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PRIMARY AFFERENT DEPOLARIZATION OF CENTRAL TERMINALS OF GROUP II MUSCLE AFFERENTS IN THE CAT SPINAL CORD

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SUMMARY

1. The origin of primary afferent depolarization (PAD) of the central terminals of group II afferent fibres of tibialis anterior and extensor digitorum longus muscles has been investigated in the cat. Changes in the excitability of the terminals to intraspinal stimuli, upon application of conditioning stimuli to muscle nerves (quadriceps, sartorius, gracilis, posterior biceps–semitendinosus, anterior biceps–semimembranosus, gastrocnemius–soleus, deep peroneal), cutaneous nerves (sural, superficial peroneal) and the posterior nerve to the knee joint, were used as a measure of PAD.

2. PAD was most readily evoked by conditioning stimuli which were maximal for group II muscle afferents. However, some PAD was also evoked from group I afferents and evidence is presented that group Ia afferents contributed. Afferents of posterior biceps–semitendinosus and sartorius muscles appeared to be most effective. PAD was also evoked by stimulation of cutaneous and joint nerves, often in the same fibres which were affected by group Ia afferents.

3. It is concluded that there are several common sources of PAD of group II and group Ia afferent terminals on the one hand, and group Ib afferent terminals on the other.

4. The properties of PAD of group II afferents are discussed in relation to the problem of how PAD affects transmission from fibres with long terminal branches of small diameter.

INTRODUCTION

Primary afferent depolarization (PAD) of the central terminals of group I muscle afferents and cutaneous afferents has been extensively investigated (for references see Schmidt, 1971; Burke & Rudomin, 1977; Lundberg, 1982; Jankowska, 1984). In particular, the pattern of PAD evoked in them from different peripheral and descending fibre systems has been documented and many of the characteristics of the underlying neuronal circuitry have been elucidated (Jankowska, McCrea, Rudomin & Sykova, 1981; Rudomin, Jimenez, Solodkin & Duenas, 1983; Brink, Jankowska

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& Skoog, 1984; Rudomin, Solodkin & Jimenez, 1986; Jimenez, Rudomin & Solodkin, 1988).

In contrast, rather little is known about the PAD of group II muscle afferent terminals. The data available are from only two previously published studies, by Eccles, Schmidt & Willis (1963) and by Carpenter, Lundberg & Norrsell (1963). Eccles *et al.* (1963) recorded intracellularly from six fibres which were tentatively classified as belonging to group II afferents because their thresholds were higher and their conduction velocities slower (no details are given) than for group Ib afferents. PAD was evoked in these fibres from group I afferents of either flexors or extensors (whether they belonged to the Ia or to the Ib subgroup of these afferents was not specified) and from cutaneous afferents. However, in a recent study (Jimenez *et al.* 1988; performed concurrently with the present study), PAD from group I afferents was found in only one of nine intra-axonally recorded group II fibres, while PAD from cutaneous afferents was not observed. Carpenter *et al.* (1963) found an increase in excitability of six of twelve tested group II afferents after stimulation of the sensorimotor cortex.

The present study has been prompted by the recent demonstration of a powerful projection of group II muscle afferents to the middle lumbar segments of the cat spinal cord. Since a number of axon collaterals of group II afferents terminate there outside the area of termination of group I afferents (Edgley & Jankowska, 1987*a*, *b*), these collaterals are much easier to activate in isolation from group I afferents than in caudal lumbar segments, and are therefore well suited for a study involving the testing of excitability of group II afferent terminals to intraspinal stimuli as a measure of PAD (see Wall, 1958). An abstract of this work has been published (Harrison & Jankowska, 1987).

METHODS

Preparation

The experiments were performed on cats anaesthetized with chloralose (70 mg kg^{-1}, supplemented with up to 5–7 mg kg^{-1} h^{-1}), continuously infused with a 100 mM solution of sodium bicarbonate containing 5% glucose, paralysed with gallamine triethiodide and artificially ventilated. The above doses of chloralose have been found to result in deep anaesthesia in non-paralysed cats kept under observation over more than 36 h. Furthermore, the level of anaesthesia was controlled while checking that the applied stimuli did not cause any pupil dilatation or increase in blood pressure or heart rate. Blood pressure and end-tidal CO_2 were kept above 90 mmHg and at about 4% respectively. During the preliminary dissection (under ether anaesthesia) laminectomies exposed the spinal cord at Th12 and from the fourth lumbar to sacral segments. The following peripheral nerves were dissected and mounted for stimulation: quadriceps, sartorius, gracilis, posterior biceps–semitendinosus, anterior biceps–semimembranosus, sural, gastrocnemius–soleus, the posterior nerve to the knee joint, the superficial peroneal and the deep peroneal nerves. The deep peroneal nerve was dissected as far distally as possible in order that fine filaments of tibialis anterior and extensor digitorum longus branches of this nerve could be separated and placed on individual pairs of recording electrodes (see *A* in Fig. 1). L6 and L7 ventral roots were cut in order to prevent any orthodromic discharges of motoneurones reaching these nerves and hence contamination of the records from sensory fibres by these discharges.

Recording and stimulation

Following stimulation of the deep peroneal nerve (*B* in Fig. 1), field potentials were recorded in the fourth lumbar segment (*C* in Fig. 1) with glass micropipettes filled with 2 M-sodium chloride.

These recordings were made simultaneously with recordings from a silver ball electrode (*E* in Fig. 1) located on the cord dorsum at the middle of the fifth lumbar segment, which was used to determine the timing and size of volleys in peripheral afferents as they reached the midlumbar segments. Following satisfactory location of field potentials of group II origin, the glass micropipette was exchanged for a glass-coated tungsten microelectrode in order to avoid changes in the excitability of the tested fibres by leakage of ions from the glass micropipette. Intraspinal

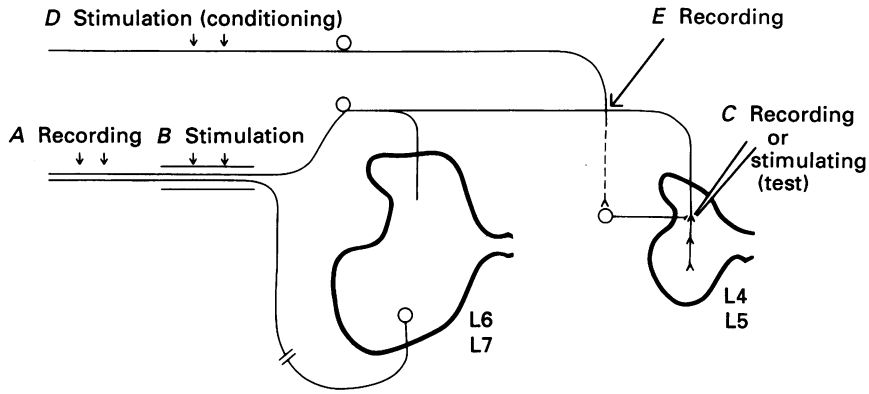

Fig. 1. Diagram of the experimental arrangement, with the various stimulation and recording sites indicated by *A–E*. For explanations see text.

electrical stimuli (10–20 μA) were then delivered down the tungsten microelectrode in order to activate collaterals of single afferent fibres of the deep peroneal nerve, and recordings were made from their distal parts in one of the branches of the tibialis anterior and extensor digitorum longus nerves (*A* in Fig. 1). Centrifugal action potentials induced by intraspinal stimuli were collided by centripetal volleys induced by stimuli applied to the deep peroneal nerve (*B* in Fig. 1). By carefully grading the stimulation of this nerve, the peripheral threshold of the centrally stimulated fibre (with respect to the lowest threshold group I fibres) could be ascertained.

In order to detect changes in the excitability of the stimulated fibres, the amplitude of the test intraspinal stimuli was adjusted to be either just subthreshold, or at threshold for antidromic firing of the fibre under test. PAD produced by the conditioning stimuli (*D* in Fig. 1) was then evidenced by a reduction of the threshold for activation of the fibre, or its more effective activation by the same intraspinal stimuli. The conditioning stimuli typically consisted of a train of five shocks with an intershock interval of 3·33 ms, commencing 25–30 ms before the test intraspinal stimulus.

The largest changes in excitability were detected by comparing the responses to the test stimuli which were set to be below threshold, and became suprathreshold when preceded by conditioning stimuli; single-sweep records were then used. Weaker changes in excitability could not be readily detected in this way, and to disclose them, an approach with greater sensitivity was adopted. The intensity of the test stimuli was adjusted so that they would activate the selected fibre in 20–50 % of trials when applied alone. An averager (Nicolet, model 1170) was then used to obtain a sum of the spike potentials generated in response to sixty-four test stimuli alone, and sixty-four test stimuli preceded by conditioning stimuli, applied alternately and fed to separate quarters of the averager's memory. These sums were compared with the sum of sixty-four spike potentials evoked by suprathreshold stimuli. Typically, the sum of spike potentials generated in response to effective conditioned test stimuli were 50–100 % larger than the sum of spike potentials evoked by the test stimuli alone. On the basis of a single test trial, such increases were usually significant at the 5 % level (χ^2). However, during data collection, a positive result of such a trial was only considered reliable if it was obtained on two or more occasions. Consequently, many other results, including a few examples where the increase was only 20 %, became significant at the 5 % level when the results of repeated testing were taken into account. All reported results were significant at the 5 % level.

RESULTS

Selection of group II muscle afferents

Because action potential conduction velocity of group I and group II afferent fibres undergoes progressive reduction as they ascend the dorsal columns, measurements of their mean conduction velocity will underestimate the peripheral

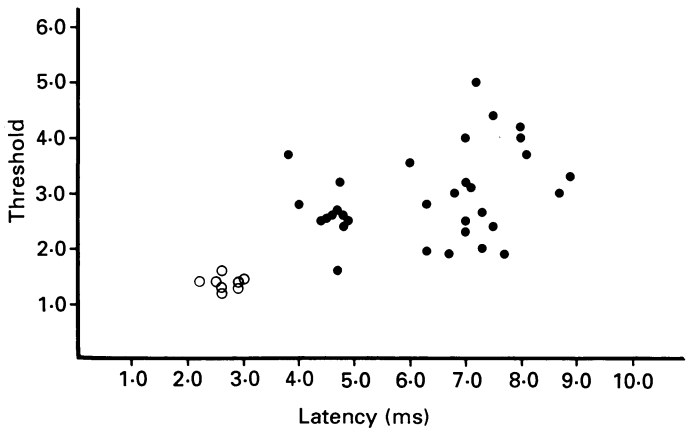

Fig. 2. Plot of thresholds to electrical stimulation of the deep peroneal nerve against latency of their activation by intraspinal stimuli for the sample of afferents classified as group II afferents (●) and of a sample of group I afferents (○) projecting to midlumbar segments.

conduction velocity of these afferent fibres leading to their incorrect classification as of group I or group II origin. However, in a recent study (Fern, Harrison & Riddell, 1988), the extent of slowing of the conduction velocity of the ascending collaterals of group I and group II fibres has been investigated. From that study, the shortest conduction times of action potentials in posterior biceps–semitendinosus fibres, which could be classified indubitably as of group II origin (peripheral conduction velocity less than 60 m/s), from L4 to their muscle nerve are approximately 3·0 ms. Allowing for the longer conduction distance to tibialis anterior and extensor digitorum longus muscles, antidromically evoked action potentials with latencies exceeding 3·7 ms (thirty-two fibres; filled circles in Fig. 2) were tentatively identified as occurring in group II afferent fibres.

Further indications of the origin of afferent fibres could be obtained from a consideration of their peripheral electrical thresholds. However, there is a great deal of overlap between the ranges of electrical thresholds of group I and group II muscle afferent fibres. Some group II fibres are reported to have thresholds as low as 1·5 times threshold (T) for the most excitable fibres and some group I fibres have been reported to have thresholds as high as 2·6 T (see esp. Jack, 1978). Those fibres with peripheral thresholds greater than 2·6 T, but not exceeding 5·0 T, could be confidently classified as group II on the basis of their excitability alone. Thus, twenty-two fibres of our sample with peripheral thresholds of 2·6–5·0 T fulfilled this criterion. In

addition, ten fibres with thresholds of 1·8–2·5 *T* were studied in order to avoid the possibility that our sample of group II fibres were limited to a narrow range of sizes. Figure 2 shows that latency and peripheral threshold distinguished the tested group II fibres from a sample of group I fibres which displayed a much narrower range of thresholds and conduction times. No differences were found in the sources of PAD evoked in the two subpopulations of fibres classified as group II fibres and all conclusions of the present study are valid for those with thresholds in excess of 2·6 *T* as well as for those with thresholds between 1·8 and 2·6 *T*.

We had, on the other hand, to limit our sample of group II fibres to those of the pretibial flexors because only these, together with group II afferents of quadriceps and sartorius, are reliably found to terminate extensively in the midlumbar segments and recording of single-fibre activity of quadriceps and sartorius is technically more difficult.

PAD evoked by muscle afferents

On commencing this study it soon became apparent that the most effective way of producing PAD of group II afferents was by stimulating muscle nerves at a strength sufficient to activate group II afferents. This was effective in all fibres tested. However, in order to compare the origin of PAD of group II afferents with that of group I afferents we have paid particular attention to the possibility of evoking PAD from group I, especially Ia afferents and from cutaneous afferents, since PAD of Ia origin characterizes Ia but not Ib afferents, while PAD from cutaneous afferents is, as a rule, evoked in Ib but not in Ia afferents (see Schmidt, 1971; Jimenez *et al.* 1988).

When the intensity of the test intraspinal stimuli was set below threshold for activating the tested fibres, the conditioning stimulus strengths generally had to be in the range of 2–5 *T* to be effective in evoking PAD. Figure 3*A*, *B* and *C*, *D* illustrates this with records from two group II fibres. Stronger conditioning stimulation of either the sartorius nerve or the quadriceps nerve increased the excitability of these fibres to the extent that they fired in response to each test stimulus, while weaker conditioning stimuli were either less effective (at 2–3 *T*) or not effective at all (at stimulus strengths below 2 *T* in *A* and below 3 *T* in *B–D*). The range of stimulus strengths, 2–5 *T*, corresponds to the range of stimulus strengths required to activate the majority of group II fibres (Eccles & Lundberg, 1959; Ellaway, Murphy & Tripathi, 1982).

However, muscle nerves were not equally effective in producing PAD. The most effective was stimulation of posterior biceps–semitendinosus (in eleven out of twelve fibres tested, 92%) and of sartorius (in fourteen out of sixteen fibres tested, 88%) nerves. Stimulation of quadriceps and of gracilis nerves was less effective (in seventeen out of twenty-one fibres tested, 81%, and in five out of eight fibres tested, 63%, respectively), that of gastrocnemius–soleus and anterior biceps–semimembranosus nerves (in five out of thirteen fibres tested, 38%, and in one out of fourteen fibres tested, 7%, respectively) being least effective. In this respect it is interesting to note that the two most effective nerves innervate flexor muscles (posterior biceps–semitendinosus and sartorius) and the two least effective nerves innervate extensor muscles (gastrocnemius–soleus and anterior biceps semitendin-

osus), which suggests that flexor muscle afferents are more effective than extensor muscle afferents in producing PAD of group II afferent fibres. This is further illustrated in Figs 3 and 4. Figure 3 shows that somewhat higher strengths of conditioning stimuli were required to produce PAD from quadriceps than from

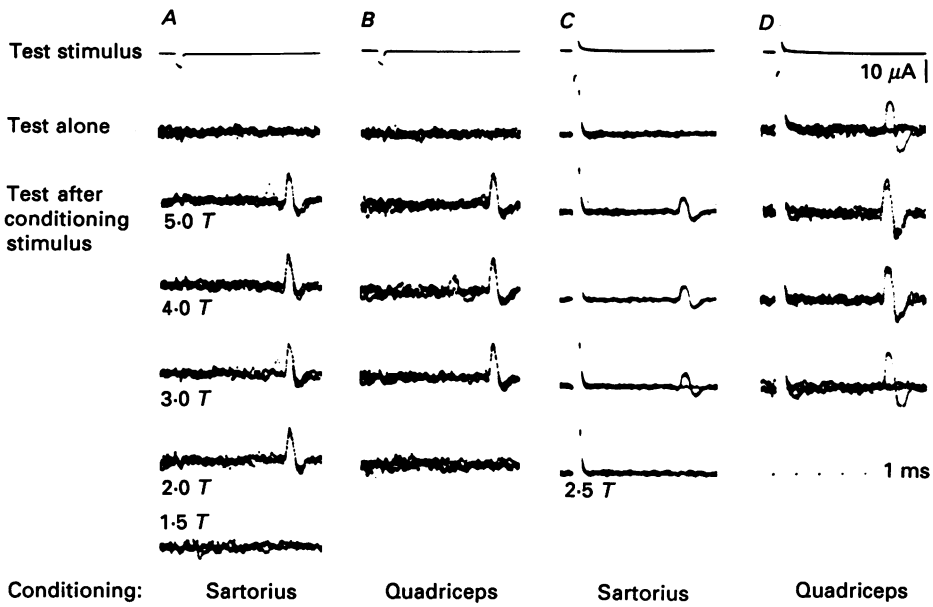

Fig. 3. PAD of two group II fibres of the anterior tibial nerve (with peripheral thresholds of 2·4 and 2·6 *T*, respectively). The top traces show the output of the current monitor, and show the timing and waveform of the test stimuli. The remaining traces in *A*, *B* and *C*, *D*, respectively, are records from two filaments of the tibialis anterior muscle nerve (at a lower gain in *C*). The uppermost records show the effect of applying the test stimulus alone through the tip of the micropipette, located in the L4 segment (at a site corresponding to *C* in Fig. 1). The remaining traces show the effect of applying a conditioning stimulus to the sartorius and to the quadriceps nerves at the strengths indicated in column *A*. With regard to the first fibre (columns *A* and *B*), a conditioning stimulus at twice threshold to the sartorius nerve, or three times threshold to the quadriceps nerve, was effective in depolarizing the fibre sufficiently that the test stimulus was effective in evoking an antidromic action potential. In the case of the second fibre (columns *C* and *D*), a conditioning stimulus of three times threshold or stronger was necessary to achieve the same effect. Each trace consists of three superimposed records.

sartorius in the first of the illustrated fibres, while Fig. 4 compares the effects from three flexors and one extensor. Stimulation of the three flexor nerves, sartorius, posterior biceps–semitendinosus or deep peroneal, evoked such an increase in the excitability of the tested fibre that with a conditioning stimulus of 2 *T* the test stimulus evoked an antidromic action potential on every trial, while stimulation of the gastrocnemius–soleus nerve (with the same intraspinal stimulus strength) failed to evoke any detectable PAD even at much higher conditioning stimulus strengths.

As shown in Fig. 3, upon reducing the conditioning stimulus strength below that

sufficient to activate group II fibres, PAD could not readily be revealed using single-sweep records, which suggests that group II fibres and not group I were the main source of PAD. In order to investigate whether group I fibres could also evoke PAD of group II fibres, it was necessary to use the more sensitive approach described in

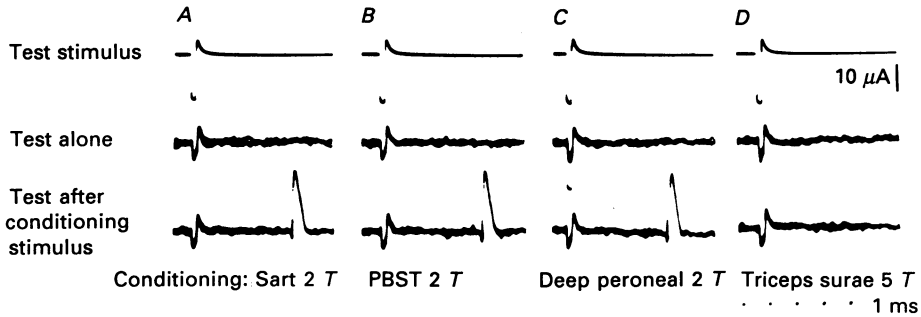

Fig. 4. As in Fig. 3, but for a fibre of the extensor digitorum longus (with peripheral threshold of 3·8 *T*). The figure illustrates the lower efficacy of stimulation of triceps surae (*D*) than of three flexor nerves (*A–C*) in evoking PAD. PBST, posterior biceps–semitendinosus. Sart, sartorius.

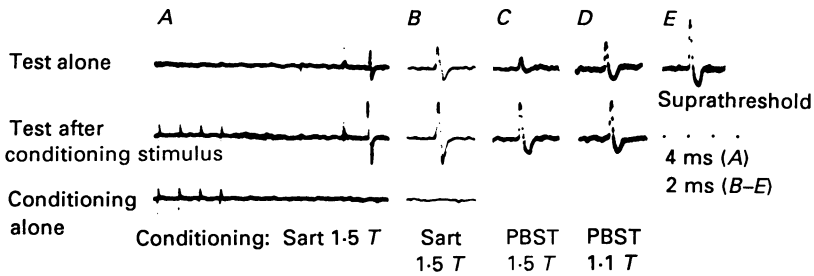

Fig. 5. An example of the actions of group I fibres in evoking PAD. In *A* the top record shows the algebraic sum of the action potentials evoked by the test intraspinal stimulus when applied alone. The bottom record shows that no action potentials were evoked in response to the conditioning train alone (to sartorius nerve, Sart). The middle trace shows the sum of the action potentials evoked by the test intraspinal stimulus when preceded by the conditioning stimulus. The amplitude is now larger and reflects the greater success of the test stimulus in evoking action potentials when conditioned by a stimulus at group I strength. In *B* are the same records as in *A*, but on a faster time scale. In *C–E* are similar records from another fibre, with conditioning stimulation of posterior biceps–semitendinosus (PBST). The fibres were of anterior tibial and extensor digitorum longus respectively, with peripheral thresholds of 2·55 and 3·3 *T*.

the Methods. An averager was used to algebraically sum the action potentials occurring as a result of sixty-four test stimuli, applied alone or preceded by conditioning stimuli. PAD was only concluded to be of group I origin when conditioning stimuli were 1·5 *T* or below.

Figure 5*A* and *B* shows an example. The top record shows a sum of the action potentials evoked by the test intraspinal stimulus when applied alone. Its amplitude is thus in proportion to the number of times the stimulus evoked an action potential,

bearing in mind that the stimulus had been adjusted so that an action potential was generated in less than 50% of the trials. The bottom record shows that no action potentials were evoked in the fibre in response to the conditioning train. The middle record shows the sum of the action potentials evoked by the test intraspinal stimulus when preceded by the conditioning stimulus. The amplitude is now larger and reflects

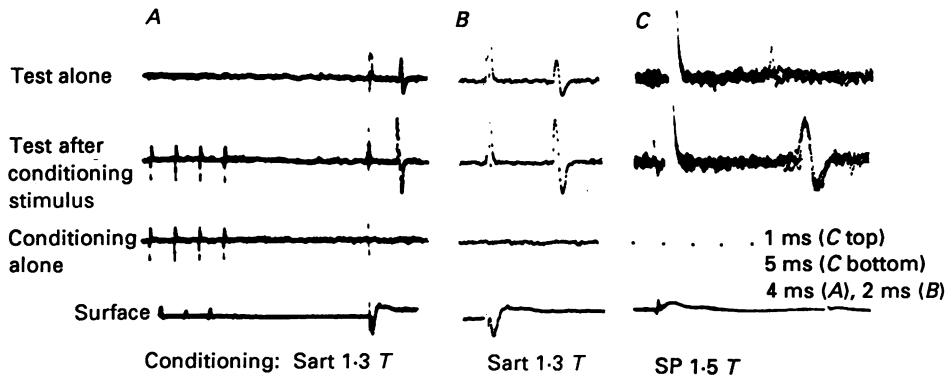

Fig. 6. An example of PAD evoked from both group Ia afferents and from cutaneous afferents in another fibre of extensor digitorum longus (with peripheral threshold of 2·55 *T*). *A*, as in Fig. 5*A*, except that the bottom trace shows records from cord dorsum. The timing between the conditioning stimulation of the sartorius (Sart) nerve and the test intraspinal stimuli can also be noted. *B*, expanded sections of the right-hand side of the traces in *A*. *C*, records following test subthreshold intraspinal stimuli when applied alone (top trace) or after a single conditioning stimulus to the superficial peroneal (SP) nerve (middle trace). The bottom trace is from the cord dorsum (at a slower time base). Three superimposed traces as in Figs 3 and 4.

the greater success of the test stimulus in evoking action potentials. Thus, we can conclude that conditioning stimulation in the case of group I afferents of sartorius was effective in depolarizing the tested group II fibre. Using such tests, we have found that conditioning stimulation of group I afferents was effective in evoking PAD in eight out of fourteen fibres tested. Of these eight fibres, PAD was found to be evoked from the group I afferents of posterior biceps–semitendinosus (in five fibres), sartorius (in five fibres), quadriceps (in four fibres) and gracilis (in two fibres), but not from gastrocnemius–soleus or anterior biceps–semimembranosus.

Figure 5*C–E* shows another example when group I fibres were effective. In the top records of Fig. 5*C* and *D* are the summed action potentials evoked by the test stimulus alone (somewhat larger in *D*), and below them are the summed action potentials evoked by the test stimulus when preceded by a conditioning stimulus at the strength indicated. On three occasions in two fibres, reduction of the conditioning stimulus strength to 1·3 *T* (illustrated in Fig. 8*A* and *B*) and even to 1·1 *T* (Fig. 5*D*) was effective. Since these stimulus strengths were below threshold for the Ib fibres in this nerve (Bradley & Eccles, 1953; Jack, 1978), we can conclude that group Ia fibres are effective in producing PAD of group II fibres.

PAD evoked by cutaneous afferents and by joint afferents

Cutaneous afferents were also found to be effective in evoking PAD in group II fibres (in 63% of twenty-three tests, in nine of eleven group II fibres tested). Two examples of their actions are shown in Figs 6 and 7. Figure 6 shows that both group

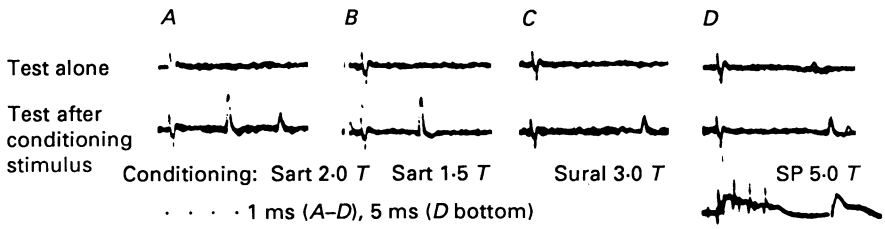

Fig. 7. Superimposed records showing the difference in sensitivity of a group I and a group II fibre of anterior tibial nerve (with peripheral thresholds of 1·45 and 2·6 *T*, respectively) to the actions of group I and of cutaneous afferents. The top records show the lack of effect of the subthreshold test intraspinal stimuli when applied alone. Preceding conditioning stimuli to the sartorius (Sart) nerve at 2 *T* were then sufficient to assist the antidromic discharge of a group I fibre (large potentials at short latency) and a group II fibre (smaller potentials, at longer latency). After the conditioning stimulus had been reduced to 1·5 *T* (which would only activate group I fibres) the test stimulus was ineffective in discharging the group II fibre, though remained effective for the group I fibre. With the same test stimulus, a preceding conditioning stimulus to the sural nerve or to the superficial peroneal (SP) nerve was effective in evoking PAD of the group II, but not of the group I fibre. Bottom trace in *D* is a record from the cord dorsum.

Ia afferents and cutaneous afferents of the superficial peroneal nerve evoked PAD in one of the tested group II fibres. Similar data were obtained in three other fibres. Figure 7, on the other hand, shows that there was a marked difference in the sensitivity of group I and group II fibres in the actions of group I and cutaneous afferents. Figure 7 shows the effects of conditioning stimuli when the test stimuli alone were below threshold for producing any antidromic discharges in this filament. However, a preceding conditioning stimulus to the sartorius nerve at 2 *T* was effective in antidromically discharging a single group I fibre recorded from the filament (after a latency of about 2·5 ms) and a single group II fibre (at about 4·7 ms latency). When this conditioning stimulus was reduced to 1·5 *T*, only the group I fibre was antidromically activated, while a conditioning stimulus applied to the sural nerve or to the superficial peroneal nerve (both cutaneous nerves) decreased the threshold of activation of the group II fibre, but not of the group I fibre.

PAD from joint afferents was found in two of the four fibres tested. In both of them PAD was evoked from Ia as well as from cutaneous afferents. In Fig. 8 are records from one of these fibres. The left-hand side of the figure shows the effects of conditioning stimulation of group Ia afferents, while records in *C* illustrate effects of stimulation of the joint nerve. When the test stimulus was applied alone, it antidromically discharged a group II fibre at 5·1 ms latency. A preceding conditioning stimulus to the joint nerve then activated another group II fibre at a latency of about 8·9 ms.

DISCUSSION

One striking feature of the PAD evoked in group II afferents was that it was more difficult to detect using intraspinal excitability testing than the PAD evoked in group I afferents. In fact, the test intraspinal stimuli usually had to be much closer

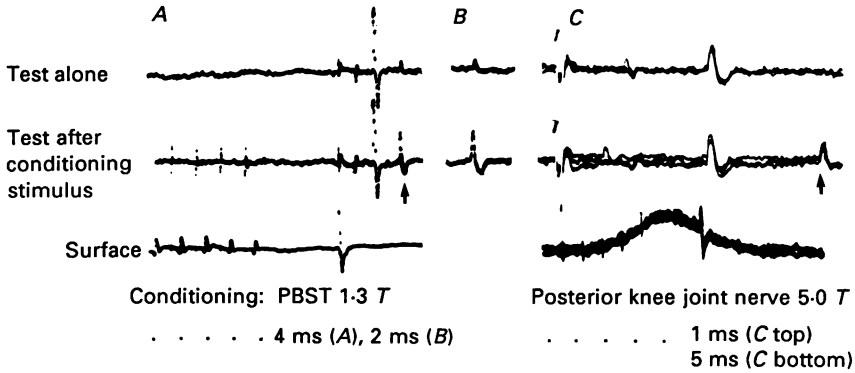

Fig. 8. An example of PAD evoked from both group Ia afferents and from joint afferents in an extensor digitorum longus group II fibre (with peripheral threshold of 3·3 *T*). *A*, as in Fig. 5, showing effects of conditioning stimulation of the posterior biceps–semitendinosus nerve (PBST) at 1·3 *T* on responses of the fibre (arrow) with the longest latency. *B* shows expanded right-most parts of records in *A*. *C*, superimposed single-sweep records of responses of the same fibres when the test intraspinal stimulus was effective in antidromically discharging a shorter latency group II fibre and was subthreshold for activating a longer latency group II fibre, which discharged only when the test stimulus was preceded by a conditioning train to the posterior knee joint nerve at 5 *T*. The bottom records are from the surface of the spinal cord to show, on a slower time base, the timing of the conditioning stimuli.

to threshold for the group II than for group I afferents, in order for the effects of conditioning stimuli to be seen. In a small number of fibres tested, the conditioning stimuli only allowed the test intraspinal stimuli to be reduced by less than 10% in order to produce the same degree of activation of the group II fibre, whereas the effect of conditioning stimuli on group I fibres was to allow a 10–20% reduction in the intraspinal stimulus. Similar changes in threshold have been found by D. Curtis and his colleagues (personal communication).

Supposing that the mechanism of PAD in group II fibres is the same as that in group I fibres, one might expect the PAD to be more difficult to detect as a natural consequence of group II fibres being of smaller diameter, and having longer and thinner terminal branches. A smaller diameter fibre will have a shorter length constant. Thus, depolarization of such a fibre by a given source of PAD will decrement over a shorter distance and, consequently, will be less readily detected while using the technique of excitability testing, unless the test stimuli are applied very close to the terminals. This does not necessarily mean that the PAD is any less effective in modulating the release of transmitter from the terminals of group II fibres. However, unless there is a corresponding increase in the innervation of the

terminal arborization, there will as a consequence of the shorter length constant be a reduction in the effectiveness of the PAD further away from its source.

Alternatively, one may argue that a shorter length constant provides the potential for much greater selectivity in limiting PAD to only some terminals. Thus the longer the length constant, the greater will be the extent of the electronic spread throughout the terminal arborization with the consequent loss of specificity. A reduced ability to detect PAD, or the disclosure of only weak PAD using excitability testing, should not, therefore, be taken to indicate that PAD is weak, as it may reflect a much more selective, highly specific organization of presynaptic control.

While the sample of group II fibres in the present study was from across the spectrum of thresholds of group II fibres in the pretibial flexor muscles, comment should be made regarding the particular species of afferent to which the studied group II fibres belong. Such discussion is particularly important for the group II component of the nerves to the pretibial flexor muscles since Boyd & Davey (1968) concluded that the group II component of these muscles contains many 'extra' fibres in addition to the secondary afferent fibres of muscle spindles. In relation to tibialis anterior, this issue was taken up by MacLennan (1972) who studied the different types of afferent in the group II range of this nerve and similarly found a number of non-spindle group II fibres (see also Jack, 1978). The question then arises as to whether or not the present sample of group II fibres is homogeneous in afferent origin and whether this population are the muscle spindle secondary afferents. We have a number of observations relevant to this. First, at sites in L4 where monosynaptic group II field potentials were seen, field potentials can be evoked by stretches that would activate spindle secondary afferents (Edgley & Jankowska, 1987*a*), but would be unlikely to activate non-spindle group II fibres (MacLennan, 1972). Second, activation of spindle secondary endings by stimulating fusimotor neurones is sufficient to evoke field potentials in this region (Harrison, Jami & Jankowska, 1988). Thus, we are confident that spindle secondary endings terminate in this region. Third, our sample of units appeared to be homogeneous in the sense that at least some sources of input (flexor muscle afferents stimulated at 5 *T*) were effective in evoking PAD in all of them. Since there are no *a priori* reasons for expecting different species of afferent to have PAD of the same origin, we are tempted to believe that all of our sample of afferents were spindle secondary fibres.

From the functional point of view it may be of interest that some groups of primary afferents, notably group Ia afferents, evoke PAD in both group II and group Ia muscle spindle afferents, while cutaneous afferents evoke PAD in both group II and group Ib tendon organ afferents. PAD of group Ia origin found in the present sample of fibres was rather weak, but its potency was increased when the number of afferents activated by the conditioning stimuli increased (when two nerves were stimulated instead of only one of them). To establish whether Ib afferents also contribute to the PAD would require that they are selectively stimulated. However, PAD evoked by stimuli maximal for both group Ia and group Ib afferents was always easier to detect than PAD evoked by electrical stimuli selective for group Ia afferents from hip and thigh muscles. The difference may thus have been due to the Ib afferents contributing, although it may also have reflected activation of more Ia afferents recruited by the stimulus. The relative contribution of group II afferents is

likewise difficult to define because PAD from group I afferents (even subthreshold) will add to PAD evoked by stimuli which activate group II afferents. Whether the latter would have much stronger effects than either Ia or Ib afferents when stimulated in isolation, remains an open question. The relative strength of PAD evoked by group I and cutaneous fibres may vary, depending both on the muscle origin of group II afferents and the segmental level, since it was found in a much smaller percentage of a sample of group II afferents from the medial gastrocnemius muscle in the L7 segment (Jimenez *et al.* 1988).

In this context it might also be of interest that PAD of group Ia afferents in Clarke's column is evoked from both the group I, cutaneous, joint and high-threshold muscle afferents (Jankowska, Jukes & Lund, 1965; Jukes, 1965), unlike that of group Ia afferents in the lumbosacral enlargement, but like PAD of group II afferents in midlumbar segments. Given the similar pattern of origin of PAD of these afferents, one might expect that the same interneurones contribute to the PAD of terminals of group II afferents in midlumbar segments and of terminals of group Ia afferents in Clarke's column. Furthermore, since there is evidence that PAD in Clarke's column is mediated by interneurones located in the same segments (Jankowska & Padel, 1984; Harrison & Jankowska, 1984), the same might be true for PAD of group II afferents. Consequently, the involved interneurones might be among those L3–L5 interneurones which are co-excited by Ia and Ib afferents, or by both group I and group II, cutaneous and joint afferents (Edgley & Jankowska, 1987*b*). If this were the case, it would be interesting to know how their activity is related to the activity of interneurones with the same pattern of input, but which exert postsynaptic actions upon motoneurones and/or other neurones. The 'presynaptic' interneurones might operate to provide negative feed-back in pathways in which there are interposed interneurones with postsynaptic actions. However, they might also serve to alter the balance between different types of input to the latter, or assist in preventing inappropriate subpopulations of 'postsynaptic' interneurones being activated. Whatever the answer, the similar origin of PAD of terminals of group II afferents in midlumbar segments and of input to midlumbar interneurones interposed in reflex pathways from group II afferents to lumbar motoneurones (Edgley & Jankowska, 1987*b*; Cavallari, Edgley & Jankowska, 1987) shows that there must be a close linkage between them.

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REFERENCES

Boyd, I. A. & Davey, M. R. (1968). *The Composition of Peripheral Nerves.* Edinburgh: Livingstone.

Bradley, K. & Eccles, J. C. (1953). Analysis of fast afferent impulses from thigh muscles. *Journal of Physiology* **122**, 462–473.

Brink, E., Jankowska, E. & Skoog, B. (1984). Convergence onto interneurones subserving primary afferent depolarization of group I afferents. *Journal of Neurophysiology* **51**, 432–449.

Burke, R. E. & Rudomin, P. (1977). Spinal neurons and synapses. In *Handbook of Physiology. The Nervous System*, section 1, vol. 1, part 2, chapter 24, pp. 877–944. Bethesda: American Physiological Society.

Carpenter, D., Lundberg, A. & Norrsell, U. (1963). Primary afferent depolarization evoked from the sensorimotor cortex. *Acta physiologica scandinavica* **59**, 126–142.

CAVALLARI, P., EDGLEY, S. A. & JANKOWSKA, E. (1987). Postsynaptic actions of midlumbar interneurones on motoneurones of hindlimb muscles in the cat. *Journal of Physiology* **389**, 675–689.

ECCLES, R. M. & LUNDBERG, A. (1959). Synaptic actions in motoneurones by afferents which may evoke the flexion reflex. *Archives italiennes de biologie* **97**, 199–221.

ECCLES, J. C., SCHMIDT, R. G. & WILLIS, W. D. (1963). Depolarization of central terminals of group Ib afferent fibres of muscle. *Journal of Neurophysiology* **26**, 1–27.

EDGLEY, S. A. & JANKOWSKA, E. (1987*a*). Field potentials generated by group I and II muscle afferents in the middle lumbar segments of the cat spinal cord. *Journal of Physiology* **385**, 393–413.

EDGLEY, S. A. & JANKOWSKA, E. (1987*b*). An interneuronal relay for group I and II muscle afferents in the midlumbar segments of the cat spinal cord. *Journal of Physiology* **389**, 647–674.

ELLAWAY, P. H., MURPHY, P. R. & TRIPATHI, A. (1982). Closely coupled excitation of gamma motoneurones by group III muscle afferents with low mechanical threshold in the cat. *Journal of Physiology* **331**, 481–498.

FERN, R., HARRISON, P. J. & RIDDELL, J. S. (1988). The dorsal column projection of muscle afferent fibres from the cat hindlimb. *Journal of Physiology* **401**, 97–113.

HARRISON, P. J., JAMI, L. & JANKOWSKA, E. (1988). Further evidence for synaptic actions of muscle spindle secondaries in middle lumbar segments of the cat spinal cord. *Journal of Physiology* **402**, 671–686.

HARRISON, P. J. & JANKOWSKA, E. (1984). Do interneurones in lower lumbar segments contribute to the presynaptic depolarization of group I muscle afferents in Clarke's column? *Brain Research* **295**, 203–210.

HARRISON, P. J. & JANKOWSKA, E. (1987). Origin of primary afferent depolarization of group II muscle afferent fibres in the cat spinal cord. *Journal of Physiology* **390**, 43*P*.

JACK, J. J. B. (1978). Some methods for selective activation of muscle afferent fibres. In *Studies in Neurophysiology presented to A. K. McIntyre*, ed. PORTER, R., pp. 155–176. Cambridge: Cambridge University Press.

JANKOWSKA, E. (1984). On neuronal pathways of presynaptic depolarisation of group I muscle afferents. *Experimental Brain Research*, suppl. 9, 72–85.

JANKOWSKA, E., JUKES, M. & LUND, S. (1965). The pattern of presynaptic inhibition of transmission to the dorsal spinocerebellar tract of the cat. *Journal of Physiology* **178**, 17–18*P*.

JANKOWSKA, E., MCCREA, D., RUDOMIN, P. & SYKOVA, E. (1981). Observations on neuronal pathways subserving primary afferent depolarization. *Journal of Neurophysiology* **46**, 506–516.

JANKOWSKA, E. & PADEL, Y. (1984). On the origin of presynaptic depolarization of group I muscle afferents in Clarke's column in the cat. *Brain Research* **295**, 195–201.

JIMENEZ, I., RUDOMIN, P. & SOLODKIN, M. (1988). PAD patterns of physiologically identified afferent fibres from the medial gastrocnemius muscle. *Experimental Brain Research* **71**, 643–657.

JUKES, M. G. M. (1965). The presynaptic inhibition of the group I pathway to the dorsal spinocerebellar tract of the cat. Thesis, Oxford University, Oxford.

LUNDBERG, A. (1982). Inhibitory control from the brain stem of transmission from primary afferents to motoneurones, primary afferent terminals and ascending pathways. In *Brain Stem Control of Spinal Mechanisms*, ed. SJÖLUND, B. & BJÖRKLUND, A. Amsterdam: Elsevier Biomedical Press.

MACLENNAN, C. R. (1972). The behaviour of receptors of extramuscular and muscular origin with afferent fibres contributing to the group I and the group II of the cat tibialis anterior muscle nerve. *Journal of Physiology* **222**, 90–91*P*.

RUDOMIN, P., JIMENEZ, I., SOLODKIN, M. & DUENAS, S. (1983). Sites of action of segmental and descending control of transmission on pathways mediating PAD of Ia- and Ib-afferent fibres in cat spinal cord. *Journal of Neurophysiology* **50**, 743–769

RUDOMIN, P., SOLODKIN, M. & JIMENEZ, I. (1986). PAD and PAH response patterns of group Ia and Ib fibres to cutaneous and descending inputs in the cat spinal cord. *Journal of Neurophysiology* **56**, 987–1006.

SCHMIDT, R. F. (1971). Presynaptic inhibition in the vertebrate central nervous system. *Ergebnisse der Physiologie Biologischen Chemie und Experimentellen Pharmacologie* **63**, 20–101.

WALL, P. D. (1958). Excitability changes in afferent fibre terminations and their relation to slow potentials. *Journal of Physiology* **142**, 1–21.