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SHARED REFLEX PATHWAYS FROM Ib TENDON ORGAN AFFERENTS AND Ia MUSCLE SPINDLE AFFERENTS IN THE CAT

BY E. JANKOWSKA AND D. A. McCREA*
From the Department of Physiology, University of Göteborg, Sweden

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SUMMARY

1. The possibility was investigated that group Ia muscle spindle afferents and group Ib tendon organ afferents influence spinal motoneurones via shared neuronal pathways. Mutual facilitation of actions of these afferents at a premotoneuronal level has been taken as evidence that they use the same interneurones to evoke post-synaptic potentials (p.s.p.s) in motoneurones.

2. Inhibitory p.s.p.s (i.p.s.p.s) or excitatory p.s.p.s (e.p.s.p.s) were evoked in motoneurones by selective activation of group Ia afferents or group Ib afferents. P.s.p.s following stimulation of both Ia and Ib afferents were then compared with the arithmetic sum of p.s.p.s evoked by each of them separately. When the former were larger the difference was used as a measure of synaptic actions mediated by interneurones co-excited by Ia and Ib afferents.

3. Both excitatory and inhibitory pathways to motoneurones have been found to be shared by Ia and Ib afferents, although the proportion of interneurones actually used in common by these afferents could not be established. The latencies of post-synaptic actions mediated by such interneurones indicated that they were evoked disynaptically or trisynaptically.

4. The study leads to two main conclusions: that group Ia muscle spindle afferents, and in consequence also fusimotor systems, may modulate the reflex action of tendon organs, and that the two groups of afferents are a source of information in a common feed-back system.

INTRODUCTION

It has recently been shown that the reflex actions of group Ib tendon organ afferents upon motoneurones are paralleled by actions of group Ia muscle spindle afferents (Fetz, Jankowska, Johannisson & Lipski, 1979; Jankowska, McCrea & Mackel, 1981*b*, *c*). While attempting a functional interpretation of this finding two possibilities might be considered. Firstly, the two groups of receptors may exert their actions independently of each other, via quite separate pathways. The second possibility is that the actions of these receptors are intimately linked and mutually interdependent, being evoked via the same neuronal systems. The latter possibility

* Present address: Playfair Neuroscience Unit, Toronto Western Hospital, 399 Bathurst St., Toronto, Ontario, Canada M5T 2S8.

has been favoured by the demonstration that a considerable proportion of Rexed's laminae V–VI interneurones are excited by both Ia and Ib afferents (Czarkowska, Jankowska & Sybirska, 1981; Jankowska, Johannisson & Lipski, 1981*a*) and, since some project to motor nuclei, may mediate the reflex actions of these afferents onto motoneurones. That these interneurones were actually involved in such actions remained, however, to be established.

The aim of the present study was to investigate whether the transmission in the pathways from Ia afferents to motoneurones can be facilitated by impulses in Ib afferents, or vice versa, as would be required for synaptic actions mediated by the same neurones. Group Ia and Ib afferents were selectively activated, each near threshold for firing the interposed interneurones and for evoking minimal excitatory post-synaptic potentials (e.p.s.p.s) or inhibitory post-synaptic potentials (i.p.s.p.s) in motoneurones. Spatial summation of actions of these two groups of afferents at a premotoneuronal level, with the enhancement of the resulting actions upon the motoneurones, was then taken as evidence for their convergence upon the same interneurones (cf. Lundberg & Voorhoeve, 1962; Lundberg, 1975, 1979). Some of the preliminary observations were made together with E. Fetz & J. Lipski (cf. Jankowska, 1979) and some of the present results have been published as a short communication (Jankowska & McCrea, 1980).

METHODS

Preparation. The successful experiments were performed on six cats under chloralose anaesthesia (50–60 mg/kg I.V. initial dose) and spinalized at Th 12–13 level; the dissections was carried out under ether. The cats were paralysed with gallamine triethiodide and artificially ventilated. Approximately 1 cm of the calcaneal bone was cut off with the triceps surae and plantaris tendons attached and was connected to a muscle puller (cf. Fetz *et al.* 1979) to allow simultaneous stretches of these muscles. The nerves to medial gastrocnemius, lateral gastrocnemius and soleus, and in some experiments also to plantaris, were left attached to the muscles but dissected free so that they could be stimulated separately. A number of other hind-limb muscle nerves were cut and mounted for stimulation to allow identification of motoneurones. Among these were usually nerves to the following muscles: quadriceps, posterior biceps and semitendinosus, anterior biceps and semimembranosus, flexor digitorum longus, plantaris, tibialis anterior, extensor digitorum longus, peroneus longus, tertius and brevis and the distal part of the tibial nerve. L7 and S1 ventral roots were cut to de-efferent muscle spindles of the stretched muscles and were mounted for stimulation to aid the location and identification of the motoneurones.

Selective activation of Ia and Ib afferents. Since the aim of the experiments was to investigate the occurrence of a mutual facilitation of actions of group Ia and Ib afferents it was essential to ensure that these groups of afferents were stimulated selectively. The group Ia muscle spindle afferents were activated by brief stretches of triceps surae and plantaris, always < 35 μm and in more than half of the trials < 20 μm. The stretches were applied at an initial tension of 5·0–5·5 N which corresponded to muscle lengths of about 2–4 mm less than the maximal physiological lengths. Under these conditions activation of Ib afferents was previously found to require more than 40 μm stretch (Fetz *et al.* 1979; Jankowska *et al.* 1981*a*). In order to differentiate Ia actions from actions of group II afferents only the shortest latency ($< 3·0$ msec) synaptic actions evoked by 30–40 μm stretches were used for the analysis (cf. Jankowska *et al.* 1981*b*, *c*): longer latencies were accepted only for p.s.p.s evoked by near threshold stimuli.

Group Ib afferents were stimulated electrically after having increased threshold for excitation of group Ia afferents by electrical stimuli above that for group Ib afferents using the method of Coppin, Jack & McLennan (1970; see also Fetz *et al.* 1979). This involved vibration of triceps surae and plantaris muscles (at 200 Hz, with peak to peak amplitudes 70–80 μm, i.e. maximal for all group Ia afferents) until the threshold for group Ia afferents was increased to about 1·3–1·4 times their

original threshold. Great precautions were taken to avoid any damage of the vibrated muscles or their nerves, since damage of even single muscle spindles or Ia afferent axons would prohibit the threshold of these axons being raised. The effectiveness of the vibration was always monitored using intracellular records from a triceps surae or plantaris motoneurone, depending on which of the muscle nerves was to be stimulated. Fig. 1 shows that when the vibration was effective, excitability of Ia afferents changed rather rapidly. In the illustrated case, the electrical stimulation at 1·1 times the original threshold failed to evoke Ia monosynaptic e.p.s.p.s after 2 min of vibration. After 4 min

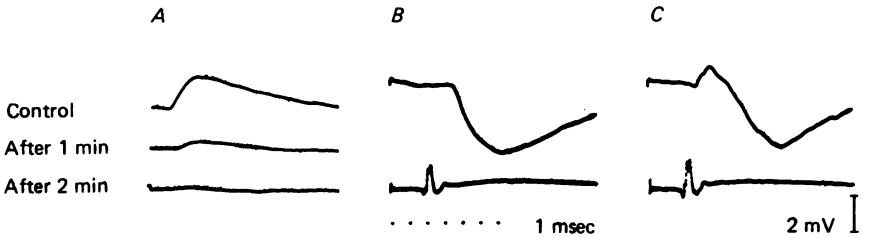

Fig. 1. Effectiveness of muscle vibration in increasing thresholds for excitation of Ia afferents by electrical stimuli. All records in *A* and upper traces in *B* and *C* are averaged intracellular records from a plantaris and a lateral gastrocnemius–soleus motoneurone, respectively; lower traces in *B* and *C* are from the dorsal root entry zone. *A* shows that increase in threshold occurs already within the first minute of vibration and becomes pronounced after 2 mins. *B* and *C* illustrate that an effective activation of Ib afferents, as judged by the size of the i.p.s.p.s may be evoked below (*B*) or near (*C*) threshold for Ia afferents. It will be noted, however, that practically no i.p.s.p.s were evoked at 1·1 times threshold (bottom trace in *A*). In this and in the following figures voltage calibrations are only for intracellular records.

1·2 times threshold stimuli also became ineffective. Such electrical stimuli evoked then only i.p.s.p.s which were attributed to Ib afferents (Fig. 1 *B*; cf. Fetz *et al.* 1979). The disappearance of Ia e.p.s.p.s in homonymous motoneurones was considered as the criterion for the lack of excitation of Ia afferents by nerve stimulation, as a very large proportion of Ia afferents of a muscle terminate upon individual homonymous α motoneurones (Mendell & Henneman, 1971; Scott & Mendell, 1976). There is no reason to expect that threshold for excitation of afferents terminating upon the impaled motoneurones would be different than that for other fibres; in fact our observation that thresholds for e.p.s.p.s recorded in different motoneurones were practically the same would speak against such a possibility.

Experimental procedure. Two micro-electrodes were introduced into two motor nuclei to allow simultaneous recording from two motoneurones. One electrode was used to penetrate a triceps surae or plantaris motoneurone (a 'control motoneurone') to monitor the effectiveness of the electrical stimulation and to ensure that it could activate a reasonable proportion of Ib afferents (as evidenced by the appearance of disynaptic autogenetic i.p.s.p.s) below threshold for any Ia afferents (as evidenced by lack of monosynaptic e.p.s.p.s). If the threshold of Ia afferents was not sufficiently raised after 30 min of vibration, prolongation of the vibration was not of much help and such cats could not be used for purposes of these experiments. Additional 5–10 min of vibration were, however, added every 15–20 min, or when needed throughout the experiment, to prevent, or slow, the return of the threshold of Ia afferents to the original level. Nevertheless, the selectiveness of Ib activation progressively deteriorated and after a few hours usually only the weakest stimuli were below the threshold for Ia afferents. When recording from the original control motoneurone was no longer possible, another motoneurone was penetrated and the threshold of Ia afferents was checked. The second micro-electrode was used to record from motoneurones to be tested. These were, as a rule, depolarized by 5–15 nA to increase the amplitude of the recorded i.p.s.p.s. Group Ia and Ib afferents were activated in the following alternating sequence: Ia afferents, Ib afferents, both Ia and Ib afferents. Thirty-two to one hundred and twenty-eight responses evoked by them were successively stored in separate quadrants of the averager's (Nicolet, model 1170) memory.

This procedure obviated problems arising from variations in recording conditions during presentation of one type of stimulus and not another; the preliminary observations made with E. Fetz and J. Lipski were considered to be not sufficiently reliable in this respect because in the averager previously available only two of the three sets of data could be stored in parallel. Single sweep records displayed on a separate oscilloscope were photographed during the collection of the averaged data. Records were then taken from both the control and the test motoneurones. Averaged records from control motoneurones were taken separately.

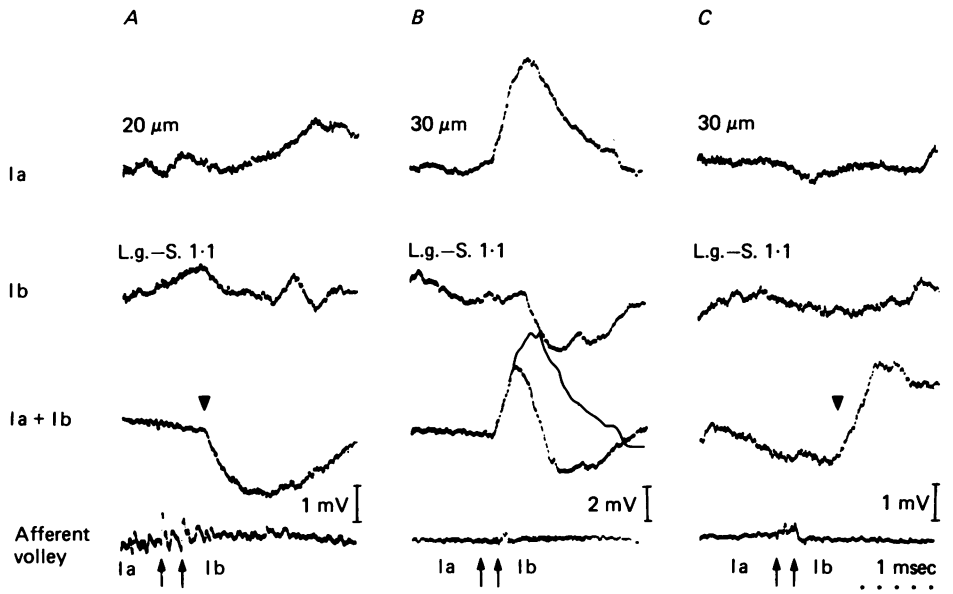

Fig. 2. Examples of separate and combined actions of Ia and Ib afferents in the motoneurones. All but lowermost traces are intracellular records from a flexor digitorum or hallucis longus (*A*), a lateral gastrocnemius or soleus (*B*) and a posterior biceps or semitendinosus (*C*) motoneurones; the latter two recorded from simultaneously. The responses were evoked by only Ia afferents (20 or 30 μm muscle stretches), only Ib afferents (1·1 times threshold electrical stimuli) or both, as indicated to the left of the records. The timing of the arrival of the afferent volleys (arrows) to the dorsal root entry zone is shown in the bottom records which were taken from the surface of the spinal cord. Records in *B* are for a control motoneurone. They were taken to ascertain that such electrical stimuli were below threshold for group Ia afferents. The i.p.s.p.s evoked by such stimuli were enhanced by the simultaneously activated Ia afferents, as appears from the difference between the first and third records (see the area below the thin line projection of the stretch-evoked response). L.g.–S., lateral gastrocnemius–soleus.

Data analysis. It was concluded that the Ia and Ib afferents converged onto the same interneurones when (i) p.s.p.s evoked by combined actions of these afferents were larger than the arithmetic sum of p.s.p.s evoked by each of them separately; (ii) when this was found in two or more independent tests and (iii) when the difference was of at least 100 μV. The additions and the subtractions were performed using the averager's internal circuitry for data manipulation. The latencies of the p.s.p.s and of the differences between them were measured from the time of arrival of the afferent volleys to the dorsal root entry zone; the records of these volleys were stored in the fourth quadrant of the averager's memory.

RESULTS

Convergence of Ia and Ib afferents in inhibitory pathways to motoneurones

The investigated inhibitory pathways were from ankle and toe extensors (plantarflexors), primarily triceps surae, to various extensor motoneurones of a hind limb. The motoneurones selected for analysis were those in which distinct disynaptic i.p.s.p.s were evoked by Ia as well as by Ib afferents of the same muscles and in which the i.p.s.p.s were not preceded by any significant e.p.s.p.s. The sample included eleven quadriceps, seven flexor digitorum or hallucis longus, one anterior biceps or semimembranosus and five plantaris motoneurones. The analysed pathways were thus those of the synergistic inhibition.

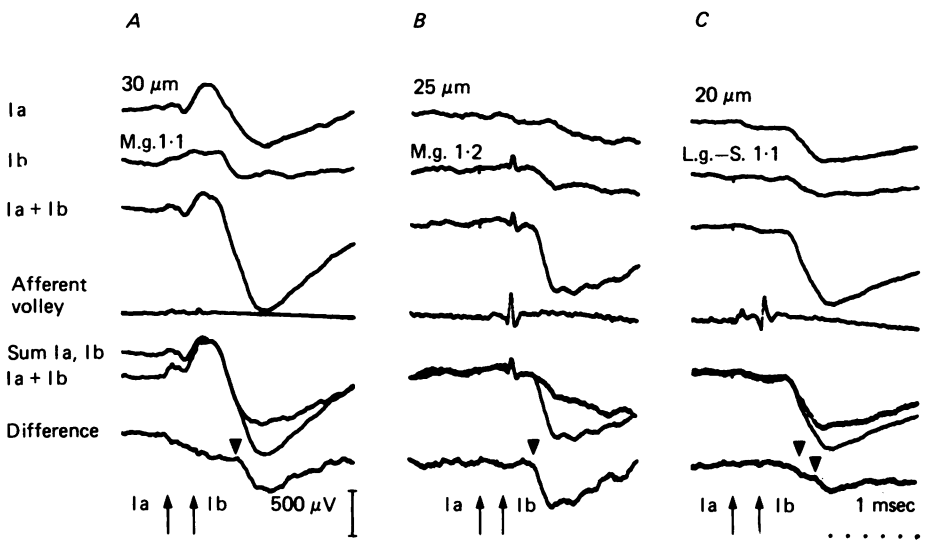

Fig. 3. Mutual facilitation of Ia and Ib inhibitory actions. Data for two plantaris (*A*, *B*) and one flexor digitorum longus (*C*) motoneurones are shown. The first three traces in each column are averaged intracellular records of p.s.p.s evoked by short muscle stretches (Ia), electrical stimuli selective for tendon organ afferents (Ib) and the two together (Ia+Ib). Middle traces are records of afferent volleys from the dorsal root entry zone. In the lower set of traces are the sums of the i.p.s.p.s evoked by separately activated Ia and Ib afferents which are superimposed on responses evoked by combined actions of these afferents to show the differences between them. The differences are displayed underneath. Arrow heads indicate onset of i.p.s.p.s evoked by Ia and Ib co-excited interneurones. Arrows indicate time of arrival of Ia and Ib volleys. M.g., medial gastrocnemius; L.g.–S., lateral gastrocnemius–soleus.

Fig. 2*A* and *B* illustrates mutual facilitation of the Ia and Ib inhibitory actions with single sweep records, while averaged records are shown in Fig. 3. In these Figures two top rows show p.s.p.s evoked by selective activation of either Ia or Ib afferents. The third row shows effects of stimulation of both these afferents; the afferent volleys are seen in the fourth row, Recruitment of additional interneurones due to combined Ia and Ib actions could often be seen in individual records, with clear i.p.s.p.s (head of arrow in Fig. 2*A*) following the two stimuli, while no or much smaller i.p.s.p.s

appeared after each of them separately. The same tendency was seen in the case of autogenetic i.p.s.p.s recorded in control lateral gastrocnemius–soleus motoneurones (compare second and third records from the top in Fig. 2*B*). Averaged records of Fig. 3 further illustrate mutual facilitation of Ia and Ib actions for three other motoneurones. In one of these a small Ia e.p.s.p. preceded the i.p.s.p.s, while in the remaining two only the i.p.s.p.s were evoked. The arithmetical sum of separately evoked Ia and Ib p.s.p.s has been superimposed on traces showing the combined Ia and Ib actions. These combined actions were much larger and the differences are shown in the bottom traces. The amplitudes of these differences greatly varied depending on, for example, the magnitude of stretches and of electrical stimuli alone, time intervals between them or a choice of the nerve used for electrical stimulation. All these factors clearly influenced the number of interneurones which remained within the subliminal fringe of separate actions of Ia and Ib afferents. The largest differences (see Fig. 4*C*) indicate that a considerable proportion of interneurones mediating the recorded i.p.s.p.s may have belonged to those co-excited by Ia and Ib afferents. However, all the estimates based on the relative amplitudes of these differences must remain underestimates since stimulation of either Ia or Ib afferents alone might result in activation of interneurones co-excited by these afferents, as well as of interneurones with a more selective input. Neither could a lack of spatial facilitation be used to exclude the contribution of common interneurones since it might have been primarily due to inadequate parameters of stimulation. In addition, a number of records were eliminated when the likely differences appeared to be of smaller amplitude than 100 μV (see Methods) or when the noise level was too great.

The optimum conditions for demonstrating the spatial facilitation of Ia and Ib actions appeared to be when stimulation of Ia afferents was suprathreshold and that of Ib afferents very close to threshold for firing the interposed interneurones, and when the Ia volleys preceded the Ib volleys. The ranges of the effective time intervals were, however, rather large as illustrated in Fig. 4*C*. Open circles show the contribution of interneurones co-excited by Ia and Ib afferents, for different time intervals between the stretches and the electrical stimuli. The zero results included in this Figure are of value, in contrast to those for other neurones, since they were obtained in the same motoneurones which showed spatial facilitation at other interstimulus intervals.

The effects of the spatial facilitation appeared with different latent periods with respect to the Ia and Ib volleys, as shown in Fig. 4*A*. About two thirds of these latent periods fell within the ranges for disynaptic i.p.s.p.s evoked by electrical stimulation of Ib afferents (up to 2·0 msec, Eccles, Eccles & Lundberg, 1957; Fetz *et al.* 1979; Jankowska *et al.* 1981*b*) or by adequate activation of Ia afferents (up to 2·8–3·0 msec, Fetz *et al.* 1979; Jankowska *et al.* 1981*b*). They are represented by data points below and to the left of the dashed lines. Some of the longer latent periods likewise might be compatible with the disynaptic coupling, considering that the interneurones responsible for the i.p.s.p.s were activated only by the latest components of the weak asynchroneous Ia volleys. As seen in Fig. 5*A*, about 1 msec may separate the earliest and the latest components of these volleys.

The data of Fig. 4*A* are replotted in Fig. 4*B* to illustrate relation between effects of the spatial facilitation with respect to the onset of i.p.s.p.s evoked by selective

activation of Ia and Ib afferents. As will be seen, i.p.s.p.s due to recruitment of additional Ia and Ib co-excited interneurones usually appeared with latencies which were equal to or shorter than the latencies of the i.p.s.p.s evoked by either Ia or Ib afferents alone. However, such a comparison could be done only for about half of the i.p.s.p.s; the remaining ones, among which were fifteen of the nineteen i.p.s.p.s with the longest latencies were not matched by i.p.s.p.s with a sufficiently sharp onset

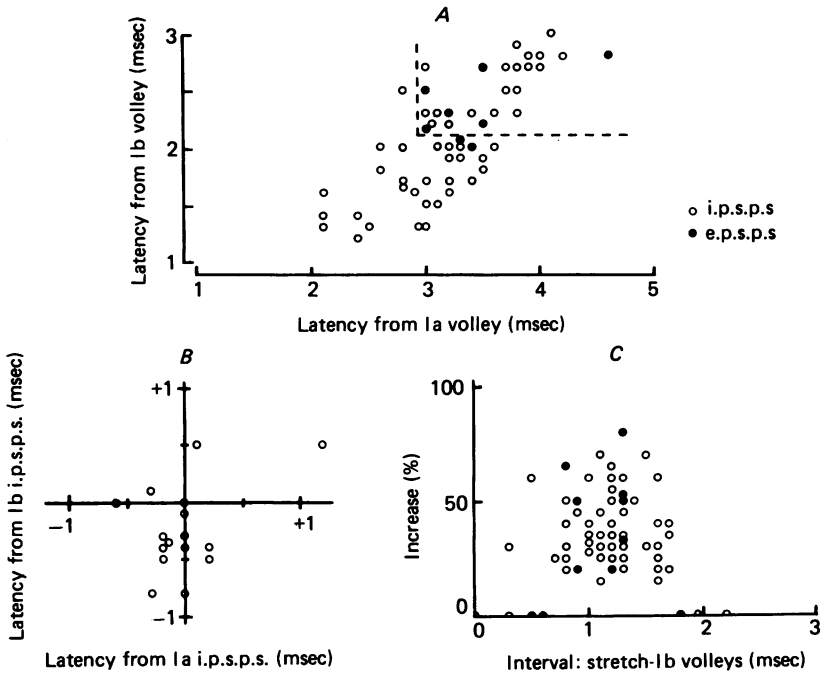

Fig. 4. A comparison of separate and combined actions of Ia and Ib afferents. *A*, latencies of synaptic actions of interneurones co-excited by Ia and Ib afferents, i.e. the 'differences' illustrated in lowermost records of Figs. 3 and 5; they were measured with respect to the Ia afferent volleys (abscissa) and Ib afferents volleys (ordinate). The number of data points exceeds the number of motoneurones recorded from since the latencies of i.p.s.p.s obtained for different conditioning testing intervals or at different stimulus intensities were counted separately. *B*, latencies of sixteen out of fifty-six p.s.p.s plotted in *A* with respect to the onset of i.p.s.p.s of Ia and Ib origin; the remaining i.p.s.p.s were too small or without sufficiently distinct onset to allow the comparison of the latencies. *C*, a comparison of the amplitudes of the 'differences' and of the amplitudes of combined actions of Ia and Ib afferents. The sizes of the former are expressed in percents of the latter, taken as 100%; they are plotted against various intervals between Ia and Ib volleys. Open circles, data for i.p.s.p.s. Filled circles, data for e.p.s.p.s.

following activation of a single afferent group. Since maximal group I stimuli evoked disynaptic i.p.s.p.s in all the selected motoneurones, the plot of Fig. 4*B* suggests that at least some of the longer latency i.p.s.p.s due to combined actions of Ia and Ib afferents may have been mediated by disynaptic pathways. However, a contribution of trisynaptic pathways would also be a possibility, especially for later components of the i.p.s.p.s. One of the clearest separate later components is indicated in Fig. 3*C* by the second arrow head.

Convergence of Ia and Ib afferents in excitatory pathways to motoneurones

Under usual experimental conditions the disynaptic excitation from group I afferents is much less frequent than the inhibition. For this reason it has been analysed in a smaller sample of seven posterior biceps-semitendinosus motoneurones recorded in three experiments. Following electrical stimuli maximal for group I afferents of medial gastrocnemius, lateral gastrocnemius–soleus or plantaris, e.p.s.p.s appeared with latencies 1·8 to 3·0 msec. Stretches of these muscles evoked e.p.s.p.s with latencies 2·0–2·8 msec. Electrical stimuli activating Ib afferents which were

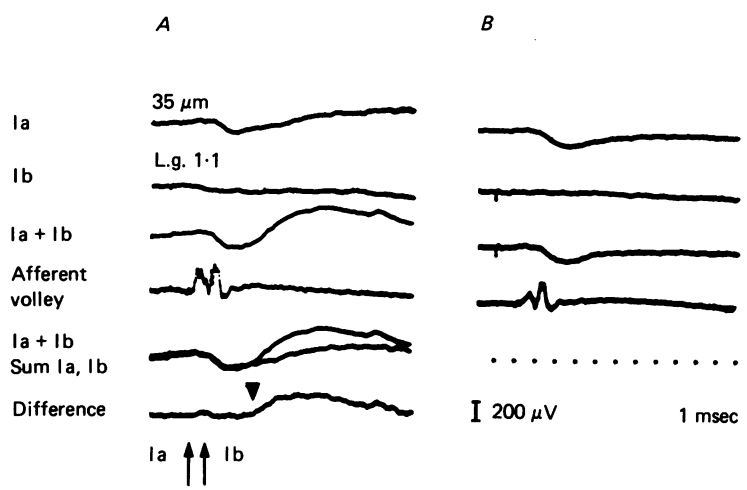

Fig. 5. Mutual facilitation of Ia and Ib excitatory actions. *A*, data for a posterior biceps or semitendinosus motoneurone. *B*, corresponding extracellular records. The layout of the records as in Fig. 3. Extracellular potentials around neurones illustrated in Fig. 3 were very similar to those in *B*. In neither case were any Ib extracellular potentials detected.

below threshold for Ia afferents evoked practically no e.p.s.p.s on their own but enhanced effects of the stretch activated Ia afferents, as illustrated with records from two motoneurones in Fig. 2*C* and Fig. 5*A*. The bottom trace of Fig. 5*A* shows, as in Fig. 3, to what extent the combined actions of Ia and Ib afferents were more effective than the sum of their separate actions. Such a facilitation has been seen in six out of the seven motoneurones. In the remaining one combined actions of Ia and Ib afferents appeared to be less excitatory than the sum of the separate ones. Occurrence of depression rather than facilitation was also seen in some preliminary observations (E. Fetz, E. Jankowska & J. Lipski, unpublished) on inhibitory pathways. However, since both e.p.s.p.s and i.p.s.p.s of group I origin may be evoked in the same motoneurones (Jankowska *et al.* 1981*b*, *c*) it was not possible to decide how much of the depression was due to a genuine inhibition at the interneuronal level (Jankowska *et al.* 1981*a*; see also Brink, Jankowska, McCrea & Skoog, 1983) and how much to a facilitation of opposite synaptic actions. The occlusion was not either easy to exclude unless one of the stimuli had practically no effect by itself.

The facilitation of e.p.s.p.s occurred at similar interstimulus intervals and with

similar segmental latencies (filled circles in Fig. 4*A* and *C*) as the facilitation of i.p.s.p.s. The observations made in a larger sample of neurones with the disynaptic inhibition may thus be valid for the excitatory pathways.

DISCUSSION

Muscle spindles and tendon organs as a source of information in a common feed-back system

The results of this study demonstrate that the reflex actions of tendon organs and of muscle spindle primaries of triceps surae and plantaris can be mediated by the same interneurones. It has been evidenced for inhibition of a few synergist extensor motoneurones and excitation of one species of flexor motoneurones. Weaker and less frequent actions of these afferents on other motoneurone species, and the autogenetic inhibition, were not tested since the experimental conditions are not as favourable in their case. In particular we tried to avoid problems of interpretation of changes of small i.p.s.p.s superimposed on large e.p.s.p.s; in all the selected motoneurones only small monosynaptic e.p.s.p.s were evoked and the i.p.s.p.s outlasted their duration. However, previous observations indicated that both Ia and Ib afferents of triceps surae and plantaris are the source of non-reciprocal inhibition and of oligosynaptic excitation of all the motoneurone species in which inhibition from group I afferents was demonstrated (Jankowska *et al.* 1981*b*, *c*) and that the same interneurones may mediate inhibition, or excitation, of several motoneurone species (Czarkowska *et al.* 1981; Jankowska *et al.* 1981*a*). We propose, therefore, to generalize the conclusions of the reported observations and to extend them to all actions of Ia and Ib afferents of triceps surae and plantaris on motoneurones. The following paper (Harrison, Jankowska & Johannisson, 1983) will show that they may be extended to actions of Ia and Ib afferents of other muscles as well.

Interneurones co-excited by Ia and Ib afferents appeared to be responsible for a considerable part of synaptic potentials evoked from these afferents. Unfortunately, the technique of spatial facilitation allows one to draw conclusions on convergence at a premotoneuronal level in a qualitative but not a quantitative way, and it does not specify the proportions of the involved interneurones. On the basis of the reported observations it is thus difficult to estimate how large is the fraction of interneurones used in common by Ia and Ib afferents among all interneurones interposed between these afferents and motoneurones. The results of the previous study (Jankowska *et al.* 1981*a*) do, however, indicate that the fraction might be considerable. More than 40 % of laminae V–VI interneurones appeared to be co-excited by Ia and Ib afferents of triceps surae and plantaris against 50 % of interneurones found to be excited by only Ib afferents of these muscles. Some of the latter, but not of the former, would be among interneurones in the pathways of the presynaptic depolarization (E. Brink, E. Jankowska & B. Skoog, in preparation); consequently in the pathways to motoneurones even more than 40 % of interneurones might be excited by both Ia and Ib afferents.

Use of the same interneurones by several fibre systems is by no means unfrequent; on the contrary, it appears to be a rule in all types of interneurones investigated (cf. Lundberg, 1979) and the interneurones mediating reflex actions from Ia and Ib

afferents are no exception in this respect. Since it has been demonstrated that transmission via these interneurones can be strongly influenced by impulses from joint and cutaneous receptors (Lundberg, Malmgren & Schomburg, 1977, 1978; Pierrot-Deseilligny, Bergego, Katz & Morin, 1981; Pierrot-Deseilligny, Bergego & Katz, 1982), as well as by descending systems (see Lundberg, 1975), these interneurones must be links in common feed-back systems from all of these afferents. With respect to the feed-back from muscle receptors, Houk and collaborators (for references see Houk, 1979) have already considered muscle stiffness as being regulated by both muscle length (with excitatory drive from Ia spindle afferents) and muscle tension (with inhibitory drive from Ib tendon organs), but proposed that the two groups of afferents contribute to two separate feed-back pathways with a site of interaction at the motoneuronal level. The results of this study demonstrate that a great part of such an interaction occurs at a premotoneuronal level, and that the actions of the Ia afferents at this level are inhibitory to motoneurones and would, therefore, subtract from their direct excitatory actions on motoneurones. Consequently, any hypothesis trying to give as faithful as possible picture of the intergratory processes in reflexes evoked from muscle spindles and tendon organs, must take into account the multiple sensory and descending, excitatory and inhibitory control of the involved interneurones (for further discussion see Harrison, Jankowska & Johannisson, 1983).

Fusimotor control of tendon organ actions

Possible direct actions on tendon organs. In contrast to muscle spindles, and a number of other receptors, Golgi tendon organs appear to lack a separate centrifugal system modifying their excitability (see Prochazka, 1981). Responses of tendon organs to changes in muscle tension could, however, be modulated also in another way: by the surrounding extrafusal muscle fibres or by contractions of intrafusal muscle fibres of spindles in close relation to them. Morphological studies have shown that a considerable proportion of muscle spindles are located in the neighbourhood of tendon organs; they may form 'tendon organ-spindle dyads', with two receptors lying side by side (Marchand, Bridgman, Shumpert & Eldred, 1971; Richmond & Abrahams, 1975), or in line, with tendon organs attached to intrafusal muscle fibres (Barker, 1948; Marchand *et al.* 1971), with observations of this kind going back to Sherrington (1894). Contractions of intrafusal muscle fibres evoked by γ and β fusimotor fibres might thus influence not only the spindles but also tendon organs. The possibility has already been considered by Granit (1950) in relation to his observations that stimulation of γ fibres increased the autogenetic inhibition of motoneurones following muscle contractions, but he could not differentiate between effects of activation of spindle afferents (primary or secondary), and effects of activation of tendon organs by contractions of intrafusal fibres. Hunt (1952), who similarly found inhibitory actions of fusimotor fibres attributed them to spindle afferents. Whether γ fusimotor neurones modulate the excitability of tendon organs, and to what extent, remains to be established but the effects of β fusimotor neurones would be the same as of α motoneurones.

Indirect actions via muscles spindle afferents. Regardless of whether or not fusimotor neurones influence tendon organs directly, the present results show that they must play an important role in modulating transmission from tendon organs at the level

of the spinal interneurones, via Ia afferents. The observations on non-reciprocal inhibition of motoneurones evoked from Ia afferents (Fetz *et al.* 1979) have shown that it is much weaker than the inhibition evoked from Ib afferents, and led to the conclusion that its main function might be to support the Ib actions, rather than to act on its own right. The weakness of the Ia non-reciprocal inhibition and of other selective Ia-like-Ib actions (Jankowska *et al.* 1981*b*, *c*) at the motoneuronal level does not imply that the effects of Ia muscle spindle afferents at a premotoneuronal level are similarly weak. Since these afferents terminate on the same interneurones as Ib afferents, as now demonstrated, they may very effectively influence reflex actions evoked from tendon organs. Any increase or decrease in the inflow of impulses from the primary muscle spindle afferents, evoked by stretch or by fusimotor neurones, must be of consequence for the interneurones which are excited both by these afferents and by the tendon organ afferents. In this context it should be remembered that impulses in Ia afferents can inhibit, as well as excite laminae V–VI interneurones (Hongo, Jankowska & Lundberg, 1966, 1972; Jankowska *et al.* 1981*a*; see also Brink *et al.* 1983), although their facilitatory action appeared to be dominant under present experimental conditions. The fusimotor systems may thus set the level of excitability of these interneurones or modulate the frequency of their discharges in both directions, as they do in the case of the muscle spindles.

In view of these considerations it becomes of great interest to know when, in which movements or phases of movements, the Ia and Ib afferents are activated in parallel. During isometric contractions, α–γ co-activation would almost certainly lead to parallel activity of both Ia and Ib afferents, as would a lengthening contraction, i.e. when the muscle is developing tension but its length actually increases (Engberg & Lundberg, 1969). The situation may, however, be quite different for a shortening contraction. Both in the cat (see Prochazka, 1981 for references) and in man (Vallbo, 1973; Burg, Szumski, Struppler & Velho, 1976; Burke, Hagbarth & Lofstedt, 1978) examples have been reported of Ia afferent activity occurring during muscle shortening. However, the arguments of Prochazka, Stephens & Wand (1979) indicate that the fusimotor systems can only maintain the Ia afferents firing so long the rate of muscle shortening is not above a certain value, or unless under particular conditions (Appenteng, Prochazka, Proske & Wand, 1982). We will, therefore, have to await further studies describing the circumstances when Ia and Ib afferents are actually co-activated. The co-activation of the afferents originating in different muscles would be of as much interest as the co-activation of afferents of the same muscle, as will be shown in the following paper (Harrison, Jankowska & Johannisson, 1983).

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