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THE EFFECT OF KNEE JOINT AFFERENT DISCHARGE ON TRANSMISSION IN FLEXION REFLEX PATHWAYS IN DECEREBRATE CATS

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SUMMARY

1. Changes in excitability of reflex arcs mediating flexion withdrawal and crossed extensor reflexes have been examined in decerebrate cats.

2. The excitability of flexion withdrawal and crossed extensor reflexes was shown to be modulated by knee joint position. Flexion withdrawal reflexes were most easily elicited when the knee was extended and crossed extensor reflexes were most easily elicited when the knee was flexed.

3. The modulation of transmission was not confined to reflex pathways to muscles acting at the knee but also included pathways to muscles acting at the hip and ankle, as well as pathways to muscles in the contralateral limb.

4. The changing excitability of reflex pathways caused by movement of the knee joint was unrelated to the stretch applied to muscles acting at the knee and to cutaneous afferent discharge. Modulation of reflex excitability by joint movement was totally abolished by local anaesthesia of the knee joint in an otherwise intact limb.

5. The results of the present experiments indicate that transmission in flexion reflex pathways can be inhibited by knee joint afferent discharge.

INTRODUCTION

Graham Brown (1911) demonstrated that the probability of appearance of reflex scratching and hopping in rabbits was determined by the initial position of the hind limb. He did not, however, establish which peripheral receptors were responsible for this modulation of reflex excitability. Earlier, Sherrington (1909) had observed that the excitability of flexion reflexes was independent of muscle length. Thus joint receptors remain as the most obvious candidates for mediating changes in reflex excitability. Indeed, projections from joint afferent fibres on to motoneurone pathways have been clearly demonstrated (Gardner, 1950; Beswick, Blockey & Evanson, 1955; Eccles & Lundberg, 1959; Ramcharan & Wyke, 1972). However, the functional significance of these connexions has remained unclear. Cohen & Cohen (1956) monitored the tension of muscles acting around the knee joint of decerebrate cats in response to flexion and extension of the knee joint and observed that tension in knee extensors increased on movement into extension. This constitutes a positive

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feed-back system driving the limb further into extension. Using monosynaptic reflex testing, Grigg, Harrigan & Fogarty (1978) also observed positive feed-back effects i.e. the amplitude of the monosynaptic reflex of cat knee extensors increased on movement into extension. However, other investigators have obtained opposite findings. Skoglund (1956) observed that the amplitude of the monosynaptic reflex of cat knee extensors decreased on movement into extension. Freeman & Wyke (1967*a*) found that electromyographic activity in the cat gastrocnemius muscle increased on dorsiflexion of the ankle, and decreased on movement in the opposite direction. Both of these are examples of negative feed-back, which would tend to prevent hyperextension or hyperflexion of the joint.

Recently, the issue has been further complicated by the findings of Rossignol & Gauthier (1980) who observed that the reflex reversal of activity in thigh flexor and extensor muscles on movement of the hip joint during the step cycle was not influenced by abolishing hip joint afferent discharge.

The present experiments were performed in order to resolve these conflicting findings, and to establish the role of joint receptors in the modulation of reflex excitability. A preliminary account of this research has already appeared (Baxendale & Ferrell, 1980).

METHODS

The experiments were performed on twelve cats weighing between 1·7 and 3·5 kg decerebrated under deep N_2O–O_2/halothane anaesthesia by transection of the mid-brain at an intercollicular level. The carotid arteries were ligated and the vertebral arteries were occluded briefly during decerebration. The forebrain rostral to the transection was removed and the anaesthesia discontinued. The rectal temperature of the animal was maintained at $37 \pm 0{\cdot}5$ °C by means of heating lamps.

Stimulating electrodes, consisting of chlorided silver wires enclosed in a Perspex cylinder, were placed around the common peroneal nerves in both hind limbs. The electromyograms of various muscles around the knee joint were recorded using silver wires inserted into the muscles through 21 gauge hypodermic needles, which were subsequently removed.

The animals were rigidly attached in the prone position to a brass frame. Steel pins were inserted into the left femur and tibia. The femur was then clamped to the frame, and the tibia to a metal rod whose axis of rotation was set to coincide with that of the left knee joint. This permitted movement between full extension (180°) and full flexion (60°).

In each experiment the following protocol was employed. A train of 5–10 stimulus pulses of width 1–2 msec and frequency 50 sec^{-1} was delivered to the right peroneal nerve. The intensity of the pulses was adjusted until the first sign of a cross extensor reflex was observed in the electromyogram of an extensor muscle in the left hind limb. This generally occurred at two to three times the group I threshold stimulus intensity. The angle of the left knee was changed and this procedure repeated. Alternatively, stimulus pulses were delivered to the left peroneal nerve and the stimulus intensity adjusted until a flexion withdrawal reflex could just be detected in the electromyogram of flexor muscles in the left hind-limb. Thus the minimum intensity necessary to elicit a threshold response for a given reflex was determined at various static knee joint angles throughout a full range of movement. This minimum intensity value was expressed as a multiple of the lowest value observed on movement of the limb from one extreme to the other. In practice it was found that the lowest value occurred at full flexion for extensor reflexes and at full extension for flexor reflexes. At each angle the minimum stimulus intensity needed to elicit a cross extensor reflex was consistently higher when measured during knee extension than when measured during knee flexion. The opposite applied for flexion withdrawal reflexes when threshold stimulus intensities were greater during knee flexion. In each Figure where only one line is shown to indicate the threshold profile each point represents the mean of four determinations of threshold made during extension for the crossed

extensor reflex and during flexion for the flexion withdrawal reflex. Once the threshold profile of a given reflex had been established, knee joint afferent discharge was abolished by injection of 1 ml. 2% lignocaine solution (Xylocaine: Astra Chemicals Ltd.) into the synovial space of the left knee joint, and the measurements of the reflex threshold repeated.

RESULTS

Effects on reflex intensity. The electromyogram of the left vasti was recorded during periods of stimulation of the right common peroneal nerve at a fixed intensity of two or three times the threshold for group I afferents. This procedure was repeated at a number of positions of the left knee between 80 and 180°. The intensity of the crossed extensor reflex, as judged by the magnitude and duration of the electromyogram activity initiated by the stimulation of the peroneal nerve, varied systematically with limb position (Fig. 1*A*). The crossed extensor reflex was most intense when the knee was fully flexed and it declined progressively as the joint was extended. The modulation of reflex intensity was not confined to the vasti. Fig. 1*B* shows the electromyogram of the left soleus during crossed extensor reflexes elicited by stimulation of the right common peroneal nerve. The ankle was held at 90° and the left knee joint angle was varied. As with the vasti, the magnitude and duration of the reflex in soleus varied with knee joint angle.

Measurements made on seven freshly killed cats showed that movement of the knee between 180 and 80° stretched the vasti by 14–18 mm. It is possible that stretching the vasti was responsible for the increased crossed extensor reflex intensity seen as the limb was flexed. However, movement of the knee does not change the length of soleus and it is unlikely that muscle stretch-induced discharge could affect reflex intensity in this case.

The action of muscle stretch in isolation was tested by repeating the stimuli to the peroneal nerves and knee joint movements after the knee joint had been anaesthetized by an intra-articular injection of 1 ml. lignocaine hydrochloride. The intensity of the crossed extensor reflex in the vasti and soleus was then observed to be substantially independent of the position of the knee (Fig. 1*C*). The change in reflex intensity accompanying movement of the limb returned after several hours, presumably due to resumption of joint afferent discharge on recovery from the effects of the anaesthetic. We conclude that the strength of the crossed extensor reflex is independent of any stretch-related muscle afferent discharge and that knee joint afferent discharge was responsible for the observed modulation of reflex intensity by limb position.

These data provided qualitative rather than quantitative information about the modification of transmission in flexion reflex pathways. Further experiments were performed to measure the change in excitability of reflex arcs instead of the change in intensity.

Reflex threshold. The excitability of flexion reflex arcs was measured by gradually increasing the intensity of the electrical stimulation of the peroneal nerve until the first signs of activity were seen in the electromyogram. The minimum stimulus intensity necesary to elicit reflex activity was designated as the threshold intensity. The threshold value was always observed to occur at the extremes of joint position.

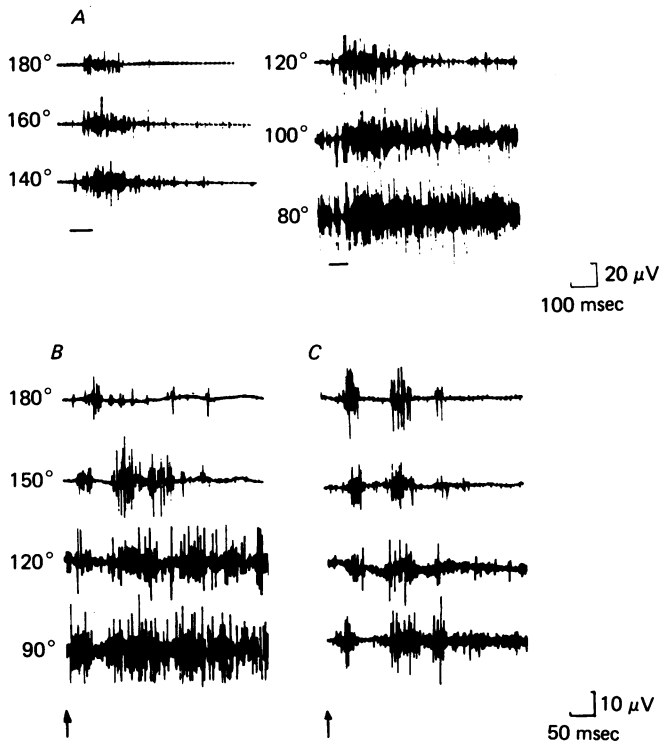

Fig. 1. *A*, modulation of intensity of the crossed extensor reflex recorded in the left vasti muscle group as the left knee joint angle is held at angles between extension (180°) and flexion (80°). The crossed extensor reflex was elicited by stimulation of right peroneal nerve at 50 sec^{-1}, at an intensity of 2·2 V and a pulse width of 1 msec. The period of stimulation is indicated by the black horizontal bar. Although the stimulus parameters remained constant the intensity of the reflex varied with changing knee joint angle. *B*, modulation of intensity of the crossed extensor reflex recorded in the left soleus as the left knee is held at angles between 90° and 180°. The angle of left ankle was kept constant at 90° throughout. The reflex was elicited by stimulation of the right peroneal nerve at 50 sec^{-1} at an intensity of 4 V. The end of the stimulus train is indicated by the arrow. The reflex intensity in soleus is modulated by knee joint position. *C*, crossed extensor reflexes recorded in the same muscles as *B* but after the left knee joint had been anaesthetized by an injection of 1 ml. lignocaine hydrochloride. Modulation of reflex intensity by changing knee joint angle is now substantially reduced. The change in background activity seen at 90 and 180° may reflect changing recording conditions at the electrodes as the limb is moved.

The threshold value for flexion reflexes was found to be located at or near full knee extension whilst the threshold value for the crosssed extensor reflex was found at or near full knee flexion (Figs. 2–4). The protocol for measuring reflex excitability was repeated at regular intervals across the full range of movement, and the minimum stimulus intensity needed to elicit the reflex at each position is expressed as a multiple of the threshold intensity. The points shown in Figs. 2–7 represent means of four determinations of the threshold intensity.

Fig. 2 illustrates the modulation of excitability of flexion reflex pathways to biceps

femoris (*A*) and tibialis anterior (*C*) by knee joint afferent discharge. The threshold was lowest for both muscles when the knee was extended and it rose progressively as the joint was flexed. The excitability of the crossed extensor reflex in the vasti (*B*) and soleus (*D*) was also affected by knee joint angle but in this case the effect was reversed and threshold was lowest when the knee was fully flexed. In each case

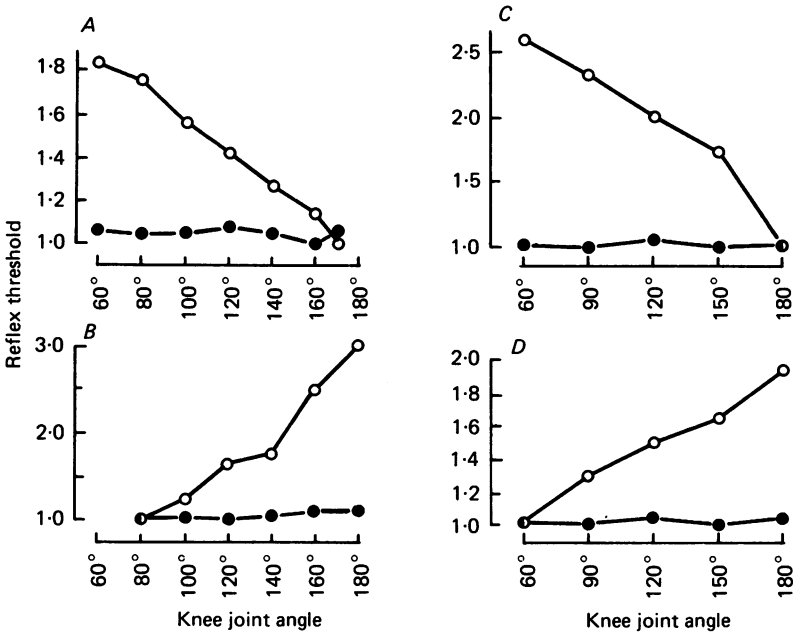

Fig. 2. Modulation of the excitability of flexion reflex pathways to *A* biceps femoris, *C* tibialis anterior and crossed extensor reflex pathways to *B* vasti and *D* soleus by changing knee joint angle. ○, normal; ●, knee joint anaesthesia. The excitability of the reflex pathway is expressed as the reflex threshold which is the ratio of the stimulus intensity needed to elicit a reflex at each angle to the lowest intensity needed at any angle. Flexion withdrawal reflexes are most easily elicited when the knee is extended and crossed extension reflexes are most easily elicited when the knee is flexed. In all cases anaesthesia of the knee joint abolished modulation of reflex excitability by movement of the knee even though the muscles acting at the knee were stretched by the movement.

injection of local anaesthetic into the knee joint abolished the modulation of reflex excitability by changing limb position (Fig. 2*A–D*, ●).

In most experiments rectus femoris was denervated and dissected away from the vasti before any recordings were made. This avoided any possible confusion of effects due to the differing actions of rectus femoris and the vasti. The former spans two joints to cause flexion of the hip and extension of the knee whilst the latter acts only to extend the knee. When recordings were made from rectus femoris (Fig. 3) it was clear that its reflex excitability was modified by knee position in just the same manner as the excitability in the vasti (Fig. 2*B*). Similar observations were made when the excitability changes in reflex pathways to medial gastrocnemius, which also spans two joints to produce knee flexion as well as ankle extension, were compared with

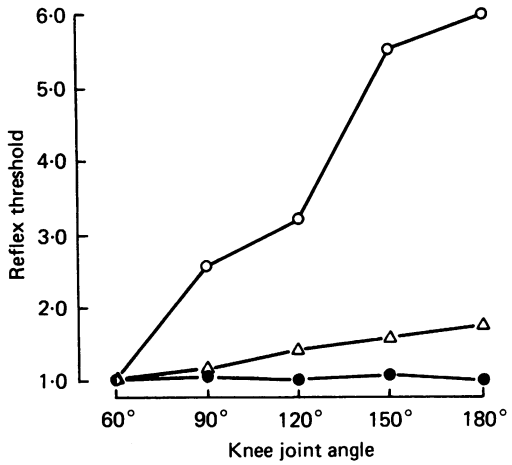

Fig. 3. Modulation of excitability of the crossed extensor reflex recorded in rectus femoris by movement of the knee before and after section of the spinal cord at T_{12}. The depth of modulation of reflex excitability increased more than threefold on transition from the decerebrate (△) to the spinal (○) state but modulation was still abolished by knee joint anaesthesia (●).

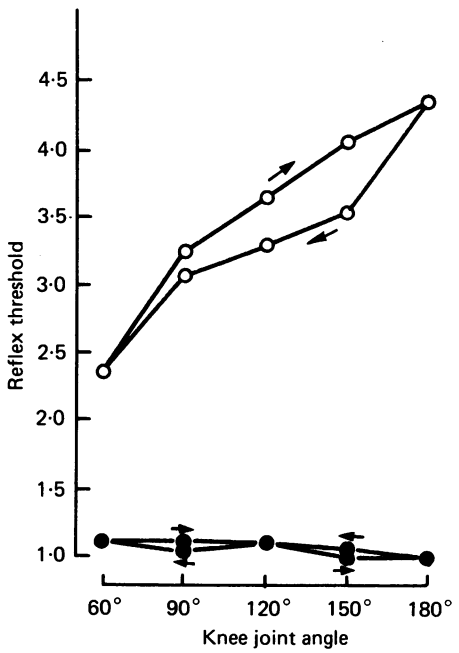

Fig. 4. Modulation of excitability of the crossed extensor reflex pathways to rectus femoris by movement of the knee joint. ○, show the stimulus intensities necessary to evoke a threshold reflex response expressed as a multiple of the minimum threshold intensity necessary after the knee joint had been anaesthetized (●). Under normal conditions threshold values are consistently higher when measured during extension of the knee than when measured during flexion. Anaesthesia of the knee joint eliminates this phenomenon in addition to lowering the reflex threshold.

those to soleus. No distinction appears to exist between pathways to muscles acting only at the knee and pathways to other muscles with more complicated actions.

The depth of modulation of reflex excitability was increased following section of the spinal cord at the level of the lowest rib (Fig. 3). In absolute terms the reflex threshold fell in flexion and was relatively little changed in extension. Anaesthesia of the knee joint again abolished any position-dependent modulation of reflex

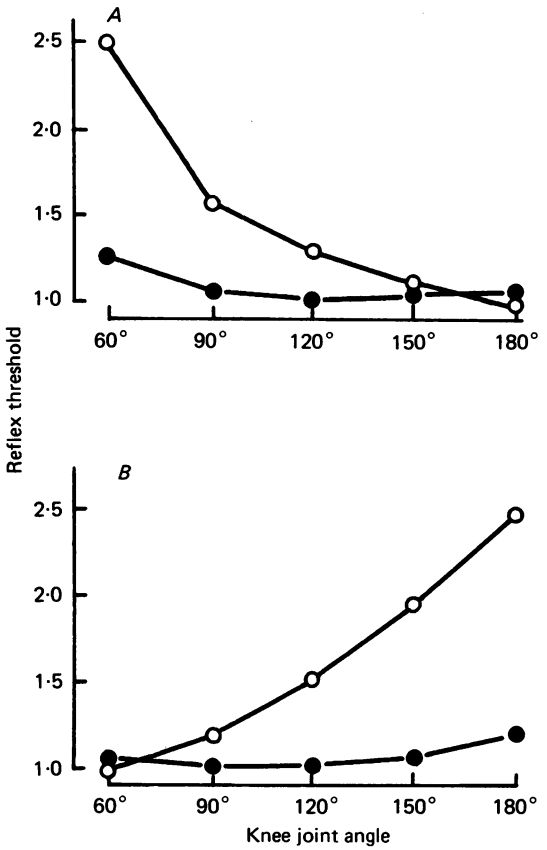

Fig. 5. Modulation of excitability of *A*, flexion withdrawal reflex in the right biceps femoris and *B*, crossed extension reflex in the right quadriceps femoris by movement of the left knee joint. ○, normal; ●, knee joint anaesthesia. Flexor responses were most easily elicited when the contralateral knee was extended, whereas extensor responses were most easily elicited when the contralateral knee was flexed. Abolition of left knee joint afferent discharge prevents movement of the left knee, modulating the excitability of reflex pathways to the contralateral biceps and quadriceps.

excitability, even with the over-all increase in flexion reflex excitability of the spinal preparations.

The reduction of threshold accompanying knee joint anaesthesia is further illustrated in Fig. 4. In this case the threshold for the crossed extensor reflex is expressed as a multiple of the minimum stimulus intensity observed after knee joint anaesthesia. The increase in excitability strongly suggests that proprioceptive joint afferent discharge exerts a predominantly inhibitory central effect.

Threshold measurements at any one angle varied consistently depending on whether the movement to that angle had been one of flexion or extension (see Methods). As seen in Fig. 4, thresholds were higher during extension of the knee. It is clear that this hysteresis phenomenon is related to knee joint afferent discharge since threshold measurements made after joint anaesthesia show no tendency to open out into a loop. It is likely that muscle afferent discharge will also be asymmetric

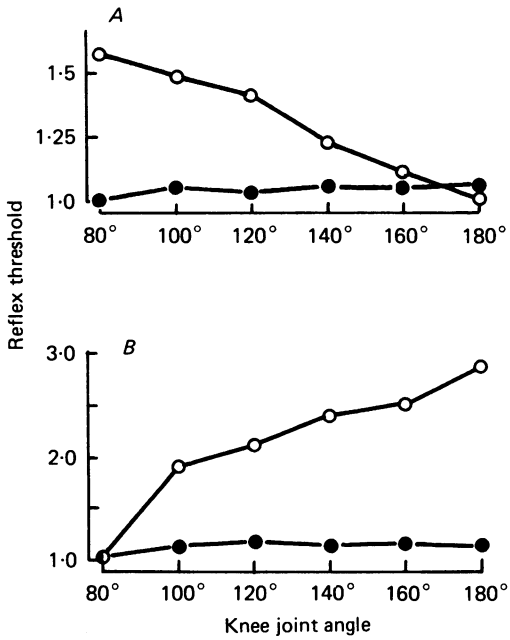

Fig. 6. Modulation of excitability of *A*, the flexion withdrawal reflex in biceps femoris and *B*, the crossed extensor reflex in quadriceps femoris as the knee joint is moved. ○, pre-anaesthesia; ●, knee joint anaesthesia. Both biceps and quadriceps had been tenotomized and were held at constant lengths corresponding to the mid-points of their normal physiological ranges. Modulation of reflex excitability by knee joint movement persisted even though muscle length remained constant and was only abolished by knee joint anaesthesia.

depending on whether the muscle is being lengthened or shortened. However, it is improbable that the hysteresis phenomenon is a consequence of stretch reflex activity since hysteresis loops were recorded in excitability changes in reflex pathways to flexor muscles and in the acute spinal state. In these circumstances there is unlikely to be any significant stretch reflex activity.

Contralateral actions. The results described thus far indicate that afferent discharge arising from the knee joint is capable of changing reflex excitability in a manner which tends to prevent excessive flexion or extension of the knee. The effect of altering the position of the left knee on the excitability of reflexes in the right hind limb was also studied. In these experiments the right hind limb was held rigid with the knee at about 90°.

Fig. 5*A* shows that the flexion withdrawal reflex in the right biceps femoris is most

easily elicited by stimulation of the right peroneal nerve when the left knee is fully extended. The crossed extensor reflex in the right quadriceps femoris is most easily elicited by stimulation of the left peroneal nerve when the left knee is flexed (Fig. 5*B*). Once again these effects were eliminated by injection of anaesthetic into the left knee joint. Thus afferents from the left knee seem capable of influencing transmission in pathways linking left side afferents to contralateral motoneurones (Fig. 5*B*) as well

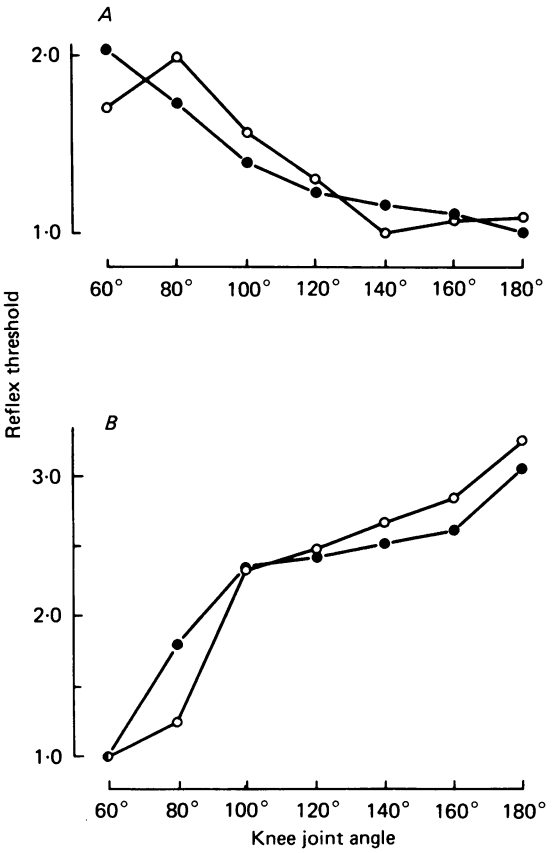

Fig. 7. *A*, modulation of the excitability of flexion withdrawal reflex pathways to biceps femoris by movement of the knee joint before (○) and after (●) anaesthesia of the skin covering the posterior and anterior aspects of the knee. *B*, modulation of the excitability of crossed extensor reflex pathways to the vasti by movement of the knee joint before (○) and after (●) injection of 1 ml. physiological saline into the knee joint.

as pathways linking contralateral afferents to contralateral motoneurones (Fig. 5*A*). Such a pattern of interaction favours the appearance of asymmetric hind-limb postures.

Effects of tenotomy. In three experiments the left vasti and biceps femoris were tenotomized and their lengths were held constant at about the mid-range of their normal physiological lengths. Despite this, subsequent movement of the left knee still modulated the excitability of flexion withdrawal and crossed extensor reflexes (Fig. 6*A* and *B*). The pattern and depth of threshold modulation persisted essentially

unaltered even though tenotomy inevitably involved some damage to the anterior portion of the knee joint as the patella was freed. Injection of local anaesthetic into the left knee joint reversibly abolished the modulation of reflex threshold.

Control experiments. Movement of the knee joint results in stretching the skin at some points on the limb. Experiments performed after the skin covering the anterior and posterior aspects of the knee had been infiltrated with lignocaine hydrochloride showed that the pattern of changing reflex excitability was unaffected by the presence or absence of cutaneous afferent input (Fig. 7*A*). It is concluded that the results reported in this paper cannot be attributed to cutaneous stimulation, though the profound reduction in reflex threshold seen when the knee was hyperflexed to 50° or beyond was probably due to cutaneous stimulation as the heel made contact with the skin at the base of the tail.

It is possible that afferents from popliteus travelling in the posterior articular nerve (Burgess & Clark, 1975) could have influenced the modulation of reflex excitability. However, this possibility can be discounted since the modulation of reflex persists after popliteus has been dissected away from the joint capsule.

The distension of the knee joint space by intra-articular injections appeared to play no part in the abolition of position-dependent excitability changes. When equivalent volumes of physiological saline were injected into the knee joint no effect was seen on the threshold changes accompanying limb movement (Fig. 7*B*). Widespread denervation of muscle and cutaneous nerves leaving only the nerves to the vasti and the knee joint intact did not influence position-dependent modulation of reflex excitability. Thus, in the unlikely event of leakage of anaesthetic from the joint space affecting nearby nerves no significant action on reflex excitabilities should result. The possibility of some central action of the local anaesthetic depressing reflex excitability can be rejected since injection of lignocaine into joints other than the left knee did not change the modulation of reflex threshold by left knee joint movement even though the local anaesthetic must have been carried centrally by the circulation at about the same rate.

DISCUSSION

The present results clearly indicate that the excitability of flexion reflexes is influenced by the initial position of a limb. This agrees with earlier observations by Graham Brown (1911) and Rossignol & Gauthier (1980). The excitability of flexion reflex pathways decreased as the knee was progressively flexed whilst the excitability of pathways subserving the crossed extensor reflex decreased as the knee was extended. In effect this constitutes a negative feed-back system, as suggested by Lundberg, Malmgren & Shomburg (1978), which acts to decrease the probability of the knee being hyperflexed or hyperextended by reflex movements.

That knee joints afferents were principally responsible for the modulation of reflex excitability was unambiguously demonstrated by the following observations. First, modulation of reflex excitability by changing knee joint position persisted when thigh flexor and extensor muscles were tenotomized and held at constant lengths. Secondly, this modulation could be totally abolished by anaesthesia of the knee joint in an otherwise intact limb. Thirdly, extensive denervation of the limb, leaving intact only

the posterior articular nerve of the knee and the nerves to the vasti, produced no change in the pattern of reflex excitability modulation. In addition, cutaneous anaesthesia had no effect. Thus the modulation of transmission in flexion reflex pathways was unrelated to stretch of the muscles under examination and cannot have been caused by convergence of afferents from other muscles or the skin. The modulation must be a consequence of joint afferent discharge.

The present results contrast sharply with earlier studies on joint afferent stimulation which showed the effects to be weak or absent (Skoglund, 1956; Grigg, 1973). Other authors have reported negative feed-back (Skoglund, 1956; Freeman & Wyke, 1967*a*) and positive feed-back (Cohen & Cohen, 1956; Grigg *et al.* 1978) from joint afferent discharge on limb position. These earlier studies all examined the effects of joint afferent discharge on motoneurone excitability. The present series of experiments examined the effect of knee joint afferent discharge on the excitability of flexion reflex arcs. Motoneurones represent only one component in such arcs and it is likely that joint afferents converge on neurones interposed in the arc rather than directly onto motoneurones (Lundberg *et al.* 1978). This could account for the relatively weak effects of joint afferents on motoneurone excitability and the much stronger actions on transmission in flexion reflex pathways.

The threshold profile for flexion withdrawal and crossed extensor reflexes was remarkably linear across a range of knee joint positions between 60 and 180°. Such monotonic profiles resemble the discharge patterns of slowly adapting mid-range receptors in the cat knee joint (Ferrell, 1980). Similarly, the hysteresis effect in threshold measured during flexion and extension movements (see Fig. 5), may be a reflexion of the known asymmetry of joint receptor discharge (McCall, Farias, William & BeMent, 1974; Fontani, Meucci & Carli, 1977).

Knee joint afferent discharge was seen to modify the excitability of reflex pathways to muscles acting at the hip and ankle as well as the anticipated actions on pathways to muscles acting at the knee. In addition, knee joint afferents also modulated transmission in pathways to contralateral motoneurones. These widespread actions are probably best viewed in the context of normal posture. When a limb flexes as the result of some noxious stimulus, the most appropriate response would be extension of the contralateral limb to support the increased load transferred to that side. However, if the contralateral limb is already almost fully extended, then it is not in a position to sustain the extra load and further extension is futile. Under these conditions the pattern of responses described in the present experiments would favour the appearance of some degree of contralateral flexion which would bring the limb forward into a position more suitable for load bearing.

Similar reflex reversals have been described by Rossignol & Gauthier (1980). They attributed the effect to afferents signalling stretch of thigh flexor muscles, and dismissed hip joint receptors from playing any significant role. In their experiments, although the hip joint was completely denervated, it appears that the knee joint innervation was left substantially intact. Now as flexion–extension at the hip was accompanied by movements of the knee (Rossignol & Gauthier, 1980, fig. 1) it is likely that the persistence of reflex reversal after hip joint denervation can be attributed to knee joint afferent discharge. Similarly, their observation that complete abolition of reflex reversal only occurred after extensive denervation of the hind limb is not

surprising, as knee joint afferents are known to run in the sciatic, obturator, common peroneal and cutaneous saphenous nerves (Skoglund, 1956; Freeman & Wyke, 1967*b*; Burgess & Clark, 1975).

In conclusion, the present results indicate that joint receptors can importantly influence flexion reflexes, that these effects are widespread, and act to limit hyperextension and hyperflexion of the joint.

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