

Needle electromyography (EMG). Three to 4 weeks after the period of ischaemia, qualitative EMG was performed by Dr K Mills (a Clinical Neurophysiologist). A concentric needle electrode was used to

explore rectus femoris (RF), vastus medialis (VM) and vastus lateralis (VL) of both legs both at rest and during voluntary activity for evidence of partial denervation. Spontaneous activity (bizzare high frequency discharges (Aminoff,1980), fibrillations and positive sharp waves) at more than 2 sites would have been considered abnormal. In addition, interference pattern was assessed during a maximal voluntary isometric contraction of quadriceps. Distal motor latency was tested by stimulating the femoral nerve at the inguinal ligament and recording evoked potentials at a measured distal point in rectus femoris. Latencies were then compared between the 2 legs. The examiner was unaware of which leg had been ischaemic.

Maximal voluntary activation of quadriceps. Quadriceps activation was measured using surface IEMG as described in Chapter 2.

Isometric quadriceps strength. Quadriceps strength was measured as described in Chapter 2.

Impulse of one-legged leaps. The force/time curve of take-off in a one-legged vertical leap was recorded by a force plate (Kistler, Type 9821A) linked to a computer (Dec PDP - 11/23). These tests were performed by Dr M Whittle (a Consultant Clinical Physiologist). The shaded area in Fig 4.1. was measured using a MOP planimeter (Reichert-Jung) and represents the impulse during take-off (N.s). Three leaps on each leg were performed using arm swing and knee flexion to assist take-off, and the best was taken on each occasion.

Height of one-legged leaps. The height of a vertical leap was measured by standing sideways to a wall and marking the wall with an inked fingertip, at full stretch, before and during a jump. Arm swing and knee flexion were again permitted. The best of 3 leaps was recorded for each leg on each occasion.

Timing of Tests

Table 4.1. is a timetable of the muscle function tests.

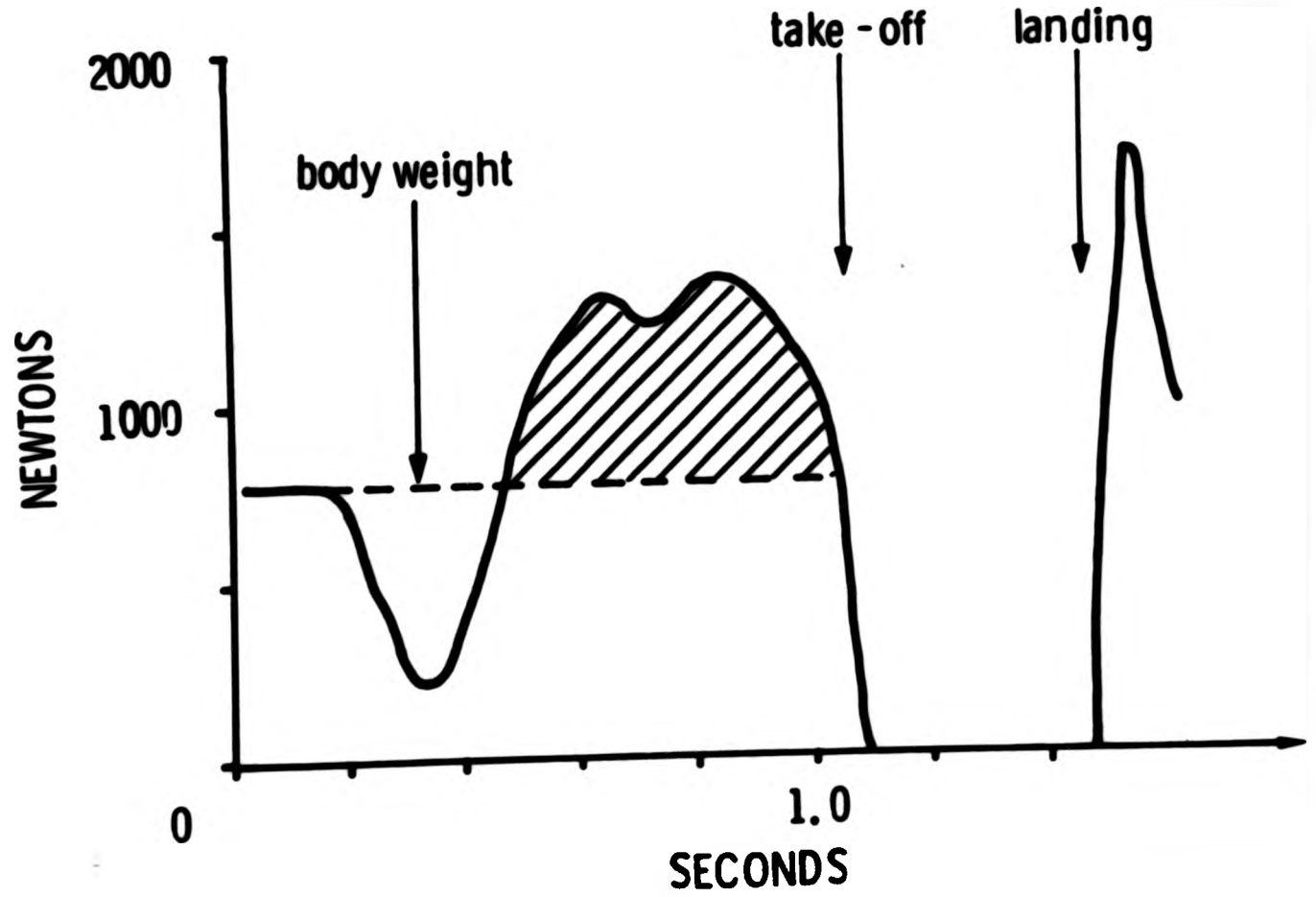

Figure 4.1. Trace obtained from the force plate showing the force / time characteristics of a one-legged leap.
▨ the impulse exerted before take-off.

	Test Leg	Control Leg	Needle EMG	MVA	MVC	Leap Impulse	Leap Height
Pre-ischaemia	+	+		+	+	+	+
Post-ischaemia							
Immediately	+			+			
2 hours	+			+	+		
24 hours	+	+		+	+	+	+
3 days	+	+		+	+		
3-4 weeks	+	+	+	+	+	+	+

Table 4.1. Timing of tests used to study quadriceps function after voluntary tourniquet ischaemia.

RESULTS

Needle EMG at 3-4 weeks showed no evidence of denervation. One high frequency discharge was detected in VM of 1 subject but this was in the control leg. In another subject there was fibrillation at 1 site in VM of the experimental leg but this was not considered to be abnormal. Distal motor latencies in the ischaemic legs, expressed as a percentage of those in the control legs were 92%. 102%, 108% and 110%, again implying no abnormality (the raw data are shown in Table 4.2.)

None of the subjects showed any evidence of impaired quadriceps function. The ranges of results for MVA, MVC and the impulse and height of one-legged leaps, expressed as percentages of the pre-ischaemia values, are shown in Table 4.3. (individual results for the 4 tests are shown in Tables 4.4.-4.7.).

DISCUSSION

Aspects of Quadriceps Function Studied

The various techniques used allowed different aspects of quadriceps function to be studied (Table 4.8.). Distal motor latency examined distal nerve conduction in the femoral nerve fibres supplying the muscle, assuming normal neuromuscular transmission. The other needle EMG test, which provided a qualitative study of activation, looked for abnormal or spontaneous activity in resting muscle and also at the interference pattern. The surface EMG was used to provide a quantitative study of MVA.

Isometric quadriceps strength was measured using the simple set up of a chair and strain gauge (Chapter 2). A "Cybex" machine can be used to measure both isometric and isokinetic force, but in the present study, dynamic strength and dynamic co-ordinated movement were measured as the impulse and height of one-legged leaps.

Subject	Distal Motor Latency (ms) Test leg	Control leg	TL/CL %
1	4.2	4.1	102
2	4.0	3.7	108
3	4.4	4.0	110
4	3.5	3.8	92

TL = Test leg
CL = Control leg

Table 4.2. Distal motor latency 3-4 weeks after voluntary ischaemia.

	Ranges of results post-ischaemia (% of pre-ischaemia value)	
	Test leg	Control leg
Activation	94 - 105	94 - 104
Isometric strength	73 - 102	79 - 99
Leap impulse	74 - 118	63 - 112
Leap height	95 - 117	98 - 119

Table 4.3. Ranges of results of muscle function tests after voluntary ischaemia.

	Time after Ischaemia									
	5 mins		2 hours		24 hours		3 days		3-4 weeks	
Subject	TL	CL	TL	CL	TL	CL	TL	CL	TL	CL
1	105	-	103	-	100	101	101	97	100	101
2	102	-	99	-	94	103	98	101	95	100
3	101	-	99	-	99	101	101	103	99	104
4	101	-	100	-	104	110	101	109	104	104

TL = Test leg
CL = Control leg

Table 4.4. Quadriceps activation after voluntary ischaemia (percentages of pre-ischaemia values for MVA).

	Time after Ischaemia							
	2 hours		24 hours		3 days		3-4 weeks	
Subject	TL	CL	TL	CL	TL	CL	TL	CL
1	100	-	102	98	95	94	94	95
2	98	-	86	91	95	99	94	96
3	89	-	85	90	97	84	96	81
4	87	-	82	87	73	79	77	86

TL = Test leg
CL = Control leg

Table 4.5. Isometric quadriceps strength after voluntary ischaemia (percentages of pre-ischaemia values for MVC).

	Time after ischaemia			
	1 day		3-4 weeks	
Subject	TL	CL	TL	CL
1*	-	-	-	-
2	118	109	112	89
3	116	63	106	71
4	74	107	85	104

TL = Test
CL = Control leg

(* Subject 1 was not tested before ischaemia. The absolute values were: 1 day TL= 706N.s, CL= 820N.s and 3-4 weeks TL= 651N.s, CL= 704N.s.)

Table 4.6. Impulse of one-legged leaps after voluntary ischaemia (percentages of pre-ischaemia values).

	Time after Ischaemia			
	1 day		3-4 weeks	
Subject	TL	CL	TL	CL
1*	-	-	-	-
2	100	105	117	119
3	95	100	95	98
4	98	106	98	103

TL = Test leg
CL = Control leg

(* Subject 1 was not tested before ischaemia. The absolute values were: 1 day TL= 27.9cm, CL= 29.0cm and 3-4 weeks TL= 28.6cm, CL= 30.8cm.)

Table 4.7. Height of one-legged leaps after voluntary ischaemia (percentages of pre-ischaemia values).

	TEST	FUNCTION TESTED
1.	Needle electromyography (EMG)	* Nerve conduction * Propagation of action potential
2.	Maximal voluntary activation	* Recruitment
3.	Isometric strength	* Force production
4.	Impulse of 1-legged leap	* Force production in dynamic contraction
5.	Height of 1-legged leap	* Force production in co-ordinated movement

Table 4.8. Different aspects of quadriceps function studied.

Possible Explanations for the Present Negative Findings

Ischaemia did not influence quadriceps MVA in the normal subjects despite the fact that the tourniquet pressure and position, and the durations of ischaemia were similar to those used for the present series of meniscectomy patients. Dobner & Nitz (1982) carried out a study in which patients underwent meniscectomy either with or without a tourniquet. They showed that tourniquet ischaemia may be associated with post-operative impairment of quadriceps function and EMG evidence of denervation. The durations of ischaemia in their experimental group were similar to those of the present normal subjects and meniscectomy patients , but their tourniquet pressures were lower (300-450 mmHg). Their patients' quadriceps were examined 6 weeks post-operatively by needle EMG, one-legged leaps and by determining the maximal resistance generated by quadriceps using a progressive resistance exercise table. All of the non-tourniquet group showed normal EMG activity but 17 out of 25 of the tourniquet group did not. Our negative results in 4 out of 4 subjects are unlikely to have been due to chance (P=0.03 Fisher's exact probability test, 2-tailed). The 2 groups of patients in Dobner & Nitz's study also showed functional differences. One-legged leap heights were significantly different, both between the 2 groups and within the tourniquet group (between those with abnormal and those with normal EMGs). Maximum quadriceps resistances (i.e. maximum weights lifted) were significantly different between the normal and abnormal EMG patients, within the tourniquet group, but not between the 2 main groups.

Tourniquet position. The site of the tourniquet was the same for the present normal subjects and meniscectomy patients (i.e. as far proximal as possible) but neither Dobner & Nitz (1982) nor a study by Saunders et al. (1979) states the precise location of the tourniquet in their patients. Perhaps this might be an important factor if their cuffs were placed more distally and were over motor points.

Re-establishment of neural transmission. An alternative explanation lies in the basic differences between normal subjects and surgical patients *viz.* the patients have received trauma to the knee and also a period of joint immobilization. During tourniquet ischaemia nerve impulses are blocked due to axonal pressure and/or ischaemia. Perhaps re-establishment of normal transmission along the nerves would not occur quickly unless normal movement patterns were performed. This might explain the negative results in the normal subjects as they were fully mobile immediately (i.e. about 5 minutes) after ischaemia. Knee surgery patients usually remain in bed with the knee immobilized for at least 2 or 3 days. Perhaps a combination of ischaemia, knee trauma and immobilization is necessary to produce significant tissue damage.

Another possibility is that the point when the normal subjects could no longer tolerate the tourniquet might be the point beyond which tissue damage would occur.

Ischaemia could not have been the main cause of the failure of voluntary activation seen in the present series of meniscectomy patients as it could be relieved by intra-articular local anaesthesia (Chapter 3).

CONCLUSION

The absence of EMG and functional abnormalities in the present normal subjects after voluntary tourniquet ischaemia contrasts with the results of previous studies (Saunders *et al.*,1979; Dobner & Nitz,1982) and this cannot be explained. It can be concluded , however, that the reduced MVA observed in the present series of meniscectomy patients being studied is unlikely to have been caused by the tourniquet alone.

CHAPTER 5

EFFECT OF JOINT ANGLE ON REFLEX INHIBITION OF VOLUNTARY QUADRICEPS ACTIVATION AFTER MENISCECTOMY

INTRODUCTION

SUBJECTS

METHODS

- Surgical Details
- Main Study
- Pilot Study I
- Pilot Study II
- Measurements

RESULTS

- Main Study: Quadriceps Activation in Extension and 40° Flexion of the Knee Joint
- Pilot Study I: Quadriceps Activation at Different Angles of the Hip and Knee
- Pilot Study II: Reciprocal Activation of Quadriceps and Hamstrings

DISCUSSION

- Clinical Implications of the Flexion Phenomenon
- Possible Mechanisms of the Flexion Phenomenon: Preliminary Tests in the Present Investigations
  - Pilot Study I
  - Pilot Study II
- Other Mechanisms to Consider
  - Intra-articular pressure
  - Capsular stretch

FURTHER STUDIES

CONCLUSIONS

EFFECT OF JOINT ANGLE ON INHIBITION OF VOLUNTARY QUADRICEPS ACTIVATION AFTER MENISCECTOMY

INTRODUCTION

Isometric quadriceps exercises are usually performed with the knee extended. The present study was prompted by the clinical observation that post-meniscectomy patients could achieve a stronger quadriceps contraction with the knee flexed.

Since conducting the present study, a very similar study has been published by Krebs et al. (1983) which reported similar results but the interpretations of the 2 studies differ (vide infra, 'DISCUSSION').

The present study was carried out to test the effect of alterations in knee joint angle on the voluntary activation of quadriceps in post-meniscectomy patients. In addition, some preliminary tests were performed on a few patients and normal subjects to see which factors might be worth pursuing in future studies to evaluate possible mechanisms for the observed effect e.g. altering muscle length and recording reciprocal activation of quadriceps and hamstrings (vide infra, Pilot Studies I & II).

Some preliminary results of this study have been published as an abstract (Shakespeare et al., 1983).

SUBJECTS

Patients

Seventeen men (aged 19-49 years) were studied. All underwent medial arthrotomy and all but one had a meniscectomy.

Normal Subjects

Two females (aged 20 and 24 years) and 1 male (aged 33 years) were studied in 1 test only (vide infra, Pilot Study II).

METHODS

Surgical Details

Duration of tourniquet ischaemia for the patients ranged from 20-71 minutes (median = 35). One of the 17 patients underwent arthrotomy only.

Main Study

Quadriceps MVA was recorded over RF in extension (0^{o}) and approximately 40^{o} of knee flexion in all patients (n = 17, only 14 pre-op).

Pilot Study I

In 3 patients, different knee angles were used with the hip both flexed and in neutral to examine the effect of changing muscle length. The order in which the different angles were tested was randomized.

Pilot Study II

Simultaneous recordings from quadriceps (over RF) and hamstrings (over St) were made in 3 of the patients (both legs) and in 3 normal subjects (1 leg) to see if the changes in quadriceps activation at different knee angles might be due to reciprocal inhibition. Activation was recorded during maximal quadriceps contractions in knee extension and 40^{o} flexion, and also during maximal hamstring contractions in 10^{o} of knee flexion. Recordings made over quadriceps during hamstring contractions were expressed as a percentage of quadriceps MVA, and recordings from hamstrings made during quadriceps contractions were expressed as a percentage of hamstring MVA. These patients were studied 4 days after surgery but only 1 of them was also studied pre-operatively.

Table 5.1. shows the numbers of patients who underwent each test.

	PATIENTS			NORMAL SUBJECTS
	Pre-op	Post-op		
		2-5 days	10 days	
	n	n	n	n
Main Study	14	16	9	-
Pilot Study I	3	3	-	-
Pilot Study II	1	3	-	3

Table 5.1. Numbers of subjects (n) undergoing each test. Recordings of quadriceps activation were made over rectus femoris, and recordings of hamstring activation were made over semitendinosus.

Measurements

Surface electrodes were used to record muscle activity (as described in Chapter 2) in both patients and normal subjects. Patients were studied pre-operatively (except 2) and post-operatively at 2-5 days (n = 16) and 10 days (n = 9).

RESULTS

Quadriceps activation is expressed as IEMG units rather than as inhibition in order to allow the data to be presented in the same way for the patients' injured and uninjured limbs and for the normal subjects. Also, not all patients were studied pre-operatively.

Main Study : Quadriceps Activation in Knee Extension and 40° Flexion

In the uninjured leg, quadriceps activation in flexion was similar to the valuesobtained in extension (Fig 5.1.a). In the injured leg pre-operatively, MVA was unaltered when the knee was in flexion rather than in extension. At 2-5 days post-operatively, MVA was reduced from its pre-operative level in extension (as expected from the study reported in Chapter 3) and increased from this value with the knee flexed ($P<0.01$, Wilcoxon paired signed ranks test, 2-tailed, n=16). At 10 days this flexion phenomenon was still present in those patients who still had reduced activation in extension (Fig.5.1.b).

Pilot Study I: Quadriceps Activation at Different Angles of the Hip and Knee

Of the 3 patients studied at 3-5 days (Table 5.1.) only 2 showed a significant decrease in MVA with the hip neutral and the knee extended. Both of these showed increased activation in flexion. Hip angle did not alter the activation recorded at the different knee angles in the unoperated limb (Fig 5.2.a) and neither did it influence the effect of knee angle on inhibition in the operated limb (Fig 5.2.b).

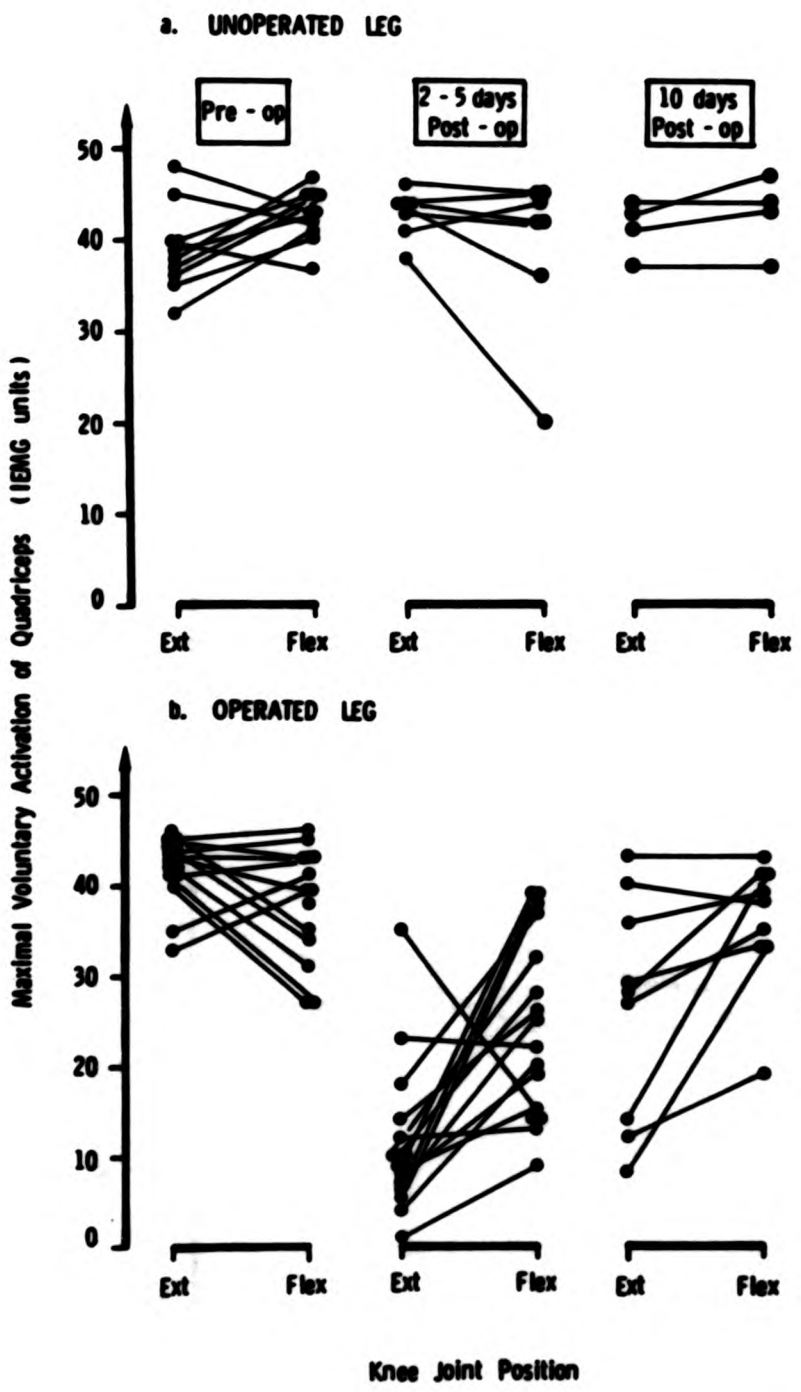

Figure 5.1. Quadriceps activation in extension and 40° knee flexion in both limbs.

a. Unoperated b. Operated

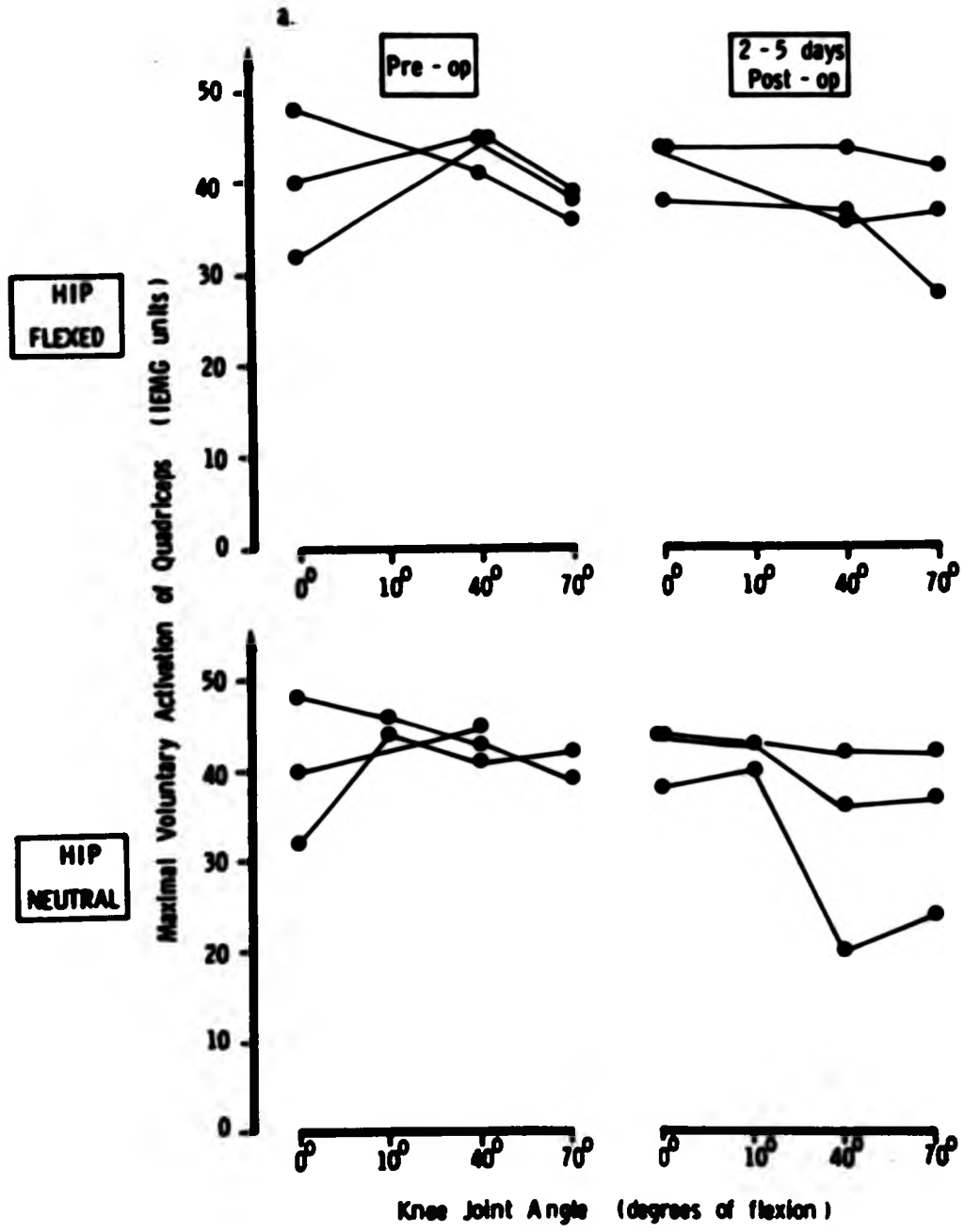

Figure 5.2.a Quadriceps activation at different hip and knee angles in the unoperated limb.

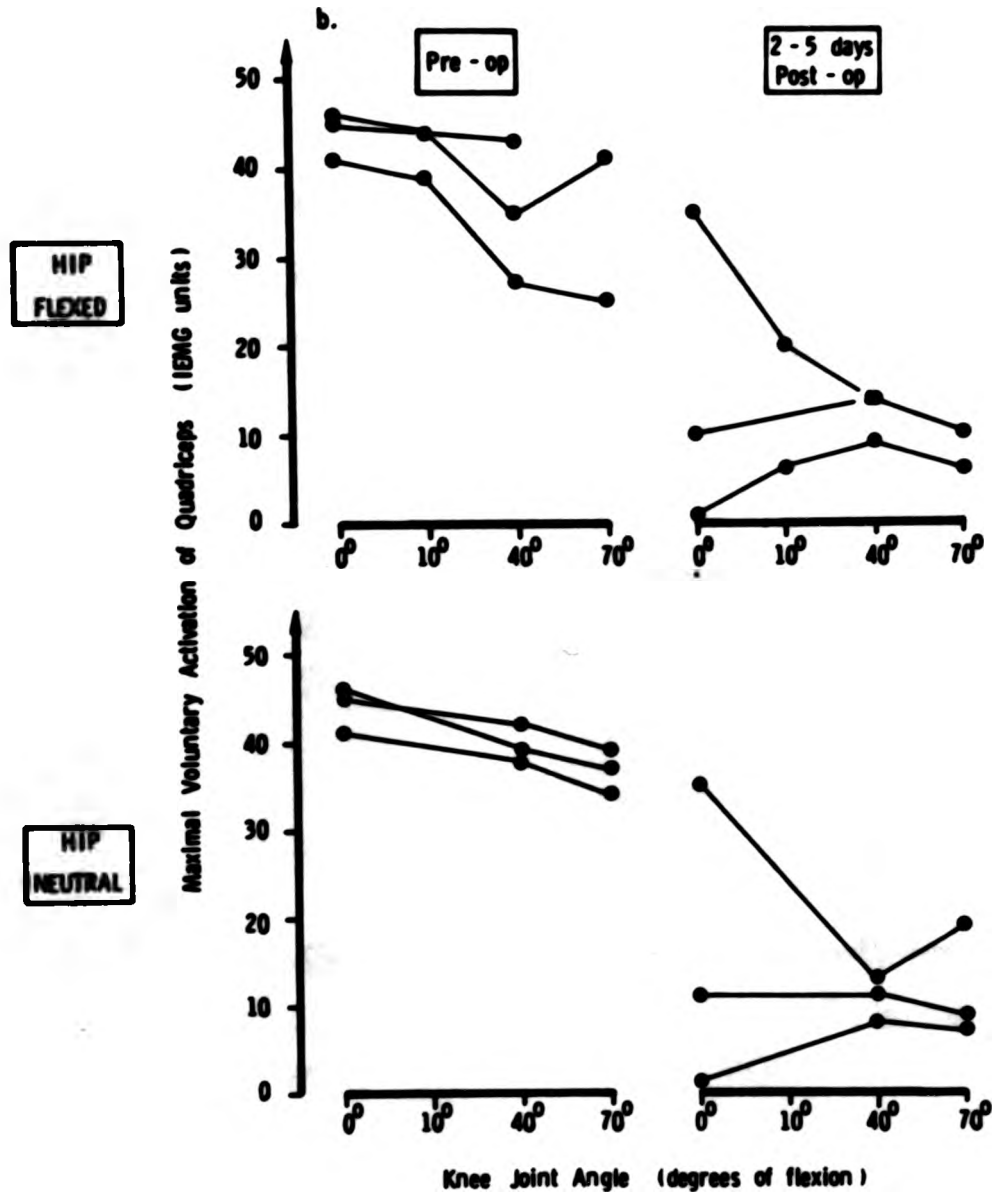

Figure 5.2.b Quadriceps activation at different hip and knee angles in the operated limb.

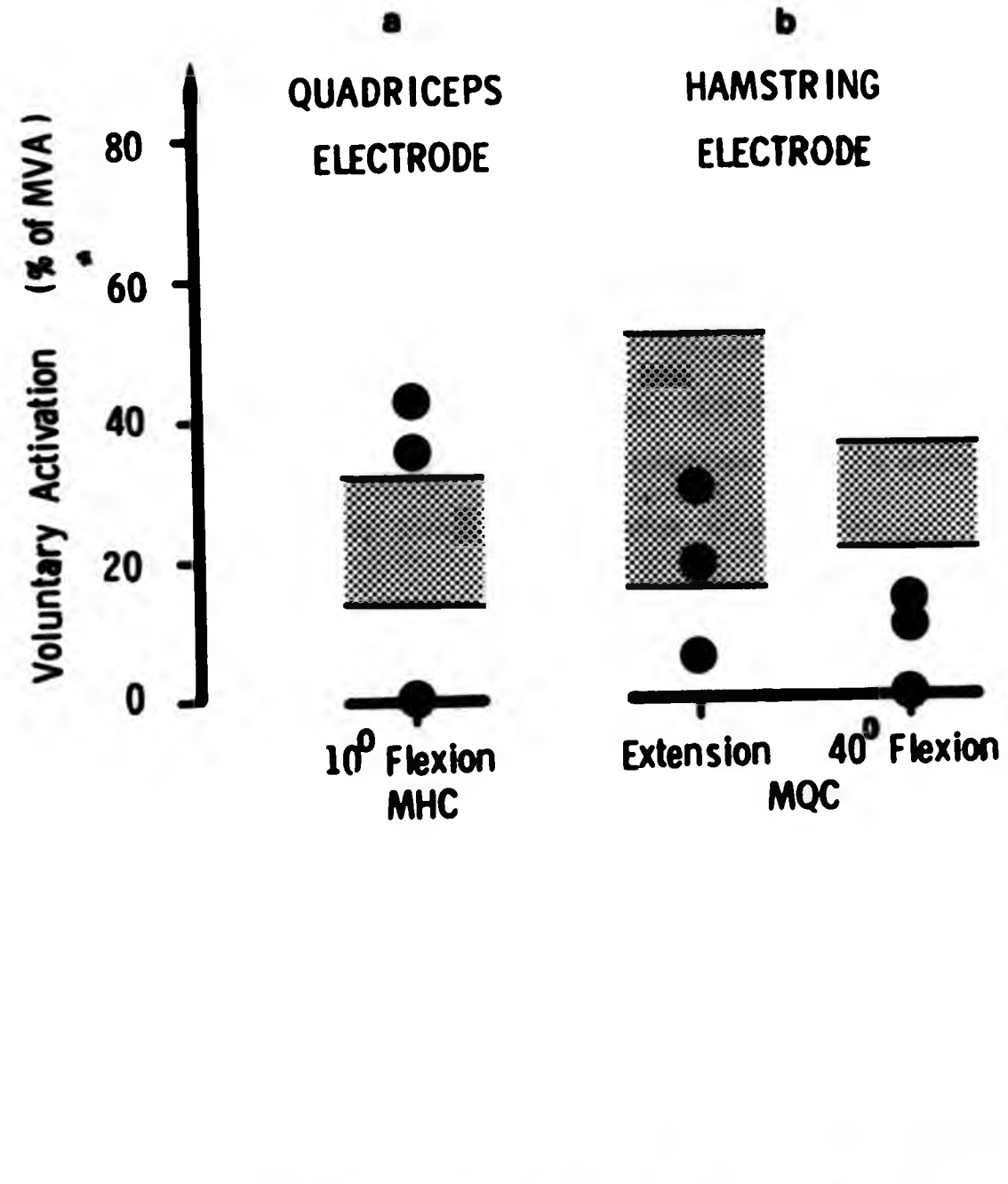

Figure 5.3. Reciprocal activation of quadriceps and hamstrings in extension and 40° knee flexion.

MHC = Maximal hamstring contraction

MQC = Maximal quadriceps contraction

▨ Normal Range ● Patients post-op

Pliot Study II: Reciprocal Activation of Quadriceps and Hamstrings

Antagonist activation during an MVC of the agonists was expressed as a percentage of the antagonists' MVA. In the normal subjects (n=3) and the uninjured limbs of the patients (pre-operatively n=1, post-operatively n=3), reciprocal activation of quadriceps was between 14% and 33%. Reciprocal activation of hamstrings was 14-51% in extension and 20-36% in flexion.

The results from the normal subjects and the patients' uninjured limbs have been used as the normal ranges in Fig 5.3. Post-operatively, quadriceps activation during maximal hamstring contractions was variable, with all 3 subjects lying outside (above and below) the normal range (Fig 5.3.a). Hamstring activation during maximal quadriceps contractions only appeared to be reduced in 1 patient in extension. In flexion however, all 3 patients showed less hamstring activation than in extension (post-operatively), and than the normal range (Fig 5.3.b).

DISCUSSION

A review of the literature revealed that various authors had studied the effect of knee joint angle on quadriceps function but with different objectives in mind e.g. Hallén & Lindahl (1967) examined the roles played by the different heads of quadriceps at different angles in order to explain quadriceps lag. They compared results obtained from post-knee surgery patients with those from normal subjects rather than with the patients' uninjured legs. The finding of the main study in this chapter, that post-meniscectomy quadriceps inhibition is reduced when the knee is in a flexed position, has also been observed in patients with other knee joint pathologies *viz*. recently, Krebs *et al*. (1983) reported similar findings in post-arthrotomy patients and Stratford (1981) demonstrated that quadriceps inhibition, in patients with knee joint efusions, was less at 30° of knee flexion than in extension.

Clinical Implications of the Knee Flexion Phenomenon

The fact that, after meniscectomy, quadriceps MVA is greater with the knee flexed than with it in the conventional extended position indicates that a greater proportion of the muscle is being activated or that the same proportion is being more completely activated. It would therefore be reasonable to suggest that, in the presence of inhibition, exercises in flexion might be more effective than exercises in extension for regaining muscle strength but this would need to be confirmed (vide infra. 'Further Studies'). This suggestion has not been tested in patients with quadriceps weakness but Lindh (1979) has studied the effect of knee angle on increases in strength after isometric strength training in normal females. She found that exercises performed at 15^{o} and 60^{o} of knee flexion , with the subject seated, produced similar increases in isometric strength which were specific to the angle of the training exercise. Dynamic force was also increased by isometric exercise but only at a low speed ($30^{o}/s$). Dynamic exercise has been shown to increase isokinetic strength (Sherman et al., 1982) and isometric strength (Young et al., 1983). On the basis of Lindh's results, early post-meniscectomy rehabilitation should include isometric exercises at different knee joint angles, and later rehabilitation should include isokinetic exercises at high speeds. However, as the relationship between joint angle and quadriceps MVA differed between normal limbs and post-meniscectomy limbs in the present study, it is necessary to study the response of the inhibited quadriceps to strength training before adopting any specific strength-training programmes. Perhaps inhibition might also alter the muscles' response to training. If this were the case, a possible explanation might lie in the fact that between the relatively brief periods of exercise in flexion, the limb rests in extension. It is already known that voluntary activation is reduced in extension after meniscectomy, so perhaps reflex (involuntary) activation is also

reduced (vide infra. 'Further Studies'). This would imply that the progress made by exercising in flexion is lost by the absence of muscle activity while the limb is rested in extension and it might therefore be beneficial to immobilize the knee in flexion.

Possible Mechanisms of the Flexion Phenomenon: Preliminary Tests in the Present Investigations

The two present pilot studies were carried out to test the following hypotheses: 1) that the knee flexion phenomenon observed after meniscectomy, occurs due to the changes in muscle length associated with changes in knee joint angle, and 2) that the knee flexion phenomenon is due to reciprocal inhibition. If the mechanism of post-meniscectomy quadriceps inhibition was by reciprocal inhibition, it would be expected that hamstring activation would be greatest in extension and reduced in flexion.

Pilot Study I. The fact that only 2 of the 3 subjects in whom the effect of hip angle on the knee flexion phenomenon was tested demonstrated increased activation in flexion does not allow any conclusion to be drawn from the data obtained. Despite the fact that hip angle did not alter any of the values for MVA with the different knee angles, more subjects would need to be studied.

Some authors have attributed the changes in quadriceps activation and /or strength associated with changes in joint angle, to changes in muscle length (Haffajee et al.,1972; Currier & Kumar,1982). Currier & Kumar (1982) studied the effect of hip angle on quadriceps strength and activation (using needle EMG) in normal subjects. They tested hip angles of 90°, 80° and 60° with the subject seated and the knee kept at a constant angle of 60° .The maximum force and EMG activity was produced at 60° hip flexion and they attributed their findings to the increase in length of RF associated with the change from 90°to 60° of hip flexion. It seems likely that, at 60° flexion, the mechanical advantage is greater due to a shift in the centre of gravity of the trunk (i.e. the subject is

leaning back rather than upright). Perhaps the position of the present meniscectomy patients (i.e. supine) might account for the lack of changes in activation (in the unoperated limb) because the centre of gravity remained constant while only muscle length was altered. The results of the 2 studies can therefore not be directly compared. The effect of hip angle on the relationship between quadriceps activation and knee angle has not previously been studied in the presence of inhibition.

Studies in the decerebrate cat, (Baxendale & Ferrell, 1981) showed that changes in the excitability of lower limb reflex pathways caused by movement of the knee joint, were unrelated the muscle stretch. They tenotomized the knee flexor and extensor muscles, and holding them at a constant length, demonstrated that changes in reflex excitability during changes in joint angle persisted. By showing that the phenomenon was abolished by intra-articular anaesthesia, they demonstrated that knee joint afferent discharge was responsible for modulating the reflexes. If these studies were repeated in the presence of inhibition, the findings would indicate whether joint afferent activity or muscle length were important factors in the knee flexion phenomenon.

Pilot Study II. Apparent reciprocal activation recorded in the normal limb could have been due to 'pick up' across the skin surface from contracting agonists, which can occur with the use of surface electrodes (Chapter 2). Reduction of apparent antagonist activity after meniscectomy might have been due to the inhibition of the contracting quadriceps which would have reduced 'pick up' over the antagonists. In order to clarify the present findings , they should be checked using needle electrodes (vide infra, 'Further Studies').

Other Mechanisms to Consider

Intra-articular pressure. It is known that intra-articular pressure varies according to joint angle in normal and effused knees (Eyring & Murray,1964) but Levick (1979) showed that in the presence of effusion, much higher pressures were reached. Highest pressures occur at the extremes of flexion and extension, with the lowest pressure occurring at about 30^{o}. Stratford (1981) demonstrated that the quadriceps was inhibited less at 30^{o} flexion than in extension in patients with knee joint effusions. They attributed this relationship between joint angle and quadriceps activation to changes in intra-articular pressure associated with changes in joint angle. If this were true, high degrees of knee flexion would be expected to produce less relief of inhibition than that seen at 30^{o} of flexion. This did not appear to occur in the present series of meniscectomy patients or in a patient with periarticular pathology (see Appendix). Stratford et al. (1981) did not measure intra-articular pressure and neither did they look at the effects at other (high pressure) angles.

Capsular stretch. Pinching the anterior aspect of the knee joint capsule in decerebrated, and decerebrated and spinalized cats caused inhibtion of the monosynaptic reflex from quadriceps (Ekholm et al.,1960). Perhaps capsular tension caused by suturing the incision produces inhibition. This suggestion is supported by some preliminary tests on some patients after arthroscopic knee surgery (Chapter 12).

When the knee is extended, the posterior aspect of the joint capsule is stretched. Perhaps posterior receptor activation is subthreshold in the normal knee, and is above threshold when combined with some other afferent activity from within or around the knee e.g. in the presence of effusion or from an incision. This hypothesis would explain why inhibition is reduced with the knee in flexion because the stretch on the posterior capsule would be

relieved and the joint afferent activity would then only involve impulses from the area of 'injury' and would become subthreshold.

Krebs et al.(1983), in their explanation of the knee flexion phenomenon, suggested that the normal facilitatory effect of anterior joint receptors is removed by surgery, and that posterior stimuli predominate, causing quadriceps inhibition. They have misquoted Ekholm et al.(1960) and stated that "stimulation of the anterior joint capsule receptors facilitates the quadriceps".

Baxendale and Ferrell (1981) demonstrated in the normal knees of decerebrate cats, that extension of the knee induced excitation of knee flexors and inhibition of quadriceps. If this were also true in man, perhaps it might explain why quadriceps inhibition was greater in extension than in flexion in the present subjects.

FURTHER STUDIES

Quadriceps Strengthening Exercises Performed in Flexion

Although it is theoretically attractive to suggest that isometric quadriceps exercises might be more beneficial if they were performed in flexion, a clinical trial to examine the effects of exercising the inhibited quadriceps at different knee angles is essential.

Muscle Length

The effects of joint afferent activity in relation to muscle length cannot be studied in man in the way in which they have been studied in the cat (Baxendal & Ferrell,1981), but if these experiments were repeated in the presence of inhibition they might help to explain the knee flexion phenomenon in man.

Reciprocal Activation

Due to the possible inaccuracy of recordings made with surface electrodes, quadriceps and hamstring activation were recorded with needle electrodes in a later study to check the occurrance of reciprocal activation (Chapter 8).

Intra-articular Pressure

Quadriceps activation needs to be studied in the presence of reflex inhibition at angles of high and low pressure to determine the role of pressure in the knee flexion phenomenon. This was attempted in the present studies but due to practical problems the experiment was only performed in 1 subject (Chapter 8).

CONCLUSIONS

1. Post-meniscectomy quadriceps inhibition is less when isometric quadriceps contractions are made with the knee in about 40^{o} of flexion.

2. The influence of hip angle on the relationship between quadriceps inhibition and knee angle requires further study, as only 2 subjects in Pilot Study I demonstrated the knee flexion phenomenon.

3. The apparent reciprocal inhibition of quadriceps observed in Pilot Study II must be confirmed with needle electromyography (see Chapter 8).

CHAPTER 6

EFFECT OF TRANSCUTANEOUS NERVE STIMULATION ON POST-MENISCECTOMY PAIN AND REFLEX INHIBITION OF QUADRICEPS

EFFECT OF TRANSCUTANEOUS NERVE STIMULATION ON POST-MENISCECTOMY PAIN AND REFLEX INHIBITION OF QUADRICEPS

INTRODUCTION

Transcutaneous nerve stimulation (TNS) dates back to the early Greeks and Romans who used live torpedo fish and organs of electric fish for afferent stimulation to control pain (Kane & Taub,1975). In the late 1700's and early 1800's, man-made devices replaced these natural sources but they were not accepted by the medical professions (Lampe,1978). In 1965 the mechanism by which TNS is thought to relieve pain was explained by Melzack & Wall's pain-gate theory (Melzack & Wall,1965; Wolf,1978). The theory states that a predominance of activity in the large diameter afferent fibres blocks activity in the small (pain and touch) fibres at the first synapse in the spinal cord. The pain-gate theory provided an acceptable scientific basis for using well designed electrical stimulators. As TNS devices also provided adequate pain relief they soon became the preferred method of therapy.

It was necessary in the present studies to find a way of reducing inhibition which was longer lasting than the effect of bupivacaine (Chapter 3) and more continuous than performing contractions with the knee flexed (Chapter 5). Smith et al. (1983), in the belief that "pain is a significant inhibitor of muscle function", used TNS to relieve pain after knee surgery. They studied 100 patients (50 post-meniscectomy and 50 post-total knee replacement) and 25 in each surgical group received TNS and the other half did not receive TNS. They found that the TNS groups showed faster recovery of function and had less pain. Jensen et al. (1984) studied the effect of TNS on pain after arthroscopic knee surgery and found a reduction in pain and faster recovery of muscle function and range of movement in those patients receiving active TNS, than in those receiving placebo TNS or no TNS.

Perhaps the faster recovery of function observed in these 2 studies was due to reduction of inhibition by TNS. The present study was therefore undertaken to see whether TNS, in addition to blocking pain, could also prevent the inhibition caused by other signals from an injured joint.

This study was presented by the author at the Society for Research in Rehabilitation.

PATIENTS

Ten men (aged 25-41 years) undergoing medial arthrotomy and meniscectomy were studied (Table 6.1.).

METHODS

Surgical Details

Periods of tourniquet ischaemia ranged from 2Ø - 75 minutes (median = 3Ø mins) and are shown for the 2 groups separately in Table 6.1. The type of meniscectomy (i.e. partial or total) performed on the patients in each group is also shown in Table 6.1. After suturing, the wound was covered with opsite spray dressing (acrylic copolymer, Smith & Nephew) to enable the TNS electrodes to be placed near the wound without risk of infection.

TNS Details

The TNS trial was performed on the second post-operative day for 2 reasons 1) because the period of severe pain was over and so voluntary inhibition was less likely to influence the results 2) the patients were still on bed rest and performing quadriceps exercises so the presence of a TNS unit did not interfere with their routine physiotherapy programme.

A Rawlings TNS unit was modified (Fig 6.1.) so that a red light could be switched on either with or without producing a stimulating current. The unit ran on rechargable batteries and the ranges of the

	Age (yrs)		Medial Meniscectomy (n)		Tourniquet Times (mins)	
	Median	Range	Partial	Total	Median	Range
CONTROL GROUP	33	25–41	2	3	45	20–75
TREATMENT GROUP	34	27–41	1	4	27.5	20–71

Table 6.1. Details of the meniscectomy patients studied using transcutaneous nerve stimulation (TNS).

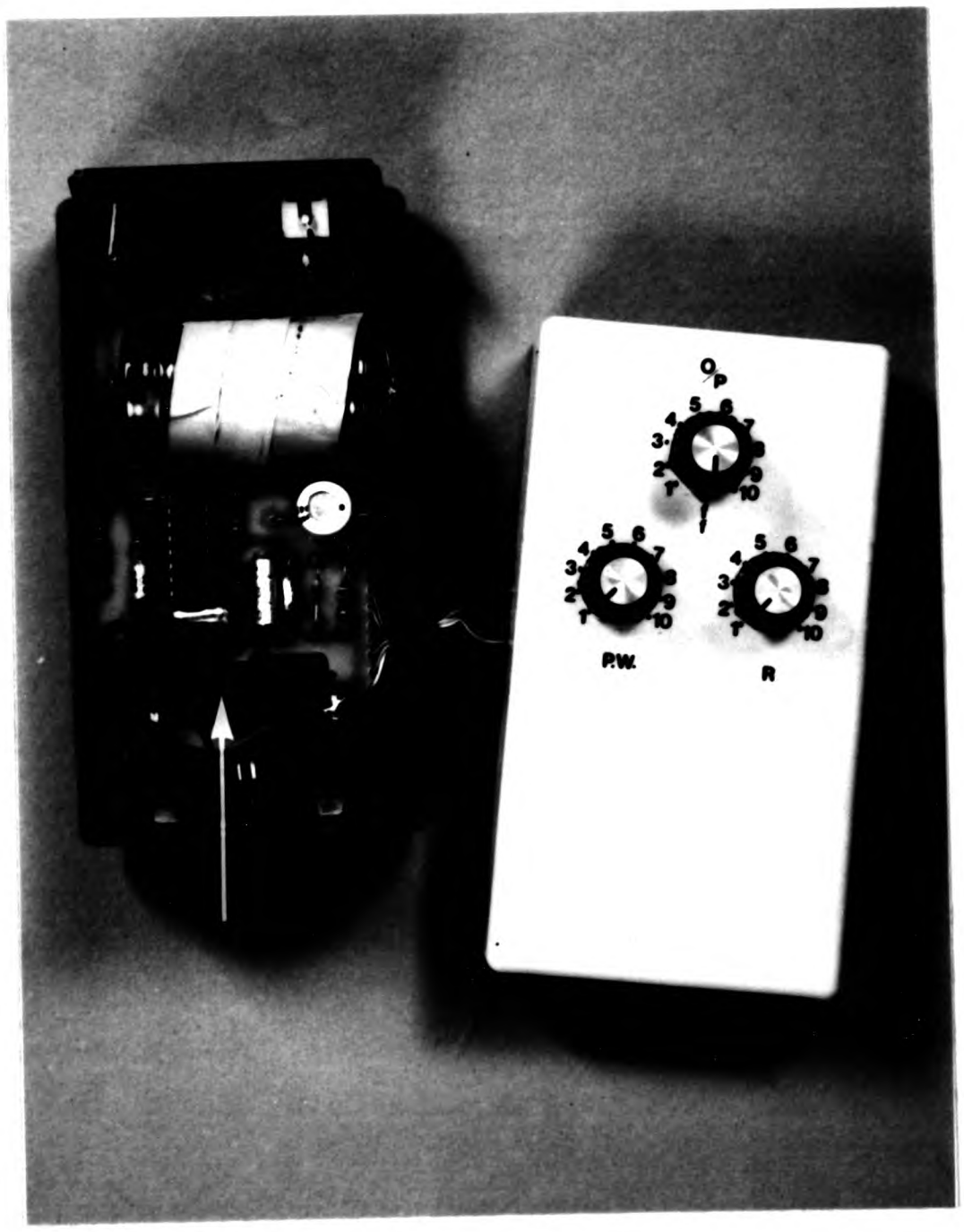

Figure 6.1. A Rawlings TNS machine showing the connection installed to allow placebo TNS to be given. When disconnected, a red light came on without producing a current when the machine was switched on.

Figure 6.1. A Rawlings TNS machine showing the connection installed to allow placebo TNS to be given. When disconnected, a red light came on without producing a current when the machine was switched on.

stimulation parameters were: frequency 12.5 - 125 Hz, pulse width 70 - 200 μs, maximum output 80V (comfortable voltage 5-10V) and the waveform was almost square (Fig 6.2.). The TNS stimulator was calibrated before and after the period of the trial and all parameters were unchanged.

Electrode placement. The knee was unbandaged , rubber pad electrodes (4.5cm * 4.5cm) were placed on either side of the wound about 10cm apart and the knee was bandaged up again.

Control group. All 3 dials on the machine were set in mid-position and a red light was visible. Patients were asked not to touch the machine at all.

Treatment group. The pulse width and rate dials were set in mid-position (200 μs and 17Hz respectively). Patients were instructed to turn up the output until a strong, but comfortable, tingling sensation was felt. If the sensation became weak during treatment, they could adjust the output accordingly.

Protocol

Quadriceps MVA and pain during contraction were recorded (as described in Chapter 2) within 5 days pre-operatively. On the second post-operative day, patients were randomly allocated to either a control group (n = 5) or a treatment group (n = 5). Quadriceps MVA and pain were re-measured before and immediately after the TNS units had been applied for 6 hours in both groups. It was thought that 6 hours should be long enough to produce an effect without disrupting the patients' physiotherapy routine. The person making the measurements was blind as to which group the patient belonged and the patient was asked not to discuss the TNS machine with that person.

Quadriceps exercises were performed hourly exactly as described in Chapter 2 but patients were asked not to exercise within in the hour preceeding the post-TNS tests.

STIMULATION PARAMETERS

Square wave-form

Output	0-80 V	(comfortable at 5-10 V)
Pulse width	200 µs	
Frequency	17 Hz	

Figure 6.2. **Stimulation parameters and waveform of the current for 'treatment' TNS.**

RESULTS

Effects of Transcutaneous Nerve Stimulation on Inhibition and Pain

Pain was reduced by TNS but this effect was similar in both the treatment and the control groups (Fig 6.3.; Table 6.2.). Considering both groups together, this reduction in pain was statistically significant (P=0.05, 2-tailed, paired Wilcoxon signed ranks test).

No consistent change in inhibition was observed in the control group. All 5 patients in the treatment group showed a reduction in inhibition (Fig 6.3.) and although this was statistically significant (P<0.05, 1-tailed Wilcoxon signed ranks test), the changes seem too small to be of clinical significance.

Analgesic Requirement

Analgesic requirement was similar in both groups during the day of the TNS trial and the subsequent 2 - 3 days while still in hospital.

DISCUSSION

Effect of Transcutaneous Nerve Stimulation on Reflex Inhibition

The values for quadriceps inhibition before TNS were similar to those observed in the patients described in Chapter 3 (approximately 40-90%). The present study failed to show a reduction in inhibition in the group receiving placebo TNS. The effect of active TNS on inhibition was only trivial and the relief was probably not enough to be of clinical importance i.e. to allow greater activation during quadriceps contractions. As the present study did not involve measurement of functional recovery, and neither Smith et al. (1983) nor Jensen et al.(1984) measured activation, the results of the previous 2 studies cannot be compared with those of the present study.

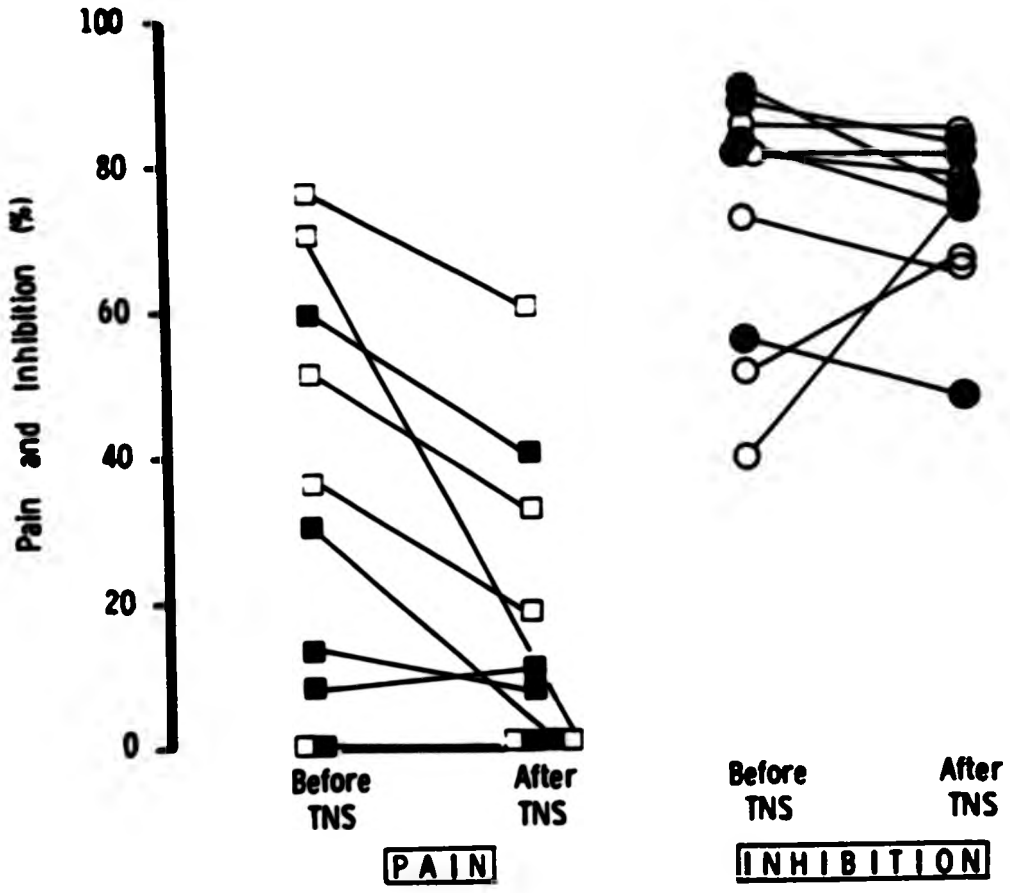

Figure 6.3. Effect of 6 hours TNS on post-meniscectomy quadriceps inhibition and pain during contractions made 2 days after surgery.

■ ● treatment group □ ○ control group

	PAIN (%)		INHIBITION (%)	
	Pre-TNS	Post-TNS	Pre-TNS	Post TNS
CONTROL GROUP				
Median	50	16	72	74
Range	0 - 76	0 - 60	39 - 95	65 - 94
TREATMENT GROUP				
Median	13	7	82	76*
Range	0 - 59	0 - 40	55 - 90	47 - 82

* P<0.05 (Wilcoxon signed ranks test, 1-tailed)

Table 6.2. Results of the severity of reflex inhibition and pain during contractions made after 6 hours of TNS.

The small effect of TNS on inhibition in the present study may have been due to the stimulation parameters used. Optimal electrode placement, stimulation parameters and duration of treatment have not been established but some have been suggested (e.g. Mannheimer,1978; Gersh et al.,1980). Perhaps experimentation with these might produce more relief.

Effect of Transcutaneous Nerve Stimulation on Pain

The similarity of the effect on pain of both active TNS and placebo TNS in the present study demonstrates the importance of a control group. Four out of 5 in the control group had substantial relief of pain and the other patient had no pain to start with. Smith et al.(1983) did not have placebo groups as their non-TNS patients did not receive any form of control treatment or even control attention. It is therefore not possible to calculate how much of the improvement in their treatment group may have been due to a placebo effect.

Dissociation of Quadriceps Inhibition and Pain

Some patients from both groups of the present study only had little or no pain despite severe inhibition, both before and after the TNS trial. These patients provide further examples of the separation between inhibition and pain which was previously observed in the patients described in Chapter 3.

Post-operative Analgesia

Analgesic requirement in both Smith's and Jensen's studies was lower in the treatment groups. In the present study, however, no such effect on analgesic requirement was seen. Pain scores in the present study were for pain experienced during contraction and those of the previous studies were recorded at rest (Smith et al.,1983; Jensen et al.,1984). In Jensen's study the placebo (TNS) group had less pain, and required less analgesia than the control group (i.e. no TNS).

FURTHER STUDIES

Perhaps a future trial, performed when optimal stimulation parameters are known, might produce a clinically useful reduction in inihibition with active TNS. It was not considered to be within the scope of the present investigations to experiment with the stimulation parameters for TNS, and therefore the present pilot study was not extended to a large treatment trial.

CONCLUSIONS

1. There was a statistically significant reduction in post-meniscectomy quadriceps inhibition after 6 hours of TNS.

2. The reduction in inhibition was only small and probably not of clinical significance.

3. Pain was reduced with both active and placebo TNS.

4. Inhibition and pain were dissociated both before and after the TNS trial.

CHAPTER 7

EFFECT OF KNEE JOINT EFFUSION ON POST-MENISCECTOMY REFLEX INHIBITION OF VOLUNTARY QUADRICEPS ACTIVATION

EFFECT OF KNEE JOINT EFFUSION ON POST-MENISCECTOMY REFLEX INHIBITION OF VOLUNTARY QUADRICEPS ACTIVATION

INTRODUCTION

The clinical observation that a knee joint effusion was often associated with quadriceps weakness and atrophy, prompted deAndrade *et al.*(1965) to study quadriceps inhibition under controlled conditions in man. They demonstrated, both in patients with joint conditions and in normal subjects, that distension of the knee joint (with human plasma) caused quadriceps weakness (assessed by lifting the heel) and that pain was not always present.

Jayson & Dixon (1970a) studied the pressure / volume relationship in the infused knees of patients with rheumatiod arthritis and normal subjects. They found that infusion with fluid (dextrose and sodium chloride) produced quadriceps weakness which was again assessed by heel lifts and so weakness was not actually quantified. Stratford (1981) used EMG to demonstrate that quadriceps activation during isometric quadriceps contractions was less in the presence of a knee effusion than without an effusion.

Large effusions do not frequently occur after meniscectomy but in patients who did develop effusions, the effect of removing the effusion was examined to determine the contribution of the effusion to post-meniscectomy reflex inhibition of quadriceps.

PATIENTS

Twelve men (aged 29-42 years) were studied before and 2-8 days after arthrotomy and meniscectomy (11 medial, 1 lateral). These included the one patient from the first study in the present series (Chapter 3) who develolped an effusion. The results for this patient reported in Chapter 3, were recorded after the effusion had been aspirated. The types of operation performed on each patient are shown in Table 7.1.

Fluid Aspirated (mls)	Type of Meniscectomy	Days Post-op	Change from Pre-asp Level Inhibition	Pain	Appearance of Aspirate
15	T. Lat.	4	- 2	-	H
30*	T. Med.	8	-27	Ø	H
40	T. Med.	2	- 2	-25	H
40*	P. Med.	2	-24	Ø	H
45	T. Med.	5	-14	4	H
45*	T. Med.	2	-32	Ø	H
45*	T. Med.	2	-27	-18	H
53*	P. Med.	3	-26	Ø	L
55	T. Med.	4	-48	-	L
65	P. Med.	4	-28	-	-
70	T. Med.	3	-66	64	H
85	T. Med.	3	-16	Ø	L

T = Total P = Partial Med = Medial Lat = Lateral
L = Lightly blood-stained H = Heavily blood-stained
* = received subcutaneous lignocaine

Table 7.1. Details of meniscectomy patients who underwent aspiration of a knee joint effusion.

METHODS

Measurements

Quadriceps MVA and pain. Recordings were made on both legs pre-operatively (within 5 days), and before and immediately after aspiration of the effusion. Aspiration was performed 2-8 days after surgery. One patient was not studied pre-operatively so quadriceps inhibition in his operated leg was expressed as the percentage difference from the value obtained from his unoperated leg.

Aspiration of the Effusion

Five patients received 0.5-1 ml of 2% lignocaine subcutaneously (Table 7.1.) prior to insertion of a 16 gauge cannula into the lateral aspect of the knee under sterile conditions. Whether lignocaine was used depended on the doctor, who performed the aspiration as he would normally have done. A 60ml syringe was used to draw off the fluid which was measured in ml and its appearance noted (e.g. viscous, clear, blood-stained).

RESULTS

The volumes of fluid aspirated ranged from 15-85 ml (median = 45 ml).

All patients showed some relief of inhibition ($P<0.01$, Wilcoxon paired signed ranks test, 2-tailed). In only 1 patient, however, was inhibition almost fully relieved and in only 3 others was it reduced to <25% (Fig 7.1.).

The degree of inhibition before aspiration was not correlated with the volume of fluid removed ($r_s=0.3$, Spearman's rank correlation, $P > 0.05$), nor was the degree of relief of inhibition related to the volume aspirated ($r_s=0.5$, Spearman's rank correlation, $P > 0.05$).

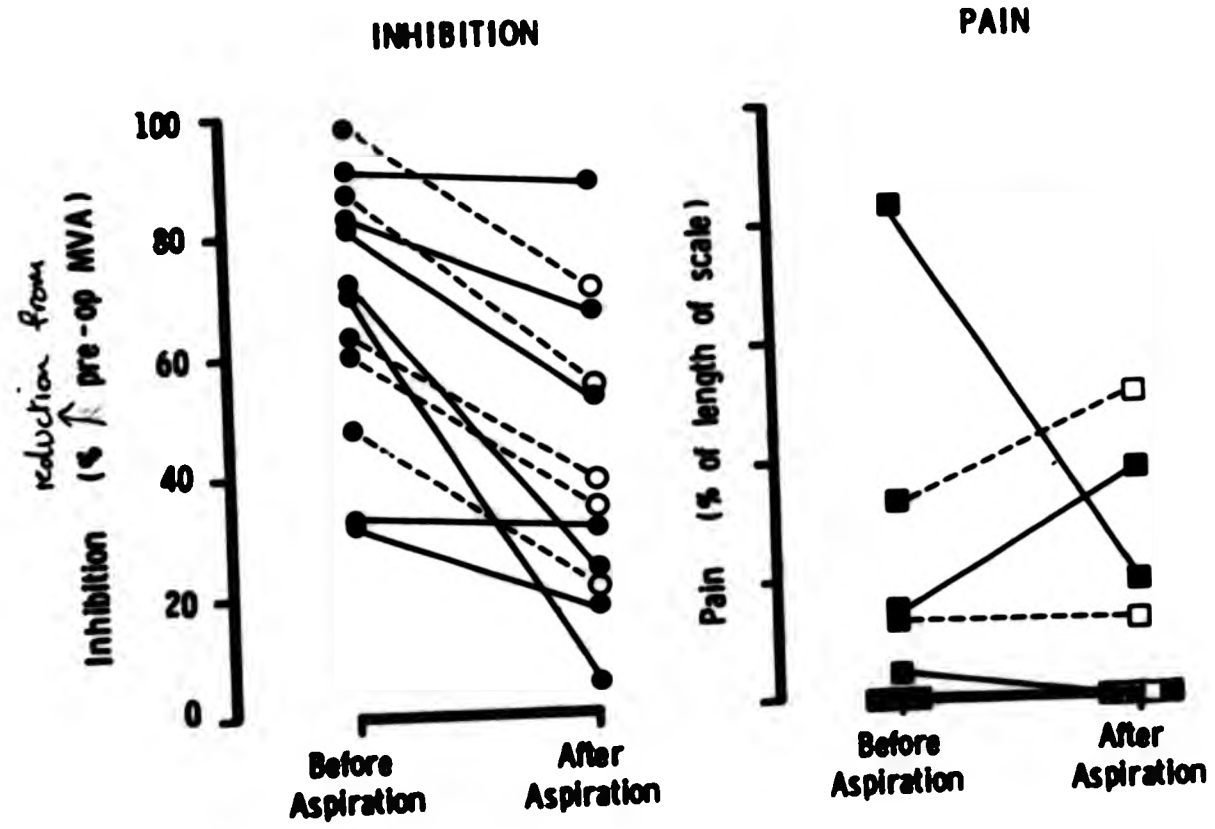

Figure 7.1. Effect of aspirating a knee joint effusion on quadriceps inhibition and pain during contraction.

● ■ Without subcutaneous lignocaine

○ □ After subcutaneous lignocaine

Inhibition was not always associated with pain as some patients with severe inhibition had little or no pain. Pain scores were not available for 3 patients.

The relief of pain was not related to the amount of fluid removed and patients who received subcutaneous lignocaine did not show more relief of inhibition or pain than those who did not receive an injection.

DISCUSSION

deAndrade et al. (1965), in their studies of patients and normal subjects who received knee joint infusions, assessed quadriceps weakness by the ability to raise the heel from the bed. In 2 of their subjects, surface EMG showed a reduction in the number and amplitude of action potentials during attempted heel lifts. They also demonstrated that the effect of joint infusion is influenced by interruption of the afferent pathway in two ways 1) by the intra-articular injection of local anaesthetic prior to joint distension, which delayed the appearance of weakness until high intra-articular pressures were reached and 2) in a patient with Charcot's arthropathy of the knee (i.e. loss of afferent activity from the joint), quadriceps weakness did not occur at all when the joint was distended at high pressure.

It was concluded from Jayson & Dixon's (1970a) studies of joint pressure / volume relationships that the quadriceps was being inhibited due to stimulation of the joint capsular stretch receptors, rather than the joint pressure receptors, and that this occurred in the absence of pain.

Increased intra-articular pressure caused inhibition of the monosynaptic reflex from quadriceps in both decerebrated and decerebrated and spinalized cats (Ekholm et al., 1960). Inhibition of reflex activation of quadriceps has been demonstrated in man by Spencer et al. (1984) in a study which was designed to refine the

earlier experiments (deAndrade et al.,1965; Jayson & Dixon,1970a)by using quadriceps H-reflex. The H or Hoffmann reflex, which is elicited by stimulation of la spindle afferents (from muscle spindle receptors) of the femoral nerve and results in a small quadriceps contraction, can be influenced by sensory stimuli in the spinal cord (Chapter 9).

Contribution of Effusion to Reflex Inhibition of Quadriceps

Although knee joint effusion clearly contributed to the quadriceps inhibition observed in the meniscectomy patients in this study, it was not its only cause because inhibition was not abolished by aspiration of the effusion.

Relief of Inhibition in Relation to Volume and Appearance of the Aspirate

The fact that inhibition was not abolished by aspiration (except in 1 patient) indicates that some other stimulus must have been causing the inhibition. It could be argued that perhaps not all of the fluid was removed, which might account for the remaining inhibition. It was assumed at the time of aspiration that all the fluid had been removed because of the appearance of the joint and the fact that no more fluid could be extracted. However, later experiments on normal subjects with knee joint infusions (see Chapters 8 & 11) revealed that the cannula could become blocked and therefore prevent all the fluid from being removed. These studies on normal subjects also demonstrated that quadriceps inhibition can occur with small amount of fluid in the joint (20-30ml) which does not produce obvious joint swelling. The degree of relief of inhibition was not related to the volume aspirated e.g. removal of a large amount of fluid did not always produce a large amount of relief (Fig 7.1.). Severity of inhibition was also not related to whether or not the fluid appeared blood-stained (see Table 7.1.).

Relief of Inhibition in Relation to Pain

It has already been established in the present series of investigations that post-meniscectomy quadriceps inhibition is not related to pain (Chapters 3 & 6). In the present study inhibition was not always associated with pain either before or after aspiration. The degree of relief of inhibition did not appear to be related to the degree of relief of pain.

Subcutaneous Lignocaine Prior to Aspiration

deAndrade et al. (1965) and Jayson & Dixon (1970a+b) used subcutaneous local anaesthesia in their subjects prior to infusing the knee with saline. Spencer et al. did not use subcutaneous local anaesthesia in their experiments which examined quadriceps H-reflex inhibition in the presence of infusion. It is not possible to determine whether inhibition, in the subjects who received subcutaneous local anaesthesia, reached levels as high as it would have done without anaesthesia because their measurements of inhibition were too crude (i.e. the ability to achieve heel lifts). Inhibition was only clearly prevented when the local anaesthetic was infiltrated into the joint (deAndrade et al.,1965; Spencer et al.,1984). In the present study, relief of inhibition was not related to the use of subcutaneous lignocaine and neither did the injection influence pain experienced during contraction. (NB The pre-aspiration pain scores reported in Fig 7.1. were recorded prior to injection of lignocaine.)

FURTHER STUDIES

Clinically apparent effusions do not often occur after meniscectomy but it is not known how small an amount of fluid is needed before inhibition occurs. Comparing the present patients' data with those from the first study in this series (Chapter 3), pre-aspiration inhibition is similar to that seen up to 3 days post-

operatively (typically 70-80%). Post-aspiration inhibition is similar to inhibition seen at 10-15 days (typically 30-40%). Perhaps effusions which are not clinically apparent contribute to the early inhibition. It is therefore important that the effect of small volumes of effusion should be investigated and this was done in some later studies in this series (Chapters 8 & 11).

CONCLUSION

Following arthrotomy and meniscectomy, a knee joint effusion increases the severity of reflex inhibition of voluntary quadriceps activation but is not its sole cause.

CHAPTER 8

REFLEX INHIBITION OF VOLUNTARY ACTIVATION OF QUADRICEPS AND INTRA-ARTICULAR PRESSURE/VOLUME RELATIONSHIPS IN NORMAL SUBJECTS WITH KNEE JOINT INFUSIONS

INTRODUCTION

SUBJECTS

EXPERIMENT I: DOSE EFFECT OF KNEE JOINT INFUSION ON VOLUNTARY ACTIVATION OF QUADRICEPS

EXPERIMENT II: INTRA-ARTICULAR PRESSURE/VOLUME RELATIONSHIPS

THE USE OF THE 'MODEL' OF NORMAL SUBJECTS WITH KNEE JOINT INFUSIONS

Tolerance of the procedure

Technical problems with the knee infusion model

FURTHER STUDIES

REFLEX INHIBITION OF VOLUNTARY ACTIVATION OF QUADRICEPS AND INTRA-ARTICULAR PRESSURE/VOLUME RELATIONSHIPS IN NORMAL SUBJECTS WITH KNEE JOINT INFUSIONS

INTRODUCTION

It has been demonstrated in the cat that a knee joint infusion increases activity in the joint nerves (Andrew & Dodt,1953). In decerebrate, and particularly spinal preparations, this results in reflex inhibition of quadriceps (Ekholm et al., 1960). Studies in man have demonstrated inhibition of voluntary quadriceps activation in patients with knee effusions (e.g. Jayson & Dixon,1970a; Stratford,1981) and in normal subjects with artificially induced infusions (deAndrade et al.,1965; Jayson & Dixon,1970a). Knee joint effusion occasionally occurs after meniscectomy and aspiration of the effusion reduces the severity of quadriceps inhibition but does not abolish it (Chapter 7).

In the present studies, two experiments were performed to attempt to find an explanation for the knee flexion phenomenon, but were abandoned due to technical problems (vide infra). The knee flexion phenomenon refers to the observation made in the menisectomy patients studied in Chapter 5, that inhibition of isometric quadriceps contractions is less with the knee in a flexed position than with it extended. The first experiment examined this phenomenon in relation to intra-articular pressure in normal subjects with knee infusions. It was thought that the reason inhibition was greatest in extension was because of high intra-articular pressure in this position, and that the lower pressures produced at 40° flexion (Ranke,1875; Eyring & Murray,1964; Jayson & Dixon, 1970b; Levick,1983) might be responsible for the reducedinhibition in flexion. This experiment involved performing maximal quadriceps contractions at 0°, 50° and 60° of knee flexion, while the maximal voluntary activation (MVA) of quadriceps the and intra-articular

pressure were recorded with and without infusion. Only 1 subject was studied (Subject 1a) as the 2 manoeuvres of changing knee angle and making maximal contractions caused the cannula to kink and become blocked, which prevented pressure from being recorded. Some results were obtained however, and they indicated that intra-articular pressure and inhibition were reduced during contractions made with the knee flexed. The other experiment examined the knee flexion phenomenon in relation to reciprocal inhibition. The apparent reciprocal inhibition in flexion and extension observed with the use of surface electrodes in subjects reported in Chapter 5, may have been due to 'pick up' from the contracting muscles. In this experiment, needle electrodes were used in Subject 1a to check the previous findings that hamstring activation, during maximal contractions of the inhibited quadriceps, appeared to be less in flexion than in extension. Hamstring activation was increased during quadriceps contractions with the knee flexed in this subject, which contrasts with the previous findings (Chapter 5). This suggests that the difference in findings was due to the type of electrodes used *viz.* the hamstring surface electrodes were reflecting quadriceps activity by 'pick up' rather than small changes in hamstring activity.

In the studies described below, the dose effect of joint infusion on quadriceps MVA was examined in Experiment I, and the pressure / volume relationships were examined in Experiment II. The aim of Experiment II was to refine earlier work (deAndrade *et al.*,1965; Jayson & Dixon,1970) by documenting the relationship at rest and comparing this to the relationship observed during some form of controlled functional activity.

SUBJECTS

Three normal males and 5 normal females, aged 27 - 40 were studied (2 of them twice) and their details are shown in Table 8.1.

EXPERIMENT I: DOSE EFFECT OF KNEE JOINT INFUSION ON VOLUNTARY ACTIVATION OF QUADRICEPS

Introduction

Although obvious effusions do not often occur after meniscectomy, the substantial reduction of early post-meniscectomy inhibition by aspiration of the effusion in the patients studied in Chapter 7, raises the question as to whether the patients studied in Chapter 3 might have had effusions which were too small to be clinically apparent. The use of a non-steroidal anti-inflammatory drug (NSAID) is said to speed recovery of function after meniscectomy (Muckle, 1984). Perhaps an NSAID reduces inhibition by preventing the development of small (i.e. not clinically apparent) effusions. It is therefore necessary to determine the minimal amount of fluid which will cause inhibition. The present study examined the dose response of quadriceps MVA to infusion of the knee joint with saline in 2 subjects (see Table 8.1.).

Methods

The dose effect of increasing volumes of infusion on quadriceps MVA were studied in Subjects 1a and 2a. Infusion of the knee with saline, recording of intra-articular pressure, and recording of quadriceps MVA using surface (Subject 2) and needle (Subject 1) electrodes were performed as described in Chapter 2. Quadriceps MVA and pressure were recorded before infusion and after eachincrement of infusion (every 10ml in Subject 2, and at 33ml and 55ml in Subject 1).

Results

Increasing volumes of infusion reduced quadriceps MVA in both the subjects studied (Fig 8.1.). In Subject 2, the cannula became disloged early on in the procedure and was replaced by a fresh cannula. Substantial inhibition (>25%) was first observed at 20 ml (Subject 2) and 30 ml (Subject 1). In Subject 2, activation returned

		EXPERIMENTS	
		I	II
Subject		Dose Effect on MVA	Pressure / Volume
1.	♀ 27 yrs		
	a	+	
	b		+
2.	♂ 36 yrs		
	a	+	
	b		+
3.	♂ 29 yrs		+
4.	♀ 36 yrs		+
5.	♀ 31 yrs		+
6.	♂ 37 yrs		+
7.	♀ 30 yrs		+
8.	♀ 40 yrs		+

Table 8.1. Normal subjects studied in experiments to test the effect of knee joint infusion on quadriceps activation and intra-articular pressure / volume relationships.

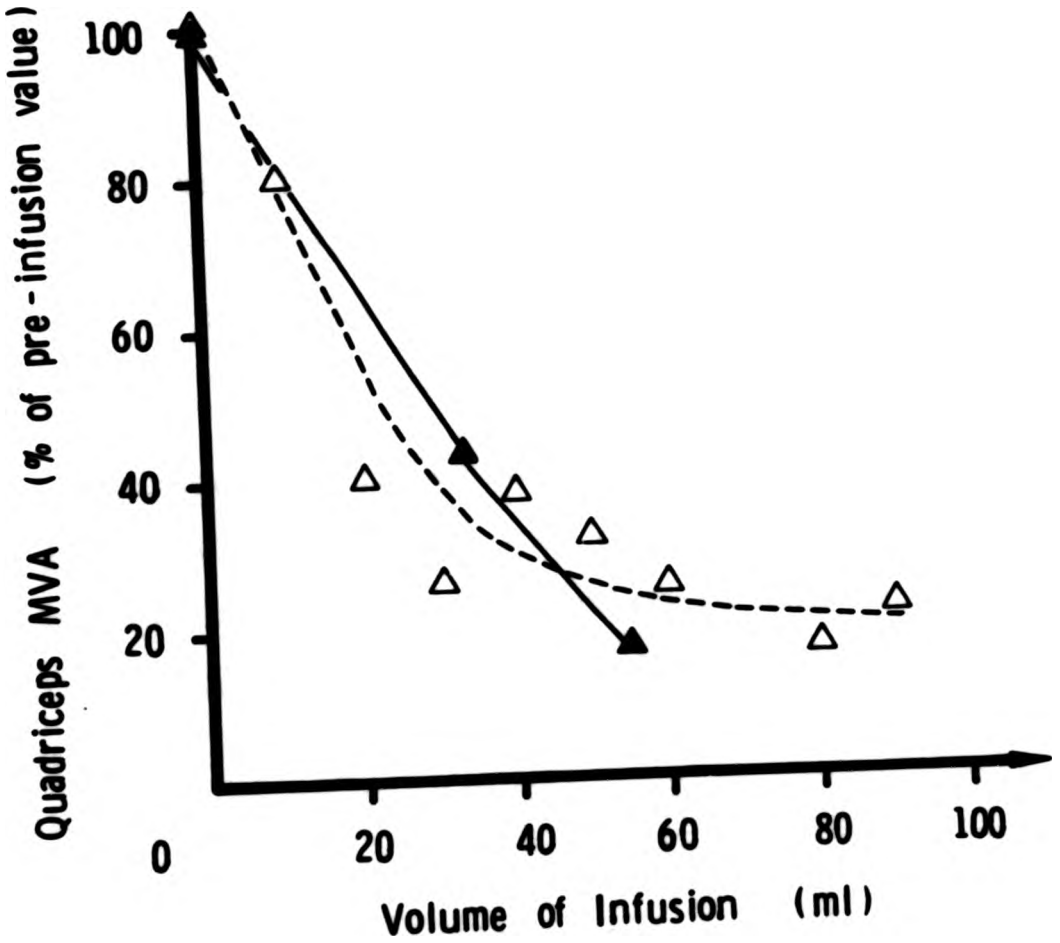

Figure 8.1. Effect of knee joint infusion on the maximal voluntary activation (MVA) of quadriceps in relation to volume.

▲ Subject 1a Δ Subject 2a

to normal (102%) after aspiration of as much fluid as possible, which was 53 ml, leaving a nominal 37 ml in the knee. In subject 2 the pressures were lower than would be expected for the volumes of infusion (probably due to leakage of fluid into the subcutaneous tissues at the site of the first cannula) and therefore the degrees of inhibition were probably also too small for each volume. Inhibition has therefore been plotted against intra-articular pressure (Fig 8.2.) and is probably a more reliable way of presenting the data as the true joint volumes are not known. Only 1 pressure reading was obtained for Subject 1a as the pressure at 55ml was higher than the top of the pressure scale used in that experiment.

Discussion

Volumes of fluid as small as 20 ml and 30 ml do not cause obvious joint swelling and yet they caused quadriceps inhibition in the 2 subjects studied. More subjects would need to be studied with smaller increments of infusion in order to establish the minimum amount of fluid necessary to cause reflex inhibition of voluntary activation. Aspiration of effusion in the early post-operative period (3-5 days) reduced inhibition to levels similar to those seen at 10 days in patients without apparent effusion (Chapter 7). Perhaps the meniscectomy patients studied in Chapter 3 may have had small effusions (i.e. not apparent) in the early post-operative period.

Inhibition increased as the volume of infusion, and hence, intra-articular pressure increased and this is in agreement with the findings of deAndrade *et al.* (1965) and Jayson & Dixon (1970a) which demonstrated increasing quadriceps weakness with infusion. Inhibition always occurred when the infusion caused apparent joint swelling and this usually occurred by 40ml in the present subjects. Apparent infusions and inhibition were not accompanied by a feeling of distension until large volumes were reached (approximately 50-60 ml). These observations made by subjective assessment of inhibition

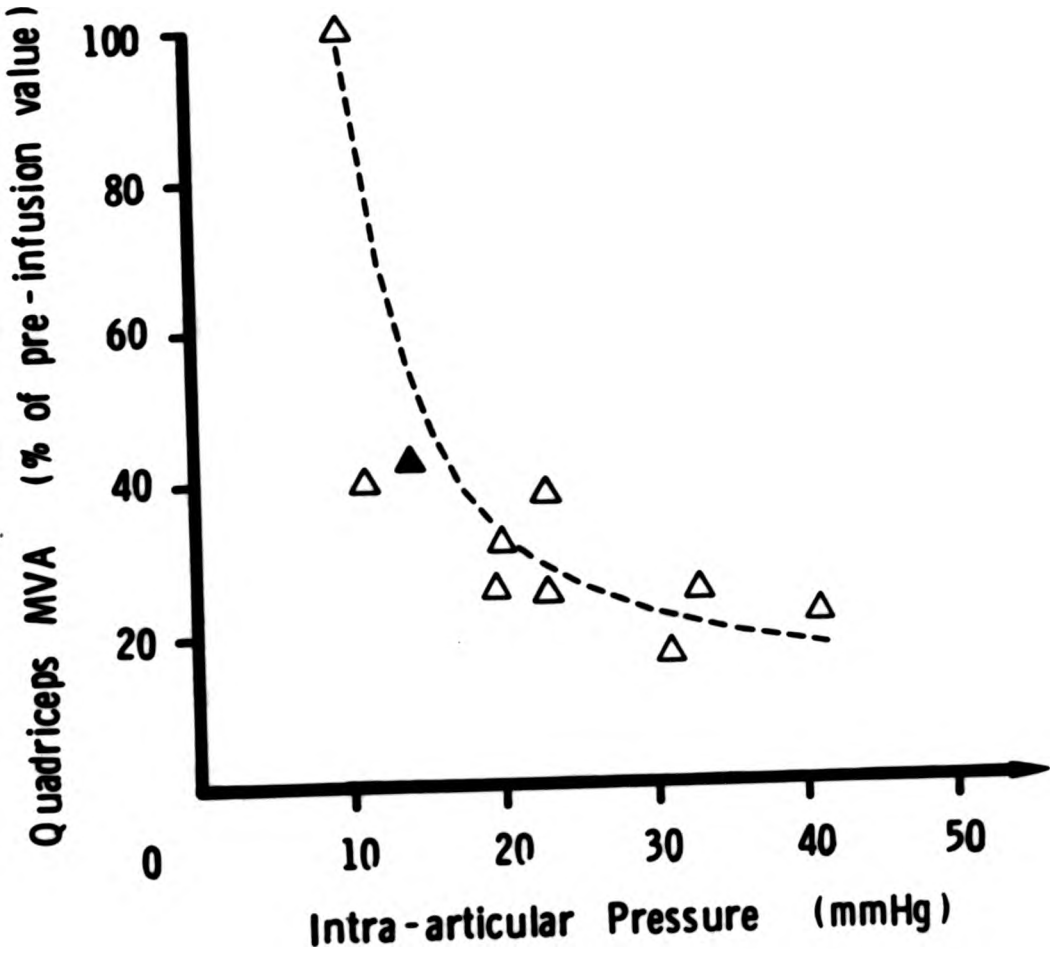

Figure 8.2. Effect of knee joint infusion on the maximal voluntary activation of quadriceps in relation to intra-articular pressure.

▲ Subject 1a (pressure transducer did not record at high pressures). △ Subject 2a

(i.e. when the subject became aware that he could no longer contract his quadriceps maximally), and of joint swelling, and were made in all the subjects included in Experiments I & II.

Conclusion

Small volumes of knee joint infusion, which are not clinically apparent, can cause inhibition of voluntary quadriceps activation. (cf. conclusions from H-reflex experiments in Chapter 11, where there is also no sign of a 'threshold'.)

EXPERIMENT II: INTRA-ARTICULAR PRESSURE / VOLUME RELATIONSHIPS

Introduction

Jayson & Dixon (1970b) stressed the inadequacy of studying the physiology of the joint at rest rather than during use, and found that pressures were much greater during use (e.g. isometric quadriceps contractions - 'quadriceps setting', walking) in the presence of effusion. deAndrade et al. (1965) demonstrated higher pressures during heel lifts than at rest, in the presence of an infusion. In the present study the pressure/volume relationship was examined at rest and during controlled contractions of quadriceps in an attempt to document the findings of the earlier studies more accurately (deAndrade et al.,1965; Jayson & Dixon,1970 a+b).

Methods

The relationship between intra-articular pressure and volume of infusion was studied in 7 subjects (Table 8.1.). Subjects lay supine with both knees extended. Tests were performed at rest and during small isometric quadriceps contractions (15-20 % maximum effort) in all subjects and during maximal contractions (i.e. Jayson & Dixon's 'quadriceps setting' in 2 subjects (Subjects 1 & 2). Infusion of the knee with saline, recording of intra-articular pressure, and control of quadriceps contraction using biofeedback

were performed as described in Chapter 2. One recording of intra-articular pressure was made at rest immediately after each infusion of saline (usually in 10ml increments), before any contractions were made. Pressure was then recorded during contraction at each volume and also after aspiration of fluid in some subjects.

Results

Resting intra-articular pressure was only slightly increased with infusion and the relationship was almost linear (Fig 8.3.). During a 15- 20% quadriceps contraction, the pressures were higher than those recorded at rest at each volume in all subjects except Subject 4 (Fig 8.4.).

Maximal quadriceps contractions were only tested in 2 subjects. In subject 1a, maximal contractions produced pressures of > 161mmHg (the top of the pressure scale during that experiment) with volumes over 55 ml. Pressure readings were not obtained for maximal contractions without infusion due to the air in the cannula. In subject 2a, the volumes of fluid infused after insertion of the second cannula did not reflect the volumes actually contained within the joint because fluid was pushed out into the extra-articular tissues during contraction. This fact was demonstrated by unexpectedly low pressures recorded at each volume during contractions. For this reason, subject 2a has been omitted from Fig 8.4. Aspiration of different volumes of fluid produced lower pressures than during infusion at the same nominal volumes.

Discussion

Resting intra-articular pressures recorded during joint infusion in the present subjects were similar to those reported previously (deAndrade et al.,1965; Jayson & Dixon, 1970a) which are illustrated in Fig 8.3. for comparison. The results for the pressure/volume relationships at rest in the present subjects are comparable with the mean values for results obtained in 12 subjects

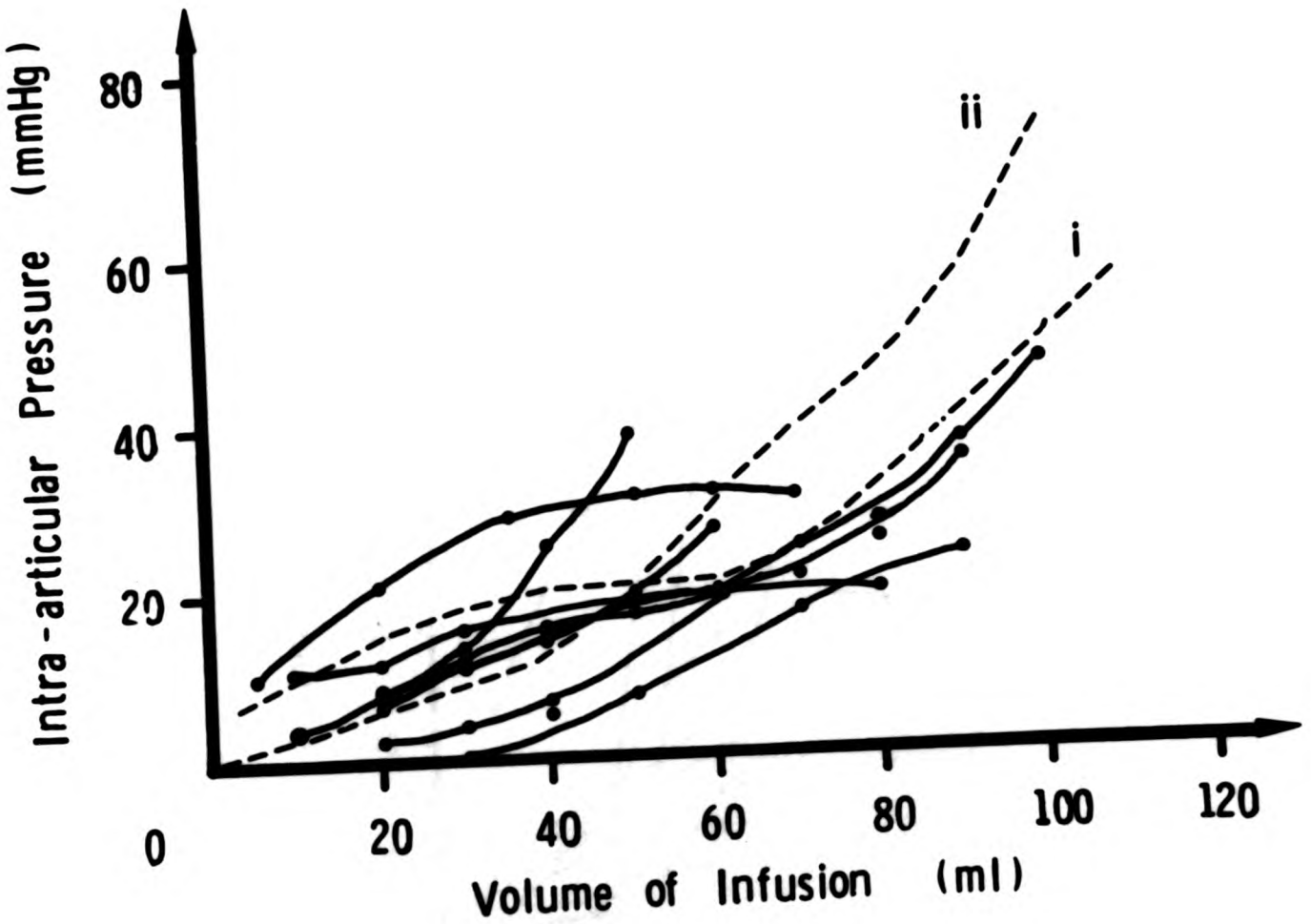

Figure 8.3. Intra-articular pressure/volume relationship at rest.
—●— present subjects ---- i deAndrade et al. (1965)
---- ii Jayson & Dixon (1970a)

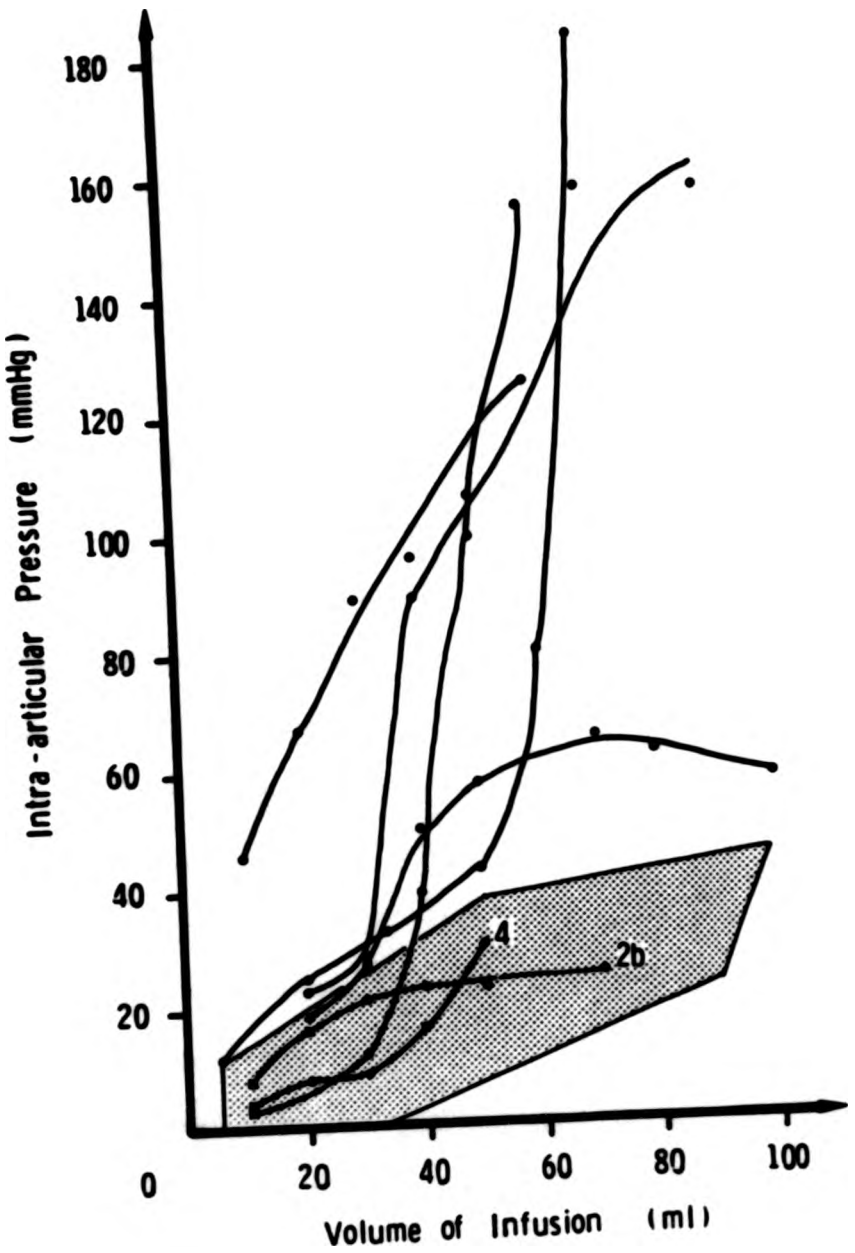

Figure 8.4. Intra-articular pressure/volume relationship during quadriceps contractions (15-20% max).
▒ range for pressure/volume relationship at rest in the same subjects. Subjects 2b and 4 are discussed in the text (see page 83).

by Jayson & Dixon (1970a). When the standard deviations (SD) is calculated from their standard errors at 100ml, the range over ± 2SD is from 0 mmHg to 160 mmHg. The SD is smaller at the lower volumes of infusion.

Intra-articular pressures were greater during voluntary contractions of quadriceps than at rest in the infused knee. deAndrade et al. (1965) demonstrated, in 3 normal subjects, that pressures were higher during heel lifts (a test which is difficult to standardise) than at rest. Jayson & Dixon (1970b) examined pressure/volume relationships during straight leg raising (SLR), resisted (SLR), and quadriceps setting (presumably attempted maximal contractions). Pressures were greater during all 3 activities than at rest and showed similar pressure/volume relationships to those observed in the present studies. The effect of contraction on intra-articular pressure in the absence of an infusion was not investigated due to the presence of air in the cannula, preventing pressure from being recorded. The pressure in healthy knee joints is normally about -5mmHg with the knee fully extended (Levick,1979).

In subject 2a, the stronger contractions produced lower intra-articular pressures than during the weaker contractions. This may have been due to more fluid being pushed out of the joint at the site of the first cannula during the stronger contraction. The experiment was repeated in Subject 2 (omitting maximal contractions) as the data from the first set of tests were unreliable. The lower pressures recorded after aspiration suggest that some fluid had either leaked out of the joint or had been absorbed.

Ferrell (1982) observed that at high volumes of infusion in the knee joint of the cat, activity in the posterior articular nerve (PAN) levelled out despite increasing intra-articular pressure. After infusion, both PAN activity and intra-articular pressure decreased. Ferrel's explanation of these findings is that there is a limit to capsular distension beyond which fluid is diverted into

other areas of the joint which are not innervated by the PAN. Knight & Levick (1982) demonstrated that, although the knee joint of the rabbit was anatomically continuous, the suprapatellar pouch and the postero-medial region of the joint acted as hydraulically separate compartments at low pressures. At high pressures, fluid passed from one area of the joint to the other *via* 2 channels.

As voluntary contractions of quadriceps are inhibited by the presence of fluid in the knee joint (deAndrade *et al.*,1965; Jayson & Dixon,1970a; Chapter 7 & Experiment I in the present chapter), the pressure volume curves recorded in the present subjects, during contraction, would have been steeper if inhibition had not occurred. During quadriceps setting in Jayson & Dixon's studies (1970b), pressure fell after 80ml of infusion presumably because the voluntary contractions were inhibited and therefore could not produce large increases in pressure. Inhibition of voluntary contractions may not have been so obvious in the present subjects because of the smaller degrees of contraction (i.e. 15-20% max).

Conclusion

The increase in intra-articular pressure with joint infusion is greater during contraction than at rest and this may increase reflex inhibition of quadriceps.

THE USE OF THE 'MODEL' OF NORMAL SUBJECTS WITH KNEE JOINT INFUSIONS

Tolerance of the Procedure

All subjects tolerated the procedure well and there were no after effects. In some cases, fluid remained in the knee after removal of the cannula and swelling had resolved within a few hours. The only discomfort experienced was a dull ache for 30s to 2 mins after insertion of the cannula. As the joint was being filled with fluid, the sensation experienced was of pressure and not of pain, indicating that the cannula was in the joint. Pain occurred in

1 subject (Subject 4) as soon as the cannula was inserted and did not ease. The suspicion that the cannula was not correctly placed was confirmed when an attempt at infusion of less than 10ml increased the pain and there was resistance to infusion. The cannula was therefore removed and a fresh cannula was inserted through the same skin wound but presumably not through the same capsular wound. Generally, manipulation of the cannula and changing the angle of the knee joint also caused discomfort.

Jayson & Dixon (1970b) reported joint rupture in some of their subjects during joint use but this did not occur in any of the present subjects studied as the joint remained in the same non-weight bearing position during contractions.

Maximal Effort during Recording of MVA

When the effect of joint infusion on quadriceps MVA is being monitored, it is essential to encourage maximal effort. This might be a problem particularly when the subject is aware of the expected effect of infusion on MVA, so naieve subjects should be studied whenever possible. The repeatability of MVA recordings made in the patients studied in Chapter 3 demonstrated that they could consistently produce maximal contractions.

Technical Problems with the Knee Infusion Model

The cannula became kinked after maximal quadriceps contractions at different knee angles were performed by subject la. This caused failure of the system to record intra-articular pressure, and produced difficulties with infusing and removing fluid from the knee joint. It was decided that the tests of MVA at different angles would not be performed on the next subject, but maximal contractions in extension also caused the cannula to become kinked. It was therefore decided to abandon experiments involving maximal contractions and changing joint angle and concentrate on other aspects of the studies.

In subjects 2a & 4, a second cannula was inserted and caused fluid to leak out of the joint into subcutaneous tissues, presumably because the cannula was not replaced into the original capsular wound. Insertion of the cannula near the mid-patellar level was more satisfactory than inserting it more proximally (halfway between the mid patella and the upper pole).

FURTHER STUDIES

The two studies mentioned in the 'INTRODUCTION' which examined the knee flexion phenomenon, indicate areas for further investigation but an infusion system which is not influenced by changes in joint angle etc. would need to be used, and more subjects should be studied.

If intra-articular pressure was responsible for the fact that inhibition is less in mid-range ($30-50^{o}$), it would be expected that inhibition would be greatest at joint angles of high pressure ($0-10^{o}$ and $70-90^{o}$). Further studies should therefore include tests at the higher degrees of flexion.

The effect of increasing joint volume on quadriceps activation need to be studied in more subjects. Smaller increments of infusion should be used (e.g.5ml) in order to determine the minimal amount of fluid which will cause inhibition.

CHAPTER 9

REVIEW OF THE CHARACTERISTICS AND USE OF THE H-REFLEX TO INVESTIGATE REFLEX (INVOLUNTARY) ACTIVATION OF MUSCLE

Examination of Reflex Activation using Quadriceps H-reflex

History of the H-reflex

Distinction between the H-reflex and the F Wave

Physiological Characteristics of the H-reflex

Inhibition of the H-reflex by Conditioning Stimulation

Effect of Voluntary Contraction on the H-reflex

Effect of Movements of Remote Parts of the Body on the H-reflex

Effect of Joint Angle on the H-reflex

REVIEW OF THE CHARACTERISTICS AND USE OF THE H-REFLEX TO INVESTIGATE REFLEX (INVOLUNTARY) ACTIVATION OF MUSCLE

Examination of Reflex Activation using the H-reflex

The H-reflex is a monosynaptic reflex which produces a small muscle contraction in response to low intensity stimulation of a mixed nerve. Group Ia fibres from the muscle spindles form the afferent arc of the reflex, and alpha motoneurones form the efferent arc (Fig 9.1.). The small muscular response which is produced can be recorded by surface EMG and is a measure of reflex or involuntary muscle activation. The purpose of this chapter is to provide a background to the H-reflex studies in the next 2 chapters *viz.* control experiments for investigating inhibition of quadriceps H-reflex in (Chapter 10) and experiments which examine the central pathway of the afferent inhibitory stimuli from knee joint receptors (Chapter 11).

History of the H-reflex

The electrically induced monosynaptic reflex was first demonstrated by Hoffmann (1918) when he stimulated the human tibial and femoral nerves submaximally. He observed that the latencies and configurations of these reflex responses in soleus and quadriceps were comparable with the action potentials associated with the ankle and knee jerks respectively. Hoffmann thus concluded that both the electrically and mechanically induced late responses must involve the same type of stretch reflex pathway. Supramaximal stimulation abolished the late response and produced an early wave which was evoked by direct stimulation of motor axons. This observation, and the fact that the late responses were at relatively short latencies (soleus 30ms, quadriceps 25ms) led Hoffmann to conclude that the afferent pathway of this reflex must consist of very fast conducting fibres and that the central delay must be very short.

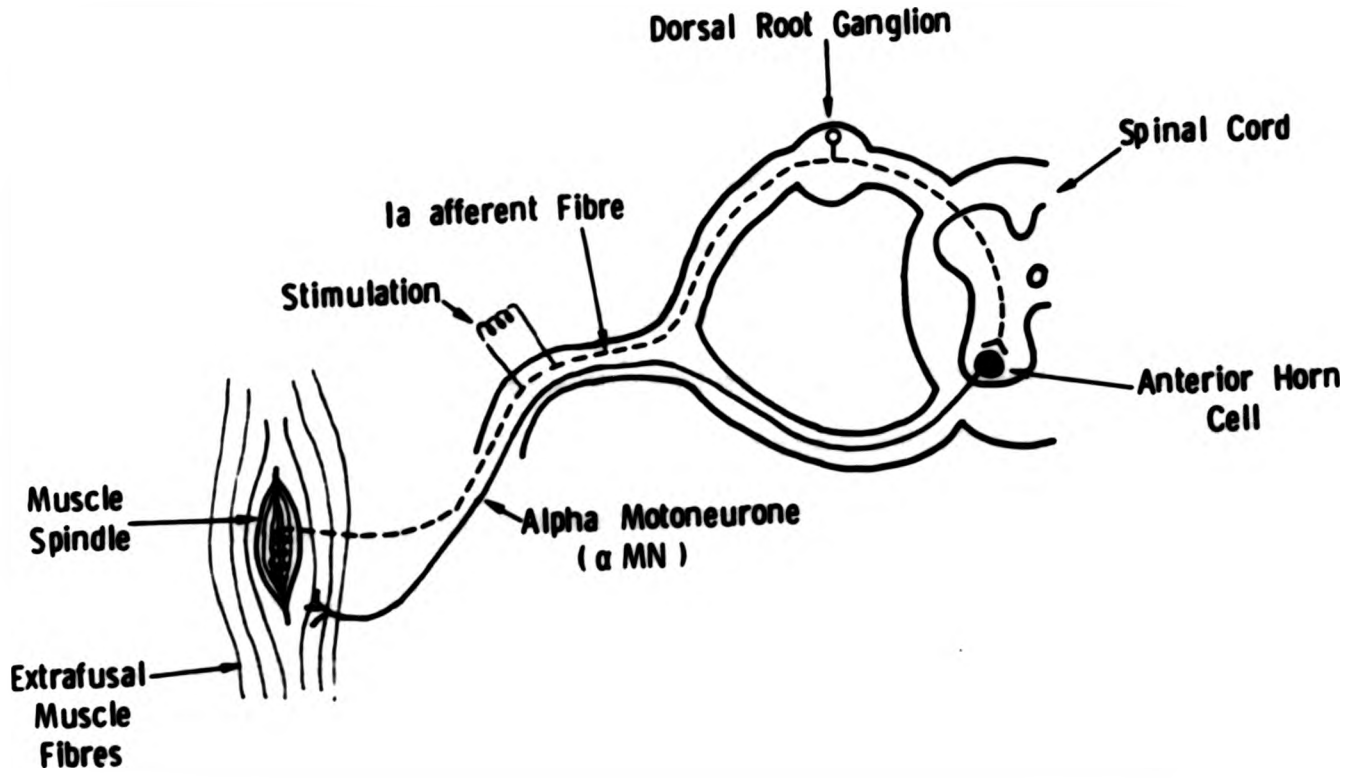

Figure 9.1. Quadriceps H-reflex arc. Low intensity stimulation of the femoral nerve excites the Ia afferent fibres from quadriceps causing excitation of quadriceps anterior horn cells. This results in a small contraction of the quadriceps muscle.

Since Hoffmann's experiments many authors have studied the late responses, particularly in animals. Lloyd (1943a) studied the reflexes in the hindlimb of the cat and demonstrated that the afferent fibres involved in the monosynaptic reflex were the low threshold, fast conducting, large diameter fibres from the annulospiral endings in muscle spindles (i.e. Group Ia fibres). The multineurone arc reflexes involve higher threshold fibres. Lloyd (1943a) also found that afferent fibres directly connected with the MN's are only found in significant numbers in muscle nerves. Hoffmann's conclusions were confirmed in humans by Magladery & McDougal (1950) who named the late response the H or Hoffmann reflex and the early muscle response the M wave or M response. Magladery et al.(1951) verified that the H-reflex was monosynaptic by making recordings more centrally, from the spinal roots and the dorsum of the spinal cord. The time interval between dorsal and anterior root potentials at the tip of the cord was 1.5ms. They calculated the central delay by deducting time for intraspinal conduction (vide infra) leaving 0.9ms which indicated that impulses could only have passed across a single synapse. Intra-spinal conduction involving 1 synapse is between 0.65-1ms (Renshaw, 1940). Magladery et al. (1951) also demonstrated that the conduction velocity in the afferent arc was 10% faster than in the efferent arc.

Distinction between the H-reflex and the F Wave

Magladery & McDougal (1950) observed another late response which had a similar latency to that of the H-reflex but was smaller in amplitude and required stronger stimulation. This response was termed the F wave and was originally thought to be a polysynaptic reflex but it is now agreed that it is not a reflex but a small centrifugal discharge from the spinal cord produced by an antidromic volley in the axon (Dawson & Merton,1956; Thorne,1965; Mayer & Feldman,1967). An antidromic volley is produced by a direct shock applied to a motor axon at or above alpha threshold, which passes

centrally and causes a centrifugal discharge of some of the neighbouring MN's in the spinal cord (Lloyd,1943b; Sherrington (In: Denny-Brown,1979c). The centrifugal impulses are repetitive discharges which are directed distally (centrifugally) and are not reflex discharges synaptically excited through recurrent collaterals (Renshaw,1941). The F wave is reliant upon an intact motor pathway and is independant of afferent nerves. The differences between the H-reflex and the F wave are summarized in Table 9.1. (after Shahani & Young,1980).

The F wave can be mistaken for the H-reflex if the characteristics of the responses are not known or understood e.g. Goldberg (1983) claimed that an H-reflex could not be elicited in the quadriceps and that results reported in a paper by Kennedy et al.(1982) must have been F waves. Kennedy (1983) defended the validity of his data in a letter in which he presented the results of some classical physiological tests (vide infra) which proved that he was recording an H-reflex. The preliminary data reported by Kennedy et al. (1982) has now been published in greater detail (Spencer et al.,1984).

Physiological Characteristics of the H-reflex

Many of the studies which have examined the parameters of the H-reflex in man have used soleus because it is easy to elicit reflexes in this muscle . Certain physiological characteristics of the H-reflex have been well documented viz. 1) the H-reflex is elicited at low intensities of stimulation 2) the reflex can be inhibited by a conditioning stimulus (vide infra) and 3) voluntary contraction of the agonist enhances the reflex and contraction of the antagonists inhibits it.

Stimulation parameters. The stimulation parameters are: 1) low intensity; 2) long duration ; Erlanger and Blair (1938)showed that sensory fibres were more responsive than motor fibres to waves of long duration and less responsive to those of short duration e.g.

H Reflex	F Response
1. A monosynaptic reflex in which the afferent arc consists of group Ia afferent fibers from muscle spindles, and the efferent arc consists of alpha motor axons.	Not a reflex. Centrifugal discharge of a small percentage of motor neurons initiated by antidromic volleys in their axons. Both afferent and efferent arc consists of alpha motor axons.
2. Stimulus threshold lower than that required to evoke a direct M response. Supramaximal stimulation for the M response blocks the H reflex.	Stimulus threshold usually higher than that required for the H reflex and the M response.
3. Mean amplitude (5–10 responses) can be up to 50%–100% of the maximal M response.	Mean amplitude small, usually less than 5% of the maximal M response.
4. Appearance and persistence rather constant at low rates of stimulation (1 every 10–30 seconds).	Appearance and persistence rather variable, even at low rates of stimulation.
5. Easily recorded from the soleus muscle and some proximal "postural" muscles in adults. Present in intrinsic hand and foot muscles in newborn infants.	Can be recorded easily from almost every skeletal muscle.
6. Single motor units activated in the H reflex are different from those in the preceding M response.	Single motor units activated in the F response are same as those in the M response.
7. Fluctuation of latency of the single motor units activated in the H reflex is greater than for those in the F or the M response.	Fluctuation of single motor unit latency in the F response is less than that in the H reflex, but greater than that in the M response.

Table 9.1. Differences between the H-reflex and the F wave. (After Shahani & Young, 1980)

0.5ms (Mongia,1972), 1ms (Hugon,1973); 3) a low frequency to avoid 'late depression' or 'high frequency depression' (Beswick & Evanson,1955; Magladery et al.,1952; Iles & Roberts,unpublished) e.g. 1 every 2s i.e. 0.5Hz (Baldiserra et al.,1983), 1 every 3s (Eccles & Rall,1951; Guiheneuc & Ginet 1974), Hugon (1973)found 1 every 5s to be the optimal rate, 1 every 20-30s (Hayes & Sullivan,1976). Any rate < 1 per minute will probably depress the H-reflex to some extent, but due to the number of reflexes needed for data collection , one has to compromise.

Electrode placement. The cathode should be placed proximal to the anode to avoid slowing or blocking of impulses, which occurs at the anode, and would prevent impulses from passing centrally.

Recruitment curves of the H-reflex and M response. The amplitudes of the H-reflex and M response vary according to the stimulation intensity and their recruitment curves can be plotted. The pattern of recruitment is as follows : as the stimulus intensity is increased from zero, the H-reflex begins to appear and becomes larger. The M wave then begins to appear and as it increases so the H-reflex peaks and then decreases. The fall in H-reflex size is due to collision between antidromic and orthodromic (reflexly elicited) impulses in motor axons (Magladery et al.,1951; Upton et al.,1971). The H-reflex is abolished when the M wave is maximal. In some instances both the H and the M waves begin to appear at the same stimulation intensities and sometimes the M wave preceeds the H-reflex (Mongia,1972; Spencer et al.,1984) but the general pattern of recruitment is always the same. The H and M recruitment curves have been demonstrated in man for the calf muscles (e.g. Mongia,1972; Hugon,1973), quadriceps (e.g. Mongia,1972; Guiheneuc & Ginet,1974; Kennedy, 1983) and muscles of the upper limb (e.g. Upton et al.,1971; Baldiserra et al.,1983).

Inhibition of the H-reflex by Conditioning Stimulation

The H-reflex can be inhibited by applying a conditioning stimulus before the H-reflex stimulus. This type of intervention is useful for studying neural pathways. The conditioning stimulus can be mechanical or electrical.

Prolonged muscle vibration. Vibration of muscle can set up a tonic contraction (tonic vibration reflex - TVR) in the vibrated muscle (Matthews,1966; Burke 1976 1981) and inhibits the phasic reflexes (i.e. tendon jerk & H-reflex) in the same muscle and other muscles. Vibration of a muscle at rest reduces the H-reflex by presynaptic inhibition (i.e. inhibits a neurone before it synapses with the AHC) which is mediated through the group Ia muscle afferents (Gillies et al., 1969). Feline studies by Brown et al.(1967) have shown that primary spindle endings (annulospiral) of Ia fibres respond to vibration when the muscle is under slight tension, and that secondary endings (flower-spray) of group II afferents are insensitive. Golgi tendon organs (giving Ib fibres) were also insensitive to vibration if the muscle was not contracting and therefore the physiological effects of vibrating a tendon are probably the result of repetitive stimulation of primary muscle spindle endings and activation of Ia afferent fibres (Brown et al.,1967). Iles & Roberts (unpublished) found that there was no difference in the effect on H-reflex whether the muscle tendon or its belly were vibrated provided sufficient pressure was applied.

A TVR does not develop in all subjects at rest (Burke,1981). The muscle spindles respond almost immediately to tendon vibration but the TVR may develop slowly (e.g. onset 3s after vibration). Once the reflex contraction occurs, muscle spindle activity decreases. The mechanisms responsible for the slow development of the TVR in normal man are not known but the properties of the TVR represent a progressive change in the gain of the stretch reflex pathways which cannot be attributed to the fusimotor system

(due to constant or reduced spindle activity).

The mechanism by which vibration of a muscle produces inhibition of itself i.e. 'paradoxical' inhibition (Desmedt & Godaux,1978) is as follows: vibration excites receptors of the muscle spindle afferent fibres (Gillies et al.,1969) which should produce autogenetic excitation of the motor neurones to that muscle i.e. TVR (deGail et al.,1966), but it also produces autogenetic presynaptic inhibition of the monosynaptic pathway (Lance et al.,1973) and this inhibition outweighs the excitation. This dual excitatory and inhibitory action of muscle spindle primary afferents has also been demonstrated in the cat soleus (Matthews,1966; Gillies et al.,1969; Magherini et al.,1972; Thoden et al.,1972).

Vibration studies in man have mainly been performed on soleus by vibration of soleus itself (Rushworth & Young,1966; Lance et al.,1973; DeGail et al.,1966; Desmedt & Godeaux,1978). Iles & Roberts (unpublished) have examined the effect of vibration in other muscles of the leg, including extensors. They have shown that flexor muscles of the leg are inhibited mainly by vibration of themselves whereas the extensors are inhibited by vibration of themselves, by vibration of flexors but not strongly by other extensors. The functional significance of this difference in effects on flexors and extensors is not known.

Brief mechanical stimulation of tendons. Tendon percussion using a high velocity vibrator has also been used to condition H-reflexes (Iles & Roberts, unpublished).

Brief electrical stimulation of muscle nerves. Electrical stimulation of antagonist muscle nerves, at or above threshold, can be used to condition the H-reflex. Renshaw (1941) used common peroneal nerve (CPN) stimulation to condition soleus H-reflex in the cat. In man, soleus H-reflex was conditioned by CPN stimulation (Braddom & Johnson,1974b; Tanaka,1974; Pierrot-Deseilligny et al.,1981; El-Tohamy & Sedgwick,1983; Iles & Roberts, unpublished),

and quadriceps H-reflex was conditioned by sciatic nerve stimulation (Iles & Roberts,unpublished). Sciatic nerve stimulation is only successful in lean subjects.

Electrical stimulation is preferable to vibration for conditioning because voluntary contraction may cause muscle spindles to become more sensitive to vibration, and a TVR reduces sensitivity. The time interval between the conditioning shock and the H-reflex stimulus determines the type and magnitude of H-reflex inhibition (vide infra). The time course of soleus H-reflex inhibition by CPN stimulation shows a curve with an early phase (peak about 4ms) and a later phase (peak about 12ms) (Iles & Roberts, unpublished). In Iles & Robert's study (unpublished) the intensity of the conditioning stimulus was weaker than 0.8 times alpha threshold, so both early and late inhibition could be attributed to stimulation of group I muscle afferent fibres or very low threshold cutaneous afferents. Iles & Roberts (unpublished) assumed that the early phase represented reciprocal Ia inhibition (postsynaptic cf.Tanaka) and the later phase represented group I presynaptic inhibition. This interpretation was supported by their observation that the inhibitory time curve obtained by conditioning tibialis anterior H-reflex by tibial nerve stimulation showed little late inhibition. This was consistant with an earlier observation that vibration of gastrocnemius/soleus had little effect on the tibialis anterior H-reflex. Reciprocal inhibition of H-reflexes by short latency shocks has also been demonstrated in the human forearm (Day & Rothwell,1983) and in the calf (Tanaka,1974).

The known pathways of inhibition of quadriceps H-reflex used in the present studies (Chapters 10 & 11) were: Ib inhibition from Golgi tendon organs, by stimulation of the tibial nerve (Pierrot-Deseillgny et al.,1981); FRA inhibition via cutaneous nerves by sural nerve stimulation (Hugon, 1969 & 1973) and Ia pre-

and postsynaptic (reciprocal) inhibition by stimulation of the sciatic nerve (Iles & Roberts, unpublished).

Effect of Voluntary Contraction on the H-reflex

The H-reflex is facilitated by voluntary contraction of the agonists (Hagbarth,1962; Delwaide & Hugon,1969; McComas et al.,1970; Upton et al.,1971; Tanaka,1974; Lundberg,1982; Baldissera,1983; Iles & Roberts,unpublished). Voluntary contraction also reduces the inhibition in both the early and late phases of the inhibitory time curve, and the inhibition produced by vibration (Iles & Roberts,unpublished). The fact that the H-reflex is under supraspinal control presents a problem when using the H-reflex for investigation of inhibition. Even at rest the H-reflex is said to be influenced by just thinking about making a contraction (Coquery & Coulmance,1971; Upton et al.,1971; Delwaide & Toulouse,1981). The changes in gain of the H-reflex pathway are not due to changes in muscle spindle activity, as these only occur after the onset of a contraction, and so must occur through a mechanism which is independent of the fusimotor system (Burke,1981). The problem of enhancement of the reflex by thinking about contracting can be overcome by recording the H-reflex during a small controlled voluntary contraction to avoid fluctuations in the descending influence (Iles,1977). Another reason for recording the H-reflex during contraction is that it is important to examine muscle during function. Contraction of the antagonists reduces the H-reflex, presumably in part by reciprocal inhibition.

Effect of Movement of Remote Parts of the Body on the H-reflex

Movements of remote parts of the body can influence the H-reflex by the same mechanism as the Jendrassik manoeuvre (Delwaide & Toulouse,1981). This classical manoeuvre involves hooking the distal phalanges of 1 hand with those of the other hand in front of the chest and pulling the hands apart from each other. Facilitation of

H-reflexes in the leg is thought to be transmitted *via* long loops comprising of peripheral afferents, spinal pathways, supra-spinal relays and descending spinal pathways (Delwaide & Toulouse,1981). The level of the supra-spinal relay and the identity of the descending pathways involved are not known. The position of the head also influences the H-reflex *via* the tonic neck reflex (Hayes & Sullivan,1976). Rotation of the head to the right increases the H-reflex in the right limb and rotation to the left reduces it. This descending influence is thought to occur by direct facilitation of the MN pool. Care was therefore taken in the present studies to record H-reflexes with the subject relaxed in a supine position and remaining there throughout the procedures.

Effect of Joint Angle on Quadriceps H-reflex

Passive lengthening of the quadriceps is said to reduce the H-reflex (Guiheneuc & Ginet,1974). In man, soleus H-reflex amplitude is increased with plantarflexion of the ankle and decreased with dorsiflexion (Davies & Lader,1983). The mechanism of the effect which joint angle has on the H-reflex might be attributed to joint afferent activity as demonstrated on the limb reflexes of the cat (Baxendale & Ferrell,1981).

Effect of Knee Joint Infusion on Quadriceps H-reflex

Quadriceps H-reflex was progressively decreased by inflation of normal knees with saline in a study which aimed to determine the minimal amount of fluid needed to produce inhibition (Spencer *et al.*,1984).

CHAPTER 10

CONTROL EXPERIMENTS FOR INVESTIGATING INHIBITION OF QUADRICEPS H-REFLEX

INTRODUCTION

SUBJECTS

METHODS

- Demonstration of soleus H-reflex
- Demonstration of quadriceps H-reflex

RESULTS

- Soleus H-reflex Characteristics
  - Prolonged vibration of tibialis anterior
  - H-reflex/M response recruitment curves
  - Ia inhibition
  - Effect of voluntary contraction
- Control Data for Quadriceps H-reflex Experiments
  - Prolonged muscle vibration
  - H-reflex/M response recruitment curves
  - Ia inhibition
  - Ib inhibition
  - Effect of knee joint angle
  - Effect of voluntary contraction

DISCUSSION

- Demonstration of the H-reflex in Soleus
- Demonstration of Quadriceps H-reflex

CONCLUSION

CONTROL EXPERIMENTS FOR INVESTIGATING INHIBITION OF QUADRICEPS H-REFLEX

INTRODUCTION

Demonstration of Soleus H-reflex.

Preliminary tests were performed using soleus as a means of practising the technique. The characteristics of the H-reflex were demonstrated by classical physiological tests.

Demonstration of Quadriceps H-reflex.

The technique was then practised on quadriceps and the same tests as those on soleus were performed (vide infra).

SUBJECTS

Three male and 5 female normal volunteers aged 27 - 40 years were studied. These were the same subjects who underwent the tests reported in Chapters 8 & 11 and each subject has been referred to by the same number as before. Most of the tests were performed on only 2 subjects (1 & 2) and H/M recruitment curves were plotted for all subjects.

METHODS

All tests were performed as described in Chapter 2.

Demonstration of soleus H-reflex.

The physiological characteristics of soleus H-reflex were examined in Subject 1 using the following techniques: 1) Prolonged vibration of tibialis anterior (TA) - Vibration was applied at 3 different intensities of tibial nerve stimulation. 2) H-reflex/M response recruitment curves were plotted . 3) Common peroneal nerve (CPN) stimulation - Ia inhibitory time curves of soleus H-reflex inhibition were plotted on 2 occasions. 4) Effect of voluntary

contraction - soleus H-reflex inhibition by single shock CPN stimulation was examined during a small plantarflexion torque (4Nm) and a larger torque (9Nm).

Demonstration of Quadriceps H-reflex. (approx 80s)

1) Prolonged muscle vibration - The effects on quadriceps H-reflex of vibrating quadriceps, semitendinosus (St), TA and gastrocnemius / soleus (G/S) was tested in Subjects 1 and 2. 2) H-reflex / M response recruitment curves - these were plotted for all subjects . 3) Sciatic nerve stimulation - the Ia inhibitory time curve for quadriceps H-reflex inhibition was plotted in Subject 1 (on 2 occasions) and Subject 5. 4) Tibial nerve stimulation - Ib inhibitory time curves for quadriceps H-reflex inhibition were plotted for Subjects 1 and 2. An experiment to see how the estimate of Ib inhibition depends on test H-reflex size was performed in Subject 2 by altering the test stimulus intensity. Tests were performed at rest so that inhibition was significant. The latency of tibial nerve stimulation was 10ms before the test stimulus and the shocks were randomised (see Chapter 2, page 33). 5) Effect of joint angle - quadriceps H-reflex was recorded in extension and at 30° knee flexion in Subject 1. 6) Effect of voluntary contraction - small (approximately 20% maximum) and large (50-60% max) straight leg isometric quadriceps contractions were performed during conditioning of the H-reflex by sciatic nerve stimulation in Subject 1, to observe the effects on pre and postsynaptic inhibition. The short latency used was 7ms and the longer latency was 15ms.

When possible, control tests were performed on a separate occasion before the knee infusion experiments (4 subjects) in order to reduce the likelihood of fatigue. If this was not possible, there was a break between control tests and the infusion experiments.

RESULTS

Soleus H-reflex Characteristics

Prolonged vibration of tibialis anterior. Soleus H-reflex was inhibited by 39%, 45% and 43% by vibration at different intensities of tibial nerve stimulation. The M responses of the control and inhibited H-reflexes were the same with each level of tibial nerve stimulation (Fig 10.1.).

H-reflex/M response recruitment curves. The curves showed the expected general pattern of recruitment (Fig 10.2.). The data for both responses (in this and subsequent H/M graphs) have been normalized by expressing the response amplitudes as a percentage of the maximum M response.

Ia inhibition. The inhibitory time curves for soleus produced by CPN stimulation showed the expected early peak of 3-4ms (postsynaptic inhibition) and a later peak of about 12ms (presynaptic inhibition) and curves were plotted on 2 occasions (Fig 10.3.). The conditioned H-reflex is expressed as a % of the test reflex in all of the graphs illustrating inhibitory time curves.

Effect of voluntary contraction. During short latency (4ms) conditioning stimulation, soleus H-reflex inhibition was reduced by stronger contraction of the plantaflexors (4Nm, inhibition =42%; 9Nm = 25%).

Control Data for Quadriceps H-reflex Experiments

Quadriceps H-reflex was easily elicited during contraction in all subjects but was only large enough to be measured at rest in 3 subjects (Subjects 2, 3 & 6). The H-reflex appeared at a latency of about 20-25ms.

Prolonged vibration of lower limb muscles. In both of the subjects studied (Subjects 1 & 2) quadriceps H-reflex was inhibited by vibration of itself and lower limb flexors, while vibration of G/S (extensors) produced little or no inhibition (Fig 10.4.a+b.). The

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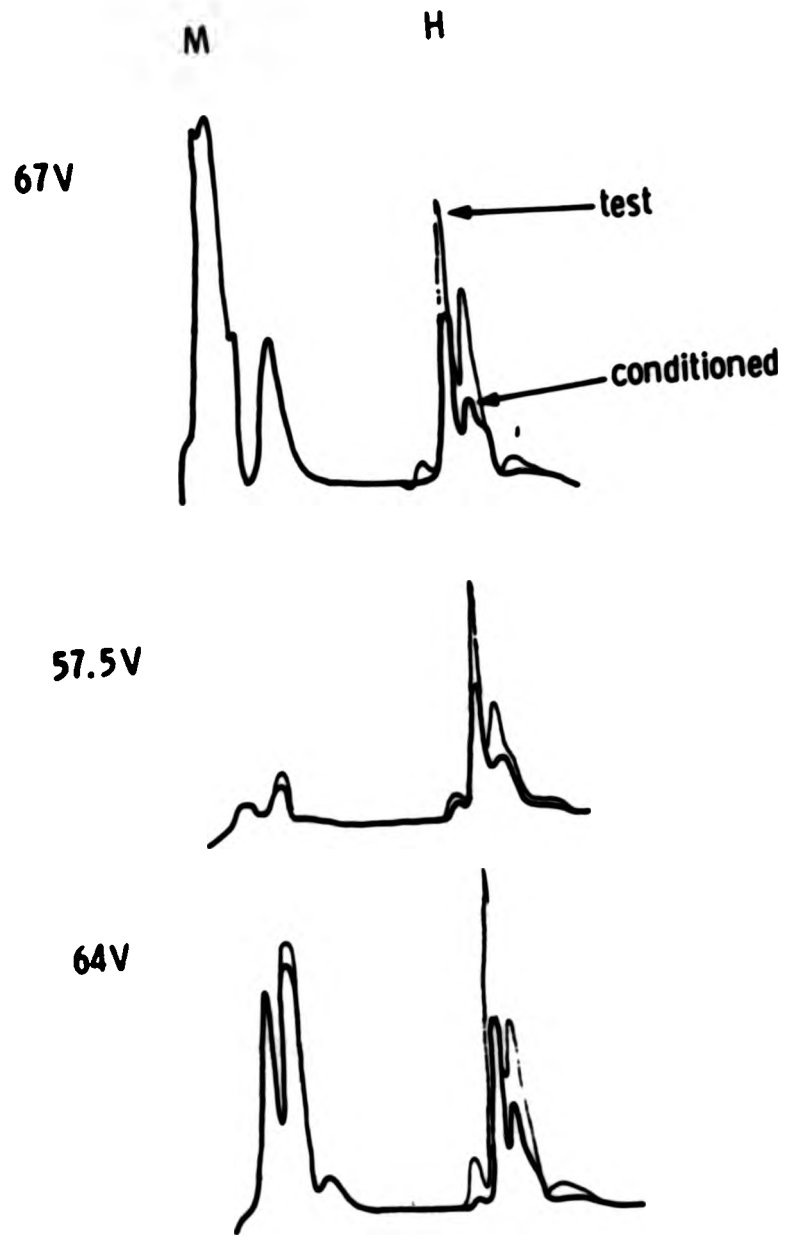

Figure 10.1. Effect of prolonged vibration of tibialis anterior on soleus H-reflex amplitude at 3 stimulation intensities. M = M response H = H-reflex

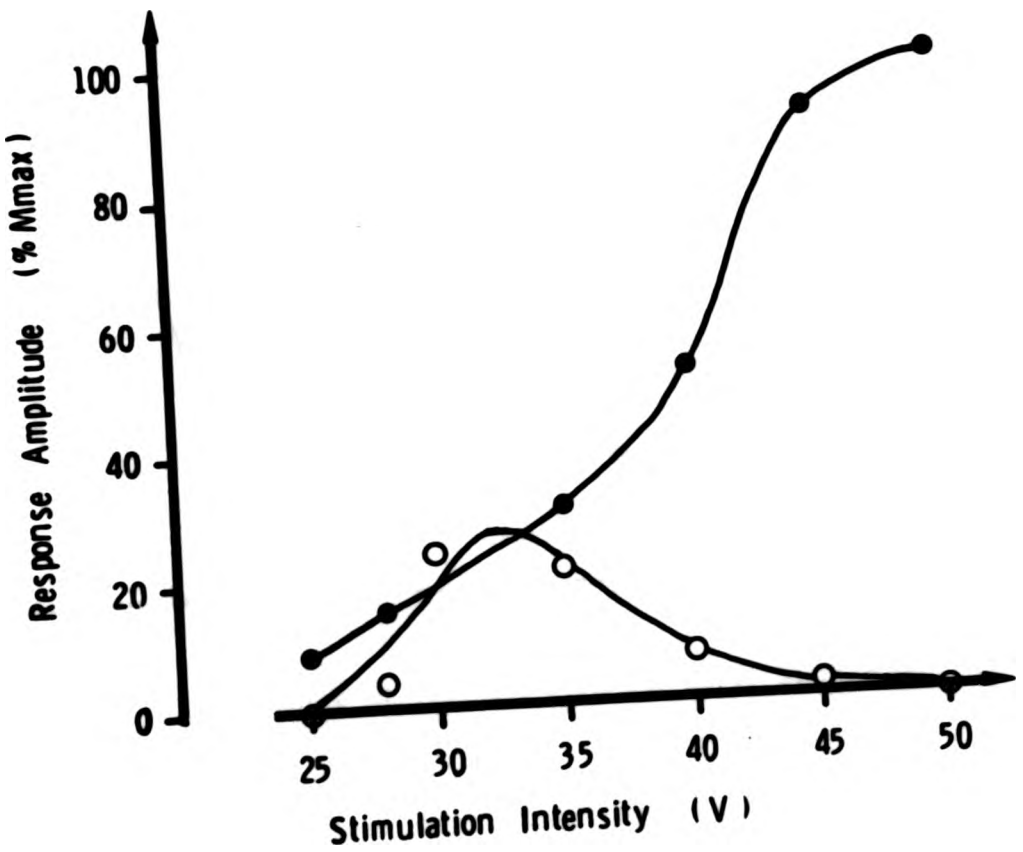

Figure 10.2. Soleus H-reflex (O)/M response (●) recruitment curves.

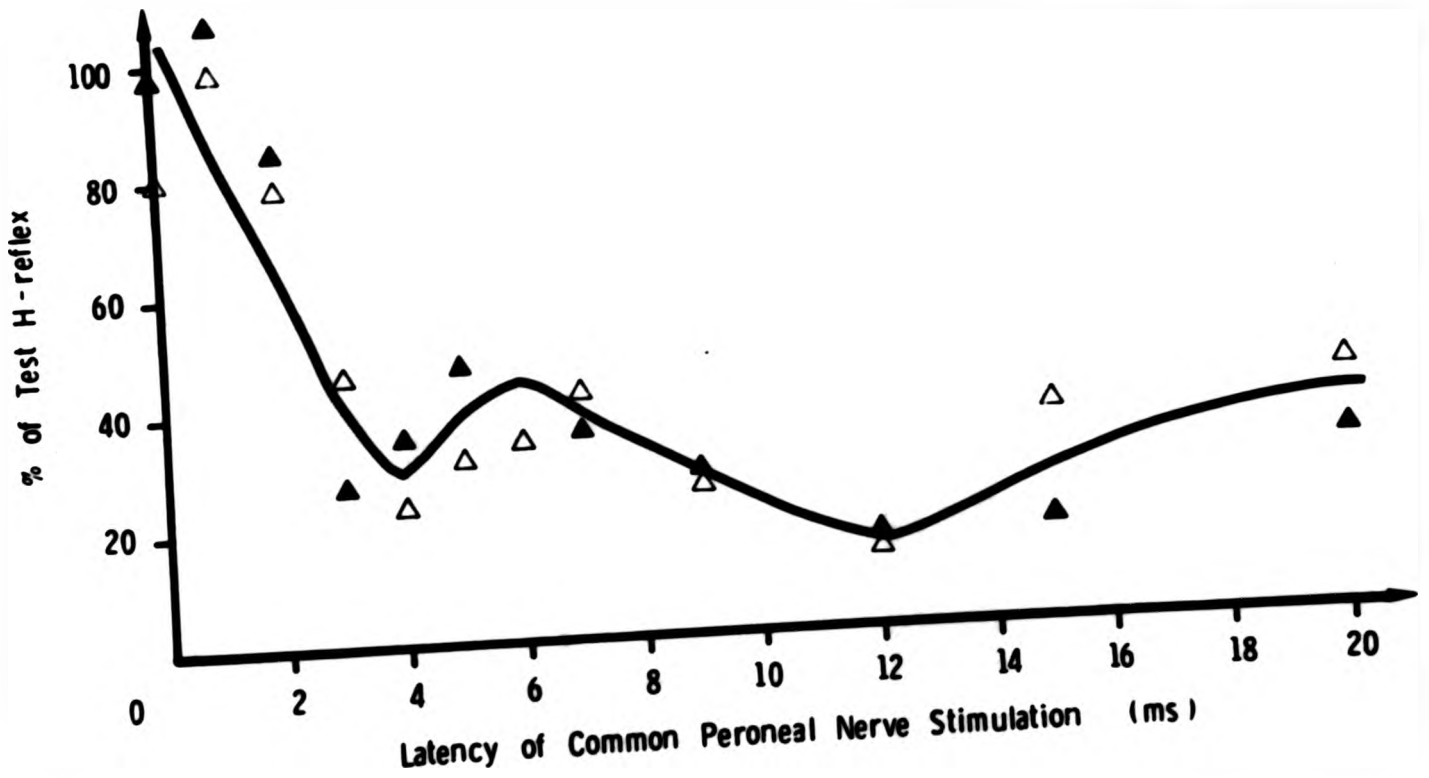

Figure 10.3. Ia inhibitory time curve for soleus H-reflex produced by common peroneal nerve stimulation in Subject 1. Peak postsynaptic inhibition occurred at 3-4ms and peak presynaptic inhibition was at 12ms.

▲ 1st occasion △ 2nd occasion

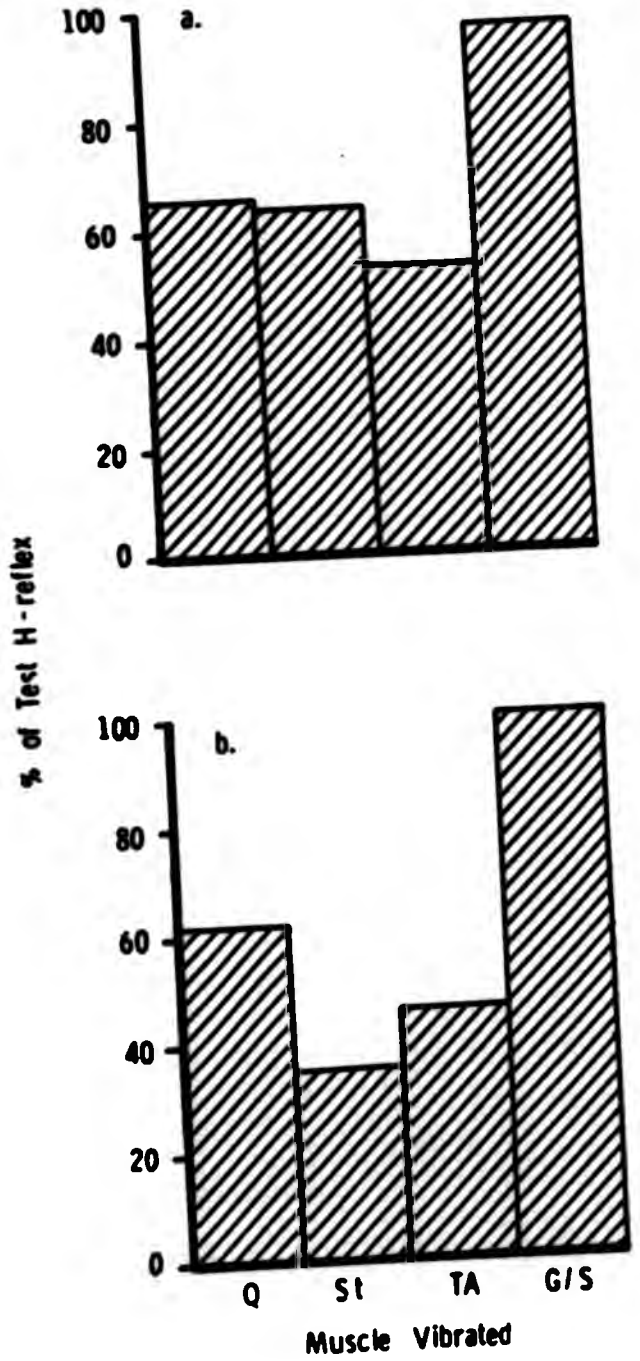

Figure 10.4. Effect of vibrating lower limb muscles on quadriceps H-reflex in a. Subject 1 and b. Subject 2. Quadriceps is inhibited by vibration of itself and the limb flexors but not of other extensors.

small effect from vibration of G/S seen in Subject 1 could have been due to vibration spread to tibialis anterior.

H-reflex/M response recruitment curves. The curves for each subject (not plotted for Subject 5) showed the expected patterns (Fig 10.5.a-e). In subjects 2 & 6, large H-reflexes could be elicited at rest so the curves were therefore plotted at rest and during a 20% quadriceps contraction (Fig 10.5.f+g). The ascending part of the H-reflex curve recorded during contraction was shifted slightly to the left due to facilitation of the H-reflex by voluntary contraction which produced larger H-reflexes at lower intensities of stimulation.

Ia inhibition. Conditioning of the H-reflex by stimulating the sciatic nerve was only achieved in 2 subjects (Subjects 1 & 5). Two inhibitory time curves were plotted for Subject 1 and showed early peaks of 6-7ms and later peaks of about 15ms, and the curve for Subject 5 showed peaks of 7ms and 11 ms (Fig 10.6.a+b). There is a large cross-over area between the peaks for the 2 types of inhibition, so mixed inhibition would occur if care was not taken to avoid the intermediate latencies when testing each type of inhibition.

Ib inhibition. Inhibitory time curves were plotted for Subjects 1 and 2 and showed peak inhibition at about 8 and 12ms (Fig 10.7.). Test H-reflexes of > 40% Mmax seem to be less inhibited than smaller reflexes (Fig 10.8.). The thin line with the slope of 1 indicates no conditioning effect. The regression line with the slope of 0.84 fits the points with test H-reflexes up to 40% M max.

Effect of knee joint angle. The H-reflex was present in both extension and flexion in Subject 1 but due to different M response sizes, the relative H-reflex sizes cannot be compared. The site of stimulation must have changed slightly during flexion. In Subject 2, the H-reflex appeared to be slightly smaller in flexion, both at rest and during a small voluntary quadriceps contraction (20% max). The results were not measured as the recording conditions were

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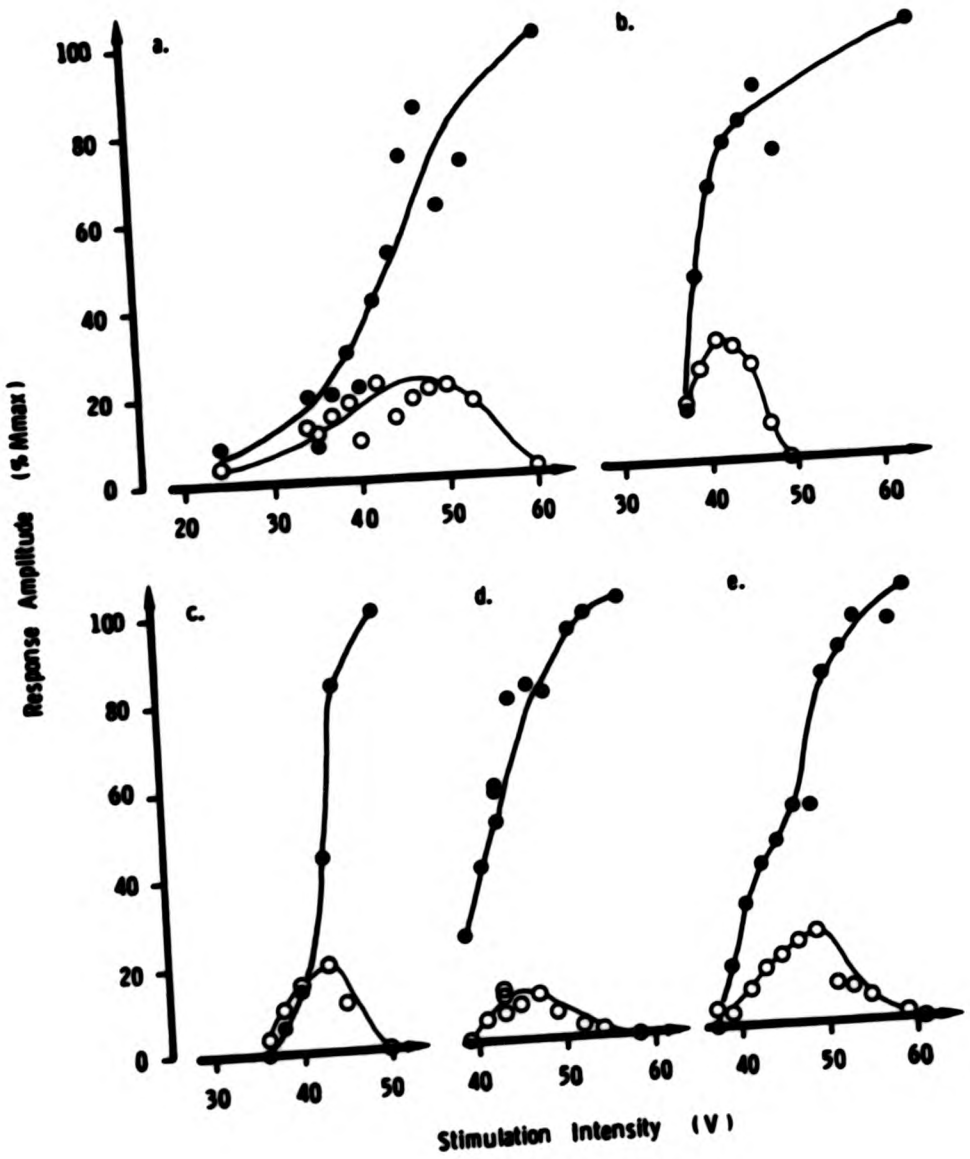
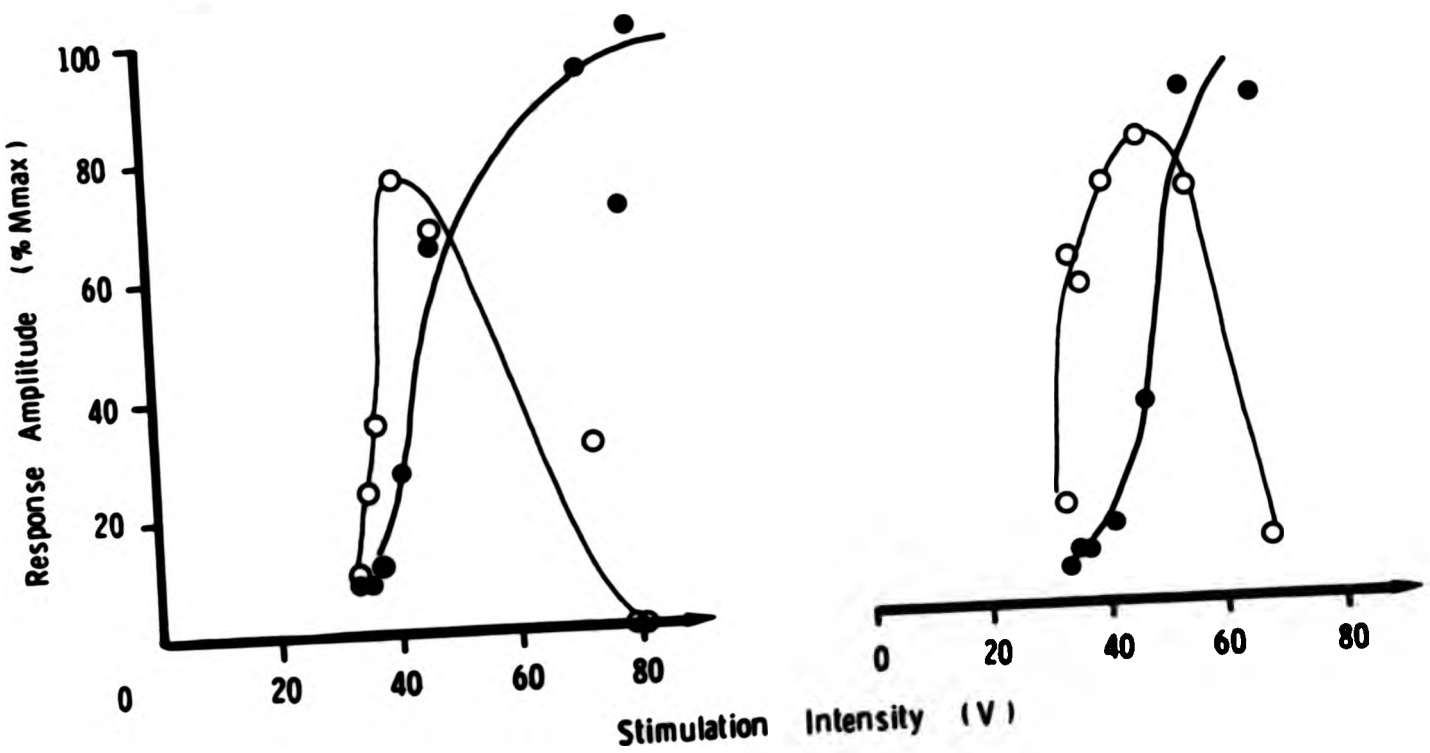


Figure 10.5.a-e Quadriceps H-reflex (O)/M response (●) recruitment curves recorded during contraction in Subjects 1,4,6,7 & 8.

f.



g.

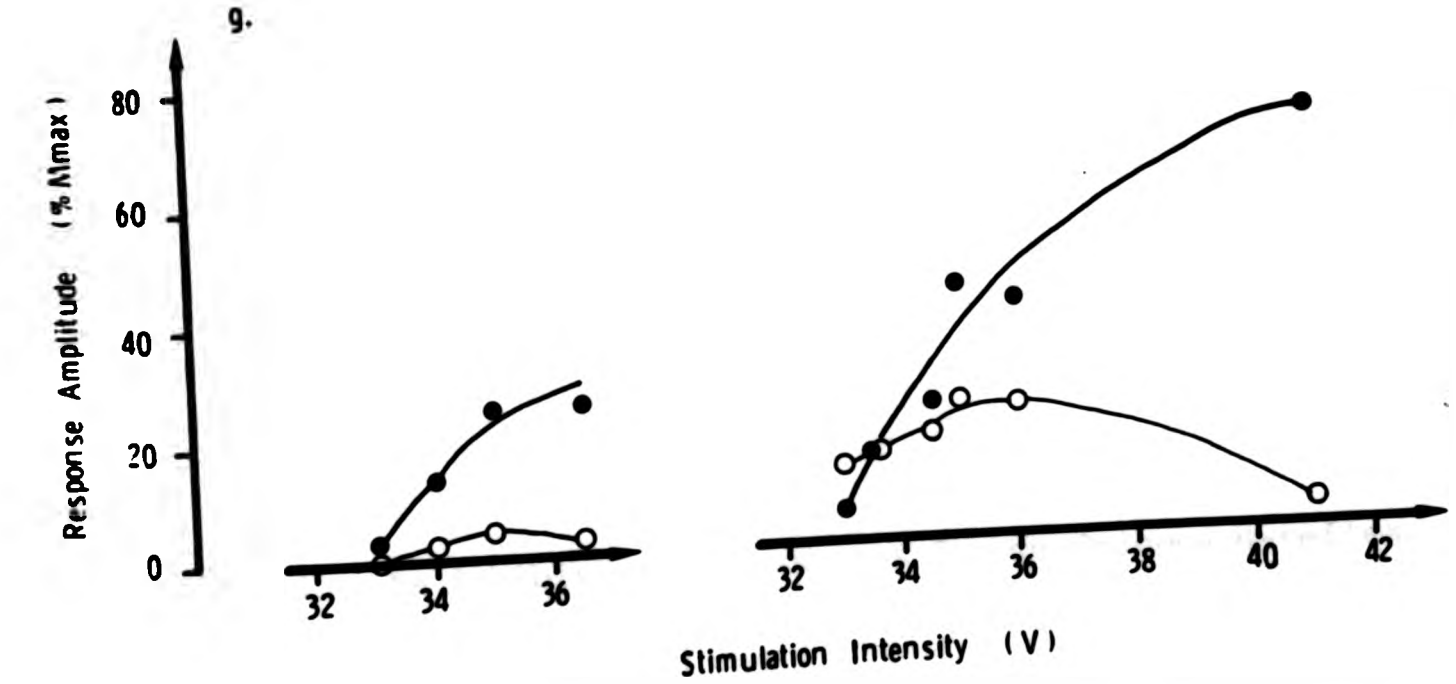


Figure 10.5.f+g Quadriceps H-reflex (O)/M response (●) recruitment curves in Subjects 2 & 3. Contraction of quadriceps produced larger H-reflexes at lower intensities of stimulation.

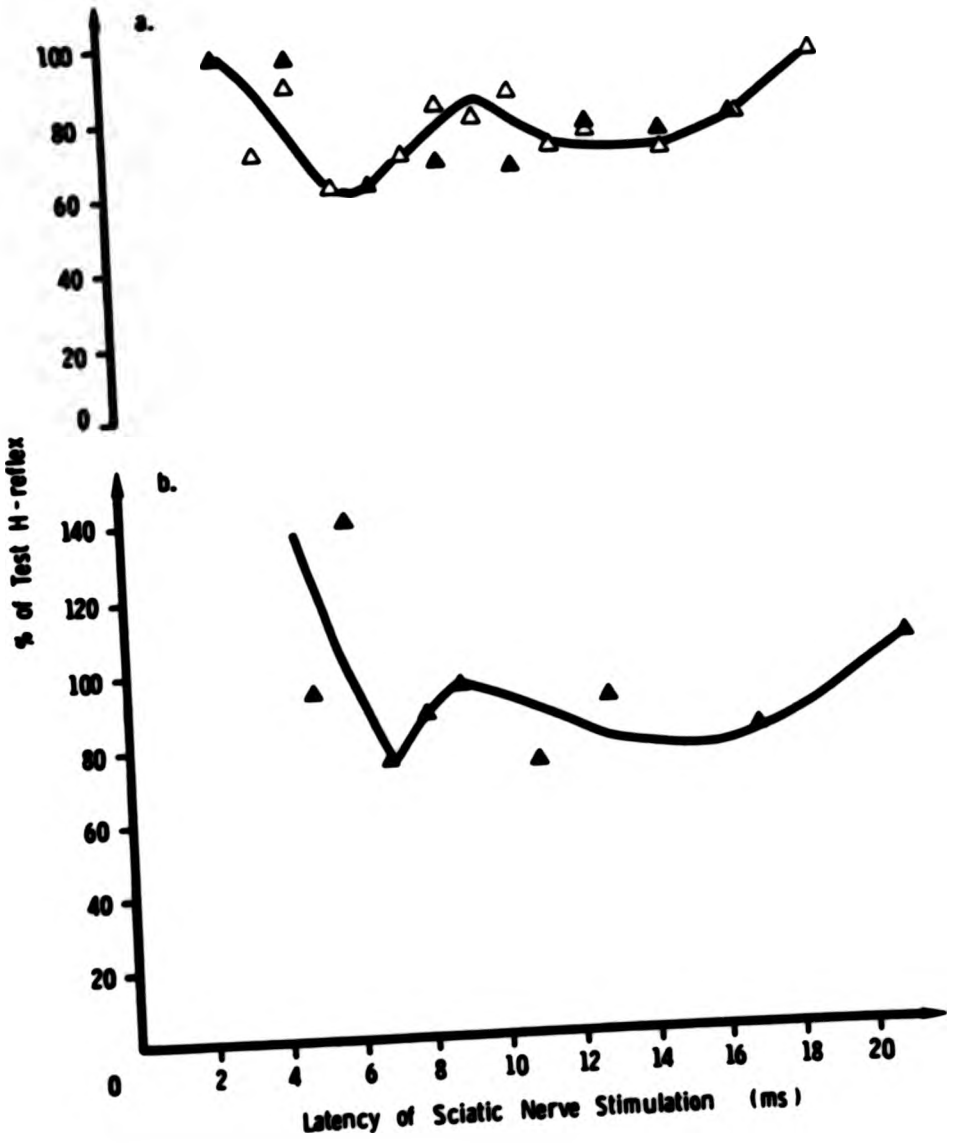


Figure 10.6. Ia inhibitory time curves for quadriceps H-reflex produced by sciatic nerve stimulation. Peak postsynaptic inhibition was at 6-7ms and the presynaptic peak was at 11-15ms.

a. Subject 1 ▲ 1st occasion △ 2nd occasion

b. Subject 5

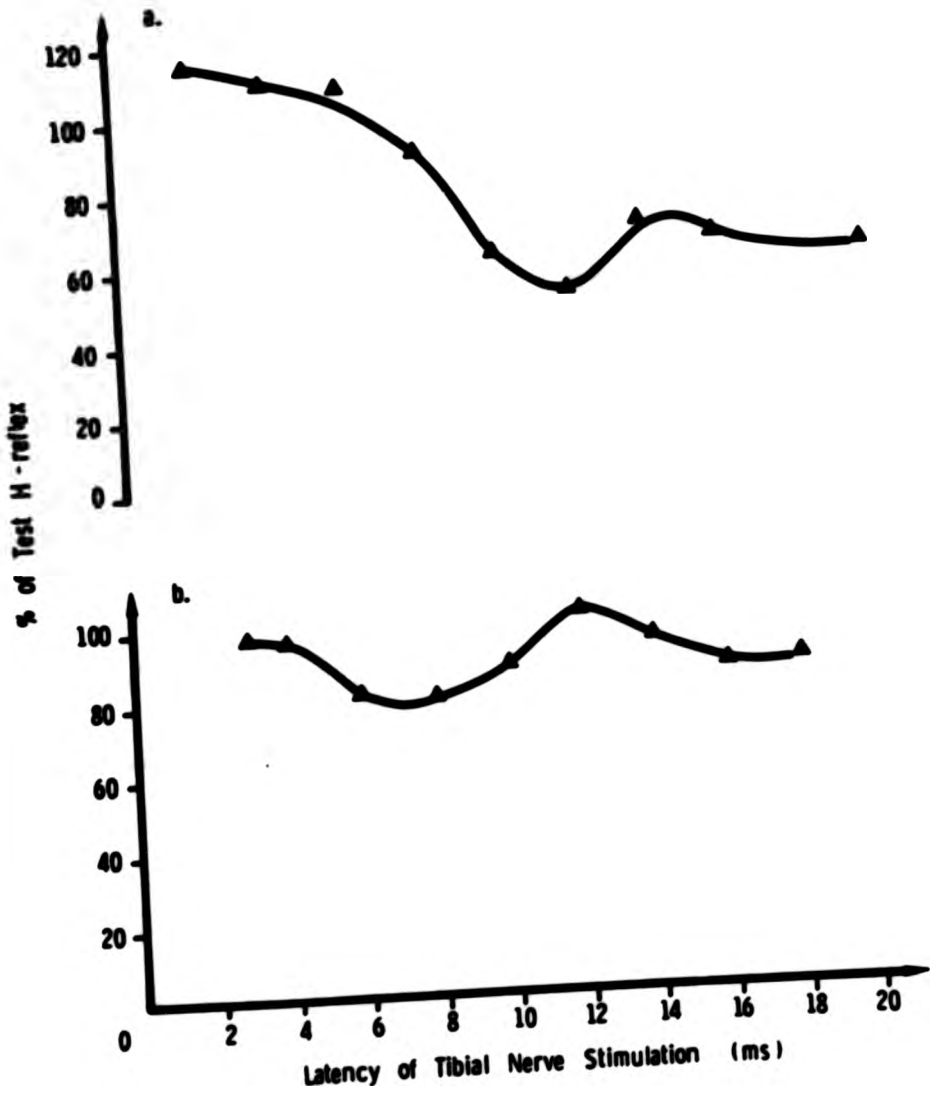


Figure 10.7. Ib inhibitory time curves for quadriceps H-reflex produced by tibial nerve stimulation. Peak inhibition occurred at 8 and 12 ms.

a. Subject 1 b. Subject 2

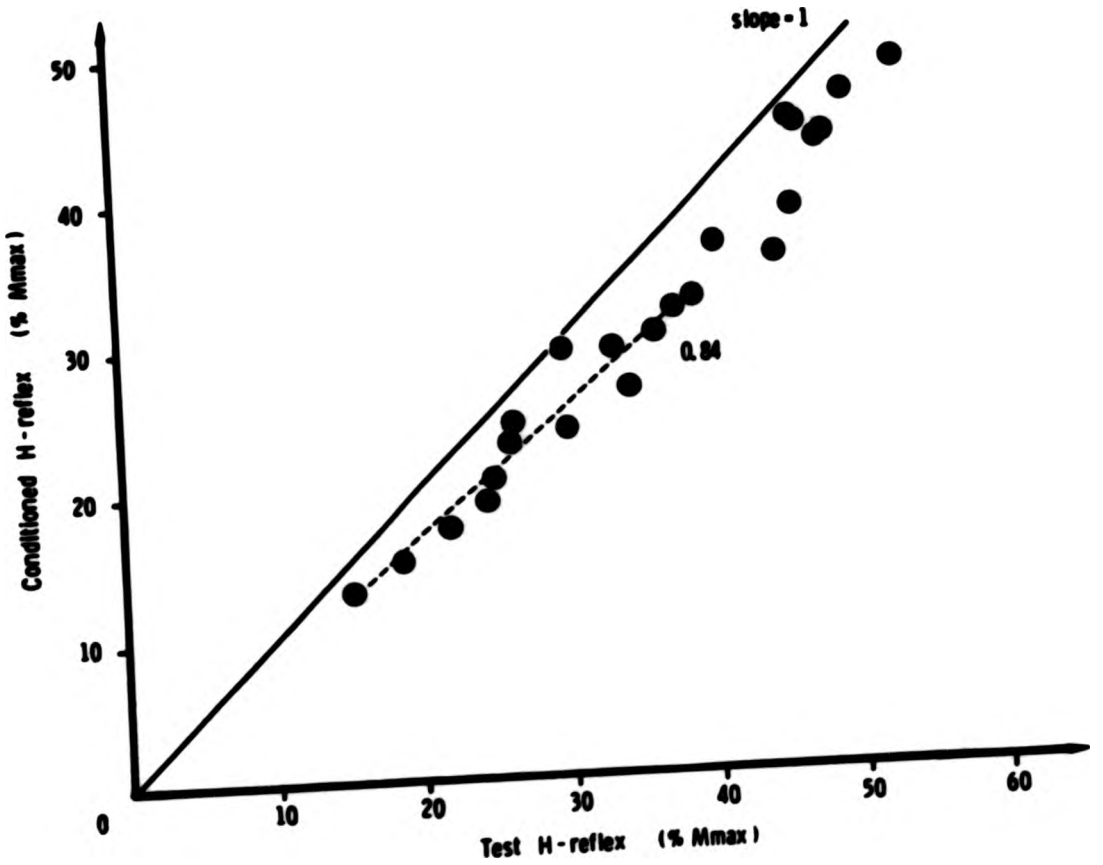


Figure 10.8. Relationship between Ib inhibition of quadriceps H-reflex and test H-reflex size. Test reflexes >40% Mmax are less inhibited than smaller reflexes. The regression line with a slope of 0.84 is for reflexes of <40% Mmax.

not strictly standardized.

Effect of voluntary contraction. In Subject 1, strong contractions of quadriceps produced an increase in postsynaptic inhibition and a slight reduction in presynaptic inhibition (Fig 10.9).

DISCUSSION

Demonstration of the H-reflex in Soleus

The presence of an H-reflex in the subject studied was confirmed by the results of the classical physiological tests *viz.* vibration of TA reduced the reflex, the H-reflex/M response recruitment curves produced the expected patterns, the H-reflex was reduced with conditioning by CPN stimulation showing similar inhibitory time curves to those previously demonstrated (Iles & Roberts, unpublished; Tanaka,1974; Bergmans *et al.*,1978), and the inhibition was reduced by voluntary contraction of the plantarflexors. These experiments were performed as a means of practice, to develop skill in the technique of producing an H-reflex, and provided a basis for attempting to produce the reflex in quadriceps.

Demonstration of Quadriceps H-reflex

Quadriceps H-reflex was easy to elicit in all of the subjects in which it was attempted despite the general opinion that the H-reflex is difficult to, or cannot be elicited in the human quadriceps (e.g. Goldberg,1983). The presence of quadriceps H-reflex was confirmed by the tests performed *viz.* the different effects on quadriceps H-reflex of vibrating the flexors and extensors were in agreement with results obtained by Iles & Roberts (unpublished), the Ia inhibitory time curves for sciatic nerve stimulation were also similar to results obtained by Iles & Roberts (unpublished), and the Ib inhibitory time curve produced by tibial nerve stimulation was similar to those reported by Pierrot-Deseilligny *et al.* (1981)

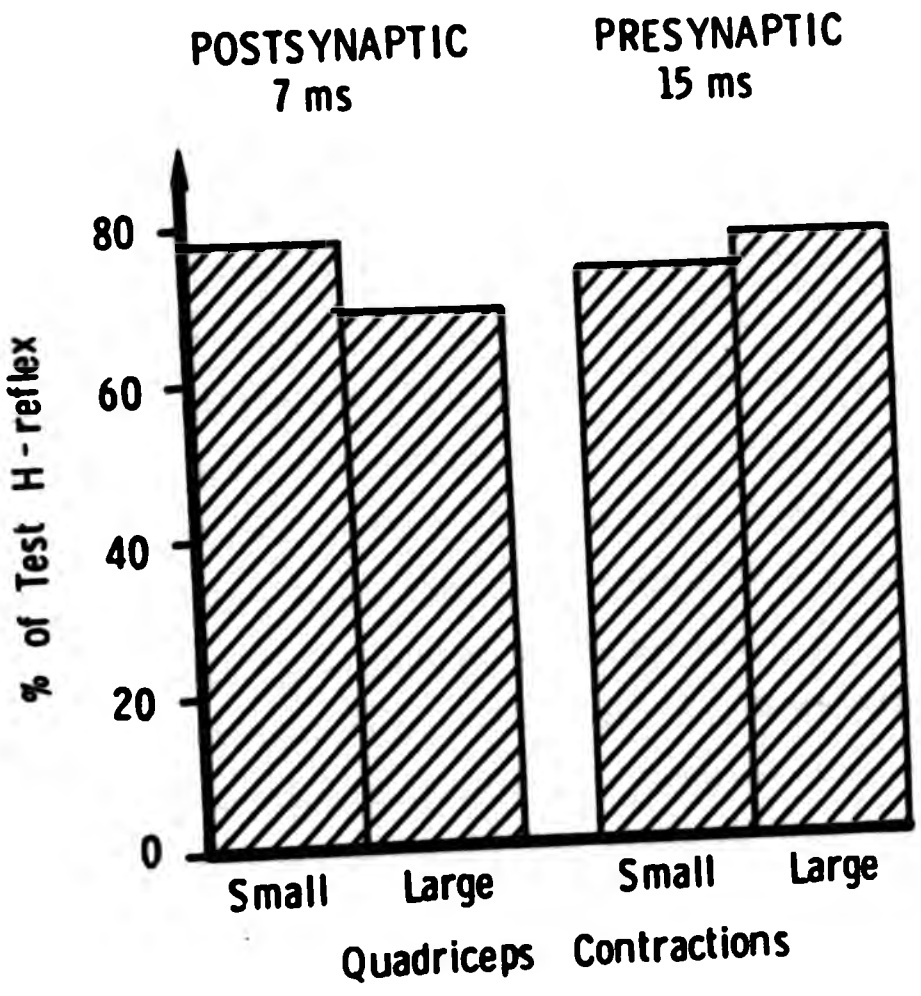


Figure 10.9. Effect of voluntary contraction on Ia inhibition of quadriceps H-reflex produced by sciatic nerve stimulation.

producing peak inhibition at similar latencies (9-12 ms).

The size of the test H-reflex influences the amount of Ib inhibition *viz.* less inhibition occurs with test H-reflexes of > 40% Mmax. This is probably because if the stimulus is large enough to make a high proportion of the motoneurone pool discharge, it must be supramaximal for many motoneurones (Renshaw,1940). These would then be 'immune' to inhibition and so the degree of inhibition would apparently be less. Similar results have been found using soleus H-reflex (J.F. Iles & T. Hale, personal communication) *viz.* with test reflexes > 40% Mmax (which are not achieved in many subjects), estimates of Ib inhibition are inaccurate. Results of experiments on Ib inhibition of quadriceps with joint infusion (Chapter 11) therefore only included test H-reflexes of < 30% Mmax. The reduction of presynaptic inhibition by a strong voluntary quadriceps contraction (in subject 1) would be expected due to increased excitation of the anterior horn cells (AHC) by descending impulses. The increase in postsynaptic inhibition, however, is difficult to explain. Perhaps co-contraction of the thigh muscles occurred with the stronger quadriceps contraction, producing some reciprocal (postsynaptic) inhibition of quadriceps by hamstrings Ia inhibitory "interneurones."

CONCLUSION

The H-reflex has been demonstrated in soleus and quadriceps and this was verified by classical physiological tests.

CHAPTER 11

INHIBITION OF QUADRICEPS H-REFLEX AND EXAMINATION OF THE CENTRAL NEURAL PATHWAY OF JOINT AFFERENT STIMULI IN NORMAL SUBJECTS WITH KNEE JOINT INFUSIONS

INTRODUCTION TO INVESTIGATIONS OF QUADRICEPS H-REFLEX INHIBITION

Dose Effect of Knee Joint Infusion on Quadriceps H-reflex Amplitude
Effect of Knee Joint Angle on Quadriceps H-reflex Inhibition
Central Neural Pathway of Knee Joint Afferent Stimuli

SUBJECTS

EXPERIMENT I: DOSE EFFECT OF KNEE JOINT INFUSION ON QUADRICEPS H-REFLEX AMPLITUDE

EXPERIMENT II: EFFECT OF KNEE JOINT INFUSION ON Ib INHIBITION OF QUADRICEPS H-REFLEX

EXPERIMENT III: EFFECT OF KNEE JOINT INFUSION ON CUTANEOUS FRA INHIBITION OF QUADRICEPS H-REFLEX

DISCUSSION OF THE CENTRAL NEURAL PATHWAY OF KNEE JOINT AFFERENT STIMULI

THE USE OF THE 'MODEL' OF NORMAL SUBJECTS WITH KNEE JOINT INFUSIONS TO STUDY QUADRICEPS H-REFLEX INHIBITION

INHIBITION OF QUADRICEPS H-REFLEX AND EXAMINATION OF THE CENTRAL NEURAL PATHWAY OF JOINT AFFERENT STIMULI IN NORMAL SUBJECTS WITH KNEE JOINT INFUSIONS

INTRODUCTION TO INVESTIGATIONS OF QUADRICEPS H-REFLEX INHIBITION

Dose Effect of Knee Joint Infusion on Quadriceps H-reflex Amlpitude

Quadriceps H-reflex was progressively decreased at rest by inflation of normal knees in a study which aimed to determine the minimal amount of fluid needed to produce inhibition (Spencer et al.,1984). The increase in intra-articular pressure with joint infusion is greater during joint use than at rest (Jayson & Dixon,1970b and Chapter 8). Spencer et al. (1984) recorded the H-reflex at rest and therefore only small degrees of inhibition would be expected from the small increase in intra-articular pressure It would therefore be helpful to know how much fluid will cause inhibition during joint use. Another reason for studying H-reflex inhibition during contraction as well as at rest is that voluntary contraction can change the strength of reflex actions (Iles, unpublished) and in some cases they can be reversed (Pierrot-Deseilligny et al., 1982). Therefore, conclusions drawn from results of experiments performed at rest cannot be directly applied to the clinical problem of inhibition of attempted maximal contractions. In the present studies, the effect of joint infusion on quadriceps H-reflex amplitude was examined both at rest (where possible) and during small contractions of quadriceps (Experiment I).

Effect of Knee Joint Angle on Quadriceps H-reflex Inhibition

It was intended to test whether inhibition of reflex activation of quadriceps was less with the knee in flexion than in extension, as observed with inhibition of voluntary activation in meniscectomy patients (Chapter 5). After studying 1 subject (Subject 1a), this experiment was abandoned because H-reflexes in flexion and extension could not be directly compared as the M responses were different.

This was probably due to movement at the hip joint underneath the stimulating electrode during flexion which produced different stimulation conditions. Also, the cannula became kinked when the knee was flexed and this caused problems with recording pressure when the knee was extended again for other experiments. However, this experiment did show the expected reduction in intra-articular pressure in flexion from that recorded in extension when the joint was infused (Stratford,1981; Levick,1983). Pressure measurements could not be made before infusion due to air in the cannula.

Central Neural Pathway of Knee Joint Afferent Stimuli

The reflex pathway from joint receptors is imperfectly understood (see Chapter 1, page 18) . Eccles & Lundberg (1959) suggested that joint, cutaneous and group II and III muscle afferents, which produced flexor facilitation and extensor inhibition when stimulated, shared a common pathway and termed them the flexor reflex afferents (FRA). These afferents as well as contributing to the FRA were also thought to have other separate reflex connections, or 'private' pathways. Activation of joint afferents by e.g. inflation of the knee joint or electrical stimulation of joint nerves in the cat, generally produces flexor facilitation and extensor inhibition (Andrew & Dodt, 1953; Skoglund, 1956; Ekholm et al.,1960; Beswick et al., 1955). Facilitation of reciprocal inhibition from joint stimulation needs to be studied in man because some FRA actions are mediated through Ia inhibitory interneurones in the cat (Fedina et al., 1975).

Lundberg etal. (1978) suggested that specific joint mechanoreceptors contribute to the FRA but also demonstrated in the cat that joint afferent activity facilitated transmission in reflex pathways from Ib afferents. They stated that the joint afferents have disynaptic connections with the Ib inhibitory interneurones. Baldissera et al. (1981) discussed the possible role of the convergence of Ib afferents with low threshold joint afferents on to

common inhibitory interneurones. It seems, therefore, that human joint afferent activity needs to be investigated and could prove to involve the FRA &/or Ib pathways and possibly some 'private' pathway.

In the present studies, electrical conditioning of quadriceps H-reflex was used to examine the central pathway of inhibition (see Chapter 9). If joint infusion increased the effect of a conditioning stimulus by spatial facilitation (Sherrington, In: Denny-Brown,1979d), a connection with that particular pathway would be indicated. The H-reflex was recorded at rest where possible to allow it to be examined entirely independantly of voluntary contraction, and also during a small voluntary contraction of quadriceps (maintained at a constant effort) for reasons already mentioned and to enable interpretation of changes in the H-reflex to be attributed to experimentally imposed influences.

Ia presynaptic inhibition. Stimulation of the FRA is known to reduce presynaptic inhibition in the cat (Lund et al.,1965) by convergence with the rubrospinal tract (Hongo et al.,1972). Brink et al. (1984) showed that electrical stimulation of joint afferents reduced Ia presynaptic inhibition in cats by recording dorsal root potentials evoked from various Ia afferents including some from quadriceps. The pathway of inhibition of presynaptic inhibition from the contralateral red nucleus is trisynaptic and the FRA converge with this pathway at the first order interneurone. Other descending influences on presynaptic inhibition are mediated through the pyramidal tract from the contralateral precentral motor area (Rudomín et al.,1983) and the ventral reticulospinal system from the ipsilateral reticular formation (Lundberg,1982).

In the present studies, Ia presynaptic inhibition in the presence of joint infusión was studied using prolonged muscle vibration in 3 subjects and sciatic nerve stimulation in 2 subjects (see Chapter 2 for methods). The results for both types of experiments were variable. The tests were performed during small

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In the present studies, Ia presynaptic inhibition in the presence of joint infusion was studied using prolonged muscle vibration in 3 subjects and sciatic nerve stimulation in 2 subjects (see Chapter 2 for methods). The results for both types of experiments were variable. The tests were performed during small

voluntary contractions which, as well as influencing the anterior horn cells (AHC) via the pathways mentioned above, also excites the AHC directly (i.e. postsynaptically). One of the problems with using muscle vibration to produce presynaptic inhibition is that the test reflexes cannot be alternated with conditioned ones as can be done with electrical conditioning. This often results in different sized M responses and therefore values obtained for inhibition are unreliable. Inhibition by sciatic nerve stimulation was only tested in 2 subjects as it is difficult to achieve. Their results were very varied but in agreement with Brink et al. (1984), presynaptic inhibition was generally reduced by joint stimulation (i.e. infusion). The conditions for testing the effects of joint afferents on Ia presynaptic inhibition in man are very critical so it was decided not to pursue this pathway any further in the present series of investigations and also because the main purpose was to examine facilitation of quadriceps inhibition.

Ia postsynaptic inhibition. Knee joint infusion did not appear to influence Ia postsynaptic inhibition but as the test reflex sizes differed (as with the Ia presynaptic experiments) the reliability of these results is questionable. This experiment was not repeated due to the same reasons given for abandoning the presynaptic tests. Care was taken to obtain similar test H-reflexes in later experiments which tested other pathways.

The methods used to produce Ia pre and postsynaptic inhibition, and the control experiments for investigating quadriceps inhibition are described in detail in chapters 2 and 10 respectively. The experiments of Ia inhibition using the infusion 'model' are not reported in full due to problems with the results as already described, and because of the small numbers studied.

Ib and cutaneous FRA inhibitory pathways. Experiments of the Ib and cutaneous FRA pathways are reported in detail in the present chapter (see Experiments II & III).

SUBJECTS

Three male and 5 female normal volunteers aged 27-40 years were studied. These were the same subjects who underwent the tests reported in chapters 8 & 10 and each subject has been referred to by the same number as before. The tests performed on each subject are shown in Table 11.1.

EXPERIMENT I: DOSE EFFECT OF KNEE JOINT INFUSION ON QUADRICEPS H-REFLEX AMPLITUDE

Introduction

The effect of knee joint infusion on the size of quadriceps H-reflex was examined during a small contraction of quadriceps in all subjects and at rest in 2 subjects (Subjects 2 & 3).

Methods

Quadriceps H-reflex stimulation and recording, infusion of the knee with saline, and recording of intra-articular pressure were performed as described in Chapter 2. After each increment of infusion (usually 10ml), intra-articular pressure was recorded at rest and then during H-reflex tests during contraction. At each volume, 16 reflexes were recorded and averaged by the computer.

Results

As the volume of infusion was increased, the size of the H-reflex decreased in 6 of the 8 subjects (Fig 11.1.). The H-reflex did not decrease with infusion in Subjects 4 & 7 (vide infra). H-reflex size has been plotted against pressure as this is probably more accurate than plotting it against volume as, for example, fluid loss through leakage would reduce pressure. This must have occurred in Subjects 2a and 4 in whom a second cannula was inserted during the same experiment (Chapter 8). H-reflex amplitude varied at each volume because the M responses also varied. Therefore, only H-

			EXPERIMENTS			
			I	II		III
			Dose Response	Ib Inhibition		FRA Inhibition
	Subject		Contr	Rest		
1.	♀	27yrs a	+			
		b	+		+	
		c			+	+
2.	♂	36yrs b	+	+	+	
		c				+
3.	♂	29yrs	+	+	+	
4.	♀	36yrs	+			
5.	♀	31yrs	+			
6.	♂	37yrs	+		+	
7.	♀	30yrs	+		+	
8.	♀	40yrs	+		+	

Table 11.1. Experiments of reflex activation of quadriceps performed on normal subjects with knee joint infusions.

reflexes with similar M responses were included in the graphs, and the ranges of sizes for inclusion are indicated in the figure legends. Although the H-reflex was reduced with infusion in Subjects 5 & 8, their results were not plotted because they were too varied.

In Subject 4, insertion of the cannula (for the second time - see Chapter 8,page 85) caused pain and the H-reflex was reduced before any saline was infused, probably due to reduced voluntary effort during H-reflex recording because of pain. In Subject 7, the submaximal voluntary contractions (15-20%) required for H-reflex recording were inhibited after infusion of 40ml of saline, to the extent that the subject could not tell whether or not she was contracting her quadriceps. Consequently, intra-articular pressure was not increased enough to inhibit the H-reflex at the larger volumes but there was some H-reflex inhibition at 20-30ml before the subject was aware of any inhibition of voluntary contractions. In Subjects 2 & 3 H-reflexes could be elicited at rest and were smaller than during contraction at all volumes as contraction facilitated the H-reflex even when inhibition occurred.

It is not possible to state the minimum volume at which reflex activation of quadriceps is inhibited because the results were so variable and tests would need to be performed after smaller increments of infusion (e.g. 5ml).

Discussion

The relationship between volume of infusion and H-reflex inhibition was almost linear over the range of volumes infused. Subjects 2 and 3 in the present series showed less inhibition at rest than during contraction and this was probably due to the greater intra-articular pressures produced during contraction. It is important to consider whether the bilateral voluntary contractions achieved a constant effort of contraction in the experimental limb. As knee joint infusion progressively inhibited quadriceps contractions, subjects reported a feeling of difficulty in

controlling contractions. This feeling was generally associated with an increased effort to contract the clearly weakened muscle. Since greater effort would increase the amplitude of the H-reflex, and therefore decrease the effectiveness of the different pathways of inhibition tested, increased effort cannot be responsible for the effects observed during infusion as it would diminish them.

Although Spencer et al. (1984) have also demonstrated that knee joint infusion inhibits quadriceps H-reflex, the accuracy of their data is questionable because they attempted to record the H-reflex without an M response where possible instead of using the M-response to monitor the stimulus size. In an illustrated example of results from 1 of their subjects in which the M response was recorded, the M response did change in size. The H-reflex amplitude with a 60 ml effusion was reported to be reduced by comparison with H-reflexes at 30 ml and 0 ml but the M-response at 60 ml was about 50% smaller than at the other 2 volumes. This casts some doubt on the validity of the percentages of inhibition reported for H-reflexes recorded without M responses . Spencer et al. (1984) stated that "there was no effect of saline introduction on the M-response amplitude" despite the obvious change in their illustrated example. This was unlikely to be due to the infusion anyway, and must have been due to different stimulus conditions. In the present studies, all H-reflexes were recorded with a small M response, and where changes in the H-reflex were being observed, only H-reflexes with similar M responses were compared (as defined in Fig 11.1.).

Small volumes of infusion (as low as 10 ml) can inhibit reflex activation of quadriceps. There was also a suggestion of the same effect on voluntary activation (Chapter 8) but this was only studied in 2 subjects. These findings have important clinical implications as the small volumes of infusion were not clinically obvious until about 40 ml (in the smallest knee). Perhaps when joint effusion is suspected in patients presenting with other joint

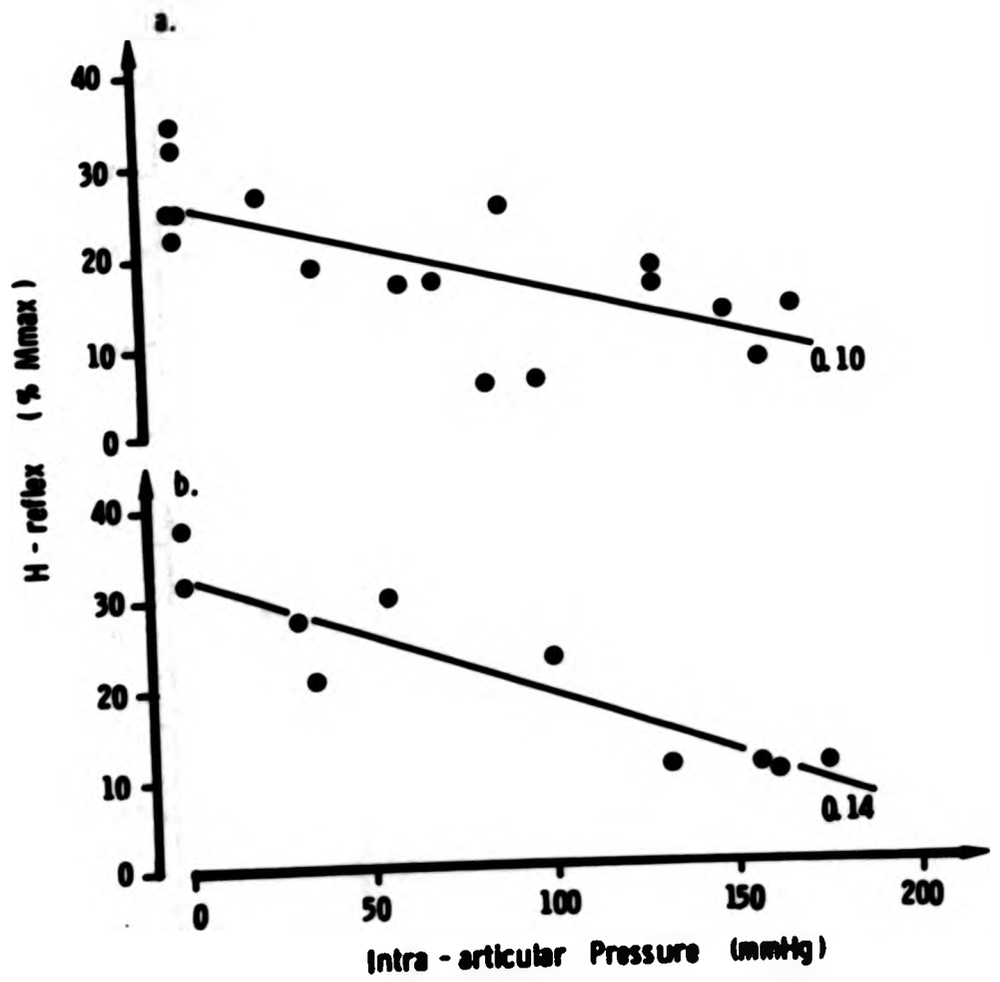

Figure 11.1.a+b Relationship between quadriceps H-reflex amplitude and intra-articular pressure during contraction.

a. Subject 6 (points for M responses 11-48% Mmax).

b. Subject 1a (M = 12-30% Mmax)

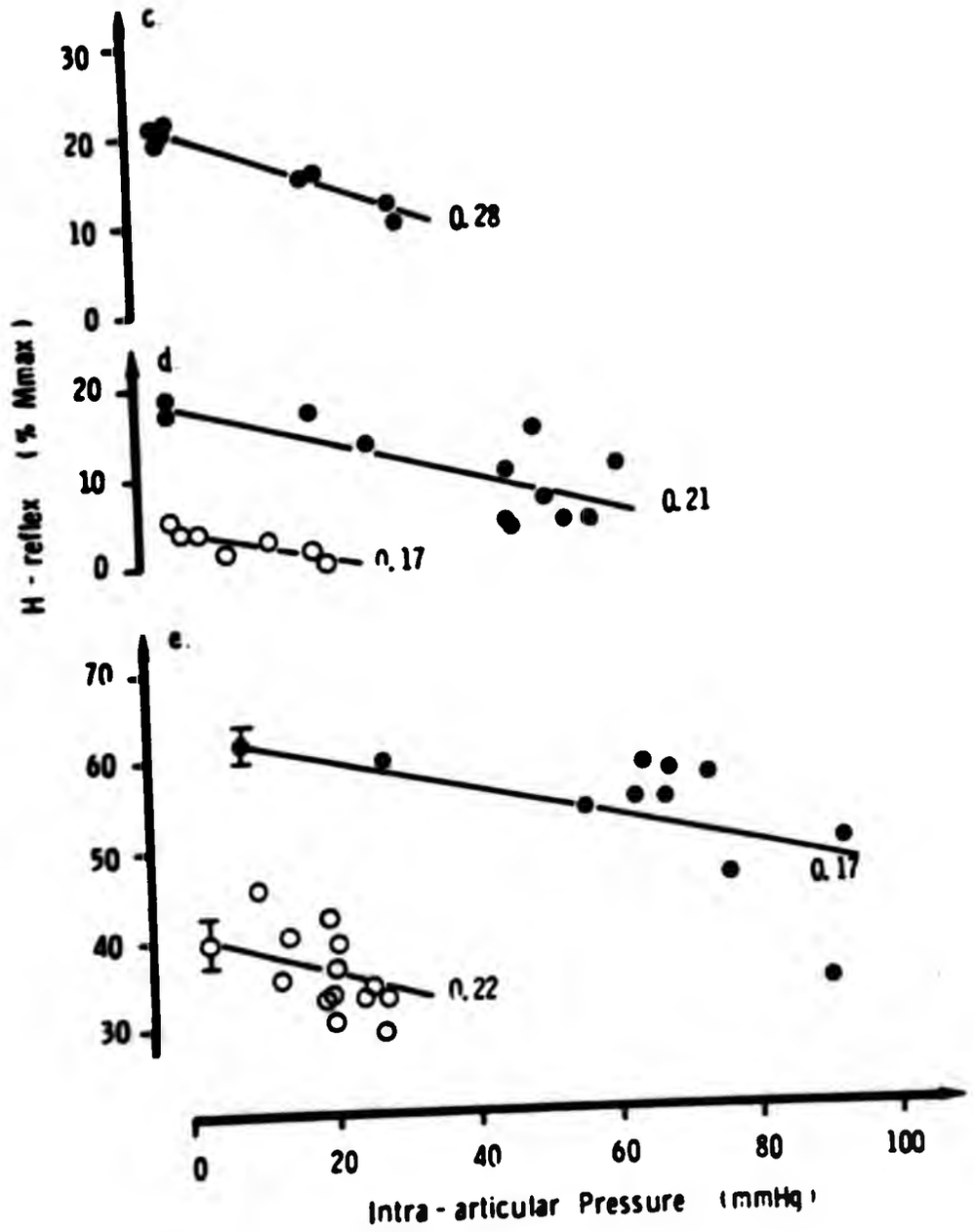

Figure 11.1.c-e Relationship between quadriceps H-reflex amplitude and intra-articular pressure (● during contraction; O at rest). c. Subject 1b (M = 1.8-12.9% Mmax) d. Subject 3 (during contraction, all data used; at rest M = 2.8-11.5% Mmax) e. Subject 2b (all data)

symptoms or quadriceps weakness (with known joint disease or history of trauma), measures should be taken to reduce effusion e.g. by oral anti-inflammatory medication. When effusion is apparent, it should certainly not go untreated.

Conclusions

1) Small knee joint infusions which are not clinically apparent (<30ml) can cause inhibition of reflex activation of quadriceps.

2) Inhibition of reflex activation of quadriceps increases as the volume of infusion and intra-articular pressure increase and is greater during voluntary contraction.

EXPERIMENT II: EFFECT OF KNEE JOINT INFUSION ON Ib INHIBITION OF QUADRICEPS H-REFLEX

Introduction

Lundberg et al. (1978) demonstrated that joint afferent activity produced by electrical stimulation, facilitated transmission in reflex pathways from Ib afferents in the cat. Quadriceps H-reflex can be inhibited via Ib pathways from Golgi tendon organs by stimulation of the tibial nerve in man (Pierrot-Deseilligny, et al., 1981).

The present study examined the effect of knee joint infusion on Ib inhibition. An increase in Ib inhibition with infusion would indicate convergence of joint afferents with Ib interneurones. Six of the normal subjects in Table 11.1. were studied (3 male, 3 female).

Methods

Quadriceps H-reflex stimulation and recording, infusion of the knee joint, recording of intra-articular pressure, and tibial nerve stimulation were performed as described in Chapter 2. The intensity of H-reflex stimulation was between 1.05 and 1.2 times

aplha threshold. In Subject 2c, nerve stimulation was randomized by a BBC Model B computer (used to control the Digitimer D4030). The latency of peak Ib inhibition seen in the control experiments was used to condition the H-reflex with and without an infusion. Inhibitory time curves were plotted for Subjects 1 and 2 (see Chapter 10, page 102), and in the other subjects control tests sought the peak latency over a range of 8-14ms. At least 3 tests of Ib inhibition were performed at different volumes of infusion. The test stimulus intensity was adjusted at each volume to ensure that similar H-reflexes were obtained to allow comparisons of inhibition at the different volumes. This experiment would have ideally also been performed at rest because Ib inhibition is greater at rest than during contraction. This was only possible in Subject 3 as the reflex was either too small or was abolished with infusion in the other 2 subjects in whom the reflex was present at rest.

Results

There was a clear increase in Ib inhibition with knee joint infusion in 4 subjects (2c,3,6 & 8). Before infusion, peak Ib inhibition of quadriceps H-reflex occurred at intervals of 8-13ms (median = 12ms) in the 6 subjects studied. The relationship between test H-reflex size and Ib inhibition (conditioned reflex expressed as % Mmax) has been plotted for test reflexes < 40% Mmax (see Chapter 10, page 102). The results for Subject 2c could not be expressed in this way as the test reflexes were too large (vide infra).

Subject 2c. The effect of increasing intra-articular pressure on Ib inhibition is illustrated in Fig 11.2. Tests were performed during contractions in this subject. Ib inhibition is expressed as the percentage reduction from the test H-reflex amplitude. Similarly sized test reflexes were obtained at the different volumes allowing reliable comparisons to be made between the degrees of inhibition observed at each volume.

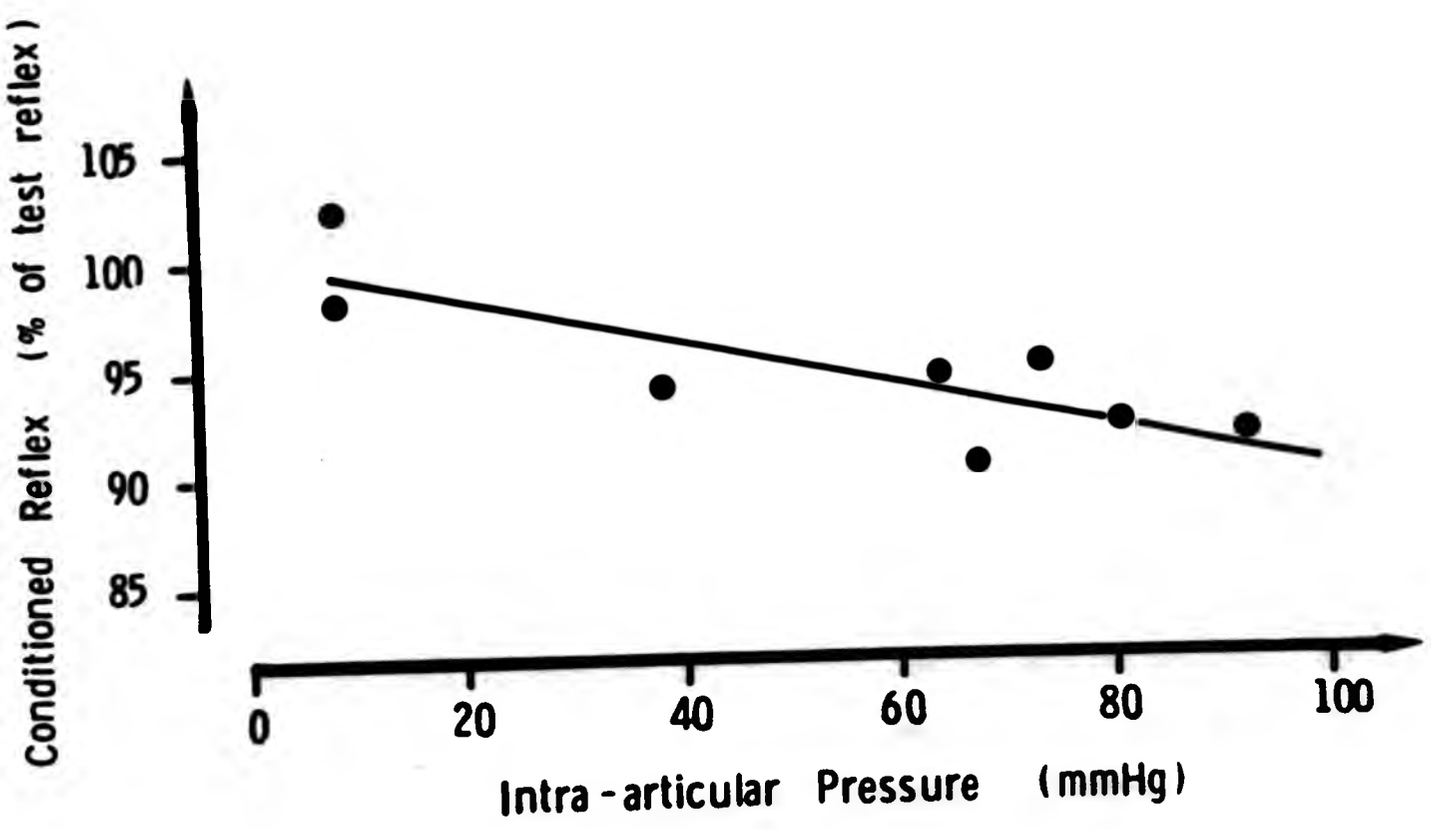

Figure 11.2. Relationship between the strength of Ib inhibition of quadriceps H-reflex and intra-articular pressure (Subject 2c).

Subject 3. Tests were performed at rest and during contraction before infusion but the H-reflex was abolished with a 70ml infusion at rest. The relationship between test reflex size and Ib inhibition is shown at different intra-articular pressures in Fig 11.3.a. During tests performed before infusion, pressure was not recorded due to air in the cannula but it was probably slightly positive because of this (approximately 8mmHg). Pressure before infusion is taken to be "zero" on the graph. The slope of the regression line at "zero" pressure was 0.96, and the slope of the line for results at 56 mmHg (54-57.9) was 0.77, demonstrating more inhibition at the higher pressures. There was no further increase in inhibition after 70-100ml infusion which may have been due to occlusion (see 'Discussion', page 115). Aspiration of fluid reduced both pressure and Ib inhibition.

Subject 6. Tests were performed during contraction and the results are illustrated in Fig 11.3.c. at 3 different pressures. Test reflexes of < 28% M max are included in the graph. At "zero" pressure the slope of the regression line was 1.04 demonstrating facilitation of the H-reflex by tibial nerve stimulation. At pressures of 70-87mmHg the slope was 0.88 and at 130-168 mmHg the slope was 0.80 demonstrating increasing Ib inhibition with increasing pressure (i.e. infusion).

Subject 8. The results for test reflexes of < 30% M max are included in Fig 11.3.b. Ib inhibition increased with joint infusion viz. at "zero" pressure the slope of the regression line was 0.92 and at pressures of 43-96mmHg the slope was 0.80.

Subjects 1 & 7. Tests were not performed before infusion in Subject 1 so results with an infusion cannot be evaluated. Results at 2 different ranges of intra-articular pressure show large Ib inhibition but the test reflexes at the 2 pressures show little overlap and do not therefore allow a comparison to be made. Knee joint infusion (maximum 50ml) did not produce a clear effect on H-reflex amplitude

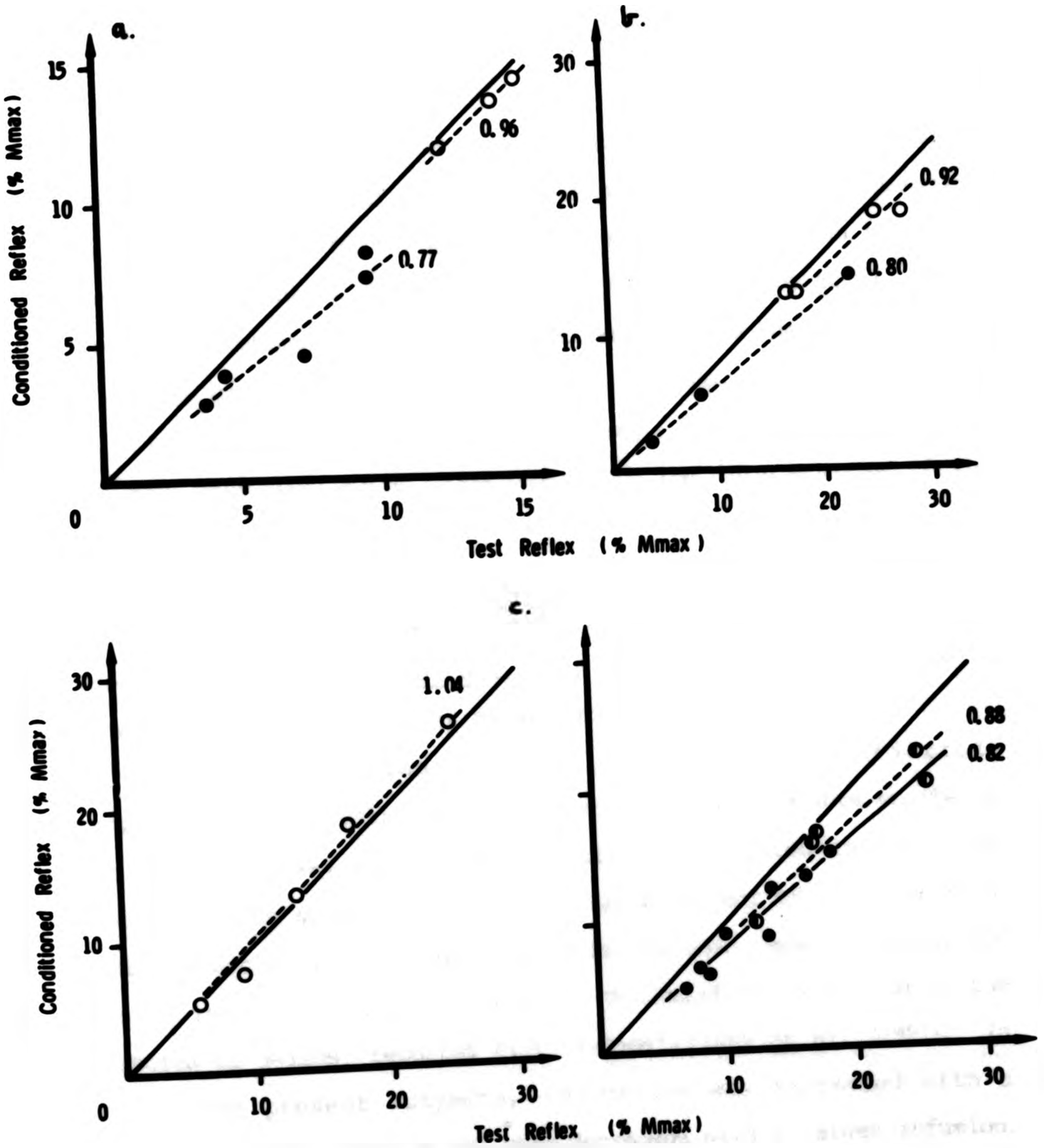

Figure 11.3. Effect of joint infusion on the relationship between Ib inhibition of quadriceps H-reflex and test H-reflex size.

a. Subject 3 O "zero" pressure, ● 56mmHg;

b. Subject 8 O "zero" pressure, ● 43-96 mmHg;

c. Subject 6 O "zero" pressure, ● 70-87 mmHg ,
● 130-168 mmHg

which might explain the small increase in Ib inhibition (vide infra).

Discussion

Joint afferents in the cat have an excitatory effect at an interneuronal level on Ib inhibitory pathways, thus influencing tension regulation from Golgi tendon organs and playing an important role in motor control (Lundberg et al.,1978). Lundberg et al. (1978) electrically stimulated the posterior nerve of the knee joint in the low spinal cat (at about 1.5 times alpha threshold) and demonstrated facilitation of the Ib inhibitory pathway. They showed that joint afferents have disynaptic connections with the interneurones of the Ib inhibitory pathway. Other authors have also discussed the possible role of the convergence of joint afferents and Ib afferents onto common interneurones from evidence obtained from cat experiments (Baldissera et al.,1981; Brink et al.,1984). The Ib pathways can be disynaptic or trisynaptic (Lundberg,1978).

Ib inhibition is known to occur in man (Pierrot-Deseilligny et al,1981) but the effect of joint afferents on the Ib inhibitory pathway has not previously been studied in man. The present results showed a clear facilitatory effect of joint infusion on Ib inhibition in 4 of the 6 subjects studied. Peak Ib inhibition before infusion occurred between 8-13ms which is similar to the latencies of 9-12ms reported Pierrot-Deseilligny et al. (1981). In some of the present subjects, inhibition was increased with a relatively small infusion and then decreased with a larger infusion. This could have been due to the larger volume producing too much activity to allow spatial facilitation causing occlusion (Sherrington, In: Denny-Brown,1979d).

In the subjects in whom the very clear effects of infusion on Ib inhibition were demonstrated, the H-reflex was clearly depressed with infusion and intra-articular pressure was increased. In some cases it was shown that the increased Ib inhibition could not have been produced indirectly by changes in test reflex amplitude.

The more varied results obtained for other subjects were probably due to less effective infusions i.e. increased pressure and H-reflex depression did not occur or were small, possibly due to leakage of fluid from the joint. Inhibition of voluntary contractions (about 20% max which were required for recording the H-reflex) at large volumes may have accounted for some of the absence of increased pressure, and therefore the lack of increased inhibition expected. Reduction of Ib inhibition occurred after aspiration to below pre-infusion levels in some subjects and facilitation of the H-reflex was also seen. Perhaps once occlusion has occurred, the Ib inhibitory pathway is not easily activated again after aspiration or that some kind of hysteresis in the behavior of the receptors occurs. Another possibility is that the reduction of inhibition of voluntary contraction after aspiration might produce greater facilitation of the AHC than before inhibition and occlusion occurred. It is difficult, however, to explain the abolition of Ib inhibition and the H-reflex facilitation seen at 0ml after insertion of the cannula before infusion in Subject 6.

Ib inhibition was greater at rest than during contraction (subjects 2c, 3 & 6). This would be expected as the facilitatory effect of voluntary contraction on the H-reflex would reduce the effect of inhibition. Ib inhibitory interneurones are influenced (both facilitated and inhibited) by descending pathways (Lundberg *et al.*,1978) so perhaps Ib inhibition is reduced *via* one of these pathways (e.g. dorsal reticulo-spinal system) during contraction. Any increased effort by the subject in a attempt to contract the weakening quadriceps would counteract the observed effect of joint infusion on inhibition.

Conclusion

Ib inhibition of quadriceps H-reflex is increased by knee joint infusion indicating convergence of the Ib and joint afferent pathways.

EXPERIMENT III: EFFECT OF KNEE JOINT INFUSION ON CUTANEOUS FRA INHIBITION OF QUADRICEPS H-REFLEX

Introduction

Stimulation of the sural nerve at rest produces a flexor withdrawal response in man i.e. facilitation of the flexors and inhibition of the extensors (Hugon, 1969 & 1973). It may be possible that sural nerve stimulation also causes inhibition of the extensors directly. This putative FRA action of sural nerve stimulation was used in the present experiment to investigate possible convergence of knee joint afferents with cutaneous fibres of the FRA. The effect of sural nerve stimulation on quadriceps H-reflex inhibition, produced by knee joint infusion, was studied in two of the normal subjects included in Table 11.1.

Methods

Quadriceps H-reflex stimulation and recording, infusion of the knee joint, and recording of intra-articular pressure were performed as described in Chapter 2. The sural nerve was stimulated inferior to the lateral malleolus and the anode was placed on the dorso-lateral aspect of the foot. The latency of sural nerve stimulation was 40ms and 10 shocks were delivered at a frequency of 300Hz. The intensity was low so as to produce a tactile sensation and not a painful sensation, and the shocks were randomized. Tests were performed during contraction in both subjects.

Results

Subject lc. There was no clear effect of joint infusion on cutaneous FRA inhibition in this subject. Test H-reflexes were reduced with infusion and became very small at relatively low intra-articular pressures (compared to records in other subjects, see Experiment I). It may be possible that occlusion could have occurred.

Subject 2c. FRA inhibition appeared to increase as pressure increased (Fig 11.4.). The results were not expressed as the relationship between test reflex size and FRA inhibition (as used for Ib inhibition results) because the test reflexes were too large.

Discussion

Although an effect of joint infusion on FRA inhibition was only seen in 1 of the 2 subjects studied it seems possible that joint afferents may contribute to the cutaneous FRA.

The sural nerve is comprised of large myelinated A alpha and A beta fibres (Group II of Lloyd,1943), small myelinated A delta fibres (Group III) and unmyelinated C fibres (Group IV). This composition was defined in man by O'Sullivan & Swallow (1968). Hugon (1973) demonstrated that increasing intensities of sural nerve stimulation excited the Group II afferents (tactile sensation) at about 0.7 times perceptual threshold, then the Group III's and lastly the Group IV afferents (pain) at about 3 times perceptual threshold (which would be unbearably strong stimulation). He concluded that only the noxious stimuli produced a flexor withdrawal response and that tactile stimulation produced a reflex which is related to exteroceptive control of foot posture. However, Kanda & Sato (1983) have reported flexor reflex effects by sural nerve stimulation at low intensities (1.5 times perceptual threshold) indicating that the larger fibres also contribute to the FRA. This was also confirmed by activity being recorded in hamstrings during low intensity sural stimulation in one of the present subjects.

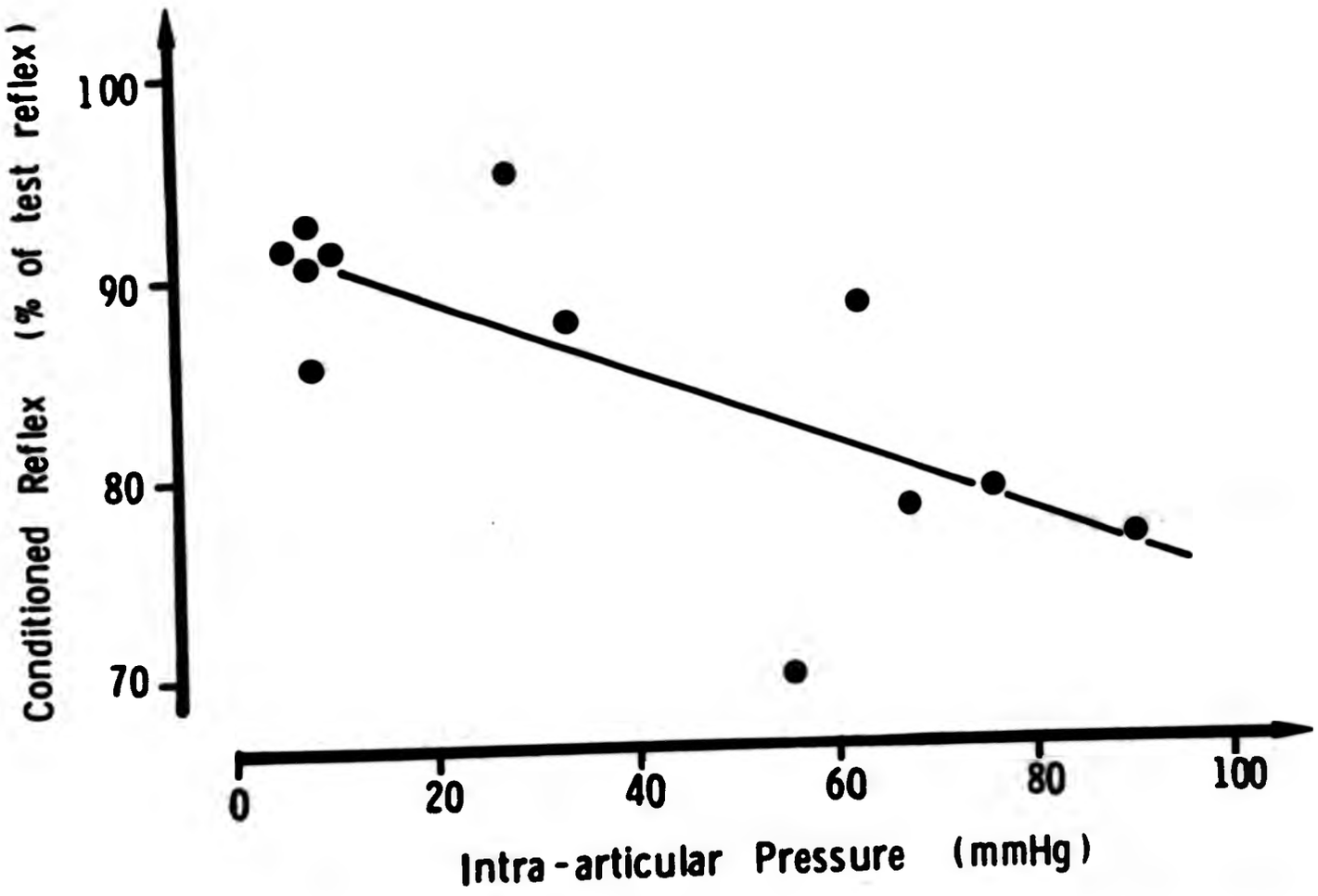

Figure 11.4. Relationship between strength of inhibition (of quadriceps H-reflex) from the sural nerve and intra-articular pressure (Subject 2c).

Lundberg et al. (1978) observed the effects of stimulating the posterior nerve of the knee and suggested that low intensity stimulation of joint afferents produced flexor effects via Ib pathways (and possibly other interneuronal pathways) and that higher intensities (but < 2 times alpha threshold) produced effects via the FRA pathways (probably because they require more spatial summation). They also stated:

> "....it does not seem likely that the contributory afferents in the joint nerve have nociceptive function".

Conclusion

It seems likely that joint afferents may converge with cutaneous fibres of the FRA pathways.

DISCUSSION OF THE CENTRAL NEURAL PATHWAY OF KNEE JOINT AFFERENT STIMULI

The present investigations did not demonstrate any clear facilitation of reciprocal inhibition by joint stimulation but they did suggest that some of the inhibitory afferent stimuli are mediated through interneurones of the Ib and cutaneous FRA pathways. The precise levels of the connections are unclear, and some authors have suggested that the different peripheral and descending pathways converge on to a common inhibitory interneurone as shown in Fig 11.5. (e.g. Lundberg et al.,1978). Only the peripheral pathways are included in Fig 11.5. for clarity.

Pierrot-Deseilligny et al. (1982) demonstrated interaction between cutaneous (not necessarily FRA) and Ib pathways. Cutaneous stimulation of the sole of the foot reduced Ib inhibition of quadriceps H-reflex at rest, and the effect was reversed during contraction. The possibility that spatial facilitation or occlusion between inhibition from the tibial and sural nerves might occur

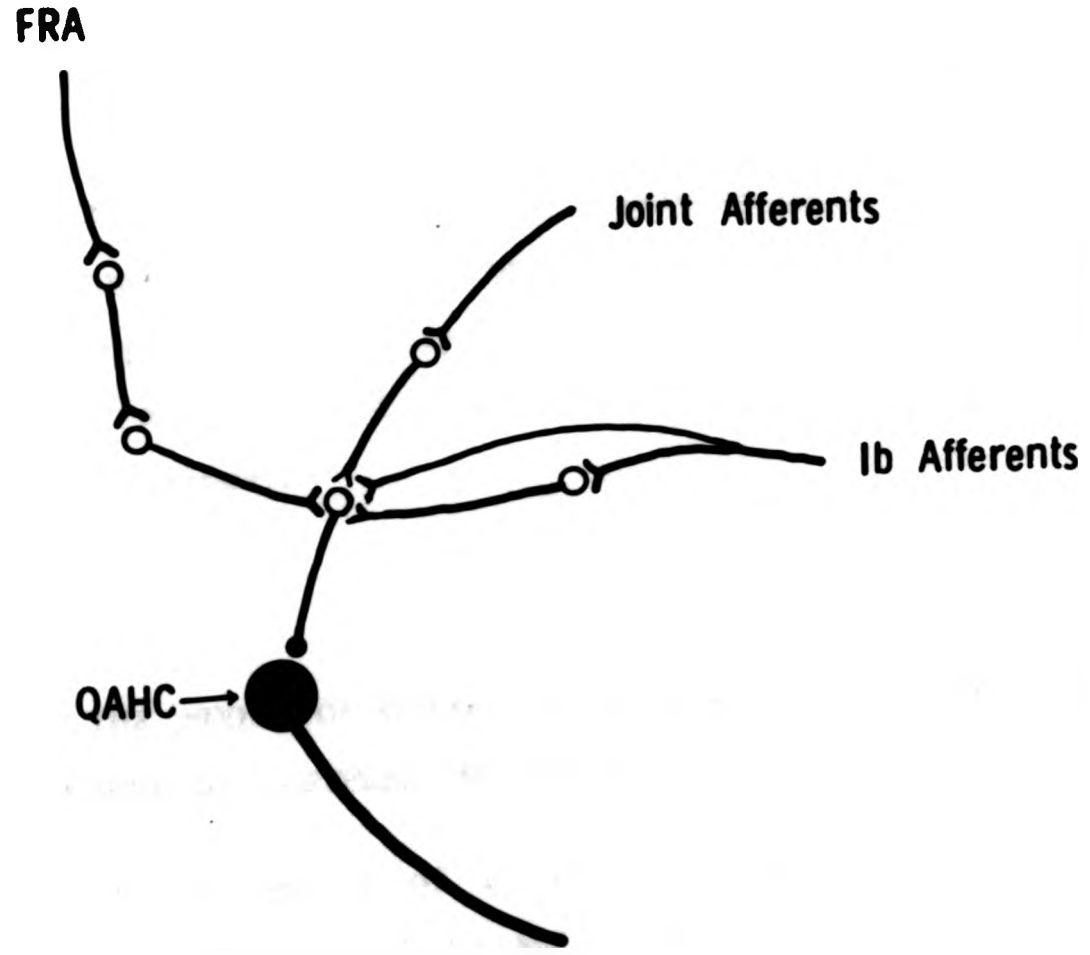

Figure 11.5. Convergence of different peripheral afferent pathwawys on to a common inhibitory interneurone as suggested by other authors (e.g. Lundberg et al., 1978).

during contraction was examined in Subject lc in a control experiment performed 4 days after the present experiment. The sural nerve was stimulated *before* each H-reflex and the tibial nerve was used to condition on each alternate shock. There was no evidence for interaction between the cutaneous FRA and Ib pathways in this experiment. This suggests that the Ib and cutaneous FRA pathways do not share common interneurones. The interaction between cutaneous and Ib pathways observed by Pierrot-Deseilligny et al. (1982) may be dependent on 'local sign' i.e. it may depend on which area of skin is stimulated.

It is proposed that joint afferents have connections with the Ib and cutaneous FRA inhibitory interneurones, and that they may also have a 'private' pathway (Fig 11.6.). Ib pathways can be tri or disynaptic and it is not known how many synapses are involved in the cutaneous pathway.

THE USE OF THE 'MODEL' OF NORMAL SUBJECTS WITH KNEE JOINT INFUSIONS TO STUDY QUADRICEPS H-REFLEX INHIBITION

The procedure of infusing the knee with saline was tolerated well in all subjects and there were no after effects. In some cases, fluid remained in or around the joint after the cannula had been removed but swelling had resolved within a few hours.

The absence of pain during the present experiments on normal subjects allowed the effects of joint afferents on quadriceps activation to be studied without the influence of nocioceptive activity.

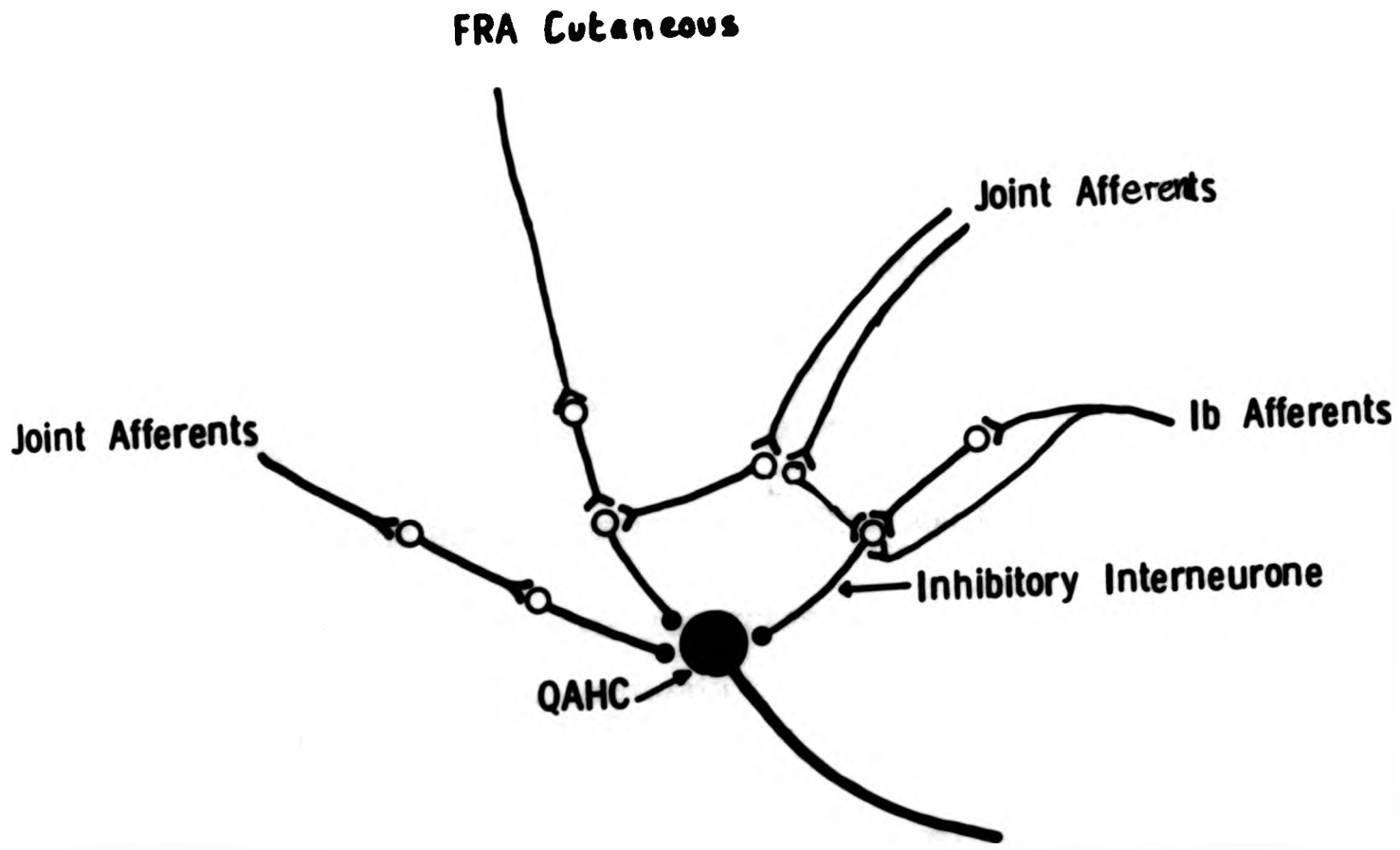

Figure 11.6. Propsoed connections of joint afferents with cutaneous and Ib inhibitory interneurones. 'Private' joint afferent pathways may possibly also exist.

CHAPTER 12

DISCUSSION OF REFLEX INHIBITION OF QUADRICEPS IN THE PRESENCE OF KNEE JOINT DAMAGE, AND CLINICAL IMPLICATIONS OF THE RESULTS OF THE PRESENT INVESTIGATIONS

SEVERITY AND DURATION OF REFLEX INHIBITION OF QUADRICEPS AFTER MENISCECTOMY

METHODS OF REDUCING REFLEX INHIBITION OF QUADRICEPS

- Local Anaesthesia
- Isometric Quadriceps Contractions Performed in Flexion
- Transcutaneous Nerve Stimulation
- Aspiration of Knee Joint Effusion

LACK OF ASSOCIATION BETWEEN REFLEX INHIBITION AND PAIN

CAUSES OF REFLEX INHIBITION OF QUADRICEPS

- Possible Role of Ischaemia
- Role of Knee Joint Effusion
- Role of Peri-articular Stimuli

CENTRAL NEURAL PATHWAY OF KNEE JOINT AFFERENT STIMULI

REFLEX INHIBITION OF QUADRICEPS IN TRAUMATOLOGY AND RHEUMATOLOGY

TOPICS FOR FURTHER STUDY

- Per-operative Bupivacaine for Post-operative Analgesia
- Effectiveness of Isometric Quadriceps Exercises Performed in Flexion
- Role of Intra-articular Pressure in the Knee Flexion Phenomenon
- Transcutaneous Nerve Stimulation
- Reflex Inhibition of Quadriceps due to Small Infusions
- Non-steroidal Anti-inflammatory Drug (NSAID) Trial
- Muscle Stimulation to Prevent Weakness and Wasting Caused by Reflex Inhibition
- Effect of Capsular Suturing on Quadriceps Activation
- Effect of Haemarthrosis on Quadriceps Activation

CONCLUSIONS

DISCUSSION OF REFLEX INHIBITION OF QUADRICEPS IN THE PRESENCE OF KNEE JOINT DAMAGE AND CLINICAL IMPLICATIONS OF THE RESULTS OF THE PRESENT INVESTIGATIONS

SEVERITY AND DURATION OF REFLEX INHIBITION OF QUADRICEPS AFTER MENISCECTOMY

Reflex inhibition of quadriceps activation occurs rapidly after arthrotomy and meniscectomy and is strikingly severe for at least 3 days (typically 70-80%). During this early post-operative period therefore, the straight leg quadriceps contractions routinely practised by patients are ineffective at strengthening the muscle as only 20-30% of the muscle is being activated. One might argue that perhaps inhibition is necessary to allow the joint to rest in the early stages, and that persistence of inhibition at 2 weeks (at 37%) may still be "useful" protection in everyday activities. Such 'protection' is counterproductive when performing therapeutic exercise and is not necessary when exercises are performed in a controlled isometric manner. It is therefore necessary to try to reduce reflex inhibition of quadriceps in order to permit effective therapeutic exercise.

The present series of meniscectomy patients were not studied beyond 2 weeks after surgery so it is not known how long the quadriceps inhibition lasts. Arvidsson et al. (1981) found that of 87 patients who had undergone anterior cruciate ligament repair, 28% still had significantly weak quadriceps 5-10 years (mean 7.8) post-surgery. They suggested that the persisting weakness occurred because the patients did not continue with their exercises for long enough, and that indefinite training for the majority of patients was necessary. This may have been true in some cases but some of their patients took part in regular sports (involving the legs and therefore, in effect, training the quadriceps) and still their quadriceps were weak. Perhaps their weakness was caused by reflex inhibition due to continuing joint problems (see Chapter 1, page 13).

METHODS OF REDUCING REFLEX INHIBITION OF QUADRICEPS

Local Anaesthesia

Reflex inhibition of quadriceps was temporarily reduced (4-5 hours) after meniscectomy by per-operative infiltration of the knee with bupivacaine (Chapter 3). Although this allows the effective performance of quadriceps contractions the effect is too brief to be clinically useful for therapeutic exercises. Per-operative bupivacaine might be useful as a post-operative analgesic because although the effect would only last for a few hours, the period of severe pain is also short.

Infiltration of the pre-patellar scar, in a patient described in the 'APPENDIX', also reduced reflex inhibition of quadriceps activation in extension and was again temporary. Although there was no control against which to assess the effect in this patient, these examples indicate that the anaesthetic must be infiltrated into the area of the joint from which the afferent stimuli originate, in order to reduce the inhibition (Chapter 3 & 'APPENDIX').

Quadriceps Contractions Performed in Flexion

Isometric quadriceps contractions are inhibited less when they are performed with the knee in a flexed position, both after meniscectomy (Chapter 5) and in the presence of peri-articular pathology ('APPENDIX'). The mechanism of this phenomenon is not known. While the cause of reflex inhibition is still present, the reduced inhibition during exercises in flexion does not appear to influence subsequent levels of activation in extension ('APPENDIX') but this has not been evaluated in post-meniscectomy patients.

The effect of joint angle on reflex (involuntary) activation of quadriceps might indicate the most suitable joint position for immobilization as well as for exercise ('APPENDIX').

Transcutaneous Nerve Stimulation (TNS)

In an attempt to find a way of reducing post-meniscectomy reflex inhibition which was longer lasting than the effect of bupivacaine, and more continuous than contracting the quadriceps with the knee flexed, the effect of TNS on inhibition was studied in post-meniscectomy patients (Chapter 6). Quadriceps inhibition was reduced in all of the patients who received 'active' TNS but although the reduction was statistically significant it was probably not clinically significant. Experimentation with the stimulation parameters might produce more relief of inhibition but such a study would be very extensive and was not feasable in the present series. Furthermore, if any treatment method is to become widely used by physiotherapists, its success in releiving inhibition should not be too dependent upon specific parameters, as these may not be adhered to or the method may be ignored if it is unnecessarily complicated.

When TNS is used to relieve knee pain, it should be remembered that despite any subsequent improvement of function, severe reflex inhibition may still be present (see page 131). In such cases, heavy weight lifting or weight-bearing exercises might cause further joint damage due to abnormal loading of the knee which occurs with quadriceps weakness (see Chapter 1, page 13).

Aspiration of Knee Joint Effusion

The severity of post-meniscectomy quadriceps inhibition in the presence of a knee joint effusion can be reduced by aspirating the effusion (Chapter 7). Effusion does not always occur after meniscectomy but it clearly contributes to inhibition and should always be treated e.g. by aspiration, anti-inflammatory medication, ice in elevation. It is not known how small an effusion will cause quadriceps inhibition, but volumes of fluid which do not cause obvious joint swelling (<30ml) can cause inhibition (Chapters 8 & 11). Therefore obvious effusions should certainly not be tolerated.

LACK OF ASSOCIATION BETWEEN REFLEX INHIBITION OF QUADRICEPS AND PAIN

The inability of patients to contract their quadriceps after meniscectomy is not due to pain but to reflex inhibition of muscle activation caused by afferent stimuli from the damaged knee joint. This is contrary to the belief of Eriksson (1981) and Smith et al. (1983). In the present studies, a lack of association between reflex inhibition and pain was seen after 24 hours following meniscectomy when some patients had little or no pain despite having severe inhibition (Chapter 3). In the early stages, this dissociation was demonstrated by the dose-related effect of bupivacaine on inhibition, while pain was reduced with both doses (Chapter 3). The patients included in the TNS study (Chapter 6) provided further examples of the dissociation between inhibition and pain viz. inhibition was severe in all patients both before and after TNS but some patients had only mild pain and others had no pain at all.

During inflation of normal knees the 8 subjects studied in Chapters 8 & 11 were unable to produce maximal quadriceps contractions. With large volumes of fluid the subjects no longer had 'control' over their quadriceps and the sensation experienced in the knee joint was of tightness and not of pain.

The patient with the pre-patellar scar ('APPENDIX') achieved full activation in flexion (i.e. equal activation to that in the uninjured limb) despite having considerable pain during contractions. She denied that the pain limited her effort to contract in both flexion and extension yet contractions were severely inhibited in extension.

CAUSES OF REFLEX INHIBITION OF QUADRICEPS ACTIVATION

Reflex inhibition of quadriceps can occur in the absence of pain and this was observed by some of the early investigators who studied quadriceps weakness in the presence of knee joint damage

(Vulpian,1875; Charcot,1889; Raymond,1890; Harding,1929). The dissociation between inhibition and pain demonstrated in the present studies (Chapters 3,6,7,8,11,& 'APPENDIX') confirms that some stimulus other than pain must cause reflex inhibition of quadriceps activation.

Possible Role of Ischaemia

Voluntary tourniquet ischaemia did not alter quadriceps function in the normal subjects studied (Chapter 4). Nevertheless, EMG changes appear to occur in the quadriceps after tourniquet ischaemia during knee joint surgery (Saunders et al.,1979; Dobner & Nitz,1982) and the possible reasons for the negative findings of the present study have been discussed (Chapter 4). If ischaemia does cause tissue damage after meniscectomy, its contribution to quadriceps weakness is only small as the majority of the reduced activation after meniscectomy was shown, by the effect of intra-articular bupivacaine, to be due to reflex inhibition (Chapter 3). Since post-meniscectomy inhibition is not due to the tourniquet, the observations made on the meniscectomy patients are relevant to non-surgical patients with knee joint injury and disease.

Role of Joint Effusion

Aspiration of effusion after meniscectomy. Reflex inhibition of voluntary quadriceps activation was always reduced by aspiration of effusion but was not abolished (Chapter 7). Therefore, although effusion clearly contributed to post-meniscectomy inhibition it was not the only cause in those patients.

Dose effect of infusion on quadriceps activation. As expected, quadriceps MVA decreased as the volume of infusion increased in the normal subjects but substantial inhibition had occurred before infusion was clinically obvious (Chapters 8 & 11). This has very important clinical implications because small effusions are probably not considered to be a serious threat to muscle and joint function

and are often left untreated. The present studies showed that 20-30 ml can produce inhibition of both voluntary and reflex activation of quadriceps. The fact that inhibition can occur with undetectably small effusions poses a problem in assessment and the effusion may go untreated. It might be possible that, after meniscectomy, small effusions or synovial swelling could cause reflex inhibition, and that the patients studied in Chapter 3 might have had undetectable effusions. Perhaps the faster recovery of function seen by Muckle (1984) could have been due to small effusions being prevented by the use of a non-steroidal anti-inflammatory drug (NSAID), hence preventing inhibition. If minor joint swelling is suspected (due to the history of injury or disease) in a patient who is not responding to treatment, perhaps it might be helpful to treat this with anit-inflammatory medication.

The patient with the peri-articular scar ('APPENDIX') did not have any intra-articular knee joint symptoms and was unlikely to have had an effusion.

Synovial hypertrophy and inflammation. Muckle (1984) showed that changes in the synovium occurred after meniscectomy and that a NSAID could prevent the changes. Perhaps synovial inflammation stimulates the joint receptors and causes reflex inhibition. It would be of interest to see whether a NSAID would also prevent reflex inhibition after meniscectomy (vide infra, 'TOPICS FOR FURTHER STUDY').

Role of Peri-articular Stimuli

Stimuli originating outside the knee joint can also cause reflex inhibition of quadriceps.

Subperiosteal tumour. Stener (1969) demonstrated how a subperiosteal tumour of the lateral epicondyle of the femur caused severe reflex inhibition of quadriceps activation .

Pre-patellar scar. The patient described in the 'APPENDIX', had similar inhibition to that observed in the meniscectomy patients in that inhibition was greater in extension than in flexion, and was

reduced by infiltration of the scar with a local anaesthetic. Despite the different sites of origin of the inhibition in the 2 pathological conditions, the nature of the inhibition appears to be the same. It is likely that the inhibition produced by inflation of a normal knee joint occurs by the same mechanism and it would therefore be reasonable to relate the findings in the normal 'model' to reflex inhibition in patients with knee joint pathology and trauma.

A neurophysiologist who examined the patient with peri-articular knee pathology ('APPENDIX') was surprised to find that the knee jerk was preserved. This was probably because the knee jerk was tested in flexion which is the position where less inhibition of voluntary activation occurs (Chapters 5 & 'APPENDIX'), and the H-reflex could only be elicited in flexion in the patient with peri-articular pathology ('APPENDIX').

Arthroscopy versus arthrotomy. Reflex inhibition of quadriceps after arthrotomy and meniscectomy might be due to stimuli arising from the capsular incision. Pinching the anterior aspect of the knee joint capsule inhibited the monosynaptic reflex from quadriceps in decerebrated and decerebrated and spinalized cats (Ekholm et al.,1960). It may be possible that tight suturing of the capsular incision would have the same effect on the quadriceps as pinching the capsule. The dexon sutures which were used to close the capsular incision after arthrotomy and meniscectomy were not absorbed until after 60-90 days. This might explain why inhibition was still present 2 weeks post-operatively.

Recovery of function after meniscectomy is faster when the meniscus is removed through an arthroscope (Pettrone, 1982; Northmore-Ball et al.,1983; Tregonning,1983). It may be possible that inhibition is less after arthroscopic surgery as some preliminary results suggest (Sherman et al., 1984; vide infra).

Measurements of quadriceps MVA were made in a small number of patients who underwent either 1) arthrotomy only (n = 2), 2) arthroscopy only (n = 2) or 3) arthroscopic meniscectomy (n = 2). Their results were compared with the pattern of reflex inhibition of quadriceps observed in the control group of patients studied in Chapter 3 who underwent arthrotomy and meniscectomy (Fig 12.1). The arthrotomy only patients showed a similar pattern and severity of inhibition to those of the arthrotomy and meniscectomy patients. Neither the arthroscopy only, nor the arthroscopic meniscectomy patients showed severe inhibition. Although these observations were not made under controlled conditions they do suggest that the method of approach into the joint might be more important as a cause of inhibition than whether or not the meniscus has been removed.

The differences between the 2 types of meniscectomy are summarized in Table 12.1. Fewer sensory nerves would be cut with the smaller incision but the relevance of this to reflex inhibition is not known. The arthrotomy would provide a greater area for both peri-articular inflammation and tension along the capsular and skin incisions. The capsular incision is left open after arthroscopic surgery, which would prevent a build up of intra-articular pressure and capsular tension from any effusion which might occur (as seen in Subject 2 in Chapters 8 & 11). The possibility of capsular tension from suturing would also be avoided. Blood is an irritant when present inside a joint and its effects might be less after arthroscopy as the joint is constantly irrigated during surgery. Even if arthroscopic meniscectomy were adopted in preference to arthrotomy, the problem of quadriceps inhibition will still exist for other knee surgery patients in whom the use of an arthroscope may not be possible e.g. ligament repair, extensive reconstruction procedures, knee replacement.

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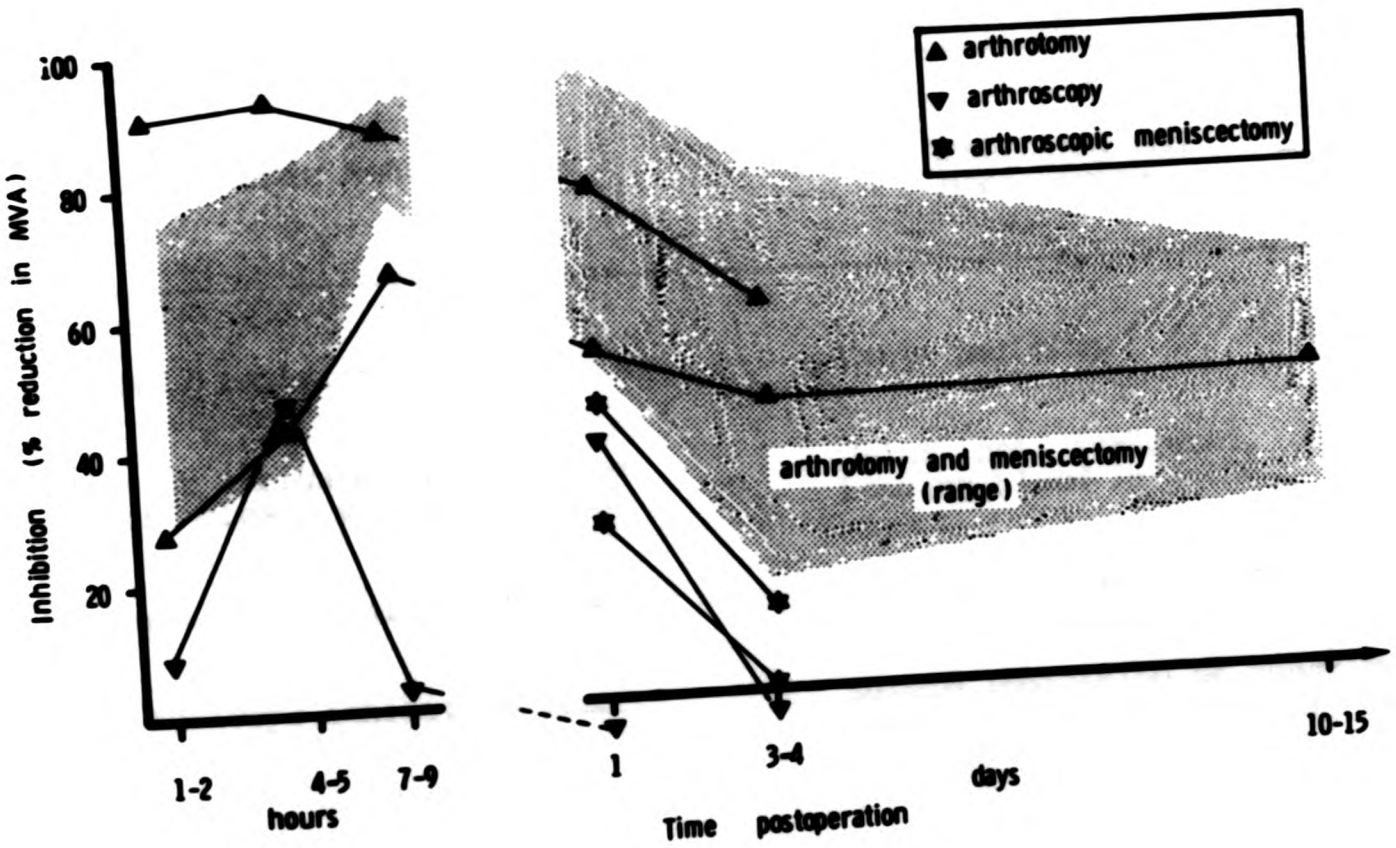


Figure 12.1. Reflex inhibition of quadriceps after different types of knee surgery.

Arthroscopic Meniscectomy	Arthrotomy and Meniscectomy
Small incision	Large incision
Joint irrigated with saline for clear vision of field	Joint not irrigated, leaving blood in joint
Capsular incision left open	Capsular incision closed
Quadriceps inhibition less and short lived	Severe inhibition of quadriceps up to 3 days after surgery and still present at 2 weeks

Table 12.1. The differences between arthroscopic and open meniscectomy.

CENTRAL NEURAL PATHWAY OF KNEE JOINT AFFERENT STIMULI

Evidence from studies in the cat suggest that joint afferents may contribute to the FRA (Eccles & Lundberg,1959; Lundberg et al.,1978) and that they also converge on Ib inhibitory interneurones (Lundberg et al., 1978; Brink et al.,1984). The relevance of the Ib connections in the cat is not clear and no previous studies have been carried out to test this pathway in man.

The present investigations suggest that knee joint afferent activity, produced by joint infusion, may inhibit the anterior horn cells via the Ib and cutaneous FRA pathways. No clear facilitatory effect on Ia postsynaptic inhibition by joint infusion was seen and was only tested in 2 subjects. It is probably unsafe to draw any conclusion from a failure to observe spatial facilitation in this type of experiment. Facilitation requires just sub-threshold activation of a substantial proportion of the shared interneurones by each of the 2 stimuli. A larger action would lead to occlusion and the facilitatory effect would be obscured.

REFLEX INHIBITION OF QUADRICEPS IN TRAUMATOLOGY AND RHEUMATOLOGY

Patients With Knee Joint Trauma

Effusion. Trauma to the soft tissues of the knee joint is often accompanied by effusion which can cause reflex inhibition of quadriceps (deAndrade et al.,1965; Jayson & Dixon,1970a; Chapters 8 & 11). The severity of quadriceps inhibition can be reduced by aspiration of effusion (Chapter 7). Volumes of fluid of <30ml can inhibit the quadriceps (Chapters 8 & 11) so even small effusions, and certainly large effusions, should not be tolerated in these patients. Inhibition of reflex activation of quadriceps is greater during contractions than at rest (Chapter 11) due to increased intra-articular pressure. This fact should be considered when advising patients with joint effusions about which activities they should

avoid. Effusion should be reduced by an appropriate method e.g. aspiration, ice in elevation, NSAID. If effusion is suspected (due to the history of trauma or other signs and symptoms) it might be reasonable to use a NSAID in an attempt to prevent or reduce inhibition.

Pain. Although pain was not associated with post-meniscectomy inhibition, patients with knee trauma often complain of pain. If voluntary inhibition due to pain (Basmajian,1970) was limiting a patients' effort during contraction,then some form of pain relief would be indicated e.g. ice, TNS, interferential, oral medication. In the light of the present series of results (Chapter 6) it should be remembered that although pain relief may appear to allow more effective exercise and improved function, severe inhibition may still be present. It would therefore be essential that pain relief should be administered with caution and that exercises and activities should be supervised in order to prevent overuse of the injured joint because excessive weight lifting or weight bearing in the presence of inhibition might produce further joint damage due to abnormal loading of the knee and patello-femoral joints (see Chapter 1, page 13).

The fact that inhibition often occurs in the absence of pain makes clinical assessment of weakness difficult. In patients whose management is proving particularly difficult it may be helpful to test for the presence of reflex inhibition.

Patients With Joint Disease

Quadriceps weakness in patients with joint disease often causes more severe and long term disability than after trauma or surgery. Quadriceps weakness is common in patients with neuritis, myositis and steroid myopathy but weakness as a result of joint disease may be complicated by reflex inhibition. Factors such as immobility, pain, effusion and irritation from damaged tissues within the joint make the causes of weakness in the presence of joint

disease very difficult to identify. It is likely that reflex inhibition contributes to the weakness and therefore makes quadriceps contractions ineffective. Methods which are already known to reduce reflex inhibition (control of effusion by e.g.aspiration, intra-articular injection of anti-inflammatory drugs, removal of hyperplastic synovium) may help with the management of such patients. Perhaps oral anti-inflammatory medication might prevent inhibition caused by small, undetectable effusions and synovial inflammation.

Our present understanding and knowledge of the causes of reflex inhibition of quadriceps is insufficient to permit effective long-term treatment for these patients. Investigations of quadriceps inhibition in surgical patients should continue. It is hoped that, while benefitting the surgical patients, the knowledge gained from such studies may be used to solve the problem of quadriceps inhibition (and therefore weakness) in rheumatology patients.

TOPICS FOR FURTHER STUDY

Per-operative Bupivacaine for Post-operative Analgesia

Pain was less severe after meniscectomy in patients who received intra-articular bupivacaine at the end of surgery. A study is necesssary to evaluate the role of per-operative intra-articular bupivacaine as a post-operative analgesic during the first few hours after surgery when pain is severe (Chapter 3).

Effectiveness of Isometric Quadriceps Exercises Performed in Flexion

Although quadriceps contractions are inhibited less when the knee is in a flexed position (Chapter 5), it is not known whether an isometric strength training programme of exercises performed in flexion would improve post-meniscectomy quadriceps activation and strength in both flexion and extension. The patient with peri-articular pathology did not respond to exercises performed in flexion ('APPENDIX'). It seems likely that increases in muscle

strength cannot be achieved while the inhibitory stimuli still operate between periods of exercise. This would need to be tested in a controlled treatment trial comparing exercises in flexion with exercises in extension. This could be carried out in meniscectomy patients during the period of severe inhibition and later when inhibition is less severe. The effectiveness of treatment could be evaluated using measurements of quadriceps size, strength and activation (Chapter 2) and assessment of functional activities.

Role of Intra-articular Pressure in the Knee Flexion Phenomenon

Quadriceps inhibition needs to be examined at angles of high an low pressure to determine whether the phenomenon of reduced inhibition in mid-range flexion (Chapter 5 & 'APPENDIX') is due to intra-articular pressure or some other factor associated with knee joint flexion. Intra-articular pressure should be recorded e.g. using a system such as that used in Chapters 8 & 11.

Transcutaneous Nerve Stimulation

Before a larger treatment trial could be carried out, (conducted in a similar way to the pilot study in Chapter 6), it would be necessary to experiment with the stimulation parameters for TNS e.g. using different pulse widths, or comparing the effects of low and high frequency TNS.

Reflex Inhibition of Voluntary Quadriceps Activation due to Small Infusions

Volumes of infusion as small as 20ml and 30ml which were not clinically apparent were shown to cause reflex inhibition of voluntary activation of quadriceps (Chapter 8). Inhibition of MVA was only studied in 2 subjects so more subjects should be studied to determine the minimal amount of fluid necessary to inhibit voluntary contractions. Perhaps infusion of the knee at 5ml increments might reveal a threshold volume after which inhibition occurs.

Non-steroidal Anti-inflammatory Drug (NSAID) Trial

Perhaps oral anti-inflammatory drugs might influence the knee joint receptors in man by reducing effusion and synovial inflammation. Muckle (1984) showed that a NSAID (Flurbiprofen, 10mg, 4 times/day) taken for 7 days following open meniscectomy, reduced pain and speeded functional recovery.

The effectiveness of a NSAID in preventing quadriceps inhibition due to joint effusion or synovial inflammation could be studied using the arthrotomy and meniscectomy 'model'. Placebo and active tablets could be given and the effects could be evaluated by measuring quadriceps size, strength and activation (Chapter 2), and by assessing functional improvement.

Muscle Stimulation to Prevent Weakness and Wasting caused by Reflex Inhibition

Eriksson & Häggmark (1979) suggested that percutaneous muscle stimulation may prevent muscle atrophy after major knee ligament surgery. They showed that electrical stimulation plus isometric quadriceps exercises produced better muscle function and higher succinate dehydrogenase activity than isometric exercises alone. Perhaps continuous stimulation of muscle would prevent atrophy due to reflex inhibition as direct stimulation of motor nerves bypasses inhibiton.

It was proposed that a muscle stimulation trial would be conducted using post-meniscectomy patients, but preliminary tests on 2 normal subjects indicated some difficulties. An Electro-Stim 180 (RDG Electro Medical) was used to determine how much of the muscle could be activated by high frequency stimulation. Isometric quadriceps force produced by stimulation was recorded with the knee at 90°, and was expressed as a percentage of the force of a MVC. Owens and Malone (1983) stated that it was generally accepted that stimulated contractions of >65% MVC were required for producing strength gains, and they reported values for normal subjects (tested

with 30° knee flexion) of 25-73% MVC. Stimulated contractions produced 56% and 90% of MVC in the present 2 subjects studied. It was intended that stimulation would be applied during the early post-operative period when the knee is immobilized in extension, but tests in the normal subjects in extension were very uncomfortable even at low intensities. Stimulation produced a feeling of cramp in quadriceps and the patella felt as though it would dislocate. It was not possible to measure the forces produced in extension but they were probably as small as those produced by conventional faradic stimulation due to the low intensities of stimulation achieved. It would therefore not be feasable to use this type of stimulation to strengthen muscle with the knee extended. Perhaps if the knee was immobilized in flexion e.g. 30°, stronger contractions might be achieved due to less discomfort in the muscle and a more stable position for the patella. This would have the additional advantage that inhibition is less with the knee in a flexed position.

Effect of Capsular Suturing on Quadriceps Activation

Reflex activation could be monitored during suturing of the incision at the end of surgery by measuring quadriceps H-reflexes. This would obviously need to be performed on a patient who demonstrated an H-reflex at rest.

Effect of Haemarthrosis on Quadriceps Activation

Blood is an irritant when present in a joint but its effect on quadriceps activation is not known. A trial could be carried out to investigate the effect of irrigating the joint during surgery. Quadriceps activation would be measured in 2 groups of patients during whose operations the joint had either been irrigated or not.

CONCLUSIONS

1. After arthrotomy and meniscectomy, reflex inhibition of voluntary quadriceps activation is severe for at least 3 days (typically 70-80%) and persists for at least 2 weeks (typically 35-40%). (Chapter 3)

2. Post-meniscectomy quadriceps inhibition persists in the absence of pain and obvious knee joint effusion. (Chapter 3)

3. Extensive infiltration of the meniscal bed and tissues surrounding the incision with an adequate dose of bupivacaine effectively reduces post-meniscectomy quadriceps inhibition, confirming that the mechanism causing inhibition involves stimuli from the knee. (Chapter 3)

4. The dose-related dissociation of the effects of bupivacaine on inhibition and pain suggests that the inhibitory impulses were not transmitted along the small pain fibres. (Chapter 3)

5. The reduction of reflex inhibition by bupivacaine is too short-lived to permit a useful period of effective post-operative quadriceps exercises. (Chapter 3)

6. The reduced quadriceps activation observed in the present meniscectomy patients could not have been caused by tourniquet ischaemia alone. (Chapter 4)

7. Post-meniscectomy quadriceps inhibition is less when isometric contractions are made with the knee in about 40^{o} of flexion. (Chapter 5)

8. Transcutaneous nerve stimulation for 6 hours only had a trivial effect on post-meniscectomy quadriceps inhibition which was too small to be clinically useful. (Chapter 6)

9. After arthrotomy and meniscectomy, a knee joint effusion increases the severity of quadriceps inhibition but it is not its sole cause. (Chapter 7)

10. Reflex inhibition of both voluntary and reflex activation of quadriceps increases as the volume of knee joint infusion increases and this occurs in the absence of pain. (Chapters 8 & 11)

11. Small infusions (20-30ml) which are not clinically obvious can cause reflex inhibition of both voluntary and reflex activation of quadriceps. (Chapters 8 & 11)

12. Increases in intra-articular pressure with joint infusion are greater during contraction than at rest and this probably accounts for the greater inhibition of reflex activation observed during contraction than at rest. (Chapters 8 & 11)

13. Facilitation of Ia reciprocal inhibition by joint stimulation (i.e. infusion) was not clearly demonstrated in the present studies so it was not possible to state whether or not joint afferents share the interneurones of the reciprocal inhibitory pathway. (Chapter 11)

14. The central neural pathway of afferent stimuli from an infused knee joint appears to have connections with Ib inhibitory pathways from Golgi tendon organs and cutaneous FRA pathways. (Chapter 11)

APPENDIX

A CASE STUDY: QUADRICEPS INHIBITION IN A PATIENT WITH PERI-ARTICULAR KNEE JOINT PATHOLOGY

INTRODUCTION

HISTORY

- Presenting Complaint
- History of Presenting Complaint
- Past History
- Current Activities
- Social History

EXAMINATION

DIAGNOSTIC INVESTIGATIONS

- Timing of Tests
- X-rays
- Biochemistry and Haematology
- Quadriceps Morphology
- Neurophysiology I
- Lumbar Myelogram
- Lumbar CAT Scan
- Neurophysiology II & III

INVESTIGATIONS FOR STUDYING REFLEX INHIBITION OF QUADRICEPS AND FOR MONITORING THE EFFECTS OF TREATMENT

- Timing of Tests
- Voluntary Activation of Quadriceps
- Quadriceps Isometric Strength
- Whole Quadriceps Cross-sectional Area
- Relationship between Quadriceps Size and Strength
- Activities and Subjective Assessment

CONCLUSIONS

A CASE STUDY: QUADRICEPS INHIBITION IN A PATIENT WITH PERI-ARTICULAR KNEE JOINT PATHOLOGY

INTRODUCTION

This case history describes a 37 year old woman with an 18 month history right knee pain and severe weakness and wasting of her right quadriceps after an operation to remove a prepatellar ossified haematoma. Investigations began in January 1983 to determine whether her knee 'injury' was causing reflex inhibition of quadriceps and whether pain was contributing to the weakness by causing voluntary inhibition.

The nature of the quadriceps inhibition demonstrated in this patient was comparable with that seen in the meniscectomy patients studied in Chapters 3 & 5. This suggests that the afferent stimuli from the knee joint which cause reflex inhibition of quadriceps may be intra- or peri-articular in origin (see Chapter 12, page 127).

HISTORY

Presenting Complaint

The patient complained of a constant background ache with superimposed sudden stabs of pain in the prepatellar region of her right knee. Her quadriceps weakness caused her to walk with a limp and she was unable to walk more than a mile due to pain. She had previously enjoyed 15-20 mile walks once or twice every weekend.

History of Presenting Complaint

Winter 1979/80	Struck right knee on the car dashboard in a road traffic accident. Experienced tingling up her right thigh for a few minutes. None since. No pain or swelling subsequently.
June 1981	Two episodes of bruise/discolouration at the right infrapatellar and medial parapatellar regions.

July 1981	Right knee began to feel stiff and a prepatellar lump appeared. An ossified haematoma was surgically removed from the prepatellar region. Symptoms worse post-operatively with right knee pain and stiffness. Patient then became aware of quadriceps weakness and wasting.
October 1981	Plaster of Paris for 2 weeks. She then developed infrapatellar pain which responded partially to an injection of hydrocortisone.
December 1981	Arthroscopy of right knee showed no abnormalities. Faradism to quadriceps for 1 month by a private physiotherapist was of "slight benefit".
March-April 1982	Nine weeks of intensive quadriceps training as an in-patient at a rehabilitation centre.
June-July 1982	Five more weeks in-patient treatment at the rehabilitation centre.
July 1982	Mixed hydrocortisone and "painkilling" injection to the infra-patellar area of pain helped for 3-4 months.
January 1983	Present investigations began.

Past History

There was no significant history of back pain or sensory disturbance and no personal or family history of joint disease. She had a history of tempero-mandibular joint pain which was cured by the dentist "realigning teeth". There was no personal or family history of joint disease. She was not on any medication.

Current Activities in January 1983

Her current activities included 2 miles cycling each day on a static bicycle, and this was painful. She was not performing regular quadriceps exercises as she found them too painful but occasionally attempted straight leg raising. Walking was painful and she wore a tight tubigrip which helped her pain. She walked with a limp and her right knee frequently gave way while walking. She found descending stairs more painful than climbing stairs. She was without pain while in bed (except if something touched her knee or when turning over) and the pain returned within half an hour of rising.

Social History

The patient was working as a secretary until January 1984 when she became a receptionist to a general practicioner. Her husband worked as a scientist in commercial genetic engineering. They had no children.

EXAMINATION

On initial examination the patient had extreme quadriceps weakness and wasting. She was barely able to straight leg raise and this caused severe pain in the prepatellar region at the inferior end of her operation scar. During the examination she experienced stabs of "nerve pain - like at the dentist", both on palpation and during active and passive knee movements. There was no apparent effusion, the knee was stable and there was no pain on patello-femoral grinding. Straight leg contractions of quadriceps were painful, particularly on relaxing, and it took her a long time to release the contraction. Knee and ankle jerks were present on both sides. The calf, hallux, toe and foot flexors and extensors were normal. Light touch was intact. Her left knee, hips and lumbar spine were normal.

The impression of her condition on initial examination was that she might have a neuroma or an undissolved suture in the prepatellar region which was causing reflex inhibition of quadriceps and that her knee joint was otherwise normal.

DIAGNOSTIC INVESTIGATIONS

Timing of Tests

The dates of the tests are shown in Table AP.1.

X-rays

X-rays of the right knee and lumbar spine were normal.

Date	Muscle Biopsy	Neuro Physiol	Lumbar Myelog	Lumbar CAT Scan	X - rays Knee	X - rays Lumb Sp	Blood & Urine
1983							
26 Jan						+	
17 Feb			+				
22 Apr				+			
10 May						+	+
18 May					+		
6 June						+	
12 Jul				+			
1984							
2 May				+			

Lumb Sp = Lumbar Spine

Table AP.1. Timing of diagnostic tests performed on a patient with peri-articular knee joint pathology.

Biochemistry and Haematology

Urine tests and blood tests were normal.

Quadriceps Morphology

Needle biopsy ofthe quadriceps was performed by Dr A Young. Muscle biopsy specimens were taken from the lateral part of the quadriceps at mid-thigh (Young et al.,1979; Edwards et al.,1980) with a UCH muscle biopsy needle (outside diameter 4.5mm) . The design of the UCH needle (Young et al.,1978) was based on the Bergstrom needle (Bergström, 1962). One specimen was taken from the right leg and 2 from the left (the results from the left were averaged). Specimens were orientated (by the author) under a dissecting microscope so that the fibres were parallel, were then mounted in gel (Tissue Tek II, OCT compound) and were rapidly frozen in isopentane cooled to its melting point by liquid nitrogen. Transverse cryostat sections were prepared from each biopsy and stained (by Ms M Reading) to show myosin adenosine triphosphatase activity at pH 9.4 (Round et al.,1980). Type I fibres showed light staining and type II fibres showed dark staining. The mean cross-sectional area of the muscle fibres in each biopsy (mean fibre area, MFA) was calculated from measurements of the mean cross-sectional area of each type of fibre and the relative frequency of the 2 fibre types. Mean cross-sectional area was calculated by measuring 100 fibres of each type with a MOPPET planimetry system which is an electronic planimeter (MOP) linked to a PET computer (Jones et al.,1980). Fibre type frequency was calculated by counting a minimum of 200 fibres. The coefficient of variation for measurements of MFA from repeated biopsies is 16% (Wiles et al.,1979). The fibre measurements were made by the author at University College Hospital, London.

Sections from the uninjured limb's quadriceps showed normal appearances (Fig AP.1.a+b; Table AP.2.). Sections from the injured limb's quadriceps showed an excess of type II fibres and

	Mean Fibre Area (μ^2)				% II	% CSA II
	I	II	All	II/I		
Left	3752	3193	3467	1.18	51	47
Right	3332	1184	1828	0.36	7Ø	45
Inj/uninj	89%	37%	53%	-	-	-

Table AP.2. Needle muscle biopsy results for mean fibre area (MFA) of quadriceps and percentage frequency of type II fibres.

i.

ii.

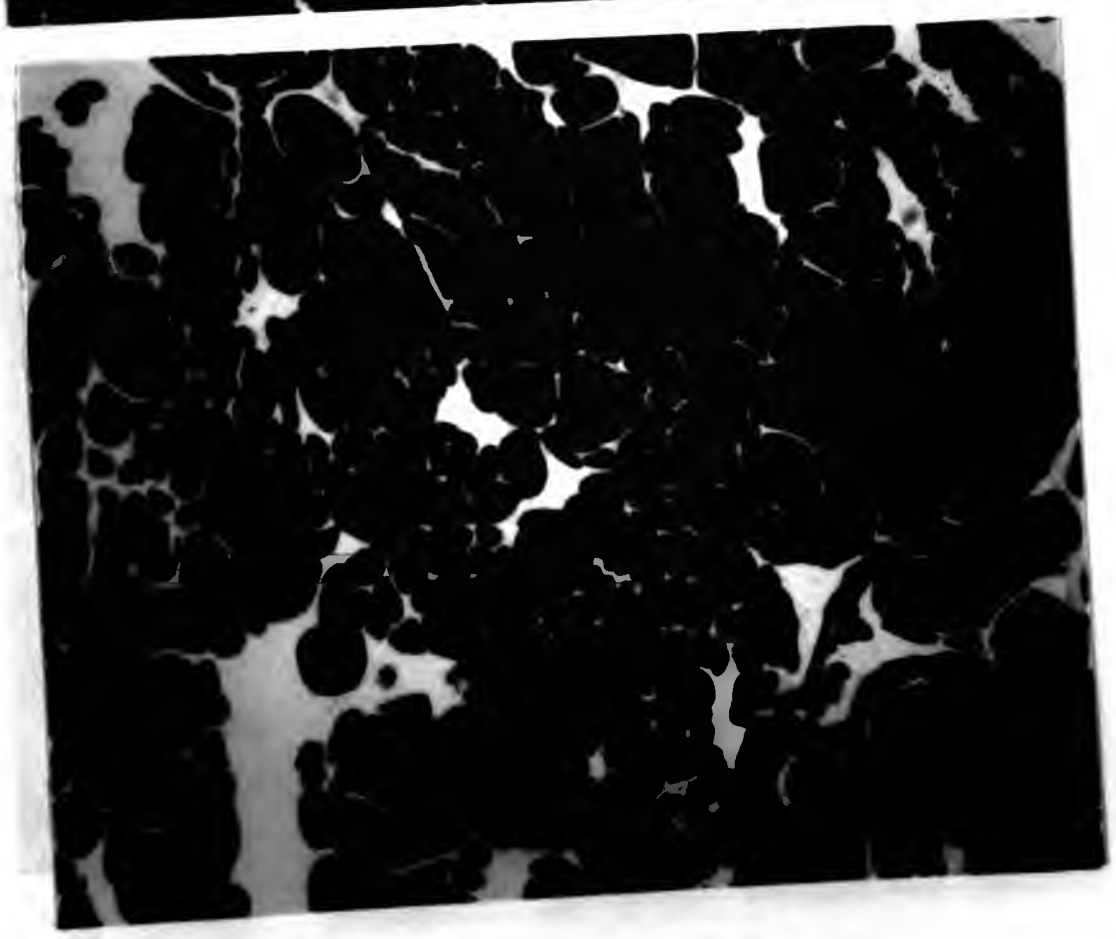
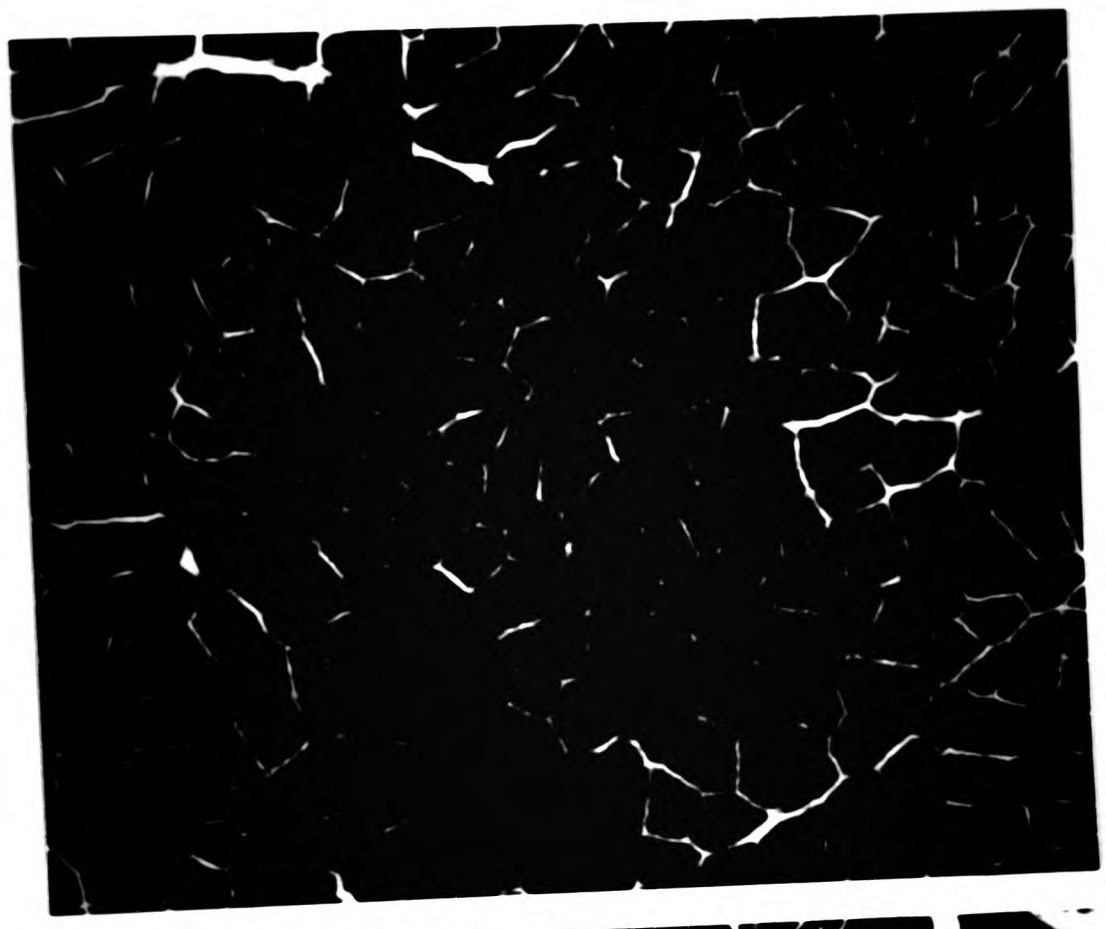


Figure AP.1.a Needle muscle biopsy specimens showing fibre type frequencies.

i. left (uninjured) ii. right (injured)

i.



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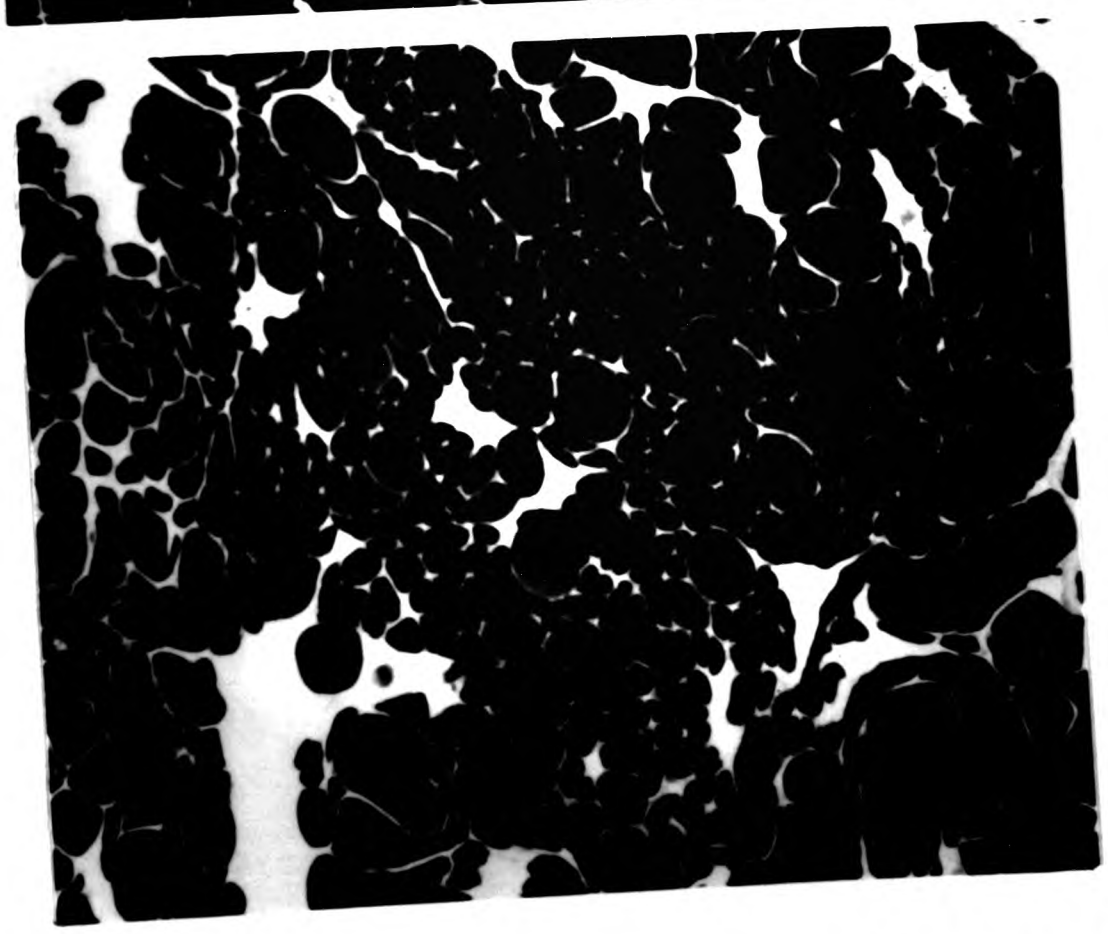
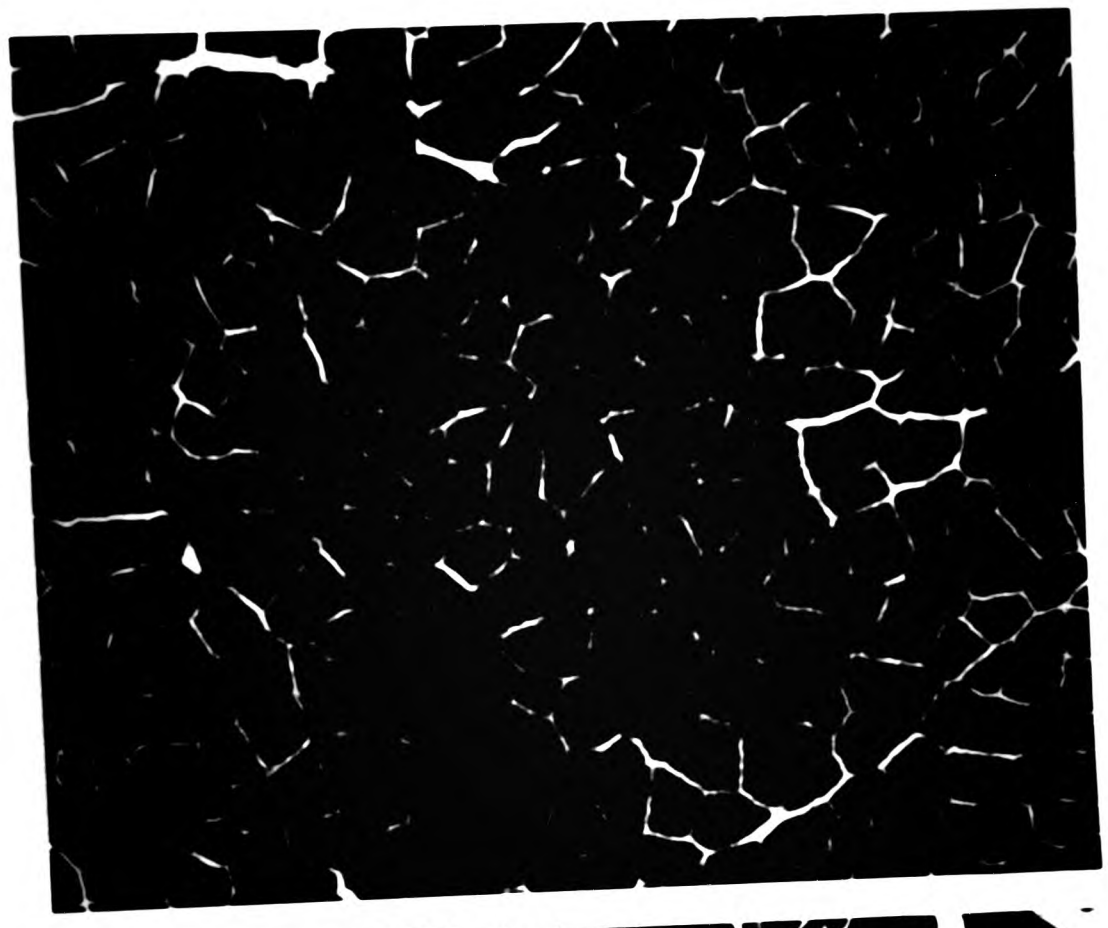


Figure AP.1.a Needle muscle biopsy specimens showing fibre type frequencies.
i. left (uninjured) ii. right (injured)

i.



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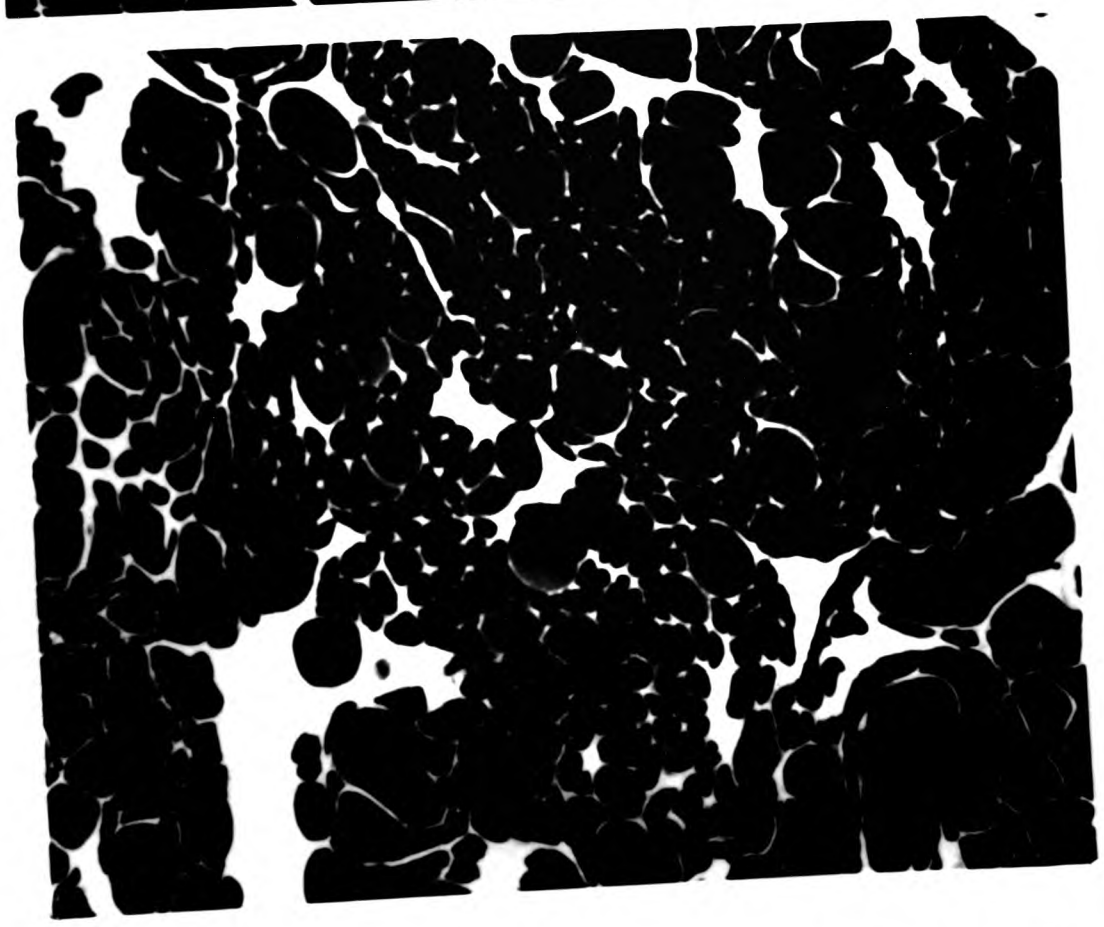
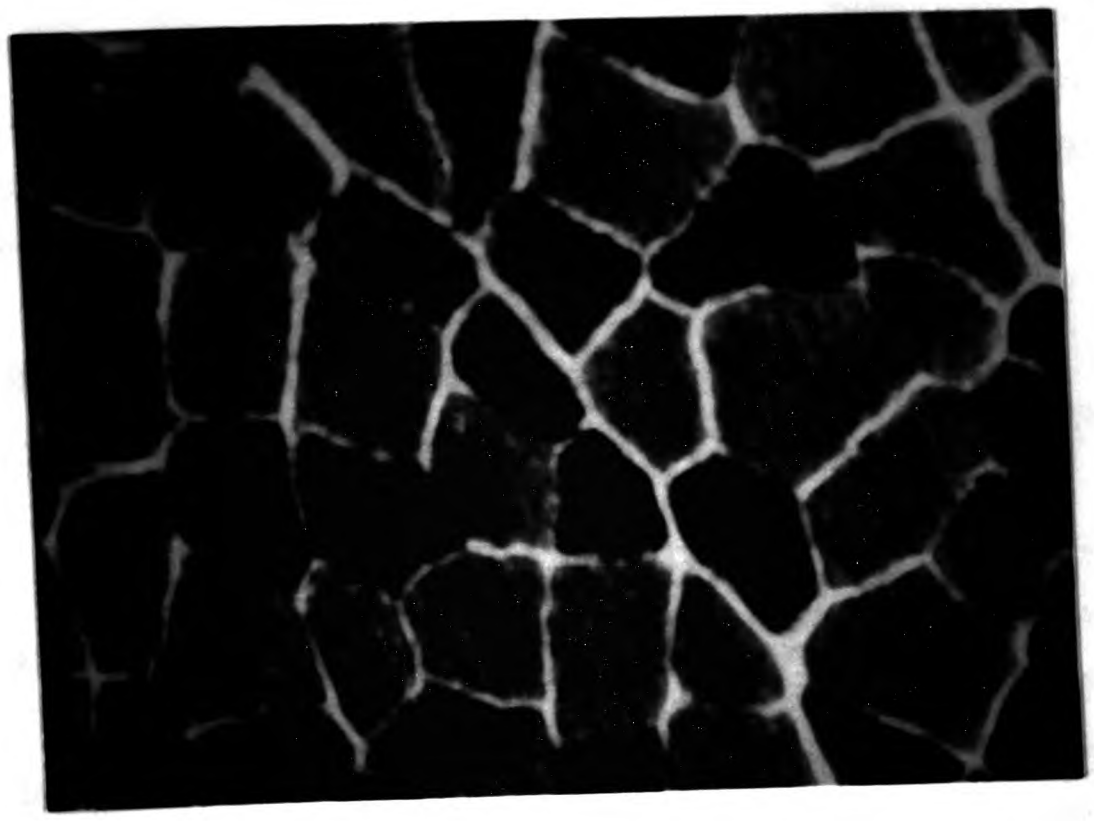


Figure AP.1.a Needle muscle biopsy specimens showing fibre type frequencies.

i. left (uninjured) ii. right (injured)

i.



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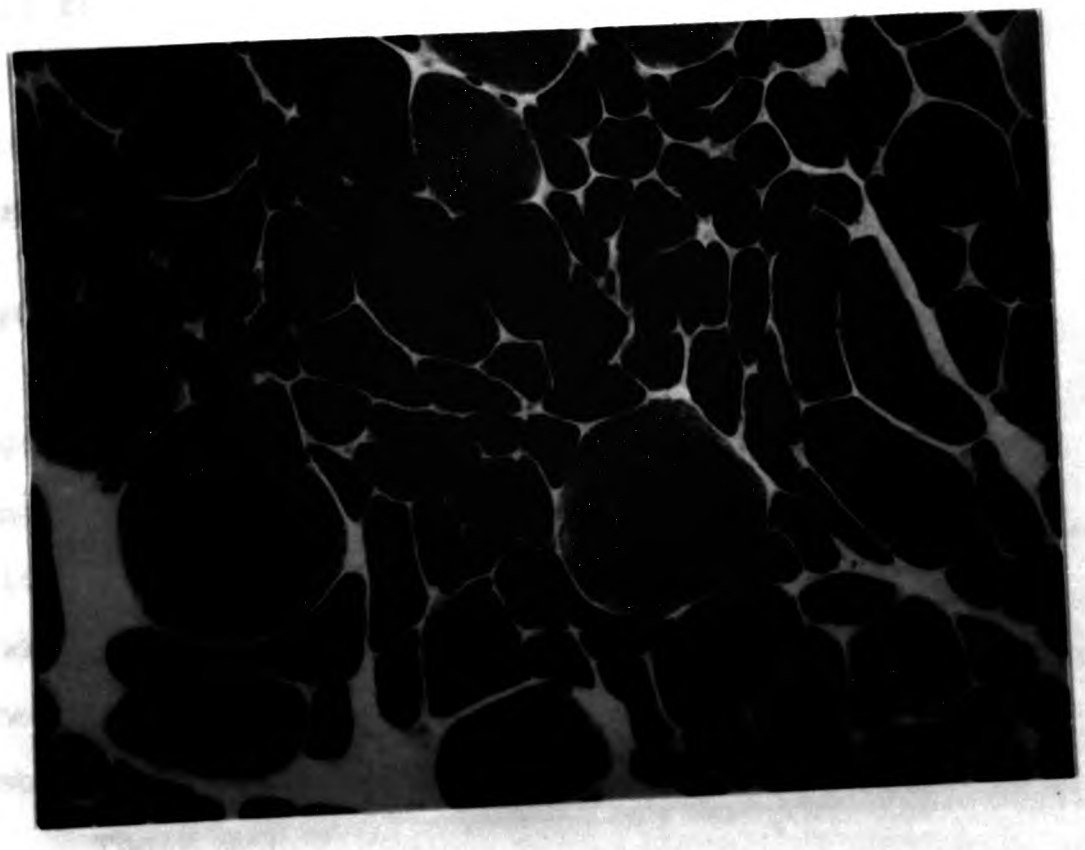
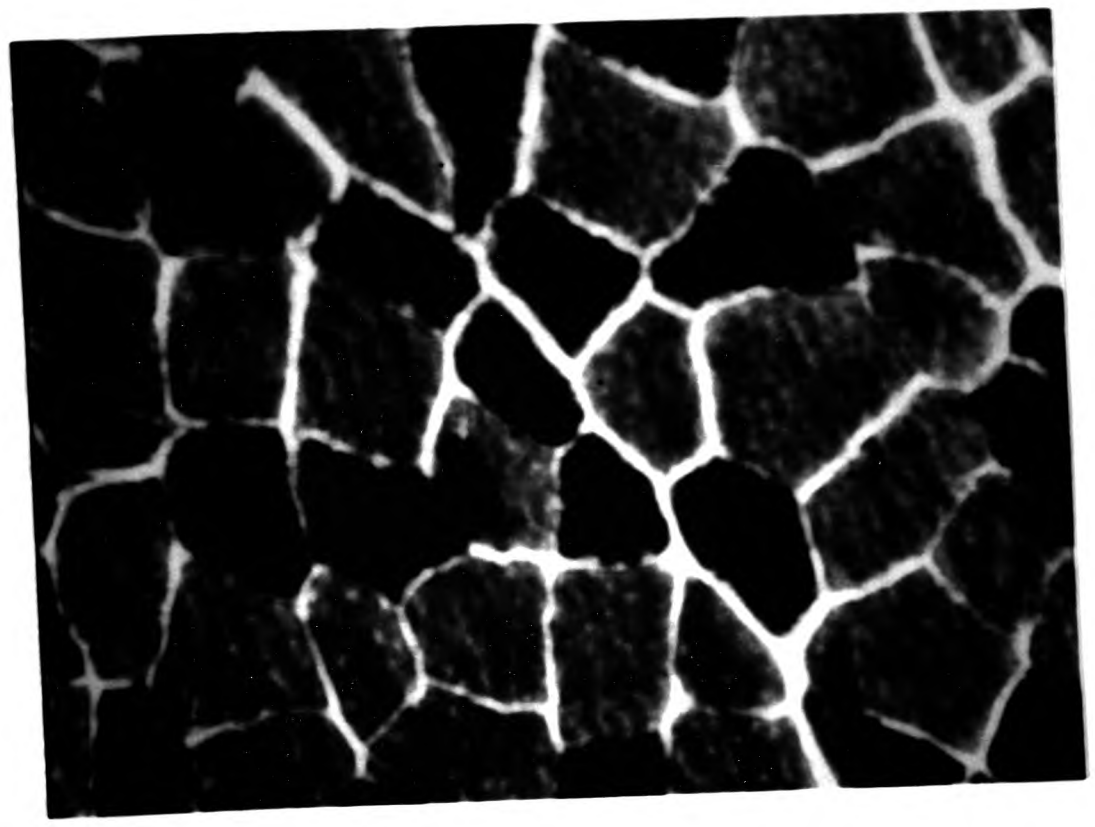


Figure AP.1.b **Needle muscle biopsy specimens showing atrophy of type II fibres.**
i. left (uninjured) **ii. right (injured)**

i.



ii.

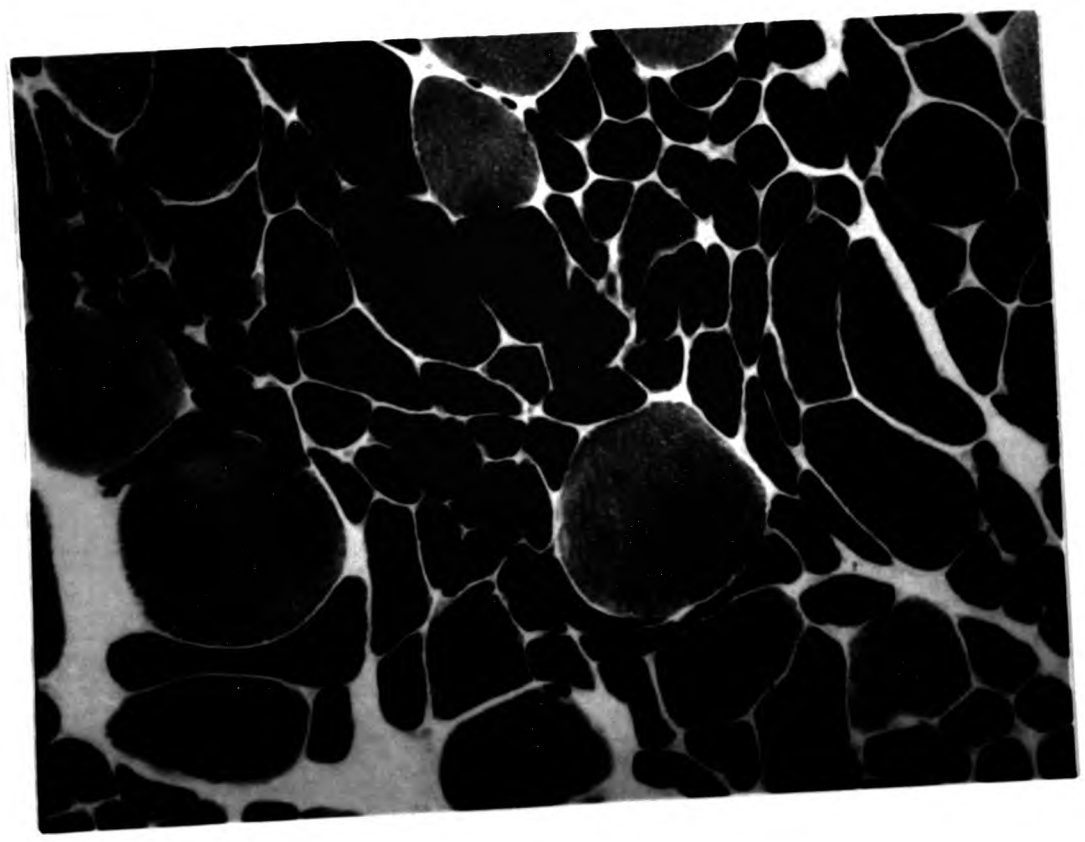
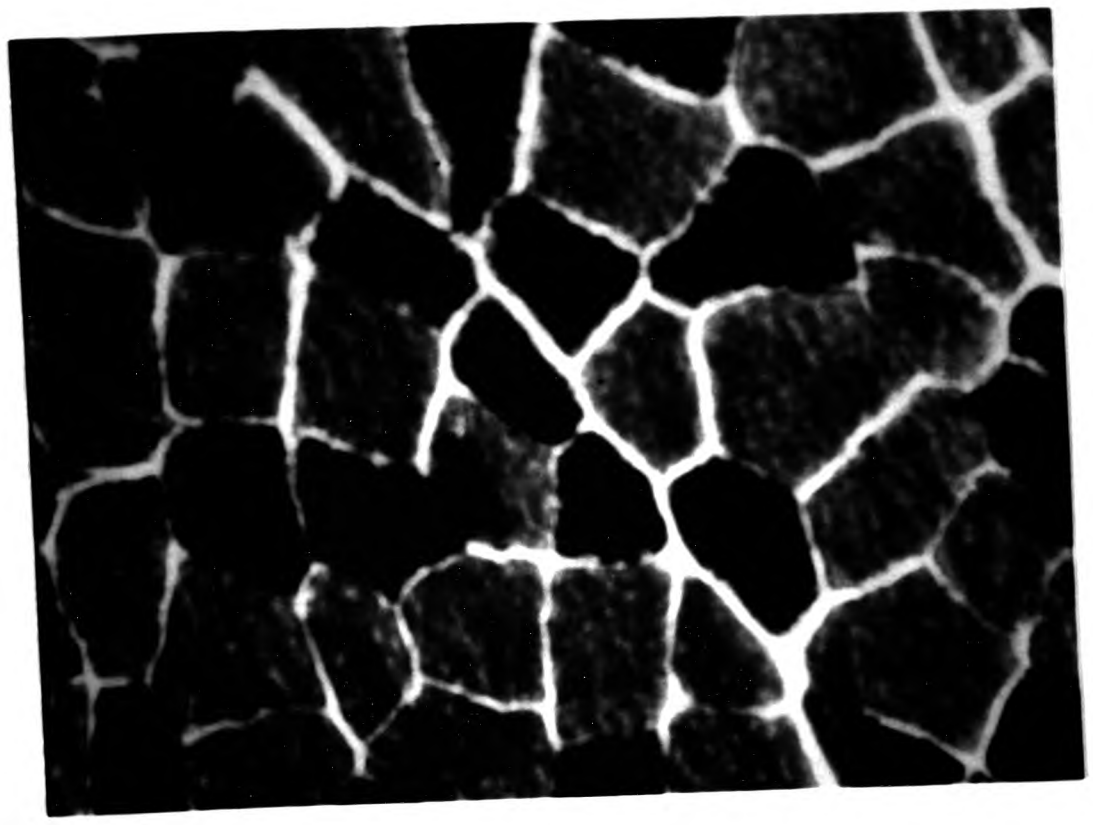


Figure AP.1.b Needle muscle biopsy specimens showing atrophy of type II fibres.

i. left (uninjured) ii. right (injured)

i.



ii.

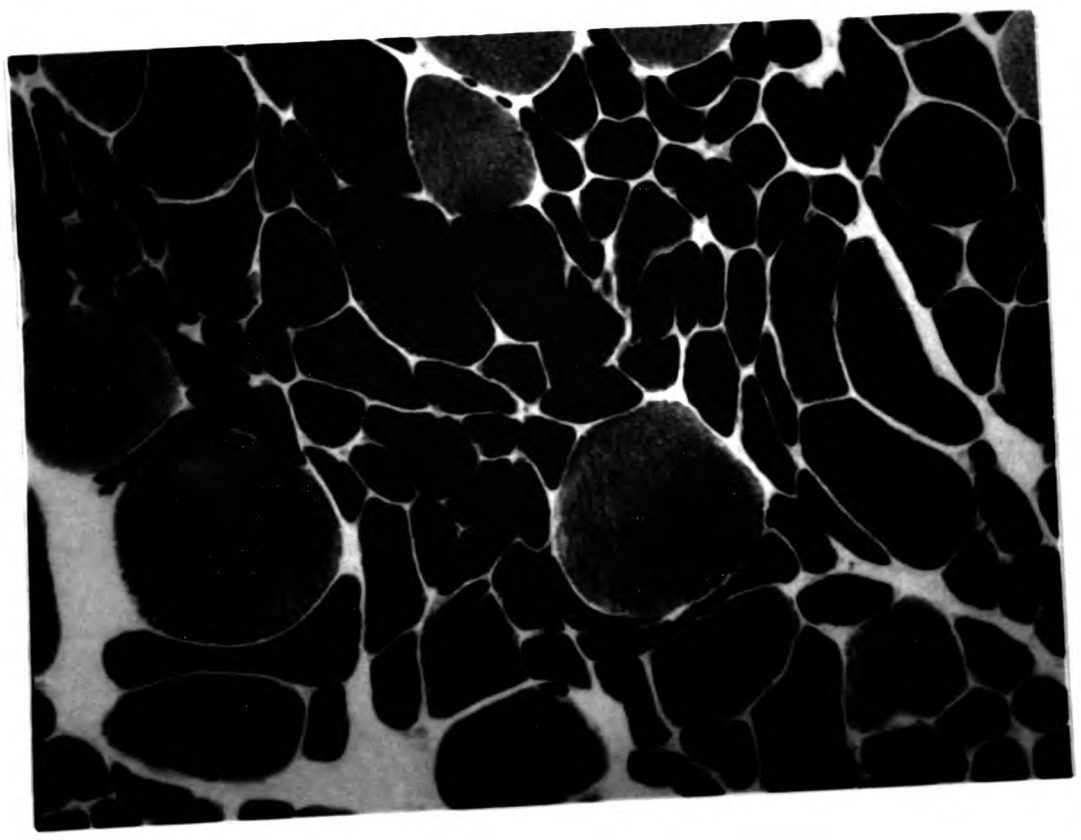
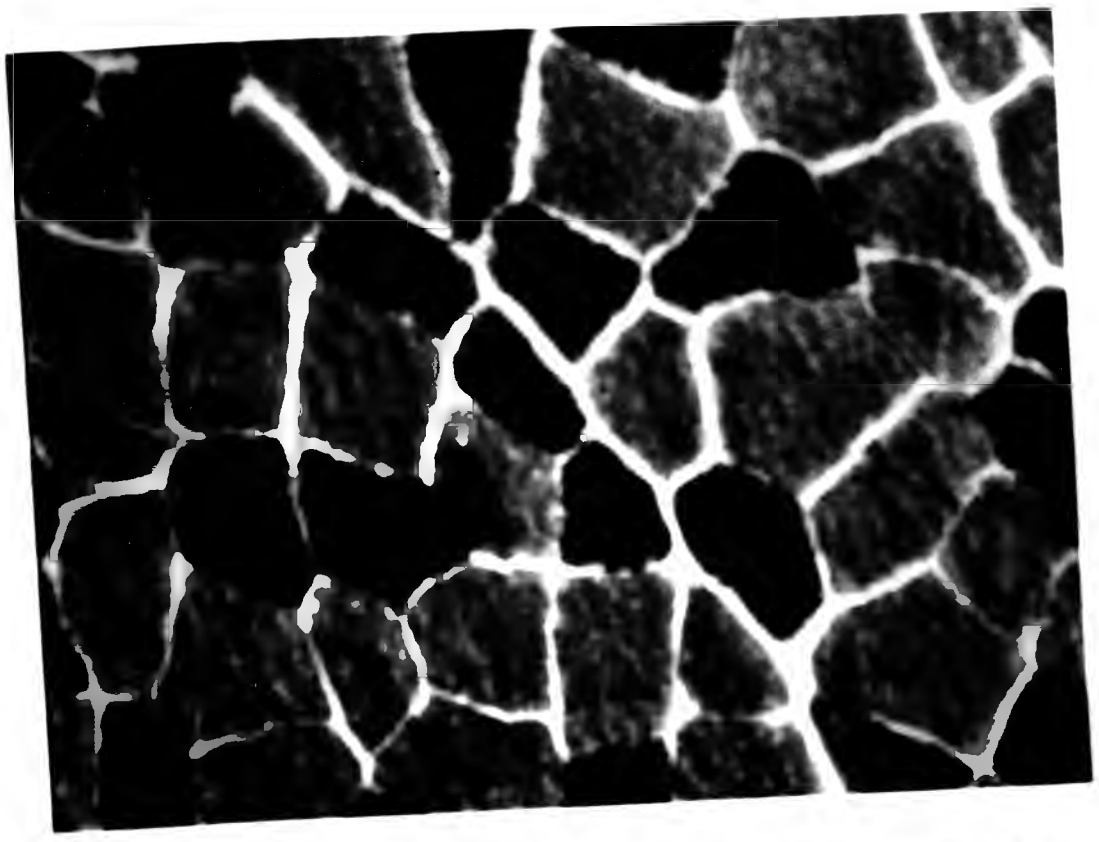


Figure AP.1.b Needle muscle biopsy specimens showing atrophy of type II fibres.
i. left (uninjured) ii. right (injured)

i.



ii.

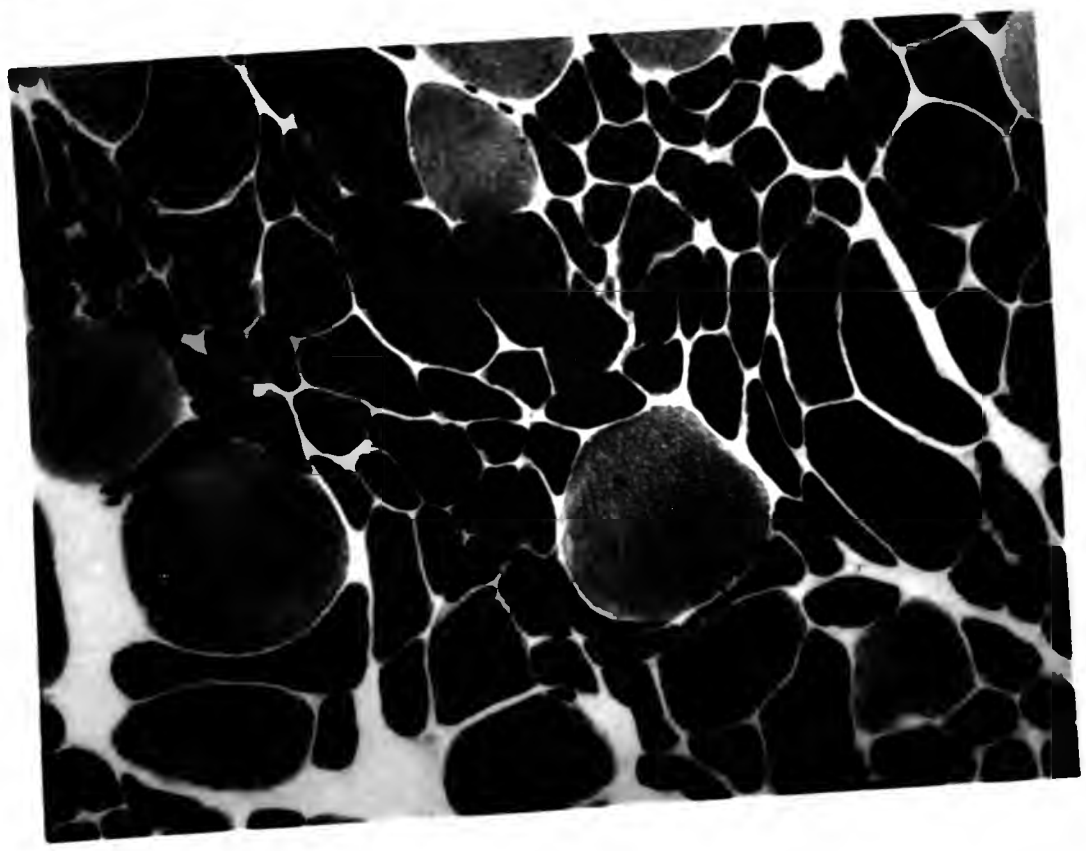


Figure AP.1.b Needle muscle biopsy specimens showing atrophy of type II fibres.

i. left (uninjured) ii. right (injured)

atrophied muscle fibres (Fig AP.1.a+b). Muscle fibre atrophy was mostly of type II fibres (62% smaller) and there was only an 11% difference in type I fibre size. The biopsy report (by Dr JT Hughes) stated that this finding was "typical of chronic denervation".

The ratios of whole quadriceps CSA to MFA (injured = 114 times 10^4 units; uninjured = 134 times 10^4 units) were similar in the 2 limbs and were similar to published values for the 2 sides of patients with unilateral knee injury (Young et al.,1982).

Selective atrophy of one or other fibre type is common in patients with arthrogenous muscle atrophy. An increased proportion of type II fibres is also common (Young et al.,1982; Ingemann-Hansen & Halkjaer-Kristensen,1983). Neither selective atrophy nor an excess of type II fibres are usually as dramatic as in the present patient. It was therefore necessary to explore the possibility of quadriceps denervation (vide infra - results of neurophysiology I and II, lumbar myelography and computed tomography).

Neurophysiology I

These tests were performed by Dr G Rushworth (Consultant Neurophysiologist, in Oxford). Conventional "qualitative" EMG was performed at rest and during maximal voluntary contractions in extension. Recordings were made from RF, VM and VL using needle electrodes. The H-reflex was tested in the right quadriceps at rest with the knee extended and could not be elicited. The H-reflex was not tested in the left quadriceps.

There was no spontaneous activity at rest in the right RF but there was a little spontaneous activity in the form of positive sharp waves and brief, low amplitude potentials (due to fibrillation) in the right VM and VL. Maximal voluntary contraction evoked a moderate number of single motor units which were not polyphasic in the right RF, and fewer single motor units which were polyphasic were evoked in VM and VL. The latencies of the right knee jerks (21ms) were longer than those of the left (19.5ms) and

total reflex amplitude was lower on the right.

Dr Rushworth concluded that the injured limb's RF was "minimally denervated" and VL and VM were "severely denervated" although the knee jerk was well maintained. Preservation of the knee jerk may have been due to the fact that it was tested with the knee flexed. If it had been tested in extension it would probably have been diminished or absent, as was the H-reflex in extension (*vide infra* - Neurophysiology III), and voluntary activation is also more inhibited in extension (see Chapter 5). The motor unit potentials of VM and VL showed evidence of an attempt at regeneration. Dr Rushworth stated that the quadriceps weakness and wasting were "of neurogenic origin" and that it was "essential to search for possible sites of a lesion, including the right L2, 3 or 4 nerve roots, even though there have been no complaints of back pain". Hence, a lumbar myelogram and CAT scan were performed.

Lumbar Myelogram

The patient was admitted to hospital for 3 days for a water soluble myelogram (16ml matrizamide), injected at L3/4. The report by Dr GS Kukowski stated: "Appearance of the lumbar sub arachnoid space is normal. There is no evidence of nerve root compression. Dimensions of the lower spinal cord are normal, it does not appear displaced or compressed. Conclusion - Normal".

Lumbar Computerised Axial Tomography (CAT) Scan

Transverse CAT scans were taken at 10mm intervals from L1 to L4. The report by Dr D Wilson stated: "The neural foramina appear normal. No soft tissue mass seen. Normal examination".

Neurophysiology II

Bilateral surface EMG tests performed by Dr K Mills (a Clinical Neurophysiologist, in London) were: sural nerve sensory action potential, saphenous nerve sensory action potential, lateral

popliteal motor study, knee jerk reflex times, distal motor latency to RF, relationship between isometric force and integrated rectified (IR) EMG, and the effect of knee angle on IREMG. The effect of local anaesthesia on IREMG was tested after injection of 1.5ml of 2% lignocaine into the painful area at the prepatellar scar. Quadriceps H-reflex was tested at rest in extension, in the normal limb and could not be elicited.

These tests confirmed that the right quadriceps' MVA in extension, was 1) less than the left 2) increased by knee flexion and 3) increased by infiltration of the prepatellar scar with local anaesthetic (Table AP.3.). The rectified IEMG was less in the right quadriceps than in the left at each force tested at 90° of knee flexion (Table AP.4.).

Important normal findings. 1) Symmetrical and normal sural and saphenous sensory action potentials ("making femoral neuropathy very unlikely") (Table AP.5.). 2) Symmetrical and normal distal motor latencies from the femoral nerve to RF (Table AP.6). 3) Symmetrical and normal motor nerve conduction velocity in the lateral popliteal nerve (Table AP.6.).

Abnormal but unexplained findings. 1) The muscle action potential over the right extensor digitorum brevis was smaller than normal and smaller than the left (Table AP.6.). 2) Asymmetry of the knee jerk reflex times (R>L near full extension; R<L at 90° flexion; Table AP.6.). (This might possibly be explained by some mechanical factor such as 'floppiness' of the quadriceps on the injured side or by differences in receptor activation.)

Neurophysiology III

Quadriceps H-reflex was tested by the author in the Department of Zoology, Oxford, using the same equipment used to study the normal subjects in Chapters 10 & 11. Recordings were made on both legs over RF in extension and at 40° knee flexion, at rest and during a small quadriceps contraction (see Chapter 2 for details).

	Knee Joint Angle		
	0°	30°	90°
LEFT IREMG (units)	1.10	1.90	1.85
RIGHT IREMG (units)			
Before Lignocaine	0.25	0.70	1.05
15 Minutes After Lignocaine	0.30	1.10	-

Table AP.3. Quadriceps activation at different joint angles and 15 minutes after infiltration of the right prepatellar scar with a local anaesthetic (lignocaine).

	Force (% MVC)			
	25	50	75	100
LEFT IREMG (units)	0.30	0.80	1.50	1.85
RIGHT IREMG (units)	0.30	0.55	1.85	1.05

Table AP.4. Relationship between integrated rectified EMG (IREMG) and force (% maximum) tested with the knee flexed to 90°.

	Amplitude V	lat peak ms	lat onset ms	cond vel m/s
LEFT				
Sural SAP	11	3.4	2.8	5Ø
Saphenous SAP	4	3.2	2.5	48
RIGHT				
Sural SAP	7	3.4	2.7	52
Saphenous SAP	4	3.Ø	2.4	5Ø

Table AP.5. Neurophysiology II Results. Sensory study of sural and saphenous nerve sensory action potentials (SAP).

	MAP mV	MAP mV	DML ms	MCV m/s
LEFT				
Lateral Popliteal	12.0	12.0	3.7	60
KJ Reflex Time				
Full Extension	15.0			
90° Flexion	15.0			
DML to RF	3.7			
RIGHT				
Lateral Popliteal	2.0	1.5	3.5	58
KJ Reflex Time				
Full Extension	17.0			
90° Flexion	12.0			
DML to RF	3.8			

Table AP.6. Neurophysiology II Results. Motor study of the lateral popliteal nerve motor action potential (MAP), distal motor latency (DML) and motor conduction velocity (MCV)), knee jerk (KJ) reflex times, and DML to rectus femoris.

The H-reflex could not be elicited in either limb at rest, which was the case with the majority of the normal subjects studied in Chapters 10 & 11. Considering the uninjured limb first, the reflex could be demonstrated in extension and flexion during a 15-20% maximal quadriceps contraction. The H/M response recruitment curves, were plotted for recordings made in extension and showed similar patterns to those plotted for the normal subjects studied in Chapter 10. In the injured limb the H-reflex was absent in extension during contraction and was present in flexion , but appeared to be much smaller than in the uninjured limb. The H-reflexes were not measured because direct comparison between reflexes recorded in extension and flexion cannot be made due to differences in recording conditions caused by movement of the tissues underneath the stimulating elctrode during knee flexion (see Chapter 10).

INVESTIGATIONS FOR STUDYING REFLEX INHIBITION OF QUADRICEPS AND FOR MONITORING THE EFFECTS OF TREATMENT

These tests were performed to investigate further the theory that this patient's quadriceps weakness was due to reflex inhibtion of muscle activation, and to monitor any changes over a 16 month period.

In view of the finding in the meniscectomy patients studied in Chapter 5, and in the present patient (vide infra), that quadriceps MVA was greater when isometric contractions were performed with the knee in a flexed position rather than in extension, the present patient underwent a 7 month period of performing quadriceps exercises in flexion. The tests described below were performed by the author and were used to examine the nature of the inhibition and to evaluate the effectiveness of treatment.

Timing of Tests

The dates of tests are shown in Table AP.7.

Date	MVC	CSA	MVA
1983			
26 Jan	+		+
16 Feb	+	+	+
22 Sep	+	+	+
10 Nov	+	+	+
15 Dec	+	+	+
1984			
25 Jan	+	+	+
7 Mar	+	+	+
2 May	+	+	+

Table AP.7. Timing of investigations of reflex inhibition and tests for monitoring treatment in a patient with peri-articular knee joint pathology.

Voluntary Activation of Quadriceps

Quadriceps MVA was tested with the subject supine for tests at knee angles of 0^{o}, 10^{o}, 40^{o} and 70^{o} of flexion. For tests at 90^{o} knee flexion the subject was seated in the chair used to measure isometric strength (Chapter 2). On 1 occasion (26.1.83) MVA was tested with a "Cho-pat" knee brace (a band placed below the patella). On another occasion (16.2.83) MVA was observed after an injection of 0.5ml 2% lignocaine into the painful area at the prepatellar scar, and after a second injection of 1.5ml given 15 minutes after the first. Quadriceps MVA was measured on 8 occasions.

Joint angle did not influence MVA in the uninjured limb. Flexion always increased MVA in the injured limb and the values at the different knee angles were also unchanged (as well as the values in extension) throughout the test period (Table AP.8.). The maximum value for quadriceps MVA (38 IEMG units) was achieved at 70^{o} of knee flexion and this was similar tho the maximum MVA of the left quadriceps (39 IEMG units) at the same angle. On 2.5.84 MVA was only measured in extension and 1 angle of flexion as the patient was complaining of a lot of pain on that day (Table AP.8.).

Quadriceps MVA in extension remained unchanged in both limbs throughout the 16 month test period (Table AP.8.) despite the 7 month period of exercises in flexion. Perhaps inhibition, which operated for a greater proportion of time than the periods of exercises, outweighed the effect of flexion and only permitted short-lived improvement in activation. The "Cho-pat" knee brace did not significantly increase MVA in extension (without brace, MVA = 14 IEMG units; with brace, MVA = 16 IEMG units) but pain during contraction was relieved. The lignocaine injections into the site of the prepatellar scar significantly increased MVA in and the highest

	Quadriceps MVA (IEMG units)				
	Degrees of flexion				
Date	0	10	30	70	90
LEFT					
26.1.83	40	–	–	–	–
16.2.83	40	40	40	39	40
22.9.83	40	41	35	39	41
10.11.83	39	42	31	24	–
15.12.83	37	38	38	36	40
25.1.84	37	39	35	35	–
7.3.84	38	37	37	34	49
2.5.84	39	35	33	33	–
RIGHT					
26.1.83	14	–	–	–	–
16.2.83	12	29	37	38	36
22.9.83	11	19	25	29	28
10.11.83	9	25	31	29	–
15.12.83	9	24	31	33	37
25.1.84	10	23	36	34	–
7.3.84	11	30	33	36	36
2.5.84	6	–	25	–	–

Table AP.8. Measurements of maximal voluntary activation (MVA) of quadriceps at different knee joint angles made over a 16 month period.

MVA recorded for the injured limb's quadriceps in extension (10 mins after the second injection) was 31 IEMG units. Some sample results of the effect of knee flexion (recorded on 16.2.83), and the effect of lignocaine on quadriceps MVA are shown in Fig AP.2.

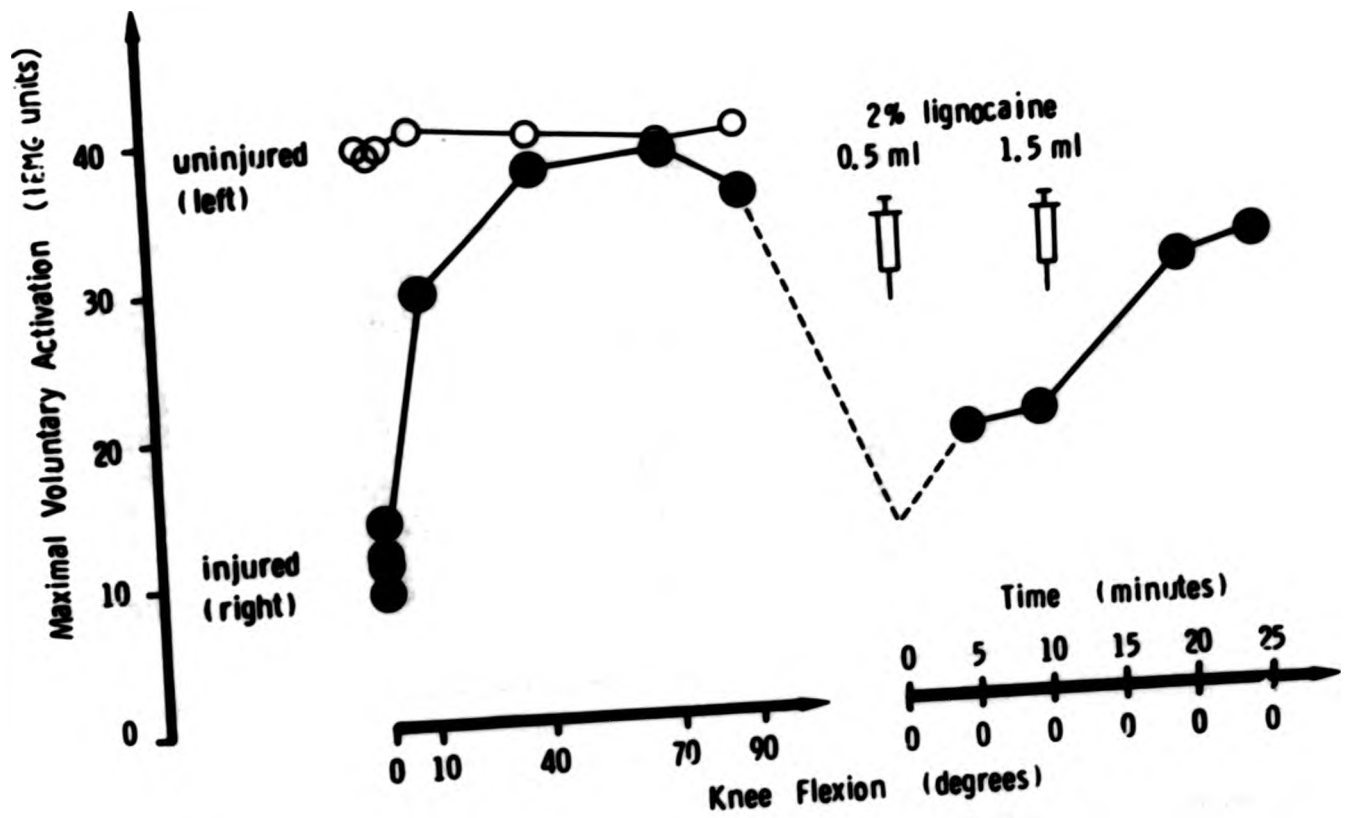
The effects of local anaesthesia and knee angle on MVA were in keeping with the results of studies of inhibition in the present series of meniscectomy patients (Chapters 3 & 5). The peri-articular injections of lignocaine confirmed that the inhibitory stimuli originated from the pre-patellar region of the knee.

Quadriceps Isometric Strength

Maximal voluntary contractions (MVC) of quadriceps were recorded as described in Chapter 2. Tests were performed on 8 occasions. On 1 occasion (26.1.83) MVC was tested with the "Cho-pat" knee brace, and on another occasion (16.2.83) MVC was tested before and 15 minutes after the second prepatellar injection of lignocaine.

During the 16 month test period the strength of the left quadriceps did not change significantly (Table AP.9.). There was no overall change in the right quadriceps' strength but on 1 ocasion (22.9.83) the MVC fell to 54 N but these tests were limited by pain. Table AP.9. shows the results for MVC in both legs on 8occasions between 26.1.83 - 2.5.84. The mean expected values for MVC in the stronger limb were calculated according to body weight and 70% or more of the expected value is considered normal (Edwards et al.,1977). The injured / uninjured limb ratios are also reported. Overall, the right quadriceps were 80% weaker than the left quadriceps.

Quadriceps MVC in the injured limb was not increased with the use of the "Cho-pat" (without brace MVC= 87 N;with brace =85 N), or the prepatellar injections of lignocaine (before injection = 87N; after injection = 77 N).



Patient: S.C. (♀37)

Figure AP.2. Effect of knee flexion and subcutaneous local anaesthesia on voluntary quadriceps activation.

Date	Weight (kg)	Quadriceps LEFT (N)	Quadriceps RIGHT (N)	MVC inj uninj (%)
26.1.83	56.5	414	87	21
16.2.83	56.5	396	87	22
22.9.83	-	416	54	13
10.11.83	56	403	76	19
15.12.83	55	368	78	21
25.1.84	-	382	89	23
7.3.84	55	450	92	20
2.5.84	53.5	414	89	21
Median	55	408.5	87	21

Table AP.9. Measurements of voluntary isometric strength of quadriceps made over a 16 month period. Overall, the right quadriceps was 80% weaker than the left.

The patient sometimes experienced pain whilst making a maximal contraction at 90° knee flexion but denied that pain limited her effort. Relaxing after a contraction was usually painful and it took her a long time (2 - 3 s) to release the contraction. Releasing the contractiuon became easier during the 16 month test period.

Whole Quadriceps Cross-sectional Area (CSA)

Compound ultrasound B-scanning produces an image of quadriceps which allows accurate measurement of its CSA (Ikai & Fugunaga,1968; Young et al.,1980). A conventional diagnostic ultrasound scanner with grey scale attachment was used (Nuclear Enterprises, 'Diasonograph NE4200').

The subject lay supine and scans were made at mid-thigh, defined as halfway between the greater trochanter and the lateral joint line of the knee (Young et al., 1980). This level was marked on a transparent sheet, together with permanent skin blemishes, to allow accurate relocation for subsequent scans (Dons et al., 1979; Young et al., 1984). Four scans were made on each thigh on each occasion. A water-based coupling medium was placed on the skin and, as the ultrasound probe was moved around the surface of the thigh, an image of a section through the thigh was built up and displayed on a screen. The image was photographed to produce an ultrasonogram (Fig AP.3.). The outline of the quadriceps was traced from the ultrasonogram and its CSA measured with a MOP electronic planimeter (Reichert-Jung). The areas were converted to life-size by a calibration factor derived from equivalent photographic records of different separations of the scanner's electronic calipers. Scans were made on 7 occasions on both legs.

The day to day variability of measuring quadriceps CSA (average of 4 scans) was reported to be 4% by Young et al. (1980). In order to verify that the variability of measurements made by the author was no worse than 4%, a small repeatability study was carried

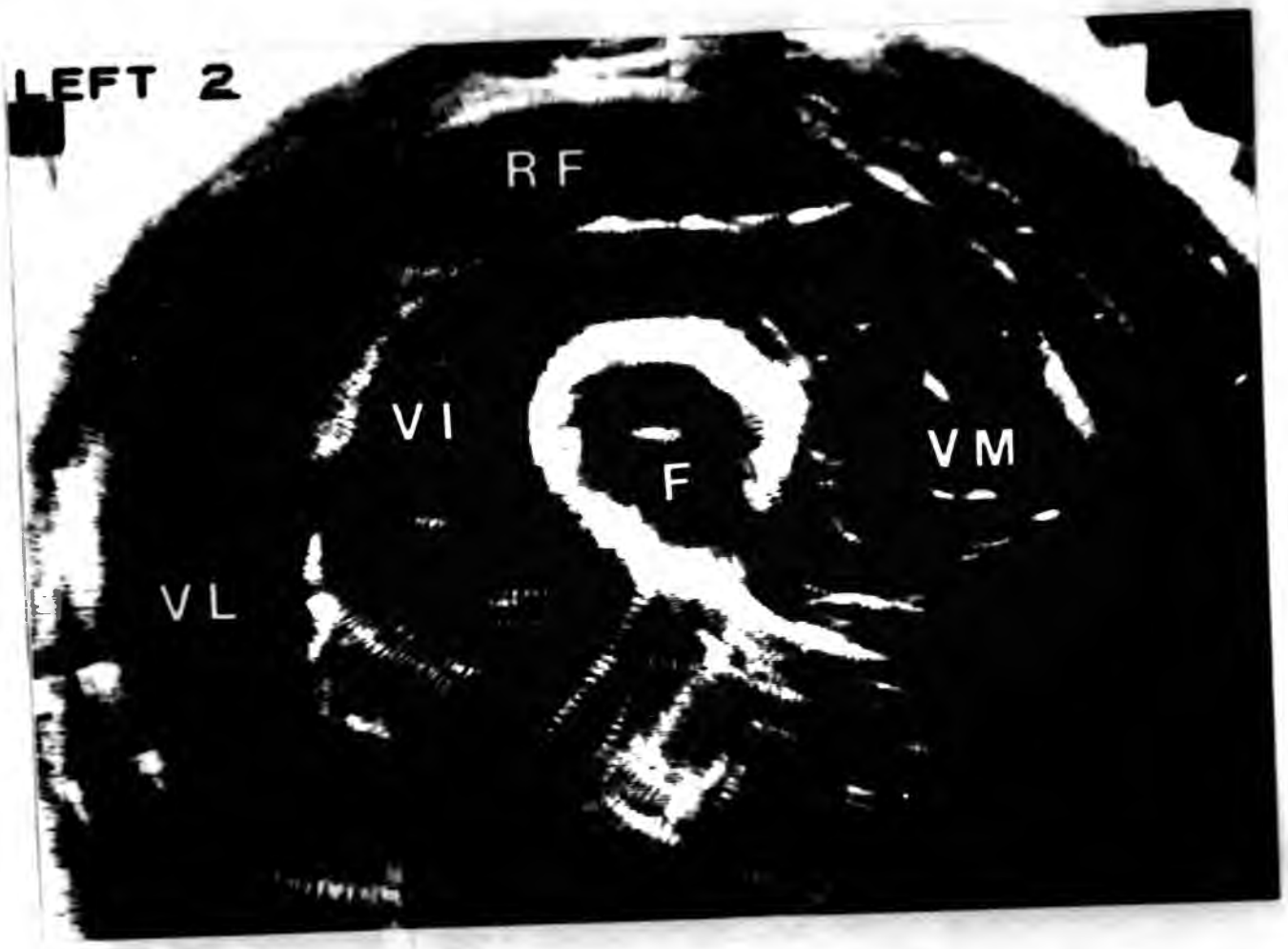


Figure AP.3. Photograph of an ultrasound scan of quadriceps made through mid-thigh. (F=femur, RF=rectus femoris, VM=vastus medialis, VL=vastus lateralis, VI=vastus intermedius).

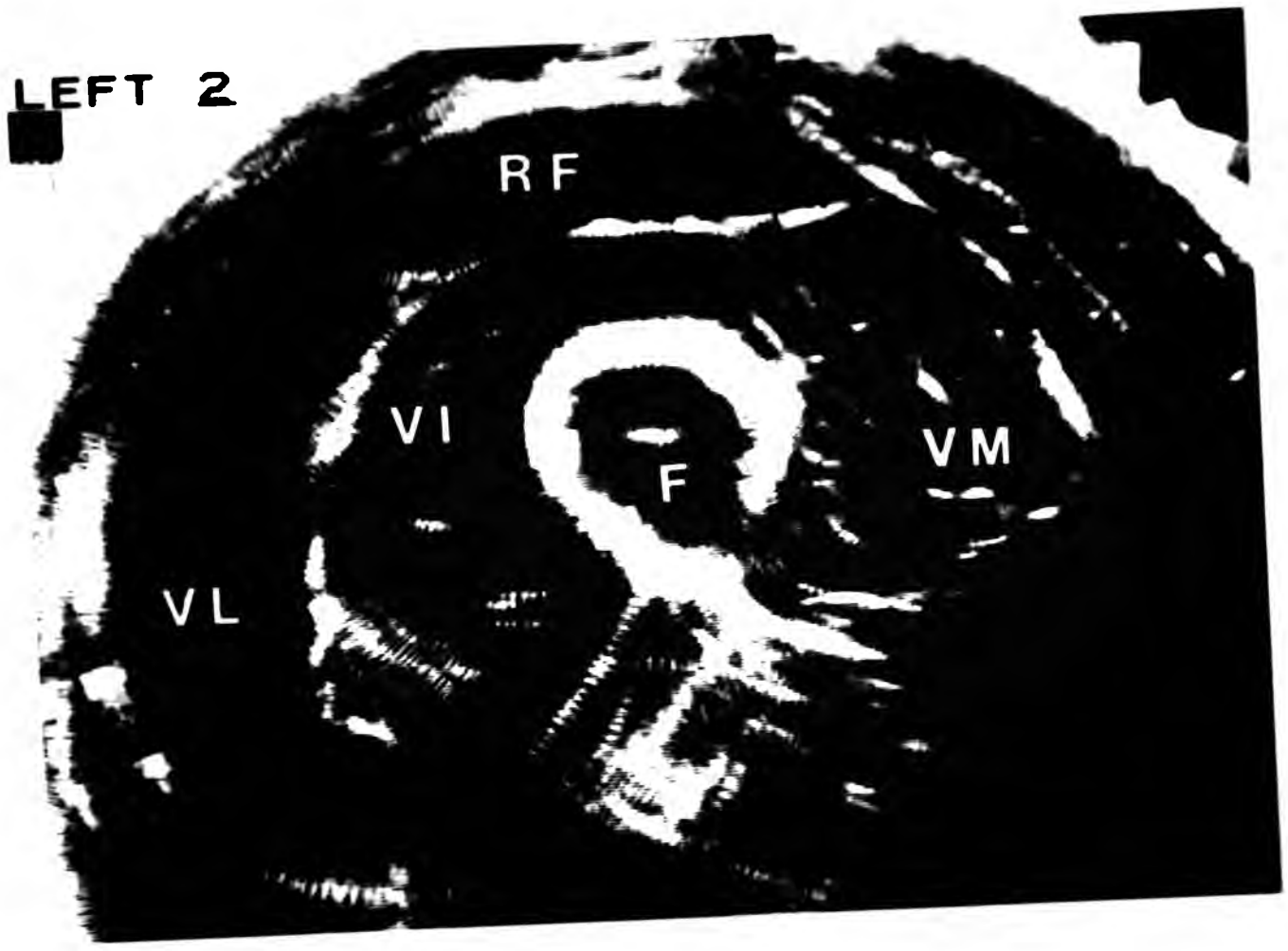


Figure AP.3. Photograph of an ultrasound scan of quadriceps made through mid-thigh. (F=femur, RF=rectus femoris, VM=vastus medialis, VL=vastus lateralis, VI=vastus intermedius).

out. Five normal female volunteers (aged 24-38 years) had both quadriceps scanned on 2 occasions. The coefficient of variation was 1.2%. This was significantly lower (P< 0.01, F max test) than the previously reported result of 4% but was probably due to the use of transparent sheets (by the author) to relocate the site for scanning. Young et al. (1980) relocated the site by measuring the height from the floor.

Quadriceps size did not change in either leg (Table AP.10.). Overall, the right quadriceps were 53% smaller than the left and the size difference is illustrated in some sample scans (Fig AP.4.). As in patients with quadriceps wasting following knee joint injury, this reduction in muscle size was not due to a loss of fibres but to fibre atrophy.

Relationship between Quadriceps Size and Strength

Measurements of the patient's quadriceps size and strength are plotted on the graph (Fig AP.5.) together with measurements obtained from normal women (Young et al.,1984). The quadriceps of the patient's uninjured limb showed normal strength for its CSA. The CSA of her injured limb's quadriceps was well below the range of sizes recorded for normal women in their 70's and the strength appeared to be approximately appropriate to the CSA (or perhaps slightly less than expected). This evaluation of strength from CSA requires extrapolation beyond the existing limits of normal data and is therefore made with reservation.

Despite the improvement in pain and function with the 7 month period of painless exercises in flexion (vide infra 'Activities and Subjective Assessments'), neither quadriceps size nor strength was altered in the injured limb. The relationship between size and strength was therefore also unaltered. When the knee was at 90 ° the wasted quadriceps appeared to be appropriately weak for its size (with the reservation mentioned earlier), suggesting that the muscle was not being inhibited in this position.

Date	Quadriceps CSA LEFT (cm^2)	RIGHT (cm^2)	inj uninj (%)
16.2.83	47	21	45
22.9.83	50	20	40
10.11.83	48	23	48
15.12.83	48	22	46
25.1.84	47	23	49
7.3.84	45	24	53
2.5.84	46	23	50
Median	47	23	48

Table AP.10. Measurements of quadriceps cross-sectional area at mid-thigh made over a 16 month period.

i

ii

Figure AP.4. Sample scans showing the difference in the sizes of the 2 quadriceps at mid-thigh level.
i. left (uninjured) ii. right (injured)

Figure AP.4. Sample scans showing the difference in the sizes of the 2 quadriceps at mid-thigh level.
i. left (uninjured) ii. right (injured)

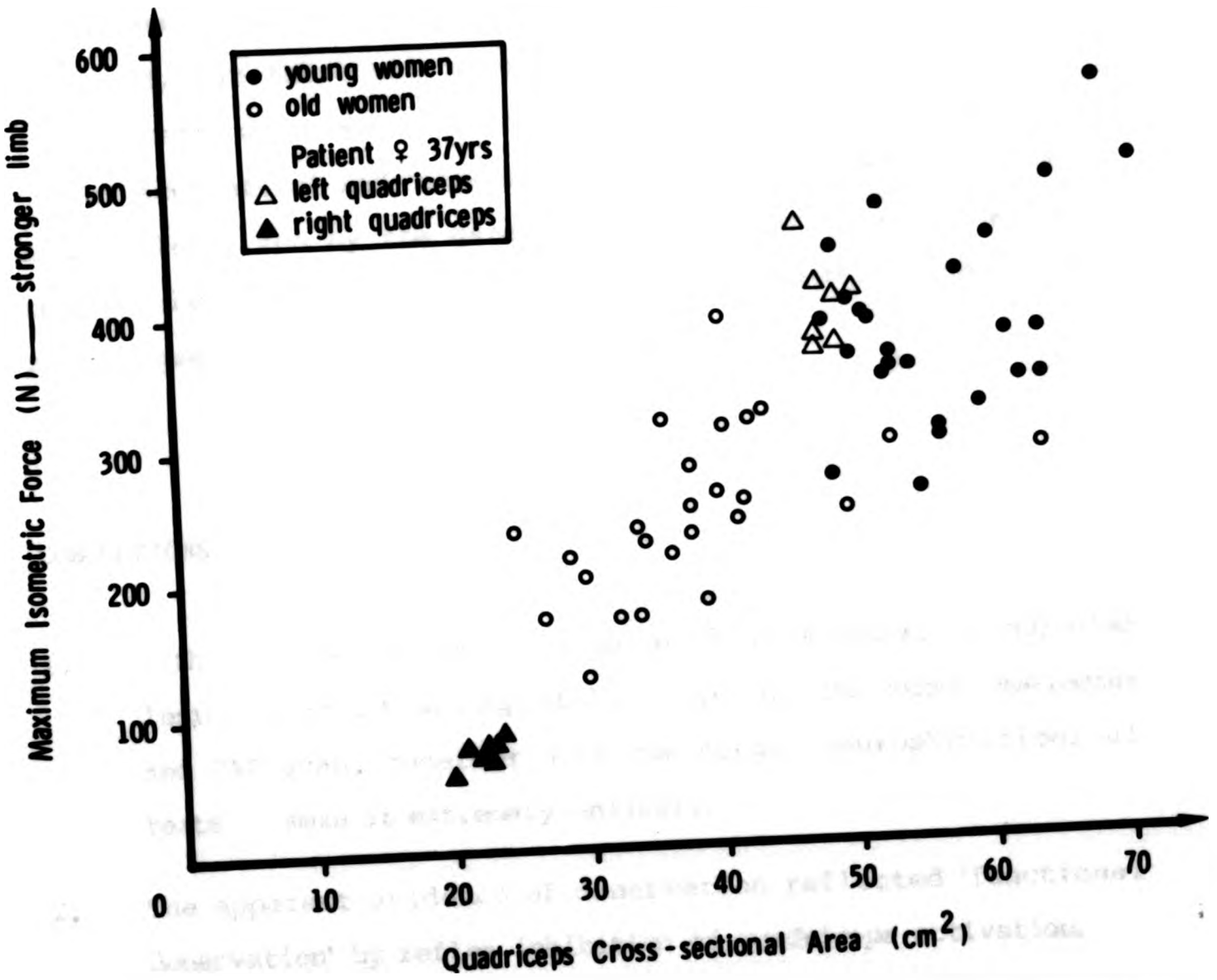


Figure AP.5. Quadriceps size and strength relationship in the present patient compared with those of normal old and young women.

Activities and Subjective Assessments

On each day that she was seen, the patient was asked to give a written record of her exercises and leisure activites (and pain experienced during these), together with her own subjective impression of her progress.

After starting the regime of isometric quadriceps exercises in flexion her right leg felt stronger and her walking improved, but there was little or no change in pain. Further improvements were seen in pain and function after she began having 'laser acupunture' and 'electro-acupuncture' treatment (details not available) given by the general practicioner to whom she was a receptionist. Despite these subjective improvements, no changes were seen in quadriceps size, strength, or activation.

CONCLUSIONS

1. Although true 'anatomical denervation' by a neural or radicular lesion could not be completely ruled out, the normal myelogram and CAT scan, together with the normal neurophysiological tests, make it extremely unlikely.

2. The apparent evidence of denervation reflected 'functional denervation' by reflex inhibition of quadriceps activation.

3. Inhibitory afferent stimuli, other than just pain, arose in her case from the prepatellar scar.

4. The effects of local anaesthesia, and the effects of knee angle on inhibition were similar in the present patient to those observed in meniscectomy patients, even though the stimuli probably originated from different areas of the knee joint.

5. The apparent preservation of the knee jerk might be explained by the fact that the test was performed with the knee flexed.

6. The lack of response of the quadriceps (regarding size, strength, and activation) to isometric exercises, despite the fact that they were performed in flexion, implied that it was not possible to improve quadriceps function while the inhibitory stimuli still operated between periods of exercise. It was therefore suggested that surgical exploration of the prepatellar scar might be performed to find the source of the inhibitory afferent stimuli (possibly a neuroma or an undissolved suture).

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COLLECTION AND ANALYSIS OF DATA

Measurement of Maximal Voluntary Activation of Quadriceps and Knee Pain

Surface EMG tests were performed by the author or one of two Orthopaedic Surgeons - Mr DT Shakespeare or Mr KP Sherman. All data were analysed by the author. (Chapters 3,4,5,6,& 7) Needle EGM was performed on 1 subject by Dr A Young (Clinical Lecturer and Honorary Consultant Physician). (Chapter 8)

Measurement of Maximal Isometric Quadriceps Strength

All tests were performed by the author except when the author acted as an experimental subject when Dr A Young performed the tests. The data were analysed by the author. (Chapter 4 & Appendix).

Application of a Pneumatic Tourniquet in Normal Subjects

Mr D Barber, a senior theatre technician, applied the tourniquet. (Chapter 4)

Impulse of One-legged Leaps

This was tested by Dr M Whittle (Consultant Clinical Physiologist) and the results were analysed by the author. (Chapter 4)

Transcutaneous Nerve Stimulation

TNS units were applied to the knee by either Mr DT Shakespeare or Mr KP Sherman and tests of MVA and pain were performed by the author. (Chapter 6)

Aspiration of Knee Joint Effusions in Post-meniscectomy Patients

Aspiration was performed by either Mr DT Shakespeare, Mr KP Sherman or Dr A Young. The results were analysed by the author. (Chapter 7)

Infusion of Normal Knees with Saline

This was performed by Dr A Young in 7 of the 8 normal subjects, and Mr KP Sherman performed the procedure on Dr A Young. (Chapters 8 & 11).

H-reflex Testing

These tests were performed, and the results analysed by either the author or Dr JF Iles.

Quadriceps Cross-sectional Area

Ultrasound scans were made and measured by the author. (Appendix)

Needle Muscle Biopsy of Quadriceps

Needle biopsy was performed by Dr A Young. Specimens were prepared for sectioning (i.e. orientated and frozen) by the author, sectioned and stained by Ms M Reading, and reported on by Dr JT Hughes. Measurements of fibre size and frequency were made by the author. (Appendix)

Neurophysiology

Needle EMG tests on normal subjects after voluntary ischeamis were performed by Dr K Mills. (Chapter 4) The patient described in the case report in the Appendix was studied by Dr K Mills, Dr G Rushworth and the author. Each examiner analysed their own results.

Illustrations

All graphs and diagrams were drawn by the author and photographs were taken by Mr P Cooper.

PROGRAMME OF ADVANCED STUDIES

In partial fulfilment of the requirements of the degree of Doctor of Philosophy, the candidate has undertaken the following programmes of advanced study :

1. Lectures on Disorders of the Musculo-skeletal System (2 years) in the Nuffield Department of Orthopaedic Surgery, Nuffield Orthopaedic Centre.

2. 'Oxford Muscle Club' seminars (2 years) at the Department of Zoology, University of Oxford.

3. Modular course (1 term) in computer programming at Oxford Polytechnic. Passed examination with Grade A.

4. Undergraduate lectures (1 term) on the physiology of motor control, at the Department of Physiology, University of Oxford.

5. Departmental seminars (1 term) on the physiological sciences, at the Department of Physiology, University of Oxford.

6. Attended and presented work at scientific meetings of the following societies :

   Alternative Muscle Club
   Chartered Society of Physiotherapy
   European Society for Clinical Investigation
   Medical Research Society
   Physiological Society
   Society for Research in Rehabilitation

PUBLICATIONS

Clinical Science

Editorial Review

This Editorial Review is from *Clinical Science*, a major monthly British journal devoted to clinical investigation and one of the world's leading publications in this field. The scope of papers published in *Clinical Science* is very wide and includes the fundamental aspects of biochemistry as it affects the clinical field. Editorial Reviews appear regularly in the journal. *Clinical Science* is published by the Medical Research Society and the Biochemical Society. Two volumes of six parts, each of about 100 pages, are published each year. Orders for *Clinical Science* should be placed with a Bookseller or Subscription Agent, or sent to the Biochemical Society Book Depot, P.O. Box 32, Commerce Way, Colchester, CO2 8PH, Essex, U.K. The subscription price in 1984 is £95.00 (U.S. $190.00) per annum, post free.

A list of the Editorial Reviews that have appeared in recent volumes of *Clinical Science* is given overleaf.

CLINICAL SCIENCE EDITORIAL REVIEWS

1982–1983

Epidemiology of hypertension: blood pressure in blacks and whites. J. K. CRUICKSHANK and D. G. BEEVERS

Interpretation of thoracoabdominal movements during breathing. M. D. GOLDMAN

Expanding views on ischaemic heart disease: a perspective for the 1980s. A. MASERI

The roles of C-apolipoproteins in the metabolism of triglyceride-rich lipoproteins. J. P. D. RECKLESS, J. STOCKS and G. HOLDSWORTH

Assessment of sympathetic nervous function in humans from noradrenaline plasma kinetics. M. ESLER

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Cholesterol transport through the plasma. N. B. MYANT

Mechanisms of gastric mucosal protection: a role for the 'mucus–bicarbonate' barrier. W. D. W. REES and L. A. TURNBERG

Dopamine and the kidney. M. R. LEE

Insulin biosynthesis and its regulation. I. L. CAMPBELL, L. N. B. HELLQUIST and K. W. TAYLOR

Measurement of cerebral blood flow and metabolism in man. N. A. LASSEN

The cell surface and disease. G. E. GRIFFIN and C. A. PASTERNAK

Biochemical assessment of adrenoceptor function and regulation: new directions and clinical relevance. S. R. NAHORSKI and D. B. BARNETT

Gastric emptying: a clinical perspective. R. C. HEADING

The role of hormones in congestive heart failure: new frontiers in therapy? G. A. TURINI and H. R. BRUNNER

Normal immune responses in the gut and liver. J. R. HODGES and R. WRIGHT

Kinetic analysis of transport processes in the intestine and other tissues. M. L. G. GARDNER and G. L. ATKINS

The natriuretic hormone: recent developments. H. E. DE WARDENER and E. M. CLARKSON

The use of stable isotopes for diagnosis and clinical research. D. HALLIDAY and M. J. RENNIE

The role of pituitary gonadotrophin-releasing hormone receptors in the physiological regulation of gonadotrophin secretion. R. N. CLAYTON

Luxuskonsumption, diet-induced thermogenesis and brown fat: a critical review. G. R. HERVEY and G. TOBIN

Luxuskonsumption, diet-induced thermogenesis and brown fat: the case in favour. N. J. ROTHWELL and M. J. STOCK

Proteolytic enzymes, their inhibitors and lung disease. R. A. STOCKLEY

Hepatic clearance of serum glycoproteins. I. G. MCFARLANE

Biochemical mechanisms of hepatic encephalopathy. I. R. CROSSLEY, E. N. WARDLE and R. WILLIAMS

Digitalis intoxication. J. K. ARONSON

Molecular variants of the lipoproteins. D. J. GALTON, J. STOCKS and A. REES

Organ culture in the study of the gastrointestinal tract in health and disease. P. D. HOWDLE

3-Methylhistidine as a measure of skeletal muscle protein breakdown in human subjects: the case for its continued use. F. J. BALLARD and F. M. TOMAS

3-Methylhistidine excretion and the urinary 3-methylhistidine/creatinine ratio are poor indicators of skeletal muscle protein breakdown. M. J. RENNIE and D. J. MILLWARD

The transport of thyroid hormones. R. HOFFENBERG and D. B. RAMSDEN

Dietary fat and platelet function. T. A. B. SANDERS

Renal function in pregnancy. J. C. ATHERTON and R. GREEN

Regulation of prolactin secretion by oestrogens: physiological and pathological significance. S. FRANKS

Protein synthesis, bodily renewal and the sleep–wake cycle. K. ADAM and I. OSWALD

Human sleep and tissue restitution: some qualifications and doubts. J. A. HORNE

Clinical Science (1984) 67, 7–14

EDITORIAL REVIEW

The contribution of reflex inhibition to arthrogenous muscle weakness

This Editorial Review is based on Dr Young's essay 'The pathophysiology of arthrogenous muscle weakness: lessons for rehabilitation', winner of the 1983 Eli Lilly Prize of the Medical Research Society.

MARIA STOKES AND ARCHIE YOUNG

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'Arthrogenous muscle weakness' is weakness of muscles acting about an injured or inflamed joint. The weakness may be due to loss of muscle or to inability to activate the muscle (Fig. 1). Weakness of the thigh muscles, and of the quadriceps in particular, is a common and important consequence of knee trauma, surgery or arthritis. Muscle weakness contributes significantly to disability and probably also renders the joint vulnerable to further damage (Fig. 1). This review starts with a brief discussion of the contribution of atrophy to weakness. It concentrates, however, on inhibition of quadriceps activation and suggests some therapeutic implications. It does not deal with the reduced oxidative capacity and increased fatigability of disused muscle since, although important, these have not been part of our programme of work.

Quadriceps wasting

Measurement

Traditionally, thigh muscle wasting is assessed from measurements of thigh circumference. The tape measure is too insensitive and inaccurate for measuring the selective wasting of the quadriceps which occurs with knee damage because it also encloses a large bulk of other muscles and a layer of subcutaneous fat. Transverse imaging techniques, such as computerized axial tomography [1, 2] and ultrasonic compound B-scanning [3], allow accurate measurement of the cross-sectional area of the quadriceps itself and should be used for any studies involving changes in quadriceps size. The inadequacy of the tape measure has been demonstrated by ultrasonic studies of selective quadriceps wasting [3] and growth [4].

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Selective wasting of heads of quadriceps

It is commonly believed that vastus medialis wastes more than the other three heads of the quadriceps. However, the few direct data that are available are more suggestive of a uniform atrophy [5, 6].

Fibre atrophy or fibre loss?

Studies of human muscle, using the needle biopsy technique [7], suggest that quadriceps wasting due to injury and/or immobilization of the

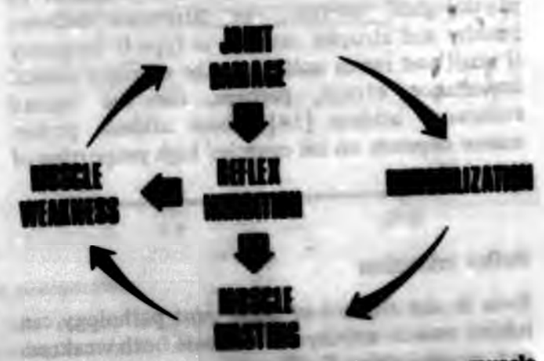

FIG. 1. 'Vicious circles' of arthrogenous muscle weakness.

knee is caused by atrophy of fibres [6]. This differs from the wasting seen in elderly muscle which is thought to involve both loss in fibre size and fibre number [8], with the latter perhaps predominating [9].

Selective atrophy of fibre types

Fibre-typing according to alkali-stable myosin ATPase activity may reveal striking changes in the relative sizes of type I and type II fibres. Atrophy of type II fibres occurs in a wide variety of conditions [7]. Since it is the high-threshold motor units that comprise type II fibres, it is generally held that selective type II fibre atrophy reflects a sick patient's general inactivity. It is no surprise, therefore, that some patients with arthrogenous quadriceps atrophy show preferential atrophy of their type II fibres [e.g. 6, 10]. Preferential atrophy of type I fibres is not a common feature in muscle histopathology. Nevertheless, in the context of arthrogenous wasting, it is as common as type II atrophy [11].

It is far from clear which clinical features determine whether there is selective atrophy of one or other fibre type [11, 12]. An understanding of the mechanisms responsible for preferential fibre-type atrophy would allow the prediction of the likely pattern of atrophy in any given clinical situation and the individual patient to be given the most appropriate treatment. For example, treatment might involve removing the cause of the selective atrophy and then performing specific exercises tailored to stimulate growth of the more atrophic type of fibre [7, 12]. The possibility that selective atrophy might result from selective inhibition is discussed later.

Irrespective of which fibre type is more atrophic, it seems that arthrogenous atrophy may be associated with an increase in the frequency of fibres with a high activity of alkali-stable myosin ATPase (type II fibres) [6, 13]. Whatever its physiological interest, the difference between healthy and atrophic muscles in type II frequency is small and seems unlikely to be of major clinical importance except, perhaps, for the injured endurance athlete [14] whose athletic performance depends on his muscles' high proportion of type I fibres.

Reflex inhibition

Even in the absence of pain, joint pathology can inhibit muscle activity and so cause both weakness and wasting (Fig. 1). The dependence of arthrogenous weakness on afferent stimuli from the joint and its dissociation from pain were documented last century, and the mechanism was judged to be a reflex [e.g. 15]. Reflex inhibition has been observed in different clinical situations, e.g. in the quadriceps after minor knee trauma [16], in the elbow flexors after elbow dislocation [17]. It is commonly seen in the quadriceps after knee surgery; patients often describe their inability to contract their quadriceps as a lack of 'control' over the muscle. Although the existence of reflex inhibition is still recognized [e.g. 18], few appreciate its potency. It is often ascribed to pain [19] and there has been little attempt to find methods for its removal other than by the relief of pain.

Measurement

Strength/cross-sectional area. Healthy women in their twenties and in their seventies show the same close relationship between the quadriceps' isometric strength and its cross-sectional area (CSA) but the older women are 35–40% weaker and their quadriceps' CSA is 35–40% less [20]. The strength of a young female patient's wasted quadriceps can therefore be judged against that of a similarly sized muscle in a healthy elderly woman. 'Excessive' weakness, whether due to pain, reflex inhibition or an intrinsic defect of muscle contractility, can therefore be quantified. Although this approach may be helpful, it has its limitations. Firstly, it cannot be used in young men; although the relationship between quadriceps size and strength in old men is similar to that in both young and old women, the quadriceps strength of some young men is substantially greater than would be expected from the size of the muscle [21]. Perhaps, even in young men, there might be a place for comparing the quadriceps strength/CSA of the injured with that of the uninjured limb (rather than with a normal range). Secondly, many patients with knee pathology are unable to flex the joint to 90°, the angle at which the standardized strength measurements were made. Thirdly, as discussed below, the severity of quadriceps inhibition may be less when the knee is in flexion than when it is in extension [22]. Finally, if type II fibres have a greater isometric specific strength than type I fibres [23, 24] severe selective atrophy of type II fibres would result in a disproportionate loss of strength/CSA.

Maximal voluntary activation. The level of quadriceps activation achieved during maximal voluntary isometric contractions can be measured by integration of the rectified electromyogram, recorded with surface electrodes at a fixed site over the quadriceps. In a patient with unilateral quadriceps wasting, the maximal voluntary activation (MVA) recorded for the two quadriceps should be almost the same. There may be a small difference in MVA due to the unequal recording conditions but major differences can be considered a measure of inhibition.

Meniscectomy as a model for investigating quadriceps inhibition

We have used arthrotomy and meniscectomy as a model of controlled knee injury, studying the magnitude and duration of postoperative quadriceps inhibition and seeking methods for its reduction or prevention.

Bilateral measurements of MVA of quadriceps were recorded over rectus femoris (at full knee extension) before surgery. All subsequent measurements were then expressed as 'percentage inhibition' (i.e. percentage reduction in MVA). Knee pain experienced during each contraction was recorded on a linear analogue scale (horizontal line without word cues) and expressed as a percentage of the length of the line.

Magnitude and duration. Inhibition in the immediate postoperative period is severe (typically 50–70%), tends to become even more pronounced over the first 24 h (80%), and by 3–4 days postoperatively is still very severe (typically 70–80%) (Fig. 2). Even 10–15 days postoperatively there is still some 35–40% inhibition despite the fact that patients have been discharged from hospital and are fully weight-bearing.

Role of pain. Dorsal root section prevents muscle atrophy secondary to experimental arthritis [15, 25] but not secondary to disuse [25]. Harding observed that her experimental animals with artificially induced arthritis did not seem to be in pain and so argued that dorsal root section was blocking an inhibitory afferent stimulus other than pain [25].

During the first 24 h after meniscectomy, pain during contraction may be quite severe (typically 50–70%) but, unlike inhibition, it is usually only

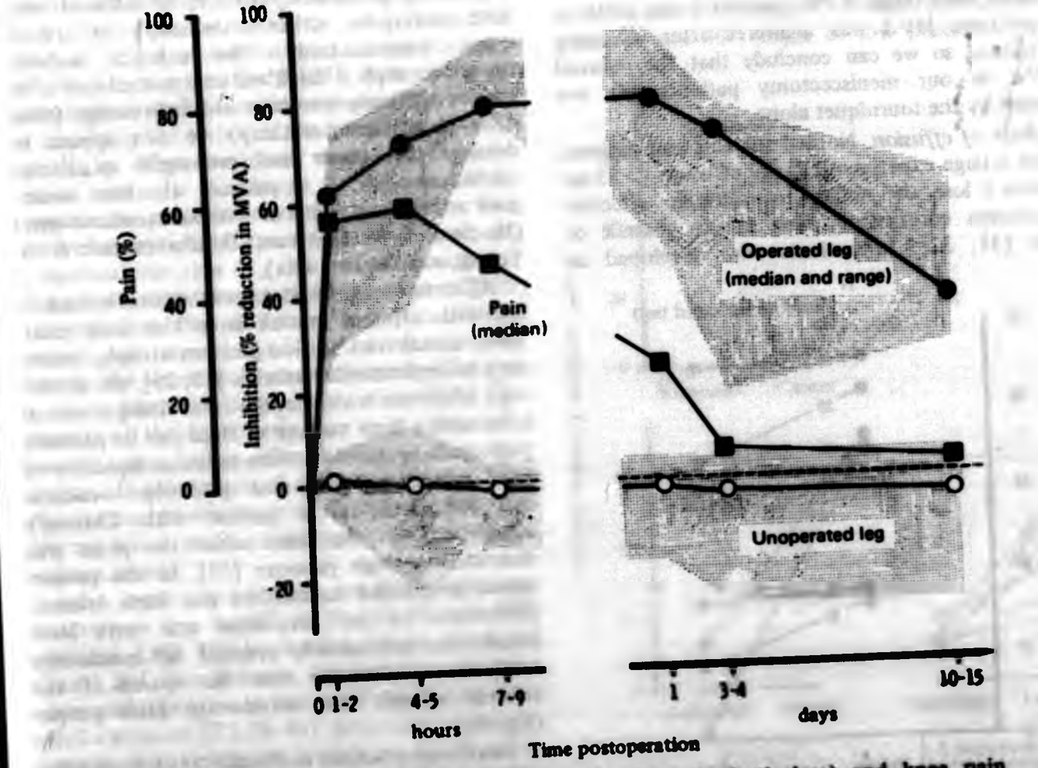

FIG. 2. Quadriceps inhibition (median and range, in both legs) and knee pain (median, in the operated leg) recorded during maximal voluntary isometric contractions (in extension), after medial meniscectomy. In the one patient who developed an effusion, recordings were made after its aspiration.

mild by 3–4 days (1–15%) [26]. Ten to 15 days postoperatively pain is mild or absent (0–10%) (Fig. 2). Another example of a clear dissociation between pain and inhibition was seen in a study in which we attempted to block the inhibitory afferent stimuli. Pre-operative infiltration of the meniscal bed and surrounding tissues with 15 ml of 0.5% (75 mg) bupivacaine temporarily prevented most of the pain and most of the inhibition. If only 10 ml were used, however, there was no change in the severity of inhibition although most of the pain was still prevented [27].

Role of ischaemia. It has been suggested that the period of tourniquet ischaemia during knee surgery contributes to postoperative quadriceps weakness [28, 29]. If this were true, our findings with the meniscectomy 'model' would be less relevant to patients with, for example, rheumatoid arthritis (i.e. with joint pathology but without tourniquet ischaemia). We therefore studied the quadriceps of four normal subjects before and after the maximal tolerable periods of unilateral tourniquet ischaemia [30]. The tourniquets were applied exactly as for meniscectomy and the durations of ischaemia (37–50 min) were comparable with those of our meniscectomy patients. Quadriceps MVA was unaltered after voluntary ischaemia so we can conclude that the reduced MVA in our meniscectomy patients was not caused by the tourniquet alone.

Role of effusion. Normal subjects and patients given a large experimental knee effusion (>55 ml approx.) lose the ability to make an effective quadriceps contraction, even in the absence of pain [31, 32]. In patients who developed an effusion post-meniscectomy, although aspiration of the effusion always reduced the severity of inhibition, it rarely abolished it (Fig. 3). Indeed, all the measurements of inhibition reported in Fig. 2 were made in the absence of any clinically apparent effusion. Perhaps congestion and inflammation of the synovium or of the capsule are also important. Nevertheless, it is clear that there are strong grounds for a less tolerant attitude to the presence of an effusion in the pathological knee.

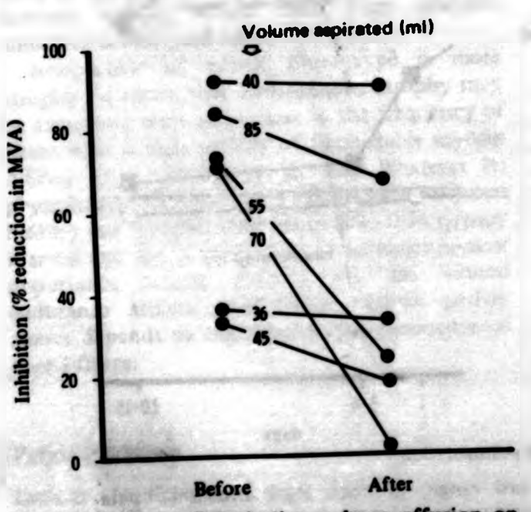

FIG. 3. Effect of aspirating a knee effusion on quadriceps inhibition, 3–5 days after meniscectomy.

Peri-articular pathology. Stimuli from peri-articular tissues have been shown to cause quadriceps inhibition [e.g. 33]. We have seen this in a patient with profound quadriceps wasting and weakness 2 years after the excision of a calcified prepatellar haemotoma (Fig. 4, and below). Perhaps inflammation around the incision causes inhibition after meniscectomy. In decerebrate cats, pinching the anterior aspect of the knee joint capsule inhibited the quadriceps stretch reflex and also inhibited quadriceps activity in the crossed extensor response [34]. Perhaps tight suturing of the capsular incision has a similar inhibitory effect. This suggestion is compatible with some preliminary studies of patients who have undergone either arthroscopy or arthroscopic meniscectomy. The capsular incision for arthroscopy is small and so is not sutured after surgery. Patients who have had arthroscopy (with or without meniscectomy) do not appear to develop the severe and prolonged quadriceps inhibition observed in patients who have undergone arthrotomy (with or without meniscectomy) (M. Stokes, K. Sherman, D. Shakespeare & A. Young, unpublished data).

Afferent block. Quadriceps inhibition is associated with afferent stimuli from the knee joint. Thus, dorsal root section prevents atrophy secondary to experimental arthritis [15, 25], the quadriceps inhibition which results from filling a normal knee with a large volume of fluid can be partially prevented by previous intra-articular injection of local anaesthetic [31], and quadriceps weakness did not occur in a patient with Charcot's arthropathy of the knee when the joint was distended at high pressure [31]. In the patient whose prepatellar haematoma had been excised, infiltration of the prepatellar scar with local anaesthetic substantially reduced her quadriceps inhibition, confirming that the source of the afferent stimuli was outside the joint proper (Fig. 4).

In clinical practice, if the afferent stimuli from a damaged knee could be blocked effectively by local anaesthetic (or some other method) for long periods, the patient would be able to achieve maximal quadriceps activation in this therapeutic

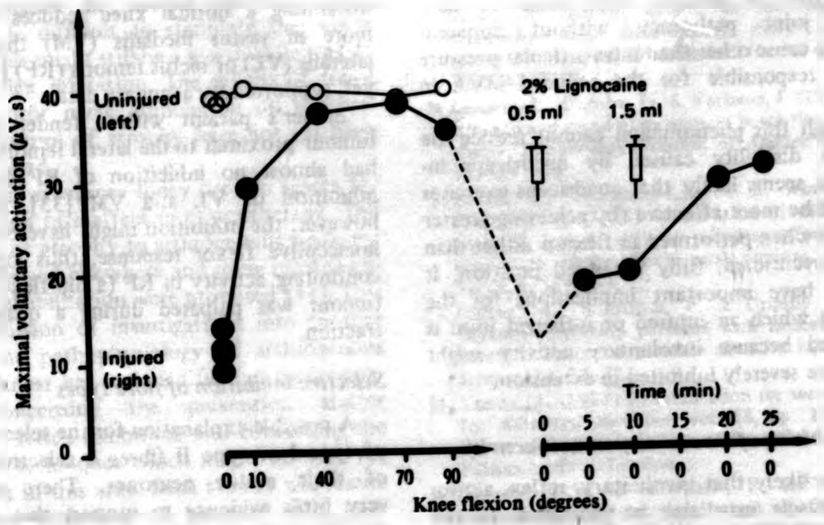

FIG. 4. Effects of knee flexion and peri-articular local anaesthesia on quadriceps inhibition in a patient 2 years after removal of a calcified prepatellar haematoma.

exercise and perhaps atrophy would be prevented. After meniscectomy, however, the protective effect of 15 ml of bupivacaine has already been lost by 5 h postoperatively [27]. Although local anaesthesia is useful for identifying inhibition and for localizing the source of the inhibitory afferent stimuli, its effect seems too short-lived to be of clinical value. I. Arvidsson, E. Eriksson & E. Knutsson (unpublished work, [19]) have described how voluntary quadriceps activation is increased, by epidural injection of a dilute local anaesthetic, in patients who have had knee surgery. They assumed that the inhibition was pain-induced because pain was blocked by the injection and because the anaesthetic was sufficiently dilute so as not to block the large motor fibres [19]. The strength of the anaesthetic may have been such, however, that other afferent fibres were also blocked. If effective in the absence of pain, light epidural anaesthesia might be useful, allowing the afferent block to be maintained for several days postoperatively.

Joint angle. An effusion inhibits quadriceps contractions less in 30° of flexion than in full extension [35]. This is probably because, in both effused and normal knees, intra-articular pressure is less with the knee in about 30° of flexion than in full extension [32, 36–38]. After meniscectomy, however, it is striking that without any clinically apparent effusion, MVA is greater during isometric quadriceps contractions in flexion than in extension [22] (Fig. 5). This also occurred in the patient with the prepatellar scar (Fig. 4). The quadriceps activation increased to similar extents at 10° and 90° (angles of high intra-articular pressure) and 40° (angle of low pressure). This

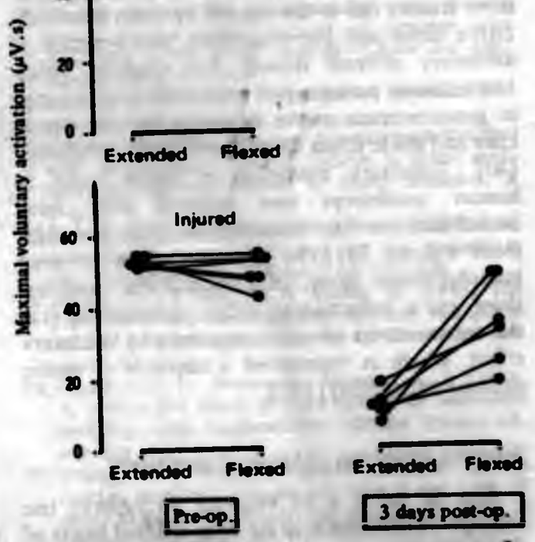

FIG. 5. Effect of knee flexion (approximately 40°) on quadriceps activation before and after medial meniscectomy.

suggests that in patients with intra- or peri-articular joint pathology without apparent effusion, a cause other than intra-articular pressure must be responsible for the reduced MVA in extension.

Although this phenomenon cannot reduce the immediate disability caused by quadriceps inhibition, it seems likely that quadriceps exercises might well be more effective (by achieving greater activation) when performed in flexion rather than in the conventional, fully extended position. It may also have important implications for the position in which an injured or inflamed joint is immobilized because involuntary activity might also be more severely inhibited in extension.

Reflex inhibition of involuntary muscle activity

It seems likely that involuntary, reflex, activation of muscle may also be inhibited. In the decerebrate cat, increasing intra-articular tension or pinching the knee capsule inhibits the monosynaptic reflex from the quadriceps [34]. The H-reflex is the muscle activation elicited, via this reflex, by electrical stimulation of Ia afferents of the femoral nerve. Reduction of the H-reflex implies reduced excitability of the motor neurone pool. In man, it has been claimed that an experimental knee effusion reduces the quadriceps' H-reflex and that intra-articular anaesthesia blocks this effect [39], but the complete data are still unpublished.

Various effects of immobilization on neural and muscular excitability have been studied but some studies fail to distinguish between immobilization alone and immobilization plus a source of inhibitory afferent stimuli. For example, group Ia excitatory postsynaptic potentials were reduced in gastrocnemius motor neurones of cats whose knee and ankle joints were immobilized by pinning [40]. Voluntary activation of motor units in human quadriceps was reduced after knee immobilization for treatment of collateral ligament injuries [41]. In a study which involved immobilization alone, of human thenar muscles, there was a reduction in reflex potentiation (i.e. the enhancement of reflex responses by voluntary effort, which is considered a function of motor neurone excitability) [42].

Selective inhibition of heads of quadriceps

Just as there is no evidence to support the concept of selective wasting of individual heads of the quadriceps, there is little published evidence to indicate that activity of a single head of the muscle can be selectively inhibited. The claim that distending a normal knee reduces the H-reflex more in vastus medialis (VM) than in vastus lateralis (VL) or rectus femoris (RF) [39] is not, as yet, supported by adequate data.

Stener's patient with a tender subperiosteal tumour proximal to the lateral femoral epicondyle had almost no inhibition of RF despite severe inhibition of VL and VM [33]. In this case, however, the inhibition might have been part of a nociceptive flexor response, thus explaining the continuing activity in RF (a hip flexor) when the tumour was palpated during a quadriceps contraction.

Selective inhibition of fibre types

A possible explanation for the selective atrophy of type I or type II fibres is selective inhibition of their motor neurones. There is, as yet, very little evidence to support this, other than the demonstration that 'painful' stimulation of the sural nerve caused selective inhibition of low-threshold motor units (presumably type I fibres) in the human biceps femoris, whereas 'tactile' stimulation caused facilitation [43]. New electrophysiological techniques [e.g. 44, 45], however, offer the exciting prospect of distinguishing changes in voluntary activity (and therefore changes in inhibition) of type I fibres from changes in activity of type II fibres. This could provide a key to understanding the clinical correlates of selective atrophy of type I or type II fibres.

Implications for rehabilitation

In the previous sections we have highlighted ways in which inhibition might be reduced in rehabilitation practice. In addition, some techniques in current use, e.g. ice, transcutaneous sensory nerve stimulation (TNS), transcutaneous motor nerve stimulation (faradic stimulation), may have an effect on reflex inhibition. Ice and TNS are used for pain relief to allow exercises to be performed comfortably but perhaps they might also be influencing inhibition. It is known that certain sensory stimuli can block other afferent sensory stimuli in the spinal cord [46]. Cutaneous sensory nerve stimulation can increase motor neurone excitability in humans [47]. Perhaps TNS might reduce quadriceps inhibition by one of these mechanisms, i.e. by preventing activation of inhibitory synapses (disinhibition), or by increasing the excitability of the anterior horn cells. We are currently testing the effect of TNS on post-meniscectomy quadriceps inhibition.

Training of the quadriceps by transcutaneous motor nerve stimulation has produced voluntary strength increases in normal subjects [48]. Although it is difficult to stimulate the whole quadriceps, this might still be a worthwhile way of bypassing reflex inhibition. The effects of transcutaneous motor nerve stimulation on the prevention or reversal of atrophy have not yet been objectively tested.

Therapeutic resources today are too scarce for the prescription of ineffective physiotherapy. The contribution of atrophy to arthrogenous weakness has been reviewed elsewhere and some recommendations for rehabilitation were proposed [11]. This present discussion of investigations into another aspect of the pathophysiology of arthrogenous muscle weakness has included further recommendations, concerning the prevention and/or reduction of reflex inhibition, and concerning the investigative techniques which might be used for further work in this area. Such studies will ensure that the rehabilitation of patients with joint damage is more scientifically based and more effective.

Acknowledgments

We are grateful to all the patients, normal subjects and co-authors who have contributed to our research programme. In particular, we are pleased to acknowledge the collaboration, at different stages, of Professor R. H. T. Edwards, Dr J. M. Round, Miss I. Hughes, Dr M. Crowe, Mr D. T. Shakespeare and Mr K. P. Sherman. We thank the Department of Health and Social Security for financial support.

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THE LANCET

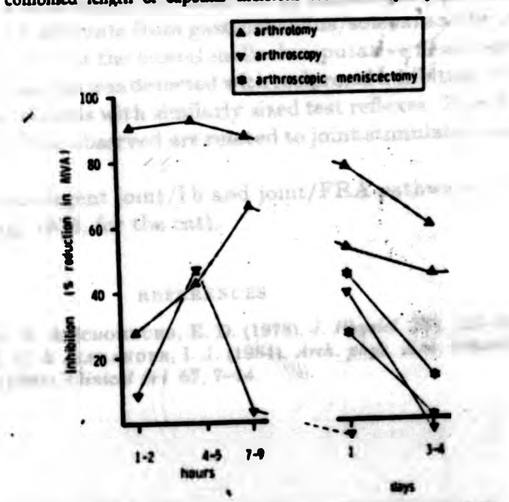
No 8403 LONDON SATURDAY 15 SEPTEMBER 1984 VOL II FOR 1984

JOINT INJURY AND MUSCLE WEAKNESS

SIR,—Your Aug 18 editorial refers to our unpublished observations comparing the severity of quadriceps inhibition after arthroscopy with that after arthrotomy. This preliminary study was intended to be followed by a more detailed investigation but nearly all meniscectomies at the Nuffield Orthopaedic Centre are now arthroscopic, so a formal, controlled comparison is no longer feasible.

Our preliminary study was on twelve male patients undergoing open meniscectomy (six), arthrotomy alone (two), arthroscopic meniscectomy (two), and diagnostic arthroscopy alone (two). Inhibition of maximum voluntary quadriceps activation (at full knee extension) was measured as the percentage reduction (from preoperative values) of the integrated rectified electromyogram recorded with surface electrodes at a constant site over the rectus femoris. The illustration shows the inhibition found post-operatively. The stippled area shows the range of postoperative inhibition for the six cases undergoing open meniscectomy. The two upper tracings show the results for the patients having diagnostic arthrotomy alone; the inhibition did not differ greatly from that in the open meniscectomy cases. The patients having arthroscopy, either alone or with arthroscopic meniscectomy showed less inhibition. For example, 24 h after arthroscopy (with or without meniscectomy) inhibition was about half that found after arthrotomy (with or without meniscectomy).

These results suggest that the arthrotomy itself is responsible for much of the inhibition. This is particularly interesting since the combined length of capsular incisions after multiple puncture



Inhibition of maximum voluntary quadriceps activation (MVA) after arthroscopy or arthrotomy.

arthroscopic techniques may equal or exceed that of an arthrotomy incision. Local tension produced by suturing of the capsule may contribute to the severe inhibition seen following arthrotomy. As you suggest, perhaps we should pay more attention to the loading imposed on the damaged capsule.

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C. 35

Reflex actions of knee-joint receptors on quadriceps in man

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Knee-joint effusion limits contraction of quadriceps. Although a fall in strength could result from reduced voluntary effort (e.g. in response to pain), there may also be spinal actions from receptors activated in the effused joint (see Stokes & Young, 1984). We have investigated the details of spinal reflex actions.

Twelve experiments were performed on eight adult subjects with informed consent. An H-reflex was elicited by stimulation of the femoral nerve and recorded over the distal part of rectus femoris. Subjects were either at rest or contracting both quadriceps (about 15% of maximum) with equal effort and the lower limbs extended. Contraction was controlled using e.m.g. biofeedback from the opposite limb. A 16-gauge cannula was inserted into the lateral aspect of the knee joint with sterile technique and without local anaesthesia. Up to 100 ml of saline were injected (and later removed). Intra-articular pressures reached more than 150 mmHg during contraction.

Increased pressure reduced H-reflex amplitude (only reflexes with similarly sized direct muscle responses were compared). This confirms and extends the observations of Spencer, Hayes & Alexander (1984) made at rest. Decline in reflex amplitude indicates inhibitory action from joint afferents on to quadriceps motoneurones. To investigate the spinal mechanisms we looked for spatial summation between joint stimulation and known pathways of quadriceps inhibition.

Summation was found with inhibition obtained by conditioning electrical stimulation of the tibial nerve (Ib afferents from gastrocnemius/soleus) and by non-noxious stimulation of the sural nerve at the lateral malleolus (putative flexor reflex afferent, FRA, pathway). No summation was detected with reciprocal inhibition. (Comparisons were made only between records with similarly sized test reflexes. This should ensure that the changes in inhibition observed are related to joint stimulation and not reflex size.)

The results suggest convergent joint/Ib and joint/FRA pathways (cf. Lundberg, Malmgren & Schomburg, 1978, for the cat).

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GALLEY PROOF

"Post-operative inhibition:" Voluntary ischaemia does not alter quadriceps function in normal subjects.

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INTRODUCTION

Quadriceps weakness following knee surgery can be due to both atrophy and reduced maximal voluntary activation (MVA). Reduced MVA, as well as producing weakness, may also contribute to atrophy. Why does this 'inhibition' of voluntary quadriceps activation occur? Pain is a possible factor (Basmajian 1970) but we have demonstrated that severe reduction of MVA can persist for several days after meniscectomy with little or no pain (Sherman *et al.* 1983) (Fig. 23.1). The presence of a knee effusion can also inhibit voluntary activation of the quadriceps (de Andrade *et al.* 1965) but large effusions are only seen in a minority of patients following meniscectomy and profound inhibition is common. It has been suggested that the use of a tourniquet during surgery may contribute to post-operative weakness. The use of a pneumatic tourniquet for meniscectomy may be associated with post-operative impairment of quadriceps function and electromyographic (EMG) evidence of minor degrees of denervation (Dobner and Nitz 1982).

The aim of the present study was to determine the contribution of the effects of a tourniquetto the post-operative reduction in MVA observed in our meniscectomy patients. We therefore studied the effects of unilateral lower-limb ischaemia on quadriceps function in a small number of normal subjects.

Although our results are essentially negative, the experiment illustrates several simple approaches to the practical evaluation of quadriceps function. The techniques used examine various aspects of the muscle's physiology, viz. nerve conduction, propagation of action potential, recruitment of motor units, and force production in isolated isometric and dynamic contractions, and coordinated dynamic movement.

SUBJECTS

The experimental subjects were four of the authors (three male, one female, aged 25-36 years). None had muscle, joint, neurological or peripheral vascular disease, or had sustained an injury to either leg sufficient to require immobilization of a joint for more than one week in the previous two years.

METHODS

Muscle function tests

Test 1

Needle electromyography (EMG) with a concentric needle electrode, was used to explore rectus femoris, vastus lateralis, and vastus medialis both at rest and during voluntary activity, for evidence of chronic partial denervation. Spontaneous activity (fibrillations, positive sharp waves, and bizzare high-frequency discharges) at more than two sites would have been considered abnormal. Interference pattern was assessed during a maximal voluntary isometric contraction of the muscle.

Distal motor latency was tested by stimulating the femoral nerve at the inguinal ligament and recording evoked potentials at a measured distal point in rectus femoris. Latencies were then compared between the two legs. The examiner was unaware of which leg had been ischaemic.

Test 2

The maximal voluntary activation (MVA) of quadriceps was measured, during maximal straight leg contractions, by integration of the rectified electromyogram (I EMG) recorded with surface electrodes at a constant site over rectus femoris (approximately mid-thigh). Skin resistance was lowered by shaving the hairs, light sand-papering of the surface to remove dead skin, and cleaning with alcohol to remove grease. The electrode sites, together with permanent skin blemishes, were recorded on transparent sheets to allow accurate relocation for subsequent testing (Dons *et al.* 1979). The subject, lying supine, was instructed to dorsiflex his foot, push his knee back towards the bed, and contract his quadriceps as hard as possible. Two two-second contractions were recorded for each leg and the maximal activity over 0.9 seconds was measured.

Test 3

Isometric quadriceps strength was measured as the force of a maximal voluntary contraction (MVC) with the subject seated and the knee at 90°. At least three contractions were made with each leg, each lasting four or five seconds. The MVC was taken to be the greatest force maintained for one second (Edwards *et al.* 1977). No visual feedback of the effort was available to the subjects.

Test 4

The force/time curve of take-off in a one-legged leap was recorded by a force plate (Kistler, Type 9821A) linked to a computer (Dec PDP-11/23). The shaded area in Fig. 23.2 was measured using a MOP planimeter (Reichert-Jung) and represents the impulse during take-off (N.s). Three leaps on each leg were performed using arm swing and knee flexion to assist take-off; the best was taken on each occasion.

Test 5

The height of a one-legged leap was measured by standing sideways to the wall and marking the wall with an inked fingertip at full stretch before and during a vertical jump. Arm swing and knee flexion were again permitted. The best of three leaps was noted for each leg on each occasion.

Period of ischaemia

A pneumatic tourniquet was applied to the thigh by a theatre technician exactly as he would do for a patient about to undergo meniscectomy at this hospital. The subject lay supine with both knees flexed over the end of the plinth. The test leg was elevated, exanguinated with a rubber Esmarch bandage, and a pneumatic cuff (9.3 cm wide) was placed around the thigh as far proximal as possible. The leg was returned to the 'meniscectomy position' (i.e. hip in neutral and knee flexed to 90°) and the cuff was then inflated to 10 lb/sq.inch (517 mm Hg). Duration of ischaemia was for as long as each subject could tolerate, viz. 37, 38, 47, and 50 minutes.

The timing of the tests is summarized in Table 23.1.

RESULTS

None of the subjects showed any evidence of impaired quadriceps function following unilateral ischaemia. The ranges of results for MVA, MVC, and impulse and height of one-legged leaps were expressed as percentages of the pre-ischaemia values, and are shown in Table 23.2.

Needle EMG at 3-4 weeks showed no evidence of denervation. One high-frequency discharge was detected in the vastus medialis of one subject, but this was in the control leg. In another subject there was fibrillation at one site in the vastus medialis of the experimental leg, but this was not considered to be abnormal. Distal motor latencies in the ischaemic legs, expressed as a percentage of those in the control legs were, 92, 102, 108, and 110 per cent, again implying no abnormality.

DISCUSSION

The various techniques used allowed different aspects of quadriceps function to be studied. Distal motor latency (measured by femoral nerve stimulation) examined nerve conduction in the distal nerve fibres supplying the muscle. The other needle EMG test, which provided a qualitative study of activation, looked for abnormal spontaneous activity in resting muscle and also at the interference pattern. The surface EMG provided a quantitative study of voluntary activation.

Isometric quadriceps strength was measured using the simple set-up of a chair and strain gauge. A 'Cybex' machine can be used to measure both isometric and isokinetic force, but in the present study, dynamic strength was measured, in dynamic coordinated movement, as the impulse and height of one-legged leaps.

Ischaemia did not influence quadriceps MVA in our normal subjects despite the fact that the tourniquet pressure and position, and the durations of ischaemia were similar to those previously used for our meniscectomy patients (Sherman *et al.* 1983). Dobner and Nitz (1982) carried out a study in which patients underwent meniscectomy either with or without a tourniquet. The durations of ischaemia in their tourniquet group were similar to those of both our normal subjects and meniscectomy patients, but their tourniquet pressures were lower. Their patients' quadriceps were examined by needle EMG, and a progressive resistance exercise table, six weeks post-operatively. The functional tests gave significantly different results for the two groups. All of the non-tourniquet group showed normal EMG activity but 17 out of 25 of the tourniquet group did not. Our negative results in four out of four subjects were unlikely to be due to chance (Fisher's exact probability test, two-tailed, $p = 0.03$).

Electromyogram abnormalities have been detected at three weeks after meniscectomy and in some patients the EMG had returned to normal before four weeks (Saunders *et al.* 1979).

The position of the tourniquet was the same for our normal subjects and meniscectomy patients (i.e. as far proximal as possible) but neither Saunders *et al.* nor Dobner and Nitz stated the precise location of the tourniquet in their patients. Perhaps this might be an important factor if their cuffs (placed more distally) were over motor points.

An alternative explanation lies in the basic differences between normal subjects and surgical patients. The patients have received trauma to the knee and also a period of joint immobilization. Following nerve damage alternative central pathways develop and re-establishment of the former pathways is delayed if normal movement patterns are not performed soon after damage. The normal subjects were fully mobile 5-10 minutes after ischaemia whereas the patients were in bed for 2-3 days. Perhaps a combination of ischaemia, knee trauma, and immobilization is necessary to produce significant damage.

CONCLUSION

We cannot explain the absence of EMG abnormalities in our normal subjects but we can conclude that the reduced maximal voluntary activation observed in our post-meniscectomy patients is unlikely to have been caused by the tourniquet alone.

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Mr David Barber (Senior Operating Department Assistant, Nuffield Orthopaedic Centre) for preparing the subjects with the apparatus for ischaemia, and the Department of Health and Social Security for financial support.

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