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THE INTERRELATION OF NEURAL DISCHARGE, INTRA-ARTICULAR PRESSURE, AND JOINT ANGLE IN THE KNEE OF THE DOG

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SUMMARY

1. Single- and multi-unit recordings were obtained from the medial articular nerve (m.a.n.) of knee joints in the anaesthetized dog. The single-unit recordings were confined to low threshold (group I and II) articular mechanoreceptors.

2. Multi-unit recordings revealed that the m.a.n. discharge was maximal in extension, submaximal in flexion, and minimal at intermediate angles, i.e. a U-shaped profile.

3. Subatmospheric intra-articular pressures do not appear to influence the m.a.n. discharge.

4. Intra-articular infusion of even small quantities of fluid, although not affecting the U-shaped profile, reversed the m.a.n. discharge pattern with maximum neural activity occurring in flexion and being submaximal in extension.

5. Recordings from single units indicated that the enhanced discharge after fluid infusion was a result of increased discharge frequency and ' recruitment ' of individual afferents.

INTRODUCTION

Most investigations of the response characteristics of articular afferent fibres have been concerned with the effects of movement and externally applied mechanical probing of the capsular tissues in the cat knee joint (Andrew & Dodt, 1953; Boyd & Roberts, 1953; Skoglund, 1956; Burgess & Clark, 1969; McCall, Farias, Williams & Bement, 1974; Clark, 1975; Clark & Burgess, 1975; Grigg, 1975; Ferrell, 1980; Schiable & Schmidt, 1983*a*, *b*). Relatively few have examined the effects of intra-articular pressure changes on joint afferent discharge (Andrew & Dodt, 1953; Grigg, Hoffman & Fogarty, 1982; Wood & Ferrell, 1984), and there has been no investigation of the response of articular afferents to simultaneous alteration of intra-articular pressure and joint movement.

It is well known from both clinical experience, and accurate measurement, that in the normal joint there are considerable changes in intra-articular pressure levels

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during movement of the joint through its normal range (Levick, 1979; Nade & Newbold, 1983; Newbold, 1984). In arthritic and injured knees the pressure may become higher than normal and may be responsible for the limitation of movement, muscle weakness, and pain which commonly occur in these conditions. It has been demonstrated that raised intra-articular pressure in the human knee joint results in reflex inhibition of muscles acting at this joint, mediated by knee joint afferents (De Andrade, Grant & Dixon, 1965; Spencer, Hayes & Alexander, 1984).

In the present study the effects of intra-articular pressure, static joint position and movement on the response characteristics of medial articular nerve (m.a.n.) fibres arising from the normal dog knee have been analysed. These experiments were performed to determine whether articular afferents respond to both joint movement and changing intra-articular pressure, and to establish the potency of each variable as a stimulus to m.a.n. fibre discharge. In addition, the effects of cannulating the joint to record intra-articular hydrostatic pressure, and the effects of infusion of fluid into the joint, on neural discharge, were also examined.

The interpretation of some of the results relies on information obtained in previous studies on the relationship between intra-articular pressure and joint position, and on synovial fluid dynamics in the knee joint of the dog (Nade & Newbold, 1983, 1984; Newbold, 1984).

METHODS

Mongrel dogs weighing between 10 and 21 kg were anaesthetized with pentobarbitone (30 mg kg^{-1}) and maintained using an anaesthetic machine (CIG Midget 3) and ventilator (Blease Manley MN2) by inhalation of an oxygen/nitrous-oxide mixture (1:2) at 4·5 l min^{-1}, with approximately 1 % halothane for the duration of each experiment. Carotid arterial blood pressure was monitored continuously, and arterial blood samples taken, at various times during the experiment, for analysis of p_{CO_2}, p_{O_2}, pH, HCO_3^- and haemaglobin. Rectal temperature was maintained between 37 and 39 °C with a heating blanket, and the hind limbs were warmed using a heat lamp.

Intra-articular pressure (I.A.P.) recordings and infusions

An 18, 16 or 14 gauge inextensible fluid-filled Teflon cannula, with small perforations around the tip, was inserted into the infrapatellar region of each joint (Nade & Newbold, 1983). This cannula was connected through fluid-filled tubing to a pressure transducer (Statham Model P23DC) via a three-way stopcock.

A second cannula, similar to that described above, was inserted in the same way as for I.A.P. recordings. This cannula was connected directly to a two-way stopcock, positioned immediately adjacent to the knee joint. This stopcock was then connected through inextensible tubing to a glass syringe, either hand held or set into an automatic infusion pump (Harvard Apparatus Compact Infusion Pump). Error introduced from leakage or distension of the infusion and recording apparatus, aggregate apparatus compliance, and base-line drift of the recorder were as measured and reported previously (Nade & Newbold, 1983).

Peripheral whole nerve recording

With the animals in a supine position both hind limbs were dissected to expose approximately 2 cm of medial articular nerve (m.a.n.) a few centimetres proximal to the medial edge of the patellar ligament. A bipolar electrode, housed within a 1·5 cm length of Perspex tubing, was sewn into place around the nerve in each limb. The muscles and skin were then sutured back over the electrodes to help hold them in place. Bone screws were placed in each femur, and the thighs attached to metal bars positioned vertically and lateral to the hip joints. The legs (i.e. knee to ankle, or stifle to hock) were supported by a horizontal bar. The knee joint angle was changed by moving the horizontal bar which was attached to and controlled by a small-animal joint servo manipulator (Mills, Newbold & Nade, 1982).

The electrical signals from the embedded bipolar electrodes were amplified and filtered (Neurolog-Digitmer Ltd), converted into standard pulses using a spike trigger, and integrated. The integrator time constant was set at values ranging from 20 ms to 1 s depending on the imposed velocity of movement. The spike trigger was also connected to an audio amplifier and a digital read-out display module. This module provided a means of calibrating the integrated neural signal. The integrated signal was then directed to a chart recorder which was calibrated to give a continuous print-out representing the whole nerve discharge rate in spikes per second. Signals from the leg mover, and from the pressure transducer, were simultaneously fed into the chart recorder along with the integrated discharge of the m.a.n. These variables were monitored under physiological conditions and after a change in I.A.P. had been induced by infusing the joint with liquid paraffin oil B.P.

The joint could either be set at one particular angle, or could be moved continuously over the full range of natural movement, or any part thereof.

Single-unit recordings

For these recordings the femur was rigidly fixed in a horizontal plane by flexion and external rotation at the hip. A paraffin pool was fashioned over the m.a.n., which was subdivided until functional single fibre filaments were obtained. These filaments were placed individually over bipolar platinum electrodes. The electrical signals were processed in the same way as described above for the whole nerve recordings. The identification of the fibres as joint afferents depended upon discharge characteristics described in the results section of this paper. This recording technique permitted examination of afferent discharge over a full range of movement. The conduction velocity of individual fibres was determined by dividing the conduction distance (measured *in situ*) by the conduction delay of an action potential evoked in the fibre by electrical stimulation (pulse width 1 ms; amplitude < 1 V; frequency 1 Hz) of the distal m.a.n. via the embedded electrodes. Based on conduction velocity and discharge characteristics, only myelinated joint afferents (groups I and II) were examined in single-fibre recordings.

RESULTS

Nature of m.a.n. afferent fibres

The status of the m.a.n. as a pure articular nerve was verified by the following procedures. Gentle probing of the joint capsule from the medial edge of the patella to the posterior edge of the medial collateral ligament elicited vigorous responses from afferents in m.a.n. Probing of other areas of the joint capsule, the patella, and the patellar ligament elicited little neural activity in m.a.n., and probing of surrounding muscles failed to produce any neural response. Intra-articular injection of fluid produced a large, sustained enhancement of m.a.n. discharge.

Intravenous injection of succinylcholine in a dose sufficient to excite muscle spindle afferents (0·1 mg kg^{-1}) produced no alteration of neural activity in m.a.n. Thus m.a.n. is a pure articular nerve and does not contain any afferents from surrounding muscles.

M.a.n. discharge pattern and I.A.P. changes during movement

Fig. 1, representing a single cycle from extension through flexion in seven dog knee joints, demonstrates the U-shaped pattern of discharge from the medial articular nerve. In most cases the discharge rate was maximum in full extension, minimum at intermediate joint angles between 60 and 110 deg, and rose again as the joint was moved towards full flexion. However, in one of seven knees there was a slight drop in discharge rate at maximum flexion. Thus the neural discharge pattern of the m.a.n. is similar in the knees of different dogs, although the absolute level may differ from dog to dog, and in some cases in both knees from the same animal. Despite variations

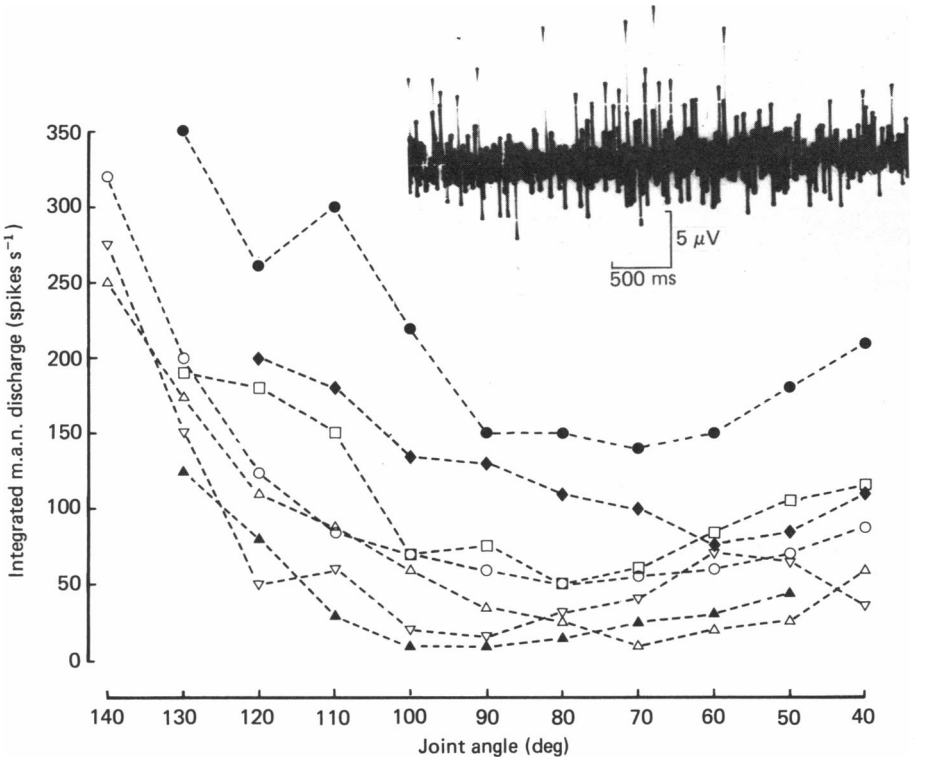

Fig. 1. Integrated medial articular nerve (m.a.n.) discharge pattern for seven knees during continuous movement of 0·04 Hz over the normal range of flexion (40 deg) and extension (140 deg). These readings were taken before cannulation of the joints for intra-articular pressure (I.A.P.) measurements. The inset depicts the appearance of the joint nerve discharge at 100 deg.

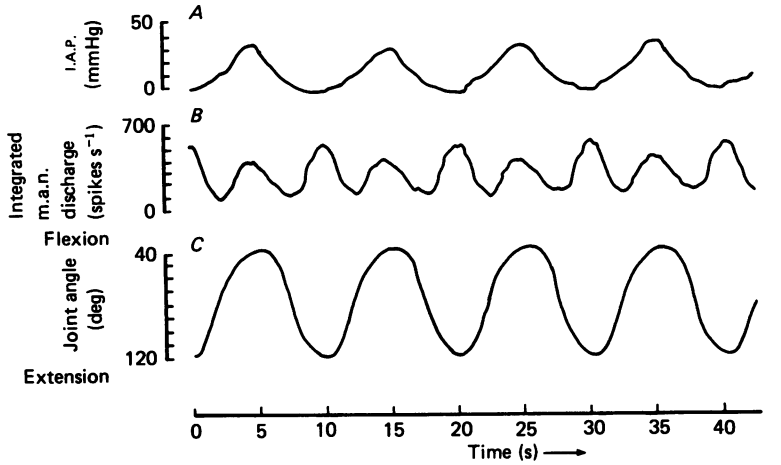

Fig. 2. Direct pen recording, on curvilinear graph paper, of *A*, I.A.P. changes and *B*, the m.a.n. integrated discharge in response to *C*, repetitive movement of the joint at 0·1 Hz over a range of 40 deg flexion to 120 deg extension.

in the absolute level of discharge in the knees of an individual animal, the discharge pattern remained similar in the two knees.

Fig. 2 demonstrates the m.a.n. discharge and I.A.P. changes to repetitive movement of the joint at 0·1 Hz over a range of 40 (flexion) to 120 deg (extension). I.A.P. parallels changes in joint angle, whereas the neural discharge peaks both in flexion and in

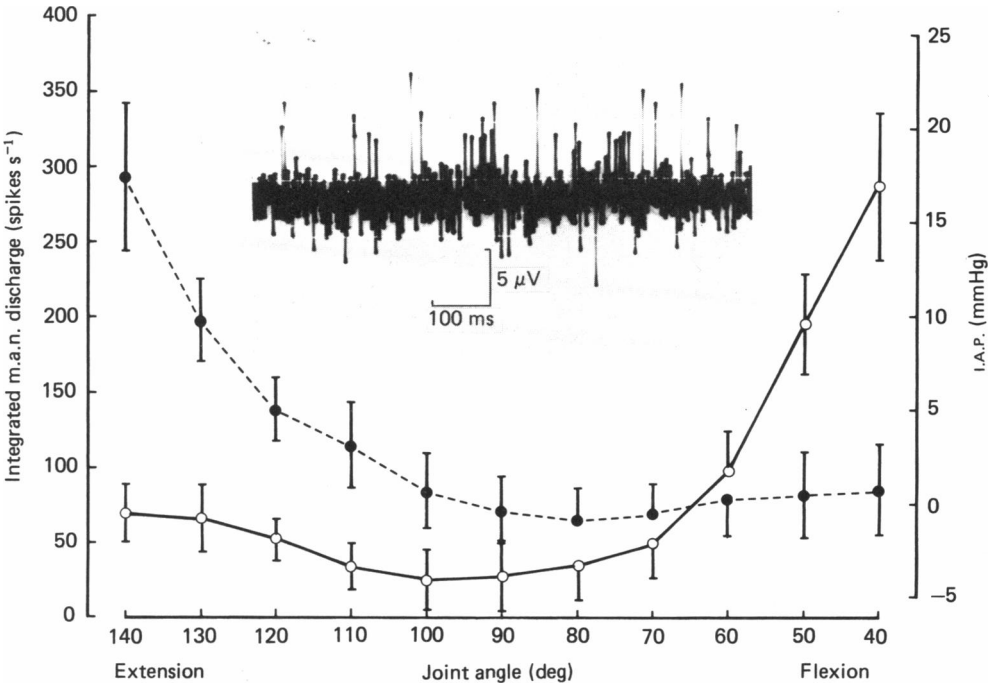

Fig. 3. The relation between m.a.n. integrated discharge in ten knees (●--●) and I.A.P. in eight knees (○——○) is such that the m.a.n. discharge does not follow changes in I.A.P. over the same movement sequence from 140 extension to 40 deg flexion. These are mean values, with standard errors. The inset depicts the appearance of the joint nerve at 100 deg.

TABLE 1. M.a.n. discharge at two different joint positions where I.A.P. is the same

Pressure (mmHg)	Position No.1 (deg)	Neural discharge (spikes s^{-1})	Position No. 2 (deg)	Neural discharge (spikes s^{-1})
−0·4	140	293	66	73
−0·7	130	198	67	72
−1·8	120	138	69	70
−3·3	110	115	83	67
−3·8	90	70	103	92

extension with each cycle. This disparity between I.A.P. and neural discharge was consistently observed in each animal (Fig. 3). The pattern of m.a.n. discharge does not follow changes in I.A.P. over all of the same movement sequence. Neural discharge is highest at maximum extension, whereas I.A.P. is highest at maximum flexion. These

results suggest that intra-articular pressure and neural discharge, within the range of pressures reached in the normal knee, do not appear to be directly related. More evidence for the lack of effect of normal intra-articular pressures on the neural discharge pattern from m.a.n. is displayed in Table 1. Over the intermediate angles

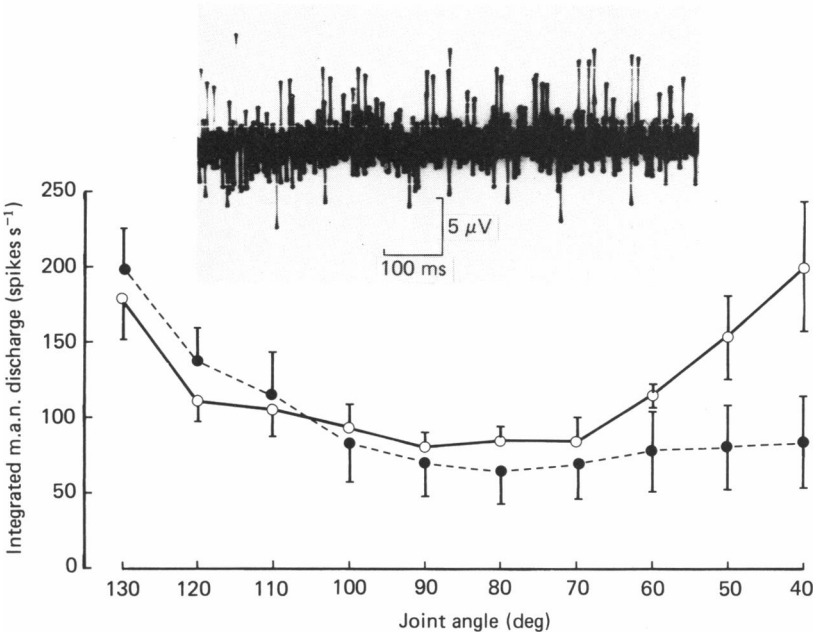

Fig. 4. There was a significant difference between m.a.n. absolute discharge levels before (●--●) and after (○——○) cannulation at the angles of flexion 40, 50 and 60 deg. The inset depicts the appearance of the joint nerve discharge at 100 deg.

where I.A.P.S are subatmospheric, I.A.P. values are the same at two different joint angles; on each side of the minimum pressure, usually around 120 deg (Fig. 3). However, the neural discharge at these two angles may differ greatly (Table 1). This would suggest that subatmospheric values of I.A.P., *per se*, do not influence neural discharge.

In Fig. 3, the pressure recordings were not from the same knees as the m.a.n. discharge recordings, but were from previous studies in the dog (Nade & Newbold, 1983). This was deemed necessary because of the effect on the absolute levels of neural discharge following cannulation of the knee joint for pressure recordings.

The effect of joint cannulation on the discharge pattern of m.a.n.

Fig. 4 shows the difference between mean absolute discharge levels before and after cannulation, and demonstrates the effect of a ' foreign body ' in the joint, and possibly puncture of the capsule by the cannulae. Cannulation only enhanced neural discharge in flexion. The magnitude of the effect in flexion depended on the size of the joint, being negligible in large joints.

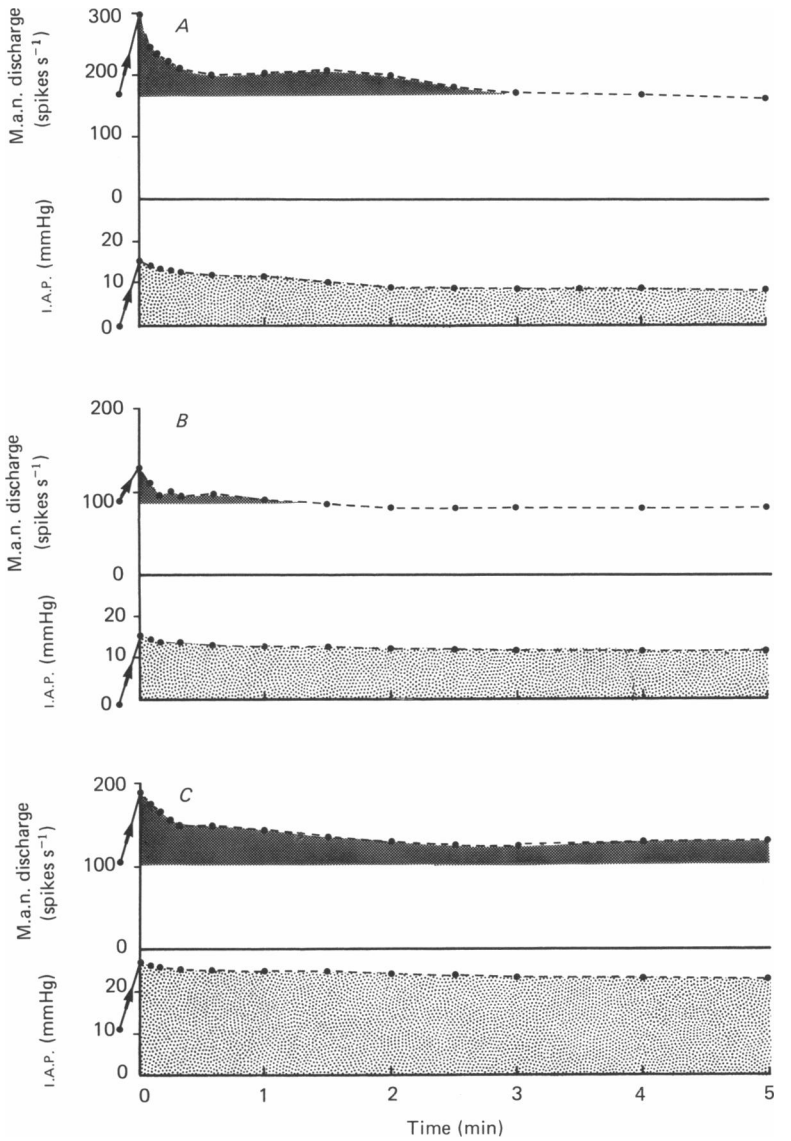

Fig. 5. M.a.n. discharge rates and I.A.P.s at time of infusion, and for 5 min after infusion of the knee with 2·5, 1·5 and 0·5 ml paraffin, at static joint angles of *A*, 140 deg extension; *B*, 90 deg mid-range, and *C*, 60 deg flexion, respectively. The shading highlights the level of fall-off, towards pre-infusion levels, of discharge rate and I.A.P. after infusion.

The effects of fluid infusions on the pattern and level of neural discharge in m.a.n.

As it appeared that normal I.A.P.s did not directly influence the discharge pattern of the m.a.n. during movement, I.A.P. was raised by infusing the joint with liquid paraffin while holding the knee in a fixed position. Infusions were carried out at three different joint angles; 140, 90 and 60 deg. Fig. 5*A* compares neural discharge with intra-articular pressure at the time of infusion and for 5 min after infusion of the knee

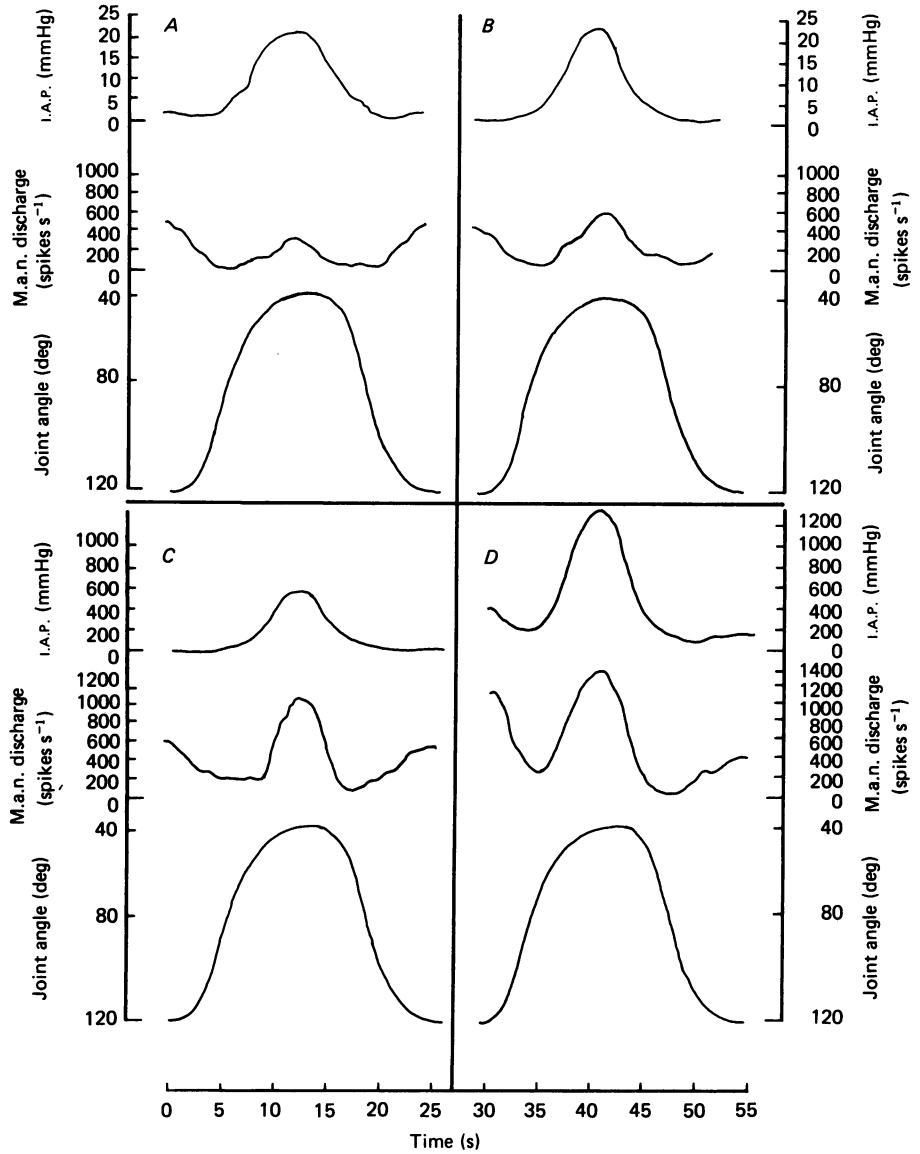

Fig. 6. Direct pen recordings, on curvilinear graph paper, of m.a.n. discharge, I.A.P. and joint position over a continuous movement sequence at 0·04 Hz from *A*, a non-infused knee, and *B*, *C* and *D*, knees infused with 1·4, 5·5 and 7 ml of paraffin, respectively.

with paraffin at a joint angle of 140 deg extension. There was an initial increase in neural discharge rate immediately on infusion, a rapid drop in rate over the first 30 s, followed by a slow decrease in discharge over the next $3\frac{1}{2}$ min. 4 min after the infusion the discharge rate had dropped back to its original level before infusion. The corresponding pressure response was typical of that observed in a previous investigation (Nade & Newbold, 1984). There was a very gradual decrease in intra-articular

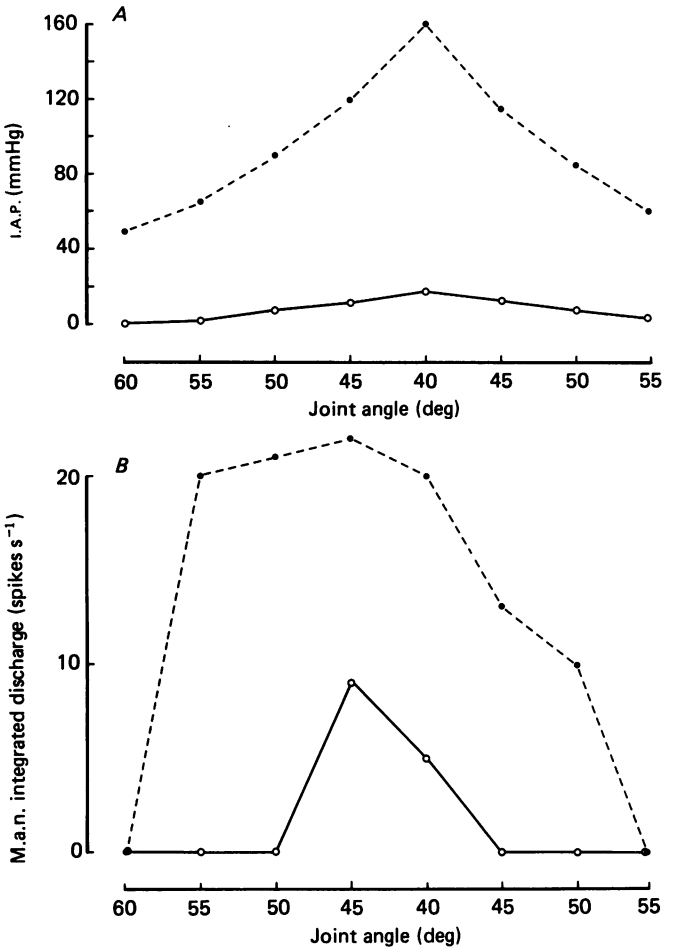

Fig. 7. The response of *A*, I.A.P., and *B*, a single m.a.n. nerve fibre discharge to continuous movement at 0·08 Hz in a normal knee (○——○), and a knee infused with 7·5 ml paraffin (●--●).

pressure over the 5 min. However, the pressure remained around 8 mmHg 5 min after infusion.

Fig. 5*B* shows the response for the same volume of infusion at joint angle of 90 deg. As would be expected from the relatively low levels of neural discharge at intermediate angles shown in Figs. 1 and 3, there was only a small initial increase in discharge rate on infusion, and this declined rapidly, returning to the original pre-infused level 1 min after infusion. The pressure level again decreased very gradually, maintaining a slightly higher level than for the infusion at 140 deg, and was still around 11·5 mmHg 5 min after infusion.

The third angle used to infuse the knee with paraffin was 60 deg flexion. The pressure is normally higher at this flexed position than at the other positions of 140 and 90 deg. Therefore, for a similar volume of fluid infused, the peak infusion pressure attained was nearly twice as high as that attained at 90 deg (Fig. 5*C*). In addition,

the pressure maintained over the 5 min after infusion was considerable, and was still at 23 mmHg at the end of 5 min. The neural discharge response to this pressure change was initially similar to the other infusions, with an initial rise in discharge rate on infusion. However, although there was some fall in discharge rate over approximately $2\frac{1}{2}$ min, the rate did not return to its original level. It appears that for intra-articular pressures above 10–13 mmHg an increase in the absolute level of neural discharge is maintained. During continuous movement (Fig. 6), after the knee has been infused with paraffin, and intra-articular pressures are higher than normally found in the joint, the discharge rate from the m.a.n. is initially affected (increases) during flexion only. It seems that only at very high intra-articular volumes and pressures is there an increase in the discharge levels at the more extended angles (Fig. 6*C* and *D*).

A striking feature of Fig. 6 is the reversal in the discharge pattern with increasing intra-articular volume. Whereas under physiological (control) conditions the discharge in extension exceeds that in flexion (Fig. 6*A*), the presence of even small quantities of fluid in the joint results in reversal of this pattern (Fig. 6*B*) which was maintained at higher intra-articular fluid volumes. It is also noticeable that at the higher volumes, apart from the obvious effect in flexion, there is a generalized increase in neural activity even at intermediate positions (Fig. 6*C* and *D*).

Single-unit recordings

Whole nerve recordings are useful in determining the effect of altered I.A.P. on a population of m.a.n. afferents in individual animals, and establishing whether a common pattern of altered neural activity occurs. However, whole nerve recordings cannot discriminate between rise in m.a.n. discharge due to 'recruitment' of afferents, or a rise in discharge due to increased firing frequency of individual afferents. In order to investigate this aspect, recordings were obtained from individual nerve fibres emanating from slowly adapting mechanoreceptors, and the response to movement and altered I.A.P. were examined as illustrated in Fig. 7.

From twelve single units studied in depth spontaneous discharge was noted in eleven, either with the leg in one position or over a range of angles during movement, while one unit could only be activated by probing the joint capsule. Of the twelve units, five responded to the mid-range of joint positions and during movement towards flexion, three fibres responded to the mid-range and during movement towards extension and one responded only at the extreme ends of the normal range of flexion and extension. Six of these units also responded to changes in I.A.P. The response characteristics (Ferrell, 1980) of the 'flexion' units were: (i) three units displayed a monotonic discharge pattern during movement, being sensitive to both movement and I.A.P; (ii) one unit had a bell-shaped discharge pattern and was sensitive to changes in velocity, but not to changes in I.A.P; (iii) one unit was rapidly adapting and responded to acceleration.

The response characteristics of 'extension' units were: (a) one unit displayed a monotonic discharge pattern to movement and was sensitive to changes in I.A.P; (b) two units displayed a bell-shaped discharge pattern during movement, with one of these units responding to changes in velocity, but non-responsive to changes in I.A.P. alone.

The firing rates of the single units varied between 2 and 73 spikes s^{-1}. Measurement

of conduction velocities was not always possible. However, those recorded fell between 30 and 50 m s^{-1}. The apparent anatomical locations of most of the receptors of the fibres studied were found to be on the medial aspect of the knee joint, next to the patellar ligament and the medial collateral ligament. Some receptors could not be located by the capsular probing method used.

It is clear that increased I.A.P. can result in both increased discharge frequency, and an increase in the angle over which the unit discharged.

DISCUSSION

The m.a.n. was used rather than the posterior articular nerve as in the dog knee it is the largest and most constant of the primary articular nerves. The territory innervated by m.a.n. previously demonstrated in anatomical studies by O 'Connor & Woodbury (1982), was confirmed electrophysiologically by probing of the capsule. The present experiments also demonstrated that m.a.n. is a pure articular nerve, free from surrounding muscle afferent ' contamination ' which has been demonstrated in the cat posterior articular nerve (Burgess & Clark, 1969; McIntyre, Proske & Tracey, 1977) although the extent of this 'contamination' is minimal (Ferrell, 1980) and therefore unlikely to be of functional significance. The use of the dog m.a.n. is appropriate as two previous investigations of the effect of the joint position on I.A.P. have been performed in the dog knee joint (McCarty, Phelps & Pyenson, 1966; Nade & Newbold, 1983, 1984).

The U-shaped relation between discharge pattern and joint position observed in m.a.n. is similar to that observed in the posterior articular nerve of the cat knee joint (Ferrell, 1980). This suggests a similarity in receptor characteristics in the two preparations. Single-unit recordings confirmed this, with a greater number of m.a.n. afferents discharging on movement towards the extremes of movement, and fewer exhibiting tonic activity at intermediate positions. This is very similar to the pattern of activity of cat knee joint afferents (Burgess & Clark, 1969; Ferrell, 1980).

It is clear from our results that under physiological conditions, I.A.P. does not significantly influence joint afferent discharge. Thus, the modulation of discharge with changing joint angle is probably a consequence of mechanical deformation of the joint capsule by movement, at least as far as the neural response in extension and at intermediate angles is concerned. However, the response in flexion could be influenced by non-physiological levels of I.A.P., as suggested by Fig. 5*C*, and the progressive enhancement of the flexion response with rising I.A.P. (Fig. 6). This enhancement could be the result of displacement of paraffin from the suprapatellar joint lumen to the infrapatellar lumen with flexion of the knee as the suprapatellar region is compressed by the overlying quadriceps muscle during flexion. Such fluid displacement would not only result in elevation of I.A.P., but would also produce greater distension and stretch of the infrapatellar portion of the joint capsule, thus increasing neural activity in fibres from that region. When a joint is artificially distended with large volumes of paraffin, then distension of the whole joint capsule occurs at all angles, which could explain the generalized increase in neural discharge seen in Fig. 6*D*.

The greater sensitivity of the flexion response to increased intra-articular volume

results in a striking reversal of the normal m.a.n. discharge pattern during movement. This was consistently observed in all the animals tested. Such a pattern reversal may form the basis of the subjective assessment in human subjects with joint effusions that, although often painless, the affected joint does not feel 'quite right'. In addition, alteration of the pattern of joint afferent discharge during movement by increased I.A.P. could have implications for joint-mediated reflexes. Freeman & Wyke (1966) observed that ankle joint mechanoreceptors were responsible for reflex regulation of postural changes in the tone of hind-limb muscles, and that these reflexes were reciprocally organized. It was suggested that articular mechanoreceptors may be operating as part of a negative feed-back system on hind-limb muscles. Baxendale & Ferrell (1981) demonstrated that cat knee joint afferents exert such feed-back on the excitability of knee extensor and flexor muscles, possibly preventing hyperextension and hyperflexion of the joint. Lundberg, Malmgrem & Schomburg (1978) have suggested that joint afferents could act to limit the terminal phase of movement. Clearly, the pattern of such reflex activity depends upon the pattern of joint afferent discharge. Thus, the asymmetric alteration of joint afferent discharge which occurs in a very swollen joint would be likely to result in alteration in the normal pattern of joint-mediated reflex activity with consequent disturbance of locomotor function. In addition to this, the results of De Andrade *et al.* (1965) and Spencer *et al.* (1984) suggest that even at a fixed knee joint angle, increasing I.A.P. produces increasing inhibition of quadriceps motoneurones. These disturbances of reflex activity could explain the tendency of acutely swollen joints to 'give way', perhaps resulting in further trauma to the joint and prolongation of the period of joint disability. This would suggest that in order to improve function, every attempt should be made to decrease the size, and pressure, of an effusion within a joint which has been injured, or is diseased. In this way the sequelae of disordered reflex activity produced by the elevated I.A.P. might be avoided.

It is suggested, as a result of our study in normal dogs, that the medial articular nerve contains afferent fibres arising solely from articular receptors which could provide information about the position of the joint. These nerves can also signal changes in pressure within the joint, but such pressures need to be greater than 10–13 mmHg to reach the threshold of activation for their receptors.

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