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COMMON INTERNEURONES IN REFLEX PATHWAYS FROM GROUP Ia AND Ib AFFERENTS OF ANKLE EXTENSORS IN THE CAT

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SUMMARY

1. Input from group I afferents of ankle and toe extensors, other muscles, skin nerves and descending tracts to interneurones of Rexed's laminae V–VI in the cat spinal cord was analysed using intracellular recording from these interneurones. Adequate stimuli (muscle stretches) were used to activate selectively group Ia muscle spindle afferents of triceps surae and plantaris while other fibre systems were excited electrically.

2. Ia and Ib afferents of ankle and toe extensors were found to co-excite, co-inhibit or exert opposite synaptic actions in 41, 33 and 50 % of the analysed interneurones, respectively. Taking into account both excitatory and inhibitory input from these two groups of afferents, 64 % of the interneurones appeared to be used in common in reflex pathways from muscle spindles and tendon organs of ankle and toe extensors.

3. Selective input from Ib afferents of triceps surae and plantaris (excitation and/or inhibition) was found in 36 % of the interneurones; there was no evidence for a similarly selective input from Ia afferents.

4. A great majority (over 90 %) of the interneurones excited by group I afferents were also inhibited by group I afferents, from either the same or other muscles.

5. Both monosynaptic and disynaptic e.p.s.p.s from Ia and/or Ib afferents from other muscles and from fibres in the ipsilateral funiculi were found in a great proportion of the same interneurones, together with disynaptic e.p.s.p.s from low threshold cutaneous afferents.

6. Intracellular staining with horseradish peroxidase revealed four different patterns of axonal projections of the analysed interneurones: (i) projections to motor nuclei and the intermediate region, (ii and iii) projections only to the intermediate region, locally or combined with projections to different rostro-caudal levels, and (iv) projections to the opposite side of the spinal cord.

7. A large proportion of interneurones projecting to motor nuclei displayed input from both Ia and Ib afferents although such an input was a feature of interneurones with other projections as well. No systematic differences in the input from group I afferents were found for interneurones with different axonal projections. In contrast disynaptic e.p.s.p.s of cutaneous origin and monosynaptic e.p.s.p.s upon stimulation of ipsilateral spinal tracts appeared predominantly in interneurones projecting to motor nuclei.

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INTRODUCTION

Observations reported in the preceding paper (Czarkowska, Jankowska & Sybirska, 1981) show that some actions of group Ia muscle spindle afferents and group Ib tendon organ afferents may be mediated by the same spinal interneurones; these interneurones were excited by Ia and Ib afferents of knee flexors and extensors. The main aim of experiments described in this paper was to find out if Ia and Ib afferents of other muscles, in particular of ankle and toe extensors, similarly converge on laminae V–VI interneurones. Since ranges of thresholds of the two subgroups of group I afferents of these muscles to electrical stimuli greatly overlap (see Jack 1978), adequate stimulation of muscle spindles by brief muscle stretches (Lundberg & Winsbury, 1960; Stuart, Mosher, Gerlach & Reinking, 1970; Lucas & Willis, 1974; Ellaway & Trott, 1978; Fetz, Jankowska, Johannisson & Lipski 1979) was used to activate Ia afferents in isolation from Ib afferents. Effects of such stimulation were compared with effects of electrical stimulation of all group I afferents in the relevant nerves, the difference indicating Ib actions. The synaptic input from the two subgroups of group I afferents to the interneurones was correlated with input from other fibre systems and with their axonal projections as revealed by intracellular staining with horseradish peroxidase (Czarkowska, Jankowska & Sybirska, 1976).

The establishment of convergence, or of lack of convergence of group Ia and Ib afferents of ankle and toe extensors, on to interneurones with projections to motor nuclei was of particular interest for further analysis of the neuronal pathways of the autogenetic inhibition and other actions of group I afferents on motoneurones, to which both Ia and Ib afferents of triceps surae and plantaris have been found to contribute (Fetz *et al.* 1979; Jankowska, Mackel & McCrea, 1980). Theoretically such actions could be mediated either by two parallel and independently operating reflex pathways, or by a shared pathway involving common interneurones (cf. Lundberg 1975, 1979). Observations on convergence of Ia and Ib afferents of knee flexors and extensors could not be extrapolated *a priori* to the afferents of ankle and toe extensors but it will be shown that the latter also converge on laminae V–VI interneurones; the degree of their convergence appeared in fact to be even higher than that of Ia and Ib afferents of knee flexors and extensors. Some preliminary observations of this study have been published (Jankowska, Johannisson & Lipski, 1978).

METHODS

The experiments were done on fifteen cats under chloralose (50–70 mg/kg) or chloralose (40–50 mg/kg) and sodium pentobarbitone (5–10 mg/kg) anaesthesia. Some of these cats were also used for the analysis of effects of group I muscle afferents on motoneurones (Fetz *et al.* 1979) and the preparation was more fully described in that paper. The technique of recording from interneurones, their identification and staining with horseradish peroxidase were those reported by Czarkowska *et al.* (1976, 1981) except for three modifications: (i) we verified that the penetrated cells did not project rostrally to the lumbo-sacral enlargement, by checking that they were not antidromically invaded on stimulation of the ipsilateral and contralateral halves of the spinal cord, as well as the dorsal columns at L1–Th13 segmental level, since it appeared that long ascending tract cells with axons in the dorsal columns have monosynaptic input from group I afferents (Jankowska, Rastad & Zarzecki 1979) and should be differentiated from segmental interneurones; (ii) axonal projections of interneurones stained with horseradish peroxidase were reconstructed after treating the spinal

cord sections with either 3,3-diaminobenzidine, as described by Graham & Karnovsky (1966) and Kristensson & Olsson (1971), diaminobenzidine after pre-incubation in a solution of cobalt chloride (Adams, 1977), or a mixture of *p*-phenylenediamine dihydrochloride and pyrocatechol (Hanker, Yates, Metz & Rustioni 1977); (iii) horseradish peroxidase was injected into the cells electrophoretically from micropipettes filled with a 15% solution in 0·3 M-NaCl.

Stimulation. Muscle spindle afferents were activated by brief stretches of triceps surae and plantaris, as described by Fetz *et al.* (1979). The amplitudes of the stretches were graded and only those post-synaptic potentials which were evoked below lowest thresholds for Ib afferents were attributed to muscle spindle afferents. Under our experimental conditions (see Fig. 1*A* in this paper

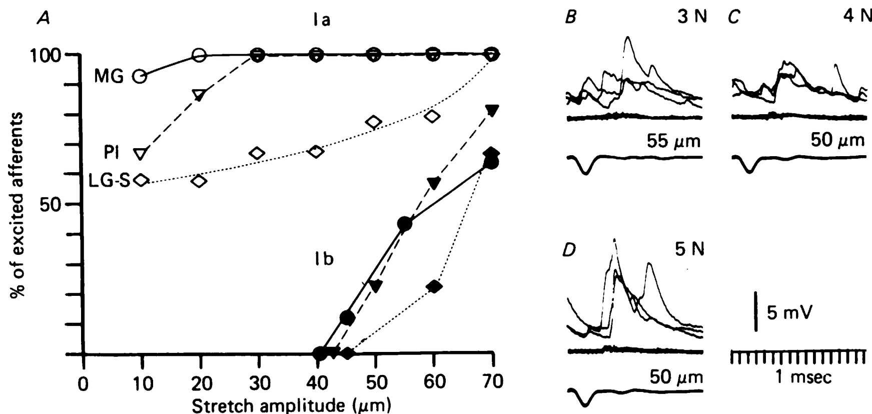

Fig. 1. Effectiveness of activation of Ia and Ib afferents from different muscles. *A*, percentage of Ia and Ib afferents from medial gastrocnemius (MG), plantaris (Pl) and lateral gastrocnemius-soleus (LG-S) excited by stretches of different amplitudes, the initial tension of the muscles set at 5 N. Data for the same sample of afferents which are illustrated in Fig. 1*A* and *B* of Fetz *et al.* (1979). *B–D*, records from a neurone monosynaptically excited by Ia and Ib afferents, illustrating effects of muscle stretch of 50–55 μm at various initial muscle tensions. Unless specified otherwise in this and in the following Figures, the upper records are intracellular records from an interneurone, the middle records are from the surface of the spinal cord near entry zone of L7 dorsal root, and the lower records show changes in muscle length (extension downward). The intracellular records were photographed together with surface records from one oscilloscope, and together with length records from another one.

and Fig. 1*A* and *B* in Fetz *et al.* 1979) these thresholds were at 45–50 μm, but the upper limit of the stretch was conservatively lowered to below 40 μm. We attributed to the tendon organs any differences between post-synaptic potentials which were evoked by electrical stimulation of the nerves with intensity maximal for group I (Ia+Ib) afferents and potentials which were evoked by muscle stretches maximal or nearly maximal for Ia afferents (60–70 μm (see Fig. 1*A* in this paper and Fig. 1*B–G* in Fetz *et al.* 1979).

In these experiments the initial tension of the muscles was 5 N, since this tension provides an optimal separation between thresholds of Ia and Ib afferents (see Fig. 1*A* and *B* in Fetz *et al.* 1979). As illustrated in Fig. 1*B–D* stretches suprathreshold for evoking Ia e.p.s.p.s in interneurones with initial tension of 5 N (*D*) were just at threshold with initial tension 3 N (*B*).

A comparison of thresholds for group Ia afferents of different muscles indicated that under our conditions muscle spindles of medial gastrocnemius and plantaris were more effectively activated than those of lateral gastrocnemius and soleus. Stretches of 30–40 μm at 5 N excited all the analysed Ia afferents of plantaris and medial gastrocnemius but only 60–70% of the Ia afferents of lateral gastrocnemius and soleus (Fig. 1*A*). Using the size of e.p.s.p.s evoked in motoneurones by stretches of these muscles as a measure of the proportion of the excited Ia afferents, the

effectiveness of the stretches appeared somewhat lower (Fetz *et al.* 1979); e.p.s.p.s evoked in plantaris and medial gastrocnemius motoneurones by stretches of their homonymous muscles attained maximal amplitudes and areas with 50–70 μm stretches, while 30–40 μm stretches evoked e.p.s.p.s of about 80% of maximal in medial gastrocnemius and plantaris as well as in lateral gastrocnemius motoneurones. In spite of the data in Fig. 1*A* we had to consider stretches of 30–40 μm as submaximal for some higher threshold group I a afferents and therefore could not differentiate between I a and I b origin of additional post-synaptic potentials evoked by larger stretches; the latter could have been due to the remaining I a as well as I b afferents. Fortunately for our analysis, when additional components of e.p.s.p.s were evoked by electrical stimulation of the nerves, the resulting e.p.s.p.s were much larger than those produced by both 30–40 μm and 50–70 μm stretches (see under Results). The presence of I b synaptic actions could therefore be established without doubt, although some errors in their quantitative estimations were unavoidable; they would be over-estimated when judged in relation to effects of 30–40 μm stretches and underestimated when compared with those of 50–70 μm stretches. The easiest to classify as I b effects were, of course, e.p.s.p.s evoked exclusively by electrical stimuli and for which even the largest stretches were subthreshold.

The differentiation between I a and I b origin of additional components of i.p.s.p.s evoked by electrical stimuli was more tentative. The differences between i.p.s.p.s evoked by 30–40 μm stretches and by larger stretches or electrical stimuli were much less pronounced than the differences between e.p.s.p.s, and in some cases additional contributions from I b afferents could have been missed. On the other hand, when the i.p.s.p.s appeared with thresholds $\geqslant$ 40 μm they were attributed to I b afferents, with a possible underestimation of the higher threshold I a effects.

Strech-evoked and electrically evoked p.s.p.s were compared at the same level of membrane, polarization, and with records taken in a close succession. The amplitudes of the e.p.s.p.s rapidly decreased with deterioration of the cells and depolarization of cell membrane, especially when the amplitudes of the i.p.s.p.s superimposed on the decay phase of the e.p.s.p.s increased (see Fig. 5).

The effectiveness of muscle stretches in activating I a afferents in individual experiments was verified by checking in a few motoneurones that monosynoptic e.p.s.p.s evoked by 50–70 μm stretches did not differ markedly from the electrically evoked ones; incoming afferent volleys by muscle stretches were also routinely monitored.

RESULTS

The input from triceps surae and plantaris muscle receptors was analysed for seventy interneurones recorded intracellularly. Twenty-four of them were subsequently stained with horseradish peroxidase. The interneurones showed seven patterns of post-synaptic potentials from group I afferent, as indicated in column 1 of Fig. 2 where white squares represent e.p.s.p.s, black squares represent i.p.s.p.s and left and right hand squares are for p.s.p.s evoked from I a and I b afferents, respectively.

Excitation by group I a and I b afferents

More than 40% of the penetrated interneurones were found to be *co-excited* by I a muscle spindle afferents and by I b tendon organ afferents (□□ in Fig. 2). Such co-excitation is illustrated in Fig. 3 with extracellular and intracellular records from one of the interneurones and also in Figs 4 and 5 with intracellular records from two other neurones. Fig. 3*A*, *B*, *D* and *E* show the effects of low and middle range threshold I a afferents (stretch amplitudes 10 and 22–27 μm). With a larger stretch (55 μm) the amplitude of the early e.p.s.p increased and later components appeared (*F*). Stimulation of lateral gastrocnemius-soleus nerve alone (*I*) evoked e.p.s.p.s larger than those evoked by 55 μm stretches of lateral and medial gastrocnemius, soleus and plantaris, which should have excited some I b afferents in addition to the great majority of I a afferents from these four muscles. With e.p.s.p.s evoked also from

Convergence from MG, LG-S, Pl				Convergence from other muscle nerves								
Patterns of input	No. of nerves: one	two	three	No. of nerves: one	two	three	four	Other muscle nerves: PBSt	Q	FDL	ABSm	DP
1 n	2	3	4	5	6	7	8	9	10	11	12	13
24*	1/3	9/4	14/7	8/8	7/	2/2	1/	(, 2, 2)/(, 1,)	(1, 3,)/(1, 2,)	13/4	4/1	2/4
4	—	—	4/	3/1	—	—	—	(, ,)/(, ,)	(, ,)/(, 1,)	3/	—	—
4	—	—	4/4	1/	1/1	—	—	(, ,)/(, ,)	(, 1,)/(, 1,)	1/1	/1	—
14	/3	3/2	11/9	6/3	2/2	2/	—	(1, 1, 1)/(, 1,)	(, 4,)/(1, ,)	7/5	3/	—
7	1/1	3/1	3/5	4/1	1/	/1	1/2	(1, 1,)/(2, ,)	(, 1,)/(1, 2,)	6/3	1/2	1/1
10	4/	3/2	3/8	4/3	1/2	1/2	—	(1, ,)/(1, 2,)	(1, 1,)/(1, 1,)	5/4	2/2	/2
6	—	/1	/5	3/3	—	1/3	—	(, ,)/(1, 2,)	(2, ,)/(1, 2,)	/2	1/4	3/
Total 70	6/7	18/9	39/38	29/19	12/5	6/8	2/2	(3, 4, 3)/(4, 6,)	(4, 10,)/(5, 9,)	35/19	8/10	3/7
Previous 96	14/10	12/16	40/45	39/30	21/16	5/9	3/3	(12, 4, 9)/(8, 4, 1)	(12, 5, 12)/(8, 14, 2)	43/41	18/7	14/16

Fig. 2. Convergence on laminae V–VI interneurones with input from group I afferents from triceps surae and plantaris. Data for seventy interneurones analysed in this study and for ninety-six interneurones previously analysed by Hongo *et al.* (1966, 1972) and by Czarkowska *et al.* (1976). Column 1: patterns of post-synaptic actions of Ia and Ib afferents. □, excitation; ■, inhibition; left and right, Ia and Ib actions, respectively. Columns 2–4: origin of the e.p.s.p.s (above) and of i.p.s.p.s (below) from one, two or all three of the stimulated nerves of medial gastrocnemius (MG), lateral gastrocnemius-soleus (LG-S) and plantaris (Pl). Columns 5–13: origin of additional e.p.s.p.s and i.p.s.p.s evoked from posterior biceps-semitendinosus (PBSt), quadriceps (Q), flexor digitorum longus (FDL), anterior biceps-semimembranosus (ABSm) and pretibial flexors (DP). Number of nerves and muscles of origin, as indicated. In columns 9 and 10, group Ia, unspecified group I and group Ib origin of p.s.p.s is indicated to the left, in the middle and to the right, respectively. Synaptic actions from other than triceps surae and plantaris nerves were tested in about 70% of the neurones; so these numbers represent and underestimate of the input from other nerves. The Figure includes monosynaptic and disynaptic e.p.s.p.s and disynaptic and trisynaptic i.p.s.p.s. *In some neurones occurrence of Ia i.p.s.p.s was uncertain; they were not counted, but their existence could not be excluded.

plantaris (*G*) and medial gastrocnemius (*H*) the total group I excitatory actions from these three nerves clearly exceeded the effects of the largest muscle stretches. According to our criteria this neurone is therefore classified as excited by both Ia and Ib afferents.

Differences between electrically and stretch evoked e.p.s.p.s were much more difficult to quantify in records from interneurones than from motoneurones. Measurements of amplitude or areas of the e.p.s.p.s would not be very reliable since they were often made up of a few large unitary e.p.s.p.s which might not show linear summation (Burke, 1967). The limited time for recording from individual interneurones and a requirement of comparison between records in as close succession

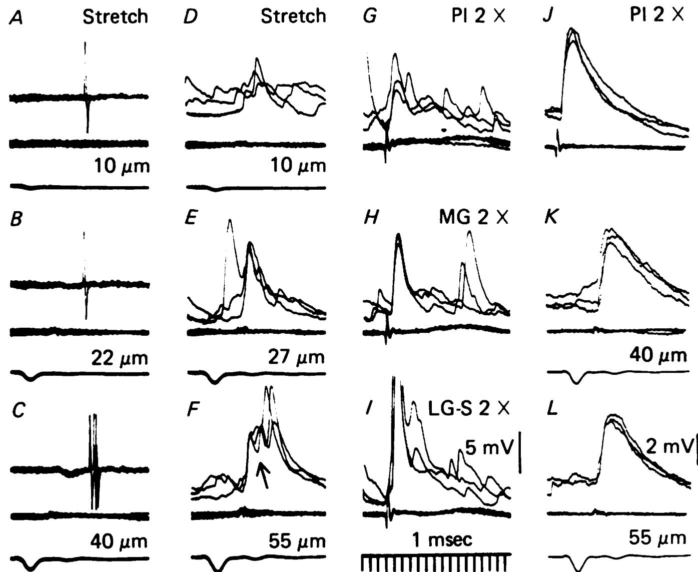

Fig. 3. Comparison of effects of different amplitudes of stretch and of electrical stimuli. *A–I*, records from the same interneurone; *A–C* extracellular, *D–I* intracellular. *A–F*, responses evoked by simultaneous stretch of triceps surae and plantaris. *G–I*, responses to electrical stimulation of the nerves to these muscles, maximal for group I afferents. Note short (< 1 msec) segmental latencies of e.p.s.p.s evoked by electrical stimuli as well as of e.p.s.p.s evoked by muscle stretches. The arrow in *F* indicates a later component most likely due to excitation of Ib afferents. (The neurone was disynaptically excited also by group I afferents from three other muscle nerves. Depolarization revealed weak inhibition from high threshold group I afferents in LG-S and MG.) *J–L*, control records of e.p.s.p.s evoked in a plantaris motoneurone in the same preparation; *J*, by electrical stimulation of Pl nerve with the intensity maximal for group I afferents; *K* and *L*, by stretches of MG, LG-S and Pl muscles. (Stimulation of MG and LG-S nerves did not evoke any e.p.s.p.s in this neurone.) Note that in spite of a more synchronized afferent volley, the amplitude of e.p.s.p.s evoked by electrical stimulation of the nerve was only about 20% higher than that of the e.p.s.p.s evoked by both 40 and 55 μm stretches. The areas of e.p.s.p.s evoked electrically and by 40 μm stretches were almost identical. The decaying slopes of e.p.s.p.s in *J* and *L* suggest the presence of small group I autogenetic i.p.s.p.s.

as possible prohibited also a routine use of averaging. For these reasons the conclusions on larger effects of electrical stimulation were based mainly on records showing that amplitudes of e.p.s.p.s evoked by muscle stretches and by stimulation of one of the nerves were approximately the same, or the latter larger, and that additional monosynaptic e.p.s.p.s were evoked from other nerves. Electrical stimulation of the nerves resulted in a more synochroneous arrival of the incoming volleys than did muscle stretch, but this higher synchronization was not a major factor in influencing the size of the electrically evoked e.p.s.p.s as indicated by control records from motoneurones. When e.p.s.p.s evoked in motoneurones by muscle stretches and by electrical stimuli had similar areas the amplitudes of these e.p.s.p.s showed only small differences, as illustrated in Fig. 3 *J–L*.

Figs 4 and 5 show that a comparison between the electrically and stretch evoked e.p.s.p.s was also complicated by i.p.s.p.s following the e.p.s.p.s. In several cells a clearly larger effect of electrical stimuli was seen in the first series of records taken soon after penetration (compare Fig. 4*C* and *D* and Fig. 5*C* and *I*). After a depolarization of the neurones and an increase in i.p.s.p.s evoked by electrical stimuli these differences diminished considerably (compare Fig. 4*C* and *F* and Fig. 5*C*, *E*,

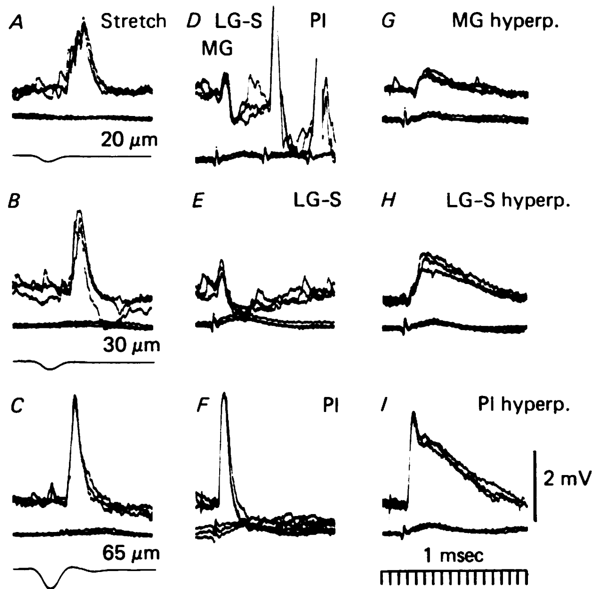

Fig. 4. Co-excitation by Ia and Ib afferents and co-inhibition by Ia and Ib afferents. Reversal of i.p.s.p.s by chloride injection. *A–D*, records taken just after penetration of the neurone, showing excitation (*A*) and excitation cut short by inhibition (*B*) evoked by Ia afferents. Stronger excitation and probably also inhibition was evoked by electrical stimuli (*D*) than by stretch of 65 μm (*C*). *E* and *F*, records taken after deterioration and depolarization of the neurone; note smaller e.p.s.p.s and pronounced i.p.s.p.s. *G–I*, records taken during hyperpolarization of the neurone and diffusion of chloride; note reversal of the i.p.s.p.s, allowing identification of the repolarization which attenuated the e.p.s.p.s in *D–F* as i.p.s.p.s. In addition the neurone was excited by group I afferents of flexor digitorum longus (not illustrated).

F and *H*). The records in severely damaged or intentionally depolarized neurones (e.g. during injection of horseradish peroxidase) could thus not be used to conclude that there were no additional e.p.s.p.s from Ib afferents, if e.p.s.p.s evoked then by electrical stimuli resembled those earlier evoked by muscle stretches. With the exception of such records, e.p.s.p.s evoked by electrical stimulation of the nerves were always much larger than e.p.s.p.s evoked by both 30–40 and 50–60 μm stretches. Consequently we have no indications of a selective excitatory input from muscle spindle afferents in our sample of interneurones. On the other hand, e.p.s.p.s apparently evoked only from Ib afferents were found in thirty-five (50%) of the interneurones. In these cases the threshold stretches were > 45 μm, i.e. above the threshold for Ib afferents, and in several cells e.p.s.p.s appeared only with nerve

stimulation (Fig. 7*E*, *F*). These e.p.s.p.s were attributed to tendon organ afferents, since there seemed to be no reason to assume a specific effect of the highest threshold I a afferents, but a contribution of the latter could not be fully excluded.

The effects of I a afferents from different muscles were not compared in view of the technical difficulty of selectively stretching single muscles without affecting the remaining ones. The muscle origin could only be established for I b e.p.s.p.s by

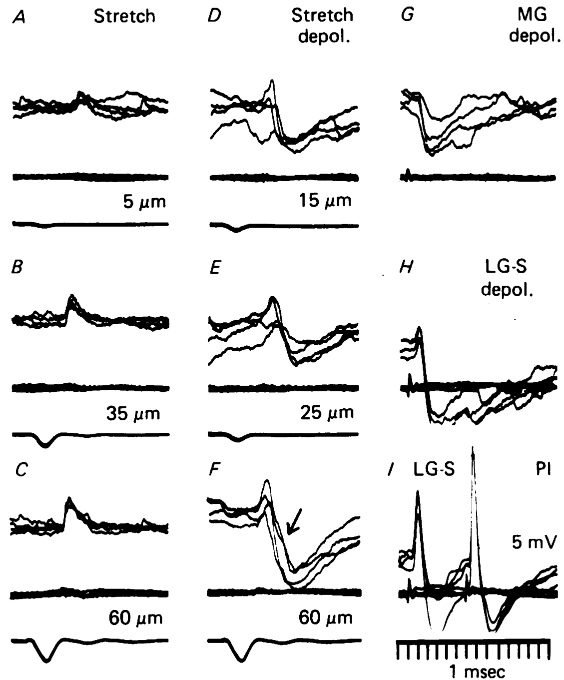

Fig. 5. Co-excitation by I a and I b afferents and co-inhibition by I a and I b afferents. Disclosure of i.p.s.p.s by depolarization. Co-excitation of the neurones by I a and I b afferents appears from records *A–C* and *I*. Depolarization disclosed i.p.s.p.s evoked from I a afferents (*D* and *E*) with some addition from I b afferents (cf. *E*, *F* and *H*; note longer duration of the i.p.s.p.s in *H*). No polarizing current in *A–C* and *I*. Depolarisation in *D–H* (9 nA). In addition this neurone was monosynaptically excited from group I afferents of flexor digitorum longus and disynaptically inhibited from group I afferents of the same muscle, as well as of quadriceps, posterior biceps-semitendinosus and anterior biceps-semimembranosus (not illustrated).

comparing effects of electrical stimulation of the individual nerves. As shown in Fig. 2, columns 2–4, out of thirty-four neurones with e.p.s.p.s from I b but not I a afferents, seventeen were excited from all three nerves, five were excited from only one nerve, and twelve from two nerves; the latter are illustrated in Fig. 7*E* and *F*.

In addition to the input from triceps surae and plantaris, the interneurones received monosynaptic or disynaptic e.p.s.p.s from group I afferents from one to four of the five other nerves regularly tested (Fig. 2, columns 5–8). Of these flexor digitorum longus contributed the most and anterior biceps-semimembranosus and pretibial flexors the least (Fig. 2, columns 9–13); e.p.s.p.s from tibial nerve, which

were evoked in a large number of neurones (monosynaptic in twenty-four neurones and disynaptic in eighteen neurones) were not included in this comparison because group I afferents of this nerve could not be stimulated separately from cutaneous afferents. The I a or I b origin of some of the additional e.p.s.p.s from posterior biceps-semitendinosus and quadriceps could be established when the two components of the incoming volleys were well separated (Bradley & Eccles, 1953; Eccles, Eccles & Lundberg 1957*a*). As in the case of e.p.s.p.s from triceps surae and plantaris they were evoked from both I a (cf. Fig. 8 *J–L*) and I b afferents.

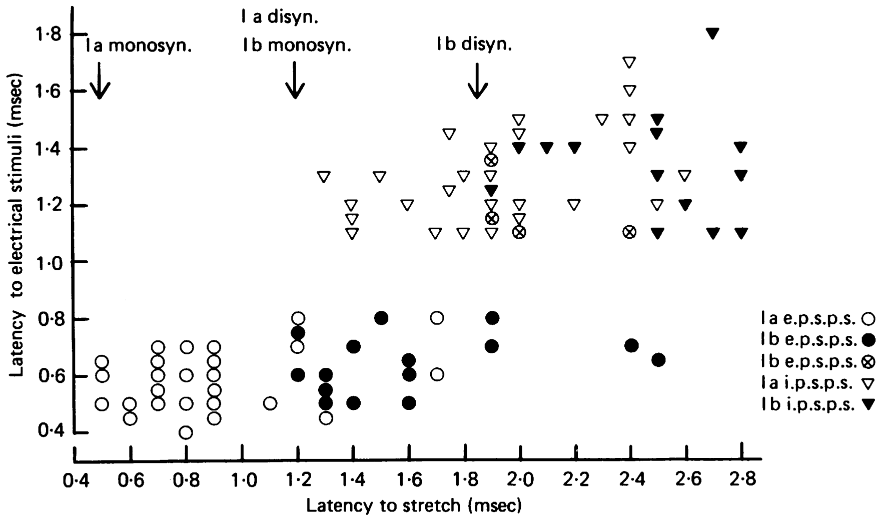

Fig. 6. Latencies of stretch-evoked post-synaptic potentials. Latencies of e.p.s.p.s and of i.p.s.p.s evoked by muscle stretches, measured in relation to onset of afferent volleys recorded from the cord dorsum near dorsal root entry zone, plotted against latencies of the corresponding p.s.p.s evoked by electrical stimulation of the nerves. ○ and: e.p.s.p.s and i.p.s.p.s evoked by stretches ⩽ 30 mm. ●, ⊗ and ▼: e.p.s.p.s and i.p.s.p.s evoked by stretches > 40 mm when smaller stretches were ineffective. Arrows indicate shortest latencies for mono- and disynaptic p.s.p.s evoked by muscle stretches. Further explanations in the text.

Segmental latencies of e.p.s.p.s evoked by stretches ⩽ 35 μm and by stretches ⩾ 45 μm (when 35 μm stretches were ineffective) appeared to distribute within different ranges, supporting the conclusion that they were evoked by I a and I b afferents, respectively.

Most of the latencies of e.p.s.p.s evoked by stretches ⩽ 30 μm (I a e.p.s.p.s, open circles in Fig. 6) fell within the range usually accepted for the monosynaptic responses (0·5–0·9 msec in relation to incoming volleys). The five or six longer ones might represent I a e.p.s.p.s evoked disynaptically, if the earliest (monosynaptic) components of e.p.s.p.s evoked by electrical stimulation of the nerves in the same interneurones were due to impulses set up in I b afferents. Alternatively these stretch–evoked e.p.s.p.s with longer latencies might have been evoked by later components of asynchronously arriving volleys, assuming that only a fraction of I a afferents would terminate on the relevant interneurones, in this case the slower conducting and/or

a higher threshold fraction. On the other hand, only the monosynaptic coupling would be possible in the case of e.p.s.p.s with threshold stretches $\geqslant 45\ \mu m$ and attributed to I b afferents (filled circles in Fig. 6); these were evoked in neurones monosynaptically excited following electrical stimulation of the nerves and lacking I a e.p.s.p.s. Longer latencies of these e.p.s.p.s fell within the same ranges as the latencies of later components of e.p.s.p.s, similarly evoked by stretches $\geqslant 45\ \mu m$ but superimposed on lower threshold and shorter latency I a e.p.s.p.s (Fig. 3 *F*, arrow). They are in keeping with observations of Stuart *et al.* (1970) that time of initiation of impulses by muscle stretches is longer for I b than for I a afferents. The differences reported by them were between 0·4 and 3·7 msec, depending on amplitude of stretch.

Whether, and how many of the interneurones monosynaptically excited by group I a or I b afferents also received disynaptic input from these afferents could not be established. Disynaptic coupling (Fig. 6, ⊗) was found in only four interneurones excited by stretches $> 45\ \mu m$ in which e.p.s.p.s evoked by electrical stimulation of the nerves appeared with latencies 1·1–1·4 msec, excluding a monosynaptic coupling. Because of their relatively high thresholds these e.p.s.p.s were classified as evoked from I b afferents, although co-excitation by I a and I b afferents of the first order interneurones which mediated them remained a possibility.

A monosynaptic coupling was found in the case of e.p.s.p.s evoked from all the tested nerves: lateral gastrocnemius soleus (85 %), plantaris (84 %), medial gastrocnemius (81 %), flexor digitorium longus (75 %), posterior biceps semitendinosus (75 %), deep peroneus (60 %), quadriceps (56 %) and anterior biceps semimembranosus (40 %), the figures in brackets giving proportions of e.p.s.p.s with latencies < 1 msec.

Inhibition by group I a and I b afferents

Of seventy interneurones analysed, sixty-six (94 %) were inhibited by group I afferents. The inhibition appeared either as an i.p.s.p. following an e.p.s.p., cutting short its decay phase (Fig. 4 *B–F*, Fig. 5 *D–F*), or without any preceding e.p.s.p. (Fig. 7 *A–C* and *G–I*, Fig. 8 *A–C*). In order to facilitate the disclosure of the i.p.s.p.s, the penetrated neurones were routinely depolarized (5–10 nA) to increase the amplitudes of the i.p.s.p.s (compare Fig. 5 *A–C* and *D–F*, Fig. 8 *N*, *O*). Reversal of i.p.s.p.s after diffusion of chloride ions from the micro-electrodes (compare Fig. 4 *D–F* and *G–I*) also helped in identifying the unusually rapid decays of the e.p.s.p.s as due to i.p.s.p.s.

As in the case of the e.p.s.p.s, a considerable proportion of the interneurones (33 % of the inhibited ones) appeared to be *co-inhibited* by group I a muscle spindle and group I b tendon organ afferents (Fig. 2, column 1 ■). However, the additional i.p.s.p.s attributed to I b afferents were usually relatively moderate. In the neurones of Figs. 5 and 7 the addition of I b effects is indicated by the appearance of later components of the i.p.s.p.s (arrows) with larger stretches and by larger amplitudes of i.p.s.p.s evoked by electrical stimulation of the nerves. Assuming that these i.p.s.p.s were mediated by interneurones co-excited by I a and I b afferents (like those described above), and that some of them were fired by I a afferents alone, one should not, however, expect a more conspicuous contribution of the I b afferents. A non-linear summation of i.p.s.p.s would also lead to an underestimation of those evoked by higher threshold afferents.

In four interneurones the i.p.s.p.s were apparently evoked only from I a afferents

(Fig. 8) as indicated by similar amplitudes of i.p.s.p.s evoked by a moderate and a large stretch on the one hand and by electrical stimulation of the nerves on the other. Thirty-nine interneurones showed no indications of Ia origin of the i.p.s.p.s, consequently attributed to Ib afferents (Fig. 2, column 1). The interneurones mediating these i.p.s.p.s might, nevertheless, have been co-excited by both Ia and Ib afferents.

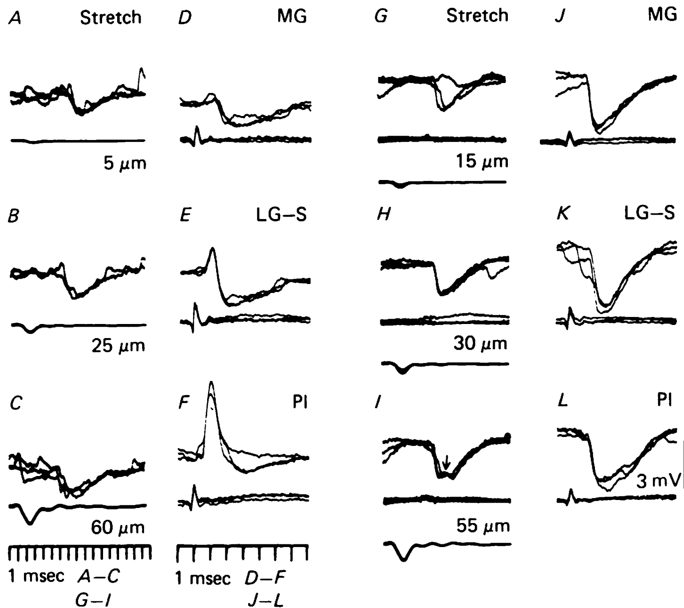

Fig. 7. Co-inhibition by Ia and Ib afferents, with or without excitation by Ib afferents from ankle extensors. Records from two interneurones in *A–F* and *G–L*, respectively. Note later components (arrow) of the i.p.s.p.s with increase of the amplitude of the stretch from 30 to 55 μm (*I*) and larger i.p.s.p.s evoked by electrical stimuli (*D*, *E* and *J–L*). The first neurone was in addition excited by group Ia afferents of flexor digitorum longus (monosynaptically) and of quadriceps, posterior biceps-semitendinosus and anterior biceps semimembranosus (disynaptically). The second neurone was excited by group I afferents of quadriceps and pretibial flexors (monosynaptically) as well as by those in flexor digitorum longus (disynaptically). It was also inhibited via Ia afferents of quadriceps and posterior biceps-semitendinosus and by group I afferents in anterior biceps-semimembranosus. Depolarization in *A–F* (6 nA). No polarizing current in *G–L*.

In more than half of the interneurones the i.p.s.p.s were evoked from the stretched muscles (Fig. 2, columns 2–4) as well as from one to four others (Fig. 2, columns 5–8), most often from flexor digitorum longus (cf. columns 9–13). Of the i.p.s.p.s evoked by stimulation of an identified subgroup of group I afferents in posterior biceps-semitendinosus or quadriceps, all originated from Ia afferents, with the examples in Fig. 8*G–I*.

The shortest segmental latencies of i.p.s.p.s evoked from Ia afferents (open triangles in Fig. 6) were 0·8 msec longer than the shortest latencies of the monosynaptic e.p.s.p.s from the same afferents. For the i.p.s.p.s evoked from Ib afferents (filled triangles in Fig. 6) the difference was 0·7 msec. The latencies of the e.p.s.p.s evoked

from the two subgroups of group I afferents overlapped more than the latencies of the e.p.s.p.s, and were distributed over a wider range. Whether Ia i.p.s.p.s with longer latencies to both stretch and electrical stimuli were evoked di- or trisynaptically is impossible to determine merely on the basis of the distribution of these latencies. On the other hand, most of the Ib i.p.s.p.s could be safely classified as disynaptic taking into account the differences between latencies of monosynaptic e.p.s.p.s evoked from Ia and Ib afferents. It will be noted that the latencies of four disynaptic Ib e.p.s.p.s (Fig. 6, ⊗) fell within the same ranges.

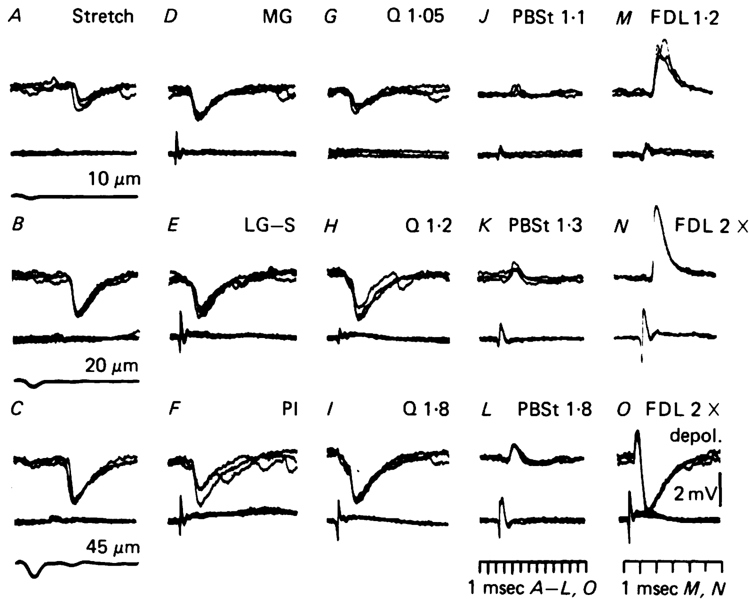

Fig. 8. Inhibition from Ia afferents of ankle extensors combined with Ia actions from other muscles. Records from an interneurone in which i.p.s.p.s of about the same amplitude were evoked by 20 and 45 μm stretches (*B*, *C*) and by electrical stimulation of medial gastrocnemius, lateral gastrocnemius, soleus and plantaris nerves (*D–F*). In addition the neurone was inhibited via low threshold afferents (probably only Ia) of quadriceps (*G–I*) and by group I afferents of flexor digitorum longus (*O*). It was monosynaptically excited by Ia afferents of posterior biceps-semitendinosus (*J–L*) and group I afferents of flexor digitorum longus (*M–O*) and pretibial flexors.

Opposite effects from group Ia and Ib afferents

One of the most striking features of the interneurones investigated was that their excitation by group I afferents was very strongly coupled with inhibition. A small proportion of the neurones showed only excitation, or only inhibition (Fig. 2, column 1). Taking into account p.s.p.s evoked from other nerves, there were in fact only five neurones with an exclusively excitatory or inhibitory group I input from all the nerves tested. Excitation and inhibition were evoked by the same or from different subgroups of group I afferents (cf. Fig. 13*D* and *E*), and from the same or by different nerves. E.p.s.p.s and i.p.s.p.s evoked on activation of *Ia afferents* by muscle stretch are illustrated in Fig. 4*A* and *B* and in Fig. 5*D–E*. The appearance of both e.p.s.p.s

and i.p.s.p.s on activation of *Ib afferents* was easiest to establish in those neurones in which stretches did not evoke any synaptic effects and in which e.p.s.p.s evoked by electrical stimuli were followed by the i.p.s.p.s, similarly as in neurones illustrated in Figs 3–5. *Ia i.p.s.p.s combined with Ib e.p.s.p.s* were similarly easy to detect, especially when e.p.s.p.s appeared exclusively on nerve stimulation (Fig. 7*E* and *F*).

Group I input patterns	Axonal projections to: motor nucl. (1)	interm. zone (3)	interm. zone loc. (5)	crossed (6)	Cutan. disyn. e.p.s.p.s.	Descending i. mono. e.p.s.p.	Descending i., co, dis. i.p.s.p.
	3	3		3	10/20	9/20	2/20
				1	3/3	1/3	0/3
		1	1		0/3	0/3	2/3
	3	1			10/12	6/12	1/12
	2	1	1	1	6/7	4/7	2/7
	2				4/7	4/6	1/6
	1				2/5	3/5	1/5
cutan. dis. e.p.s.p.s.	11/11	2/6	1/2	3/5	e.p.s.p.s.. Ia Ib i.p.s.p.s.		
i. desc. mono. e.p.s.p.s.	11/11	1/6	0/2	3/5			

Fig. 9. Correlation between axonal projections and input to laminae V–VI interneurones. Patterns of group I input (column 1) as in Fig. 2, according to the key in the right lower corner. Columns 2–5 show numbers of neurones with projections to ipsilateral motor nuclei, intermediate zone, intermediate zone locally and with crossed projections; these correspond to 1st, 3rd, 5th and 6th patterns of axonal projection of Czarkowska *et al.* (1976). The input from cutaneous afferents and from long spinal tracts indicated below is for the stained neurones. The three columns to the right give proportions of all analysed neurones with disynaptic e.p.s.p.s from one of the cutaneous nerves, monosynaptic e.p.s.p.s from long ipsilateral spinal tracts and disynaptic i.p.s.p.s from either ipsilateral or contralateral spinal tracts stimulated at Th13. Further explanations in the text.

Input from other than group I fibre systems

It has been established previously by indirect methods (see Lundberg, 1975) that interneurones which mediate the excitation and inhibition of motoneurones from tendon organ afferents are di- or trisynaptically excited by cutaneous afferents (Lundberg, Malmgren & Schomburg, 1977), oligosynaptically from joint afferents (Lundberg, Malmgren & Schomburg 1978), monosynaptically from the red nucleus Hongo, Jankowska & Lundberg, 1969) and mono- or oligosynaptically from the pyramidal tract (Lundberg & Voorhoeve, 1962; Illert, Lundberg & Tanaka, 1976).

Since disynaptic excitation from cutaneous afferents and monosynaptic excitation from ipsilateral spinal tracts might be a distinguishing feature of those interneurones which terminate on motoneurones, we tried to correlate their occurrence with either the group I input or the axonal projections (see below) of individual interneurones, E.p.s.p.s from cutaneous afferents (in superficial peroneal or sural nerves or both) were found in the majority (61 %) of the neurones. The segmental latencies of the e.p.s.p.s varied between 1·0 and 1·8 msec indicating a disynaptic coupling, although the longest ones might be compatible with a trisynaptic coupling as well.

Only in one neurone the e.p.s.p.s from a cutaneous nerve were evoked with a shorter (0·7 msec) latency. This neurone was unfortunately lost before it was tested for antidromic invasion from the dorsal columns. Thus we cannot exclude the possibility that it was wrongly classified as an interneurone, and that it was a tract neurone projecting to the dorsal column nuclei (cf. Jankowska *et al.* 1979).

As shown in Fig. 9, the disynaptic e.p.s.p.s of cutaneous origin appeared in neurones with various patterns of convergence from group I afferents. Similarly monosynaptic e.p.s.p.s from long spinal tracts were evoked in neurones with various inputs, but mainly in those which received disynaptic e.p.s.p.s from cutaneous afferents. In three interneurones e.p.s.p.s from the ipsilateral funiculi appeared with somewhat longer latencies (1·1–1·2 msec.). Mono- or disynaptic e.p.s.p.s with the same latencies were evoked also from the contralateral funiculi and disynaptic i.p.s.p.s from either the ipsilateral or the contralateral funiculi.

None of the laminae V–VI interneurones excited by I a afferents showed any signs of the recurrent inhibition, in agreement with previous observations (Hultborn, Jankowska & Lindström, 1971, Lucas & Willis, 1974); the effects of impulses in ventral roots were tested in all the seventy interneurones recorded intracellularly, as well as in a few interneurones recorded extracellularly. Neither have we found any depression of stretch-evoked i.p.s.p.s in laminae V–VI interneurones following conditioning stimulation of the ventral roots. Together with observations of S. Lindström (personal communication) and Czarkowska *et al.* (1981) on i.p.s.p.s evoked by near-threshold electrical stimuli, these observations indicate that I a inhibition of laminae V–VI interneurones is mediated by inhibitory interneurones other than those which mediate reciprocal inhibition of motoneurones.

Correlation between the input and axonal projections of the interneurones

Thirty-one of the interneurones described above were stained with intracellularly injected horseradish peroxidase and in twenty four it was possible to reconstruct projections of both their stem axons and of at least some initial axon collaterals. These fell into four (1st, 3rd, 5th and 6th) of the six patterns of axonal projections previously described for group I interneurones in lamina V and VI (Czarkowska *et al.* 1976, 1981), as summarized in Fig. 9.

No correlation has been found between patterns of group I synaptic actions and patterns of axonal projections. On the other hand, disynaptic excitation from cutaneous afferents and monosynaptic excitation from fibres in ipsilateral spinal fascicles appeared to be more characteristic for interneurones projecting to the motor nuclei than for their other subgroups (Fig. 9, bottom). All but one neurone of the first type sent the stem axon to the lateral funiculus. The axon of the exceptional neurone bifurcated close to the soma, sending one branch to the lateral and another to the ventral funiculus (Fig. 10). Axons of four neurones bifurcated into an ascending and a descending branch, while only an ascending or only a descending projection

was found for five and two interneurones, respectively. Initial collaterals of the interneurones of the first type branched both in the intermediate zone and within the dorsally located motor nuclei (Fig. 10). Axon collaterals of different axonal branches often projected towards the same cell groups and terminated within the same regions. This is shown in Fig. 11*A* for axon collaterals of one of the neurones. Those given off by the laterally directed secondary axonal branch are indicated by

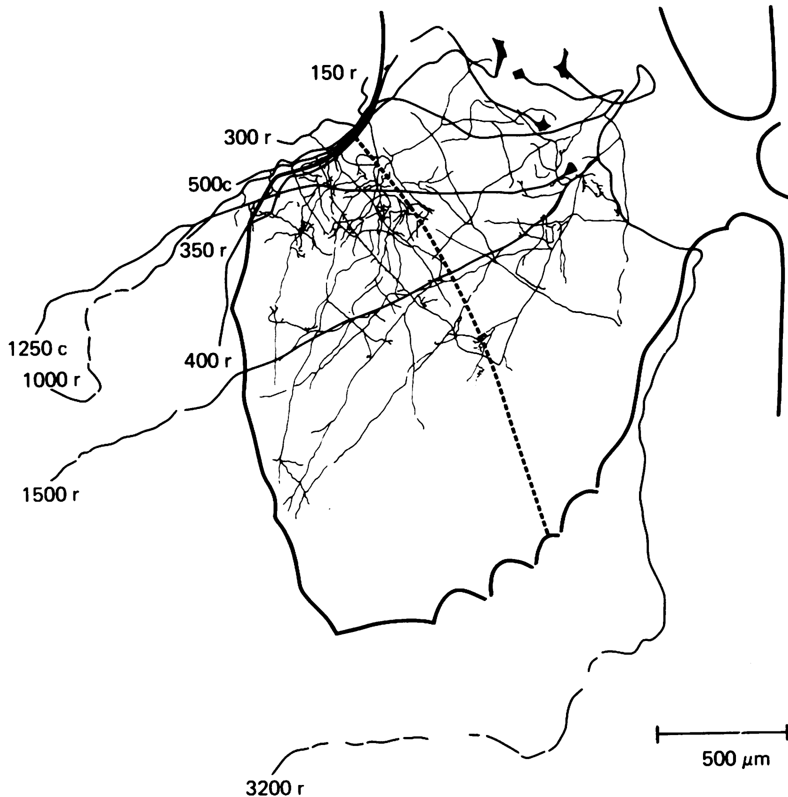

Fig. 10. Reconstruction of axonal projections of five interneurones with terminal branching in motor nuclei in rostral L7. Somata with truncated dendrites are in black, stem axons are in thick continuous lines and their collaterals in thin lines. The dashed line indicates the border between laminae VII and IX. Note densest branching within the dorsally located nuclei. Numbers at the ends of the axons in the white matter indicate their distance from soma and ascending (r, rostral) or descending (c, caudal) direction. The illustrated neurones were not influenced by group I afferents from knee flexors or extensors.

continuous lines while those of the medio-ventrally directed branch are drawn as dashed lines. Fig. 11 shows that different terminal axonal branches would actually make synaptic contacts within the same areas of the ventral horn. The terminal swellings of the terminal branches in Fig. 11*B* were found in the neighbourhood of twelve large cells in the motor nuclei, within 30 μm of their somata. Most of these cells, if not all, would be α-motoneurones. This figure illustrates our strongest evidence to date that interneurones of the first type may be the last order interneurones of reflex pathways from group I afferents to motoneurones.

Correlation between the input and location of the interneurones

Interneurones analysed in this study were located within spinal segments L5–S1, most being found in L6–L7. Usually a given part of the spinal cord was explored only until an interneurone was successfully penetrated and injected with horseradish peroxidase. Thereafter attempts to penetrate a new neurone were made 0·8–1·5 mm more rostrally or caudally. In this way in each experiment records were taken from interneurones distributed over about 15 mm of the spinal cord.

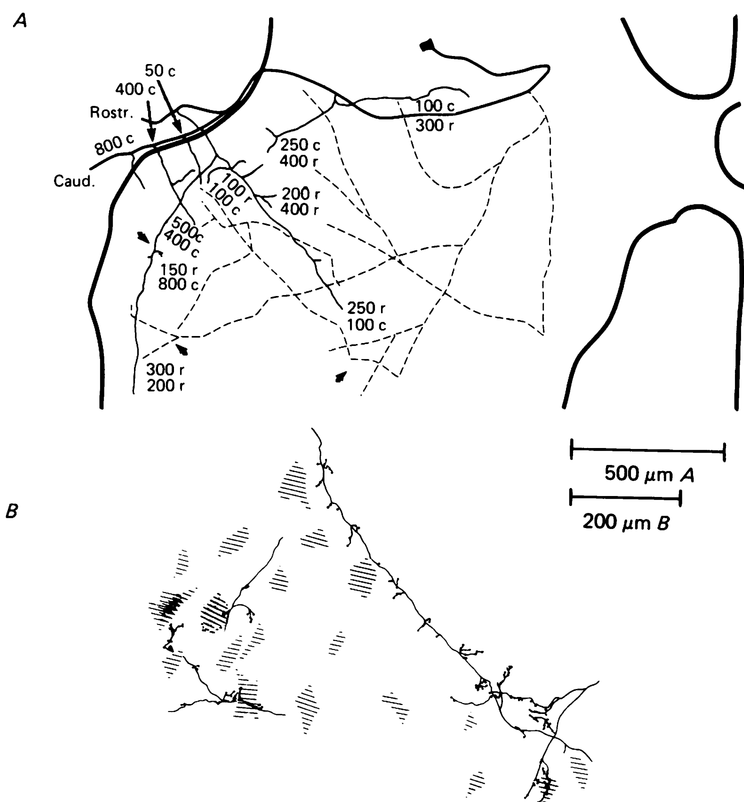

Fig. 11. Reconstruction of axonal projections of an interneurone included in Fig. 10. *A*, semi-schematic reconstruction of main axonal branches illustrating common target of various axon collaterals. Axon collaterals of laterally and ventrally directed secondary axonal branches are drawn in continuous and dashed lines, respectively. Numbers at the ends of the collaterals indicate their distance in μm from the soma, caudally (c) and rostrally (r). *B*, a detailed reconstruction of segments of axon collaterals indicated by arrows in *A*. Dots indicate terminal swellings. Hatched are somata of large neurones (most likely motoneurones) in the same sections.

In two preliminary experiments we compared the distribution of field potentials evoked in the intermediate zone by muscle stretches subthreshold for Ib afferents and by electrical stimuli maximal for group I afferents. The largest stretch-evoked Ia field potentials were found within the areas of largest electrically evoked responses. Therefore there was no indication of a separate location of neurones with Ia and Ib

input, either transversely or longitudinally. Stimulation of group I afferents from either medial gastrocnemius, lateral gastrocnemius-soleus or plantaris, resulted in field potentials whose areas overlapped to a large extent. There was thus neither evidence for a distinct location of interneurones with input from these muscles, although clusters of a few neurones with a predominant input from one or two of them were occasionally seen. Field potentials evoked by electrical stimulation of group I

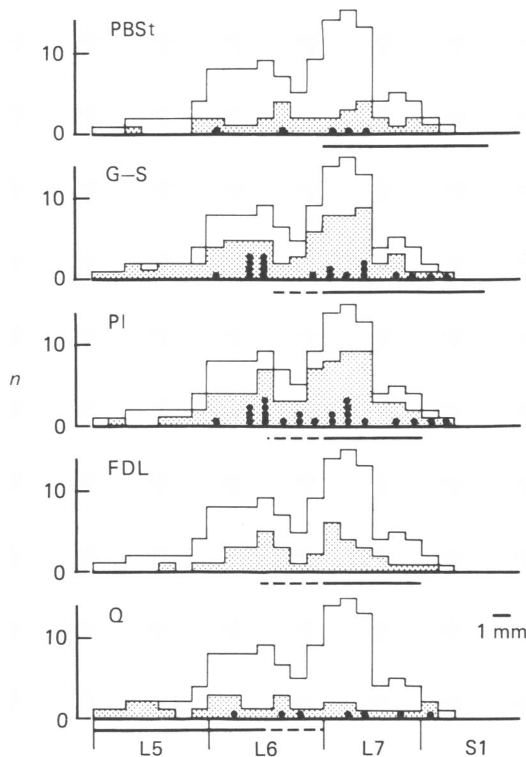

Fig. 12. Location of interneurones with monosynaptic e.p.s.p.s from group I afferents of different nerves. The unshaded histograms give the location of the entire sample of interneurones examined both in this study and by Czarkowska *et al.* (1981). The average length of L5 and L6 segments was 7 mm and that of L7 6 mm. The distances of the electrode tracts from the borders between the segments were measured during the experiments; the location of the interneurones is indicated for 1 mm distances. The stippled histograms are for neurones with input from a given nerve; e.p.s.p.s evoked selectively by Ib afferents are indicated by black circles. Continuous lines under the histograms show the usual extent of the corresponding motor nuclei. Dashed lines show their extensions in extreme cases of pre-and post-fixed cats.

afferents from triceps surae and plantaris were recorded over the whole explored length of the spinal cord, from middle L5 to middle S1; their amplitudes were maximal in rostral L7 or in caudal L6, depending on the cat.

The distribution of interneurones with monosynaptic e.p.s.p.s from triceps surae, plantaris, posterior biceps-semitendinosus, flexor digitorum longus and quadriceps is shown in Fig. 12 (stippled area). It will be noted that these interneurones were

encountered over the whole length of the spinal cord, consistent with the distribution of the field potentials in the intermediate zone, but different from the longitudinal extent of the motor nuclei. The latter are indicated by thick continuous and dashed lines below the corresponding histograms; the dashed lines indicate the extent of the motor nuclei in pre-fixed and post-fixed spinal cords (cf. Romanes, 1951). There were no indications for separate location of interneurones with a selective input from I b afferents (●).

DISCUSSION

To what extent are laminae V–VI interneurones shared by reflex pathways from muscle spindle and tendon organ afferents of ankle extensors?

Adequate stimulation of muscle spindles of triceps surae and plantaris revealed that impulses in group I a afferents of these muscles evoke widespread excitatory and inhibitory effects in interneurones of Rexed's laminae V and VI. Thus our results confirm previous conclusions on the I a input to these interneurones based on effects of graded electrical stimulation of the nerves (Eccles, Eccles & Lundberg, 1960, Hongo, Jankowska & Lundberg, 1966, 1972), There is, on the other hand, only an apparent discrepancy between our observations and those of Lucas & Willis (1974) who first applied adequate stimulation of muscle receptors to the study of interneurones in the intermediate nucleus. These authors limited their observations primarily to extracellularly recorded responses. They found that discharges of about 90% of interneurones appeared only when the stretch of the muscles was suprathreshold for tendon organ afferents; extracellular recording could not resolve whether these discharges were a cumulative effect of nerve impulses in both I a and I b afferents, or were evoked only by I b afferents. However, two of the four interneurones they recorded intracellularly showed e.p.s.p.s attributable to I a afferents.

A comparison of the input from group I a and group I b afferents to laminae V–VI interneurones revealed an extensive convergence of the two groups of afferents on individual neurones. None of the properly tested interneurones were in fact excited by I a afferents from triceps surae and plantaris without also being excited by I b afferents from these muscles. Co-excitation by muscle spindle and tendon organ afferents was found in 41% of our sample of interneurones (Fig. 13*B*). Co-inhibition by I a and I b afferents appeared in 33% (Fig. 13*C*) and excitation by the one group and inhibition by the other was evoked in 50%.

Only a minority (36%) of the laminae V–VI interneurones appeared to subserve reflexes from tendon organ afferents without being influenced by group I a muscle spindle afferents. Thus most of them would be used to mediate reflexes evoked by combined actions of impulses from muscle spindle and tendon organ afferents from these muscles; only if these two receptors were activated in isolation from each other, or if transmission from one of them were preferentially depressed by presynaptic inhibition, would these interneurones mediate only one type of reflex. If and how often such conditions occur remains a question to which we have no answer at present. On the basis of this and of the preceding study (Czarkowska *et al.* 1981) we might assume that group I a and I b afferents operate, as a rule, in concert (see Lundberg, 1979); their selective actions would be an exception in all reflexes evoked from tendon

organ afferents and muscle spindle afferents, except the Ia reciprocal inhibition which is mediated by another group of interneurones (Hultborn *et al.* 1971).

The polymodal character of reflexes mediated by these interneurones is further indicated by their co-excitation by impulses in other afferents (low threshold cutaneous and joint afferents, flexor reflex afferents) and by descending fibre systems, as described previously (Lundberg & Voorhoeve, 1962, Hongo *et al.* 1969, Lundberg *et al.* 1977, 1978) and confirmed in the present experiments.

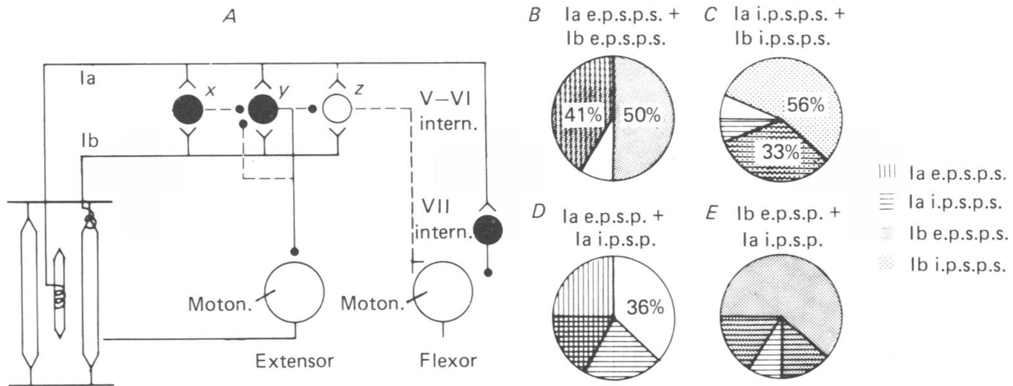

Fig. 13. Convergence on laminae V–VI interneurones. *A*, some of the connections between Ia and Ib afferents and laminae V–VI interneurones suggested by results of this study, as compared to a selective Ia input to laminae VII interneurones mediating the Ia reciprocal inhibition of antagonists. ○—< excitatory neurones; ●—< inhibitory neurones; —— established or highly likely connexions; --- hypothetical connexions. *B* and *C*, proportions of interneurones co-excited or co-inhibited by Ia and Ib afferents of ankle and toe extensors (hatched and stippled), or affected by only one group of the afferents (hatched or stippled). *D*, proportions of interneurones influenced by Ia afferents (excited, vertical hatching; inhibited, horizontal hatching; excited as well as inhibited, cross-hatching) and of interneurones selectively influenced by Ib afferents (blank area). *E*, proportion of interneurones excited by Ib afferents and inhibited by Ia afferents (hatched and stippled).

Functional differentiation of laminae V–VI interneurones with input from group I afferents

In contrast to laminae VII interneurones which have been found to mediate only one type of reflex from Ia afferents, the reciprocal inhibition between flexors and extensors, the group I excited laminae V–VI interneurones may be interposed in several reflex pathways. Among these may be (i) interneurones projecting to motoneurones and mediating their di- and trisynaptic inhibition or excitation from Ib afferents (Laporte & Lloyd, 1952, Eccles *et al.* 1957*a*, *b*) and di- or trisynaptic autogenetic and synergistic inhibition from Ia afferents (Fetz *et al.* 1979) (ii), interneurones mediating presynaptic depolarization of primary afferents from group Ia and/or Ib afferents (for references see Schmidt, 1973) (iii) interneurones subserving crossed excitatory and inhibitory reflexes from group I afferents (Perl, 1958; Holmqvist, 1961, Baxendale & Rosenberg 1976, 1977) as well as (iv) interneurones transmitting information to long ascending tract cells (Eccles, Hubbard &

Oscarsson, 1961, Lundberg & Weight, 1971). One of the main questions to consider with regard to the function of the laminae V–VI interneurones is, therefore, whether individual neurones mediate only one or several of these reflexes. Previous physiological studies have given evidence for parallel actions of some of the interneurones on motoneurones and on ascending tract cells (for references see Baldissera, Hultborn & Illert 1980). Our observations showed further that practically all the stained interneurones which projected to the ventral horn also showed terminal branching in the intermediate zone and therefore might terminate both on motoneurones and on other interneurones. Of other interneurones, some of those with crossed projections had a few axon collaterals branching ipsilaterally and some of those with predominantly ipsilateral projections sent a few collaterals to the opposite side. Thus these two groups of neurones might also be involved in more than one type of reflexes.

Another question is to what extent interneurones interposed in various reflex pathways differ in their input and how they might be recognized. In this respect our observations show that neither co-excitation nor co-inhibition by group Ia and Ib afferents differentiate interneurones which project to motor nuclei from other laminae V–VI interneurones with group I input. The only feature systematically found in the former and only occasionally in the latter was disynaptic excitation from cutaneous afferents and monosynaptic excitation from ipsilateral descending tracts.

Negative feed-back/feed-forward in reflexes from group Ia and Ib afferents

Occurrence of i.p.s.p.s accompanying e.p.s.p.s from group I afferents is such a common feature of the laminae V–VI interneurones that it deserves some special comments. Since such i.p.s.p.s are not depressed by a preceding stimulation of the ventral roots, as found also by S. Lindstrom (personal communication), and Czarkowska *et al.* (1981), it eliminates interneurones mediating Ia reciprocal inhibition of motoneurones (Hultborn *et al.* 1971) as being responsible for these i.p.s.p.s. Neurones evoking them should thus be among laminae V–VI interneurones with input from Ia and/or Ib afferents. As indicated in Fig. 13*A* such inhibitory interneurones might belong to those terminating only on interneurones (x) or to those which mediate inhibition of motoneurones (y), and one could imagine every possible combination of connexions between them, including self-inhibition of some interneurones (e.g. y), and their projections onto excitatory interneurones (z). Only some such connexions are indicated in Fig. 13. Inhibition of laminae V–VI interneurones from group I afferents might thus be considered in terms of a negative feed-back in the pathways activated by muscle contractions, analogous to that of the recurrent inhibition of motoneurones, or in terms of mutual inhibitory interactions between inhibitory neurones, as in the case of Renshaw cells (Ryall, 1970) and interneurones mediating Ia reciprocal inhibition of motoneurones (Hultborn, Illert & Santini, 1976). An understanding of the possible feed-back or feed-forward role of this inhibition and of the organization of relations between the laminae V–VI neurones generally will require identification of the cells of origin of this inhibition, as well as identification of reflex pathways in which the inhibited interneurones are interposed.

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