

INHIBITORY INTERACTIONS BETWEEN INTERNEURONES IN REFLEX PATHWAYS FROM GROUP Ia AND GROUP Ib AFFERENTS IN THE CAT

By E. BRINK*, E. JANKOWSKA, D. A. McCREA† AND B. SKOOG

From the Department of Physiology, University of Göteborg, P.O. Box 33031, S-400 33 Göteborg, Sweden

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SUMMARY

1. A hypothesis has been verified that laminae V–VI interneurones which mediate non-reciprocal inhibition of motoneurones from group I muscle afferents have collateral actions on other laminae V–VI interneurones. Stimulation within the areas of projection of these inhibitory interneurones in motor nuclei and in Clarke's column would be expected to give rise to monosynaptic i.p.s.p.s in interneurones with disynaptic i.p.s.p.s from group I afferents if the hypothesis were correct.

2. Intracellular records were made from eighty-five laminae V–VI interneurones with input from group Ia muscle spindle and/or group Ib tendon organ afferents. Weak intraspinal stimuli applied in motor nuclei in L7 and S1 segments, or in the lateral funiculus just caudal to Clarke's column in L4, were found to evoke monosynaptic i.p.s.p.s in seventy-two interneurones. These i.p.s.p.s were systematically correlated with disynaptic inhibition from group Ia or Ib afferents but not from other fibres.

3. Such monosynaptic i.p.s.p.s evoked by intraspinal stimuli were seen in forty-two interneurones which themselves projected to the level of Clarke's column and therefore (on the basis of previous evidence) should mediate inhibition of motoneurones. For seven of these interneurones it was also shown directly that they projected to motor nuclei. The inhibition of such interneurones demonstrates mutual interactions between those interneurones which are interposed in inhibitory pathways from group I afferents. Only indirect indications have been obtained for inhibition of interneurones in the excitatory pathways.

INTRODUCTION

Observations described in previous papers (Hongo, Jankowska, Ohno, Sasaki, Yamashita & Yoshida, 1983*a, b*; Brink, Harrison, Jankowska, McCrea & Skoog, 1983) have shown that interneurones with group I input in Rexed's laminae V–VI may

* Present address: Neurologische Klinik der Technischen Universität München, Möhlstrasse 28, D-8000 München 80, F.R.G.

† Present address: Department of Physiology, University of Manitoba, 770 Bannatyne Street, Winnipeg, Manitoba, R3E 0W3, Canada.

inhibit both motoneurons and cells of origin of the dorsal spinocerebellar tract (d.s.c.t.). The experiments now reported demonstrate a third type of action of these interneurons: on their fellow interneurons.

When laminae V–VI interneurons with group I input, which most probably included such inhibitory interneurons as well as other inhibitory or excitatory interneurons, were stained by intracellular injection of horseradish peroxidase (HRP), reconstruction of their axonal projections (Czarkowska, Jankowska & Sybirska, 1976, 1981; Jankowska, Johannisson & Lipski, 1981*a*) revealed that they often terminate both on motoneurons and on other laminae V–VI interneurons. Taking into account this observation and the fact that disynaptic inhibitory post-synaptic potential (i.p.s.p.s) of group I origin are evoked in a high proportion of laminae V–VI interneurons (Hongo, Jankowska & Lundberg, 1966, 1972; Jankowska *et al.* 1981*a*), a hypothesis was put forward (Jankowska *et al.* 1981*a*, see their fig. 13) that interneurons which mediate non-reciprocal inhibition of motoneurons may have collateral inhibitory action on transmission in other pathways from muscle spindle and tendon organs, as indicated by small arrows in Fig. 1. This hypothesis has now been tested by verifying some of its implications. One of the implications is that if an inhibitory interneurone which projects to the motor nuclei (neurone labelled X in Fig. 1) inhibits both motoneurons and other laminae V–VI interneurons, then stimulation of its axon within the motor nucleus should give rise to monosynaptic inhibition of some of the interneurons (e.g. neurones Y and Z). Stimulation of the rostrally projecting collaterals of interneurons X in Clarke's column (Hongo *et al.* 1983*b*) or in the lateral funiculus at L3–L4 (see Brink *et al.* 1983) should have the same effect. Another implication of the hypothesis is that the monosynaptic inhibition evoked by these intraspinal stimuli should be associated with disynaptic inhibition from group I afferents and with inhibition from all the fibre systems which converge onto the same interneurons (Lundberg & Voorhoeve, 1962; Hongo, Jankowska & Lundberg, 1969, 1972; Illert, Lundberg & Tanaka, 1976; Lundberg, Malmgren & Schomburg, 1977, 1978; Jankowska & McCrea, 1983; Harrison, Jankowska & Johannisson, 1983; Harrison & Jankowska, 1982). Only the occurrence of inhibition of group Ia and group Ib origin has been tested systematically in this series of experiments and inhibitory input from other fibre systems will be reported separately.

METHODS

The study is based on intracellular records from eighty-five laminae V–VI interneurons in fourteen cats anaesthetized with chloralose (50–70 mg/kg initial dose). Five of these cats were spinalized at the Th13–L1 level. The cats were also used for other experiments (Brink *et al.* 1983).

Experimental procedures

The preparation, the general techniques of intracellular recording from laminae V–VI interneurons, and criteria for defining group Ia and group Ib input to the interneurons were as described by Jankowska *et al.* (1981*a*). The detailed procedure adopted for the purposes of the present study was as follows.

(i) A micro-electrode, usually filled with 3 M-K citrate solution, was placed in the intermediate zone in L7 or L6, at a site where large monosynaptic field potentials were evoked by group I afferents of triceps surae and plantaris. The electrode was used subsequently for intracellular recording from interneurons within a 2–8 mm length of the spinal cord.

(ii) Another micro-electrode filled with 2 M-NaCl solution was placed in triceps surae or plantaris motor nuclei in L7, at the site of the largest monosynaptic field potentials from Ia afferents of one of these muscles; it was left there or moved only up and down. It was used for stimulation in the motor nuclei and was shielded as described by Eide (1971) and Jankowska & Roberts (1972*a*) to reduce the stimulus artifacts. The stimulus intensity was monitored as a voltage drop across a 1000 Ω resistor.

(iii) A third electrode (a glass micropipette filled with 2 M-NaCl, a tungsten electrode with a 2–3 μm tip protruding from a glass micropipette (Stoney, Thompson & Asanuma, 1968), or a tungsten electrode varnished except for its tip) was introduced into the lateral funiculus between L3 and L4 segments, about 2.5 mm from the surface. The electrode was used to stimulate ascending axonal branches of the interneurons which mediate autogenetic inhibition of motoneurons and

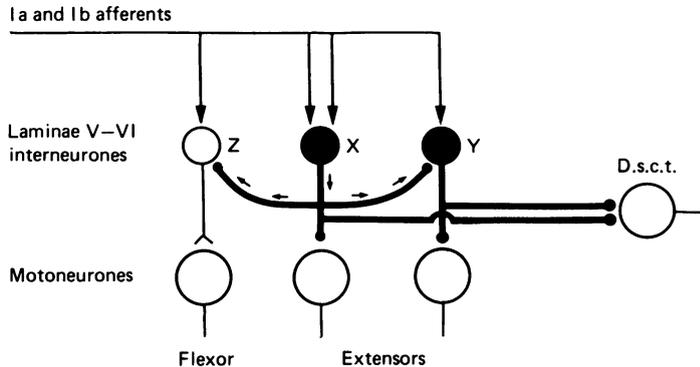


Fig. 1. Diagram of the hypothesized interactions between laminae V–VI interneurons. Excitatory and inhibitory interneurons with input from group Ia and Ib afferents of triceps surae and plantaris are represented by open and filled medium-sized circles, respectively. Motoneurons innervating the same muscles and their synergists (extensors) or antagonists (flexors) and dorsal spino-cerebellar tract cells (d.s.c.t.) are represented by large circles. The pathways under investigation are indicated by small arrows. Input from other afferents (cutaneous and joint) is indicated only for one of the interneurons. Further explanations in the text.

inhibition of dorsal spinocerebellar tract cells from group I afferents (Hongo *et al.* 1983*a, b*; Brink *et al.* 1983). The ascending branches were stimulated in the funiculus rather than in the Clarke's column in order to maximize the number activated. The position of the third electrode was adjusted by monitoring antidromic responses of the first few laminae V–VI interneurons encountered in L7, to find a site where the thresholds for their activation were minimal. The effects of constant-current stimuli applied through this electrode (100–200 μA) were always compared with effects of a strong (0.5–1 mA) stimulation of the lateral funiculus at Th13, to differentiate actions of neurones projecting only within the lumbar cord from actions of long ascending and descending tract fibres.

(iv) After the second and third electrodes were positioned, the first electrode was moved until it penetrated an interneurone with group I input. The group I input was tested using electrical stimulation of various nerves, or adequate activation of group Ia afferents of medial gastrocnemius, lateral gastrocnemius-soleus or plantaris, the nerves of these muscles being dissected in continuity. The four muscles were stretched together using single muscle stretches below threshold for Ib afferents (< 35 μm ; see Fetz, Jankowska, Johannisson & Lipski, 1979 and Jankowska *et al.* 1981*a*). Input from Ib afferents was determined by comparing the effects of electrical stimulation of the nerves at a strength near maximal for both group Ia and Ib afferents but subthreshold for group II afferents (1.4–1.5 times threshold for Ia afferents), with the effects of muscle stretches maximal for group Ia afferents (60–70 μm ; see Fetz *et al.* 1979 and Jankowska *et al.* 1981*b*).

Criteria for monosynaptic i.p.s.p.s evoked by intraspinal stimuli

I.p.s.p.s evoked from the motor nuclei. These were classified as monosynaptic when they appeared with a latency of up to 1.2 msec after a near-threshold stimulus. The latency was expected to be

made up by: (i) 0.1–0.3 msec of the latent period between the beginning of the stimulus and the generation of the action potentials in the stimulated axons (see Jankowska & Roberts, 1972*a*; Gustafsson & Jankowska, 1976). (ii) 0.2–0.5 msec of *conduction time along the terminal branches* in the motor nucleus and in laminae V–VI, allowing for distances of 2–5 mm and conduction velocity of 10 m/sec (see Eccles, Fatt & Landgren, 1956; Jankowska & Roberts, 1972*a*; Wall & Werman, 1976; Munson & Sypert, 1979) plus 0.1–0.2 msec of *conduction time along the stem axons* in the lateral funiculus, allowing for the distances of 5–10 mm in the rostrocaudal direction and 40 m/sec conduction velocity (see Czarkowska *et al.* 1976, Hongo *et al.* 1983*b*). (iii) 0.3–0.4 msec of *synaptic delay* (Eccles *et al.* 1956; Jankowska & Roberts, 1972*b*; Rapoport, Susswein, Uchino & Wilson, 1977; Hikosaka, Igusa, Nakao & Shimazu, 1978; Munson & Sypert, 1978). An alternative criterion was that the latencies of the monosynaptic i.p.s.p. should exceed the latencies of the earliest antidromic activation of laminae V–VI interneurons by not more than 0.9 msec, to allow one but not two synaptic delays; the earliest antidromic latencies were 0.4–0.5 msec (see Fig. 3, Δ), raising the upper limits of the latencies of monosynaptic i.p.s.p.s.

I.p.s.p.s evoked from LA. These were classified as monosynaptic when their latencies exceeded by not more than 0.9 msec the shortest latencies of antidromic invasion of laminae V–VI interneurons with group I input which were located in the same region of the spinal cord. I.p.s.p.s were also considered to be monosynaptic when their latencies in relation to the descending volleys, recorded at the surface of the lateral funiculus at L7, were 0.9 msec or less. As in the case of actions evoked from the motor nuclei, such latencies should allow one but not two synaptic delays. Using these criteria, 1.3 msec was set as the upper limit for the monosynaptic i.p.s.p.s.

RESULTS

Monosynaptic inhibition of laminae V–VI interneurons by stimuli applied in motor nuclei

Figs. 2 and 4 demonstrate that monosynaptic i.p.s.p.s may be evoked in laminae V–VI interneurons by stimuli applied in the motor nuclei. I.p.s.p.s which appeared with latencies 0.65–1.2 msec in relation to the onset of the stimulus (Fig. 3, Δ) and which were classified as monosynaptic according to the criteria discussed in the Methods, were seen in twenty-six out of thirty-six interneurons tested. Only one stimulating electrode was introduced into the triceps surae or plantaris motor nuclei, and the stimulus intensity (20–30 μ A) was adjusted to be effective within about 0.2–0.3 mm radius (Jankowska & Roberts, 1972*a*) to avoid spread of current to interneurons located outside lamina IX. These stimuli were thus likely to excite axonal branches of only a relatively small proportion of interneurons projecting to the motor nuclei studied, which may extend over a distance of several mm (see e.g. Burke, Strick, Kanda, Kim & Walmsley, 1977). Consequently only positive results are considered of value. Records of Fig. 2*A* show to what extent presence or absence of i.p.s.p.s in a given interneuron might have depended on the position of the stimulating electrode. With 30 μ A pulses, i.p.s.p.s followed stimuli applied at four out of seven electrode locations 100 μ m apart and failed to be evoked from three others; the track passed through tibial (depth 1.1–1.2 mm), gastrocnemius (depth 1.2–1.4 mm) and hamstring (depth 1.5–1.6 mm) motor nuclei. Since the i.p.s.p.s were superimposed on the tail of the shock artifact, one or a few of the following tests were used to ascertain their presence: a comparison with records taken at another position of the stimulating electrode (as in Fig. 2*A*), a comparison with extracellular records (as in Fig. 4), enhancement of the i.p.s.p.s after depolarization of the cell membrane, or reversal of the i.p.s.p.s after hyperpolarization (as in Fig. 2*B*).

Monosynaptic inhibition of laminae V–VI interneurons by stimuli applied in L4

I.p.s.p.s fulfilling criteria of monosynaptic i.p.s.p.s were found in sixty out of sixty-eight interneurons tested for inhibition from L4. Examples of such i.p.s.p.s are in Figs. 4–6. In fourteen interneurons i.p.s.p.s were evoked from both the motor

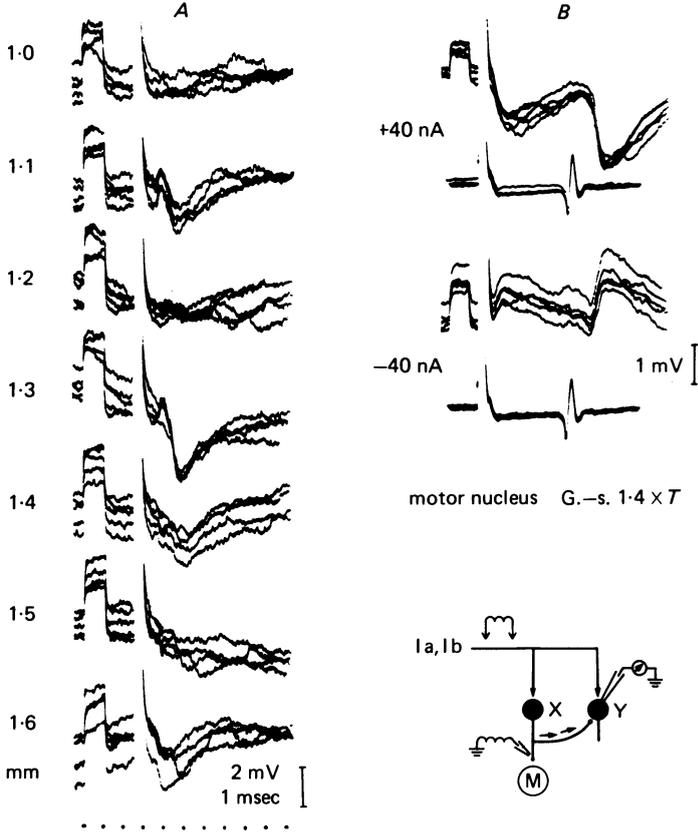


Fig. 2. Monosynaptic i.p.s.p.s evoked by intraspinal stimuli applied in motor nuclei. *A*, intracellular records from an interneurone excited by Ia afferents in tibial, plantaris and triceps surae nerves; note corresponding monosynaptic e.p.s.p.s following stimuli in tibial and medial gastrocnemius motor nuclei at depths 1.1 and 1.3 mm. The i.p.s.p.s were evoked from within tibial (depth 1.1 mm), medial gastrocnemius (depth 1.3 mm) and hamstring (depth 1.6 mm) motor nuclei. *B*, intracellular records (upper traces) from another interneurone and records from the cord dorsum (lower traces). I.p.s.p.s were evoked from the motor nucleus and from group I afferents of gastrocnemius–soleus (G.-s.): the upper set of records was taken when the interneurone was depolarized and the lower set when it was hyperpolarized, with resulting reversal of the i.p.s.p.s. The diagrams in this and in the following Figures show parts of the diagram in Fig. 1 with the experimental arrangement used to establish the illustrated interactions.

nuclei and from L4, so that i.p.s.p.s from either of these sites were seen in a total of seventy-two of the eighty-five interneurons investigated.

Fig. 3 shows the distribution of latencies of i.p.s.p.s (○) and of antidromic invasion from L4 (●) of our sample of interneurons. The latencies are plotted against

distances from the location of the interneurons to the stimulating electrode in the lateral funiculus. The latencies may be compared with latencies of responses evoked from the motor nuclei, which are indicated by triangles. The Figure shows considerable ranges of latencies of both the antidromic invasion and the i.p.s.p.s evoked from the two sites; their variability obscures any expected increase with distance. The conduction velocities of the stimulated fibres, calculated for antidromic invasion from

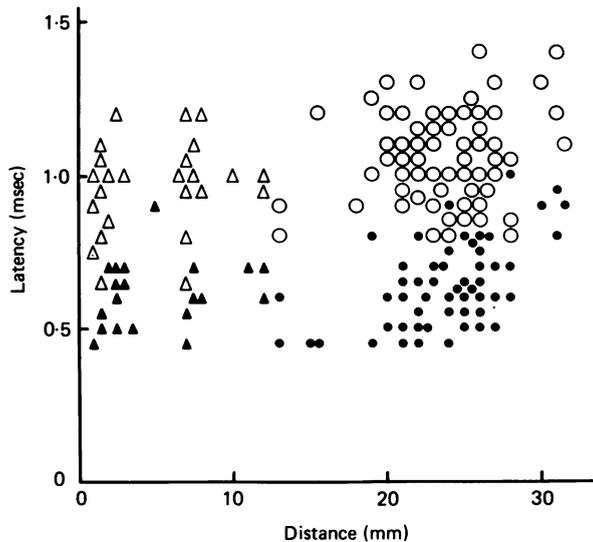


Fig. 3. Distribution of latencies of monosynaptic i.p.s.p.s evoked by intraspinal stimulation. Open symbols, latencies of i.p.s.p.s evoked from motor nuclei (Δ) and from the lateral funiculus at L4 (\circ). Filled symbols, latencies of antidromic activation, similarly from motor nuclei (\blacktriangle) and from L4 (\bullet). They were obtained for the same and for different interneurons but only those recorded intracellularly. The latencies are plotted against distance between the stimulating and recording electrodes but without correction for differences in depth and for the length of the terminal axonal branches in the motor nuclei.

distances of 10–30 mm, appeared to be as high as 65–70 m/sec and as low as 20–25 m/sec. Since the length of the collaterals stimulated in the motor nuclei was an unknown factor, the conduction velocities were not calculated for shorter distances as such calculations would be unreliable.

When stimulation of long ascending and descending tract fibres at Th13 evoked i.p.s.p.s (in two interneurons), these i.p.s.p.s appeared with much longer segmental latencies than the i.p.s.p.s evoked from the lateral funiculus at L4 and were much smaller.

Evidence for mutual inhibition between inhibitory interneurons

According to the hypothesis presented in Fig. 1, both the excitatory and inhibitory interneurons in pathways from Ia and Ib afferents to motoneurons might be subject to collateral actions of inhibitory interneurons, but we have had a possibility to verify this only for the inhibitory interneurons. Monosynaptic excitation from group I afferents and axonal projection to upper lumbar segments, in addition to

inhibition of group I origin and excitation by cutaneous afferents, were used as the distinguishing features of interneurons mediating autogenetic and synergistic inhibition of motoneurons (Hongo *et al.* 1983 and Brink *et al.* 1983). Seven of these interneurons were indeed found to be antidromically activated from both L4 and the motor nuclei (Fig. 4A and B). Monosynaptic i.p.s.p.s evoked in such interneurons

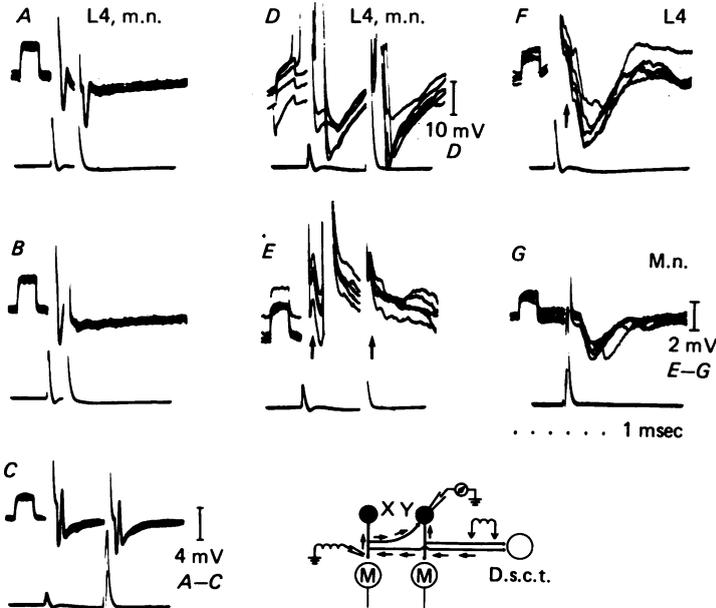


Fig. 4. Monosynaptic i.p.s.p.s evoked from motor nuclei (m.n.) and from the lateral funiculus in L4 in an interneurone projecting to the two stimulation sites. Upper traces, extracellular records before (A-C) and intracellular record, after (D-G) its penetration. Lower traces, records from the cord dorsum. D-G illustrate subsequent changes in responses to the stimuli: progressive blockade of the antidromic invasion with barely visible M spike in some of the traces in F and monosynaptic i.p.s.p.s which become more conspicuous after the cell stopped firing and became depolarized.

by stimuli applied in L4 or in motor nuclei were, therefore, considered as evidence of mutual inhibition between interneurons in the inhibitory pathways to motoneurons. They were seen in forty-two interneurons.

It should be stressed in this context that the number of interneurons of our sample projecting to the upper lumbar segments might have been larger than counted since the spike generation mechanism often deteriorated after cell penetration, and antidromic activation became difficult to assess as illustrated in Fig. 4. Fig. 4A and B show unquestionable antidromic spikes from L4 and from motor nuclei (with a collision at a shorter interval in B) while recorded extracellularly. Records in Fig. 4C and D are just before and just after cell penetration, respectively, with clear IS spikes in D; only M spikes can be detected in the next records (E and F, arrows) but even these disappeared (G) with progressive cell depolarization. The depolarization favoured, on the other hand, disclosure of i.p.s.p.s. When distinct spikes like those in Fig. 4D, or all-or-nothing M spikes as in Fig. 5A and E were not seen, we did not classify the interneurons as antidromically invaded.

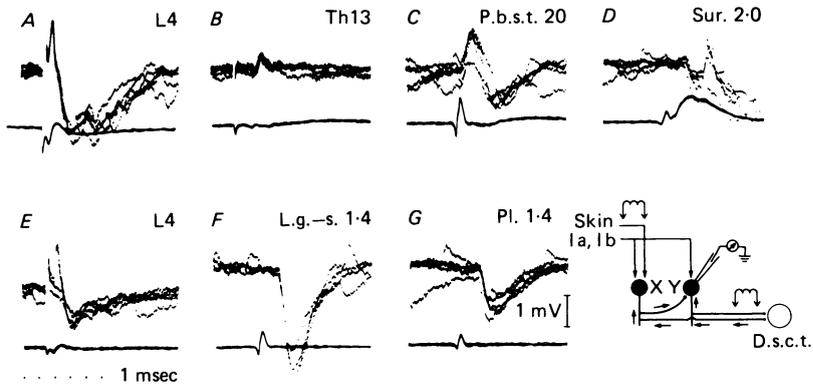


Fig. 5. Monosynaptic i.p.s.p.s evoked from the lateral funiculus (*A* and *E*) in an interneurone antidromically invaded from L4, with disynaptic i.p.s.p.s from group I afferents in three nerves stimulated electrically (*C*, *F* and *G*). Predominant inhibition was evoked from cutaneous afferents (*D*). Upper traces, intracellular records. Lower traces, records from the cord dorsum. P.b.s.t., posterior biceps-semi-tendinosus; Sur. sural; Pl., plantaris; L.g.-s., lateral gastrochemius-soleus.

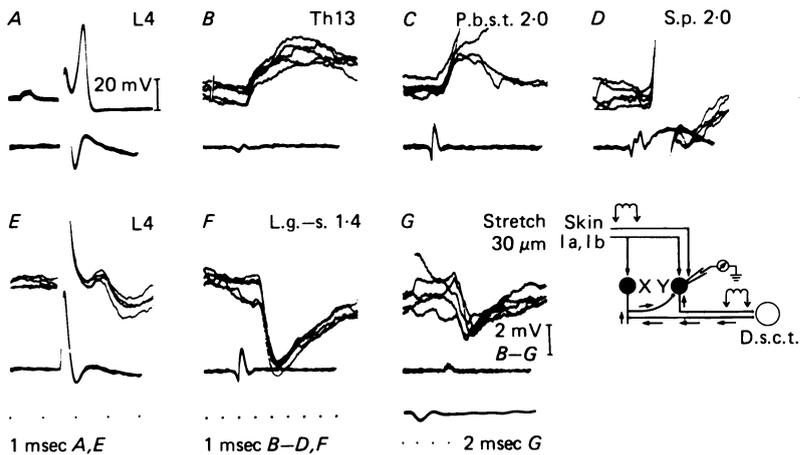


Fig. 6. Monosynaptic i.p.s.p.s evoked from the lateral funiculus in an interneurone antidromically invaded from L4, with disynaptic i.p.s.p.s from group I afferents (*F*, *G*). The i.p.s.p.s were evoked by electrical stimulation of both Ia and Ib afferents and by selective activation of Ia afferents by muscle stretch. Predominant excitation was evoked from cutaneous afferents (*D*). Upper traces, intracellular records. Lower traces, records from the cord dorsum. Lowermost trace in *G*, record of changes in length of triceps surae and plantaris stretched together. Stimulus intensity was lower in *E* than in *A*.

Correlation between monosynaptic i.p.s.p.s following intraspinal stimuli and i.p.s.p.s evoked from group I afferents

If i.p.s.p.s described in the preceding sections were evoked by interneurons mediating autogenetic inhibition of motoneurons, they should be matched by disynaptic i.p.s.p.s following stimulation of group Ib or both group Ia and Ib afferents (see Fig. 1). Fig. 5 and 6 show examples of such disynaptic i.p.s.p.s in two interneurons with monosynaptic i.p.s.p.s evoked by intraspinal stimuli. Such group

I.i.p.s.p.s were seen in sixty-two out of seventy-two (86%) interneurons with i.p.s.p.s from either the motor nuclei or L4. In about half they were evoked by Ia afferents activated by muscle stretches, as in Fig. 6*G*, as well as by Ib afferents. In ten interneurons only monosynaptic or disynaptic e.p.s.p.s were seen from the tested nerves, or the cells were lost before it was possible to check if any i.p.s.p.s were superimposed onto these e.p.s.p.s.

Stimulation of cutaneous, joint or high threshold muscle afferents sometimes evoked i.p.s.p.s, alone or combined with e.p.s.p.s (Fig. 5*D*), but evoked apparently only e.p.s.p.s in about half of the cells (Fig. 6*D*).

Comments on e.p.s.p.s evoked upon stimulation of the lateral funiculus at L4

Stimuli applied at L4 and Th13 were often followed by e.p.s.p.s, usually monosynaptic, as illustrated in Figs. 5*B* and 6*B*. In contrast to the i.p.s.p.s the e.p.s.p.s evoked by L4 (intraspinal) stimuli were usually smaller than those of thoracic origin. They were, therefore, interpreted as being due to activation of the same descending fibres, most likely rubro- or cortico-spinal, but possibly also propriospinal (Jankowska, Lundberg & Stuart, 1983), and so do not contradict previous evidence that ascending projection characterizes only inhibitory interneurons with group I input (Hongo *et al.* 1983*a, b*; Brink *et al.* 1983). However, the e.p.s.p.s could also be due to some excitatory interneurons with descending or ascending projection and input from other than group I afferents.

The previous evidence that no group I excited interneurons send ascending collaterals *with excitatory action* to L3–L4 segments consisted in showing that only i.p.s.p.s are evoked in lower lumbar motoneurons upon stimulation of single group I excited laminae V–VI interneurons with ascending projection (Brink *et al.* 1983), or by stimuli applied in Clarke's column (Hongo *et al.* 1983*b*). Stimulation of group I afferents after transection of the dorsal columns similarly evoked only i.p.s.p.s in d.s.c.t. cells (Hongo *et al.* 1983*a*). In a supplementary series of experiments we have now extended the latter observations to see whether we find other neurons in L3–L4 segments to be excited by lower lumbar interneurons with group I input.

Intracellular records were made from motoneurons ($n = 22$) and other neurons (mainly laminae V–VI interneurons, $n = 19$) in L3 and L4 segments in four cats following a complete lesion of the dorsal columns at the border between L4 and L5 segments (as in experiments of Hongo *et al.* 1983*a*). The completeness of the lesion was verified histologically and by recording from the peripheral nerves during stimulation of the dorsal columns above the lesion, or from the surface of the dorsal columns on one side of the lesion, while the stimuli were applied on the other side. Any post-synaptic potentials evoked in the tested neurons following stimulation of peripheral nerves could then be mediated only by interneurons located caudal to the lesion. The neurons selected for testing were those with membrane potential of at least 50 mV or in which some e.p.s.p.s were evoked by stimulation of the dorsal roots at the same level, or of the dorsal columns above the lesion. Motoneurons were identified by antidromic activation following stimulation of spinal nerves just outside the dura, and ascending tract cells by antidromic activation following stimulation of the spinal cord at Th12. In no case was there any evidence for di- or oligosynaptic e.p.s.p.s evoked on stimulation of hind-limb muscle nerves below threshold for group

II afferents, even when two or more shocks were used to increase the probability of appearance of the tested e.p.s.p.s. If such stimuli were effective, they produced only i.p.s.p.s. The i.p.s.p.s were evoked by stimulation of both low- and high-threshold group I afferents, or by group Ia afferents (in four motoneurons). Stimulation of quadriceps nerve was most effective in evoking these i.p.s.p.s, although they appeared also upon stimulation of triceps surae and plantaris. The i.p.s.p.s were found in eight motoneurons and in three interneurons. They appeared with latencies 1.4–2.8 msec with respect to incoming volleys recorded from the surface of the spinal cord at L6 and are classified as disynaptic, using the same arguments as used by Hongo *et al.* (1983*a*) for classifying latencies of i.p.s.p.s recorded in the dorsal spinocerebellar tract cells.

DISCUSSION

The observations reported are fully compatible with the hypothesis that the same interneurons mediate group I inhibition of both motoneurons and laminae V–VI interneurons, even if each observation considered separately might be given an alternative explanation. For instance, stimuli applied in the motor nuclei might have evoked inhibition of laminae V–VI interneurons by exciting axons of interneurons other than those with group I input. Similarly, inhibition evoked from the L4 segment might have been evoked by other types of interneurons with ascending axon collaterals, or be mediated via propriospinal neurons located in the upper lumbar segments and projecting caudally (see Hongo *et al.* 1983*b*). However, we consider it unlikely and too coincidental that these alternative explanations are correct rather than the single hypothesis which explains all the reported observations after having predicted them.

In regard to the problem of which laminae V–VI interneurons are inhibited in parallel with motoneurons, we propose that interneurons which excite motoneurons and those which inhibit motoneurons do not differ in this respect. However, our positive evidence is so far only for inhibitory interneurons; it is based on the appearance of i.p.s.p.s evoked by intraspinal stimuli in cells which were antidromically activated from L4 or L3 segments (Hongo *et al.* 1983*a, b*; Brink *et al.* 1983). Failure to activate the neurons from the L4 segment could not be used as evidence that they were excitatory but it will be noted that monosynaptic i.p.s.p.s were also found in such interneurons.

Parallel inhibition of motoneurons and of other interneurons by laminae V–VI interneurons might be useful in several ways. First of all it might help to select the required pattern of Ia and Ib actions (see Hongo *et al.* 1969, 1972). As repeatedly shown, actions of group I afferents of a given muscle may be either excitatory or inhibitory, and involve many motor nuclei (Granit, 1950; Laporte & Lloyd, 1952; Eccles, Eccles & Lundberg, 1957*b*; Hongo *et al.* 1969; Jankowska, McCrea & Mackel, 1981*b, c*; Harrison *et al.* 1983). The patterns of motoneurons excited and inhibited must thus be adjusted for each movement (Hongo *et al.* 1969, 1972) and it is postulated that these movements are subserved by separate sets of interneurons (Harrison & Jankowska, 1982). Inhibition of some subsets of these interneurons could serve to eliminate the unnecessary Ia and Ib actions or those actions that could interfere with the intended movements. The release from inhibition of motoneurons which should be activated (e.g. via interneurons Y in Fig. 1) might serve a similar

purpose. To adjust the degree of the negative feed-back of motoneurones might be another task of the inhibitory interactions between laminae V–VI interneurones. Following activation of motoneurones the negative feed-back would be subserved first by Renshaw cells and the disynaptic inhibition evoked by these cells. The subsequent contraction of a muscle, followed by activation of laminae V–VI interneurones, would supplement the Renshaw effects by i.p.s.p.s mediated via these interneurones, while a release of motoneurones from their actions might leave only recurrent inhibition operating. The interactions between interneurones may thus be considered as a subsidiary negative feed-back in the pathways of the main feed-back systems.

The parallel inhibitory actions of laminae V–VI interneurones on motoneurones and other interneurones can be integrated into larger entities of motor behaviour via several systems of control and selection. The significance of the involved afferent and descending fibre systems has already been discussed extensively (Hongo *et al.* 1969, 1972; Lundberg, 1975; Lundberg *et al.* 1977, 1978; Jankowska & McCrea, 1983; Pierrot-Deseilligny, Bergego, Katz & Morin, 1981; Pierrot-Deseilligny, Bergego & Katz, 1982). It will, therefore, only be pointed out that parallel inhibition of motoneurones and of other interneurones by the same laminae V–VI interneurones should contribute to the control of motor synergies much more effectively than if there were two subgroups of interneurones acting either on motoneurones or on interneurones. A third group of interneurones would then be needed to co-ordinate their activity. Reasoning along these lines, one might expect there to be a similar possibility that interneurones in excitatory pathways between group I afferents and motoneurones might influence other interneurones and be responsible for disynaptic e.p.s.p.s of group I origin in laminae V–VI interneurones (Czarkowska *et al.* 1981; Jankowska *et al.* 1981*a*) in addition to trisynaptic e.p.s.p.s evoked in motoneurones (Eccles *et al.* 1957*a, b*; Jankowska *et al.* 1981*c*). However, the occurrence of such collateral excitatory actions of laminae V–VI interneurones remains to be investigated.

Inhibitory interaction between laminae V–VI interneurones which subserve the non-reciprocal inhibition of motoneurones is not the first case of such interactions. Mutual inhibition has been found within both previously identified groups of spinal inhibitory interneurones: Renshaw cells (Ryall, 1970) and interneurones mediating the Ia reciprocal inhibition (Hultborn, Illert & Santini, 1976). If one may generalize features of these three groups of neurones, mutual inhibitory interactions may be expected also between other inhibitory neurones, both in the spinal cord and in the brain.

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