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PATTERN OF 'NON-RECIPROCAL' INHIBITION OF MOTONEURONES BY IMPULSES IN GROUP Ia MUSCLE SPINDLE AFFERENTS IN THE CAT

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SUMMARY

1. Inhibitory post-synaptic potentials (i.p.s.p.s) evoked by adequate stimulation of group I a muscle spindle afferents of triceps surae and plantaris and by near-threshold electrical stimulation of quadriceps and hamstring nerves were recorded in a number of motoneurone species. The aim of the study was to compare the pattern of non-reciprocal I a inhibitory actions on hind-limb motoneurones with the pattern of inhibition evoked from group I b tendon organ afferents.

2. In all the motoneurone species analysed in which i.p.s.p.s were evoked by electrical stimulation maximal for both group I a and I b afferents of triceps surae and plantaris, they were also evoked when these muscles were stretched and the amplitude of the stretch (10–35 μm) was below threshold for I b afferents; 70–100 % of motoneurones with I b i.p.s.p.s showed stretch-evoked i.p.s.p.s. The stretch-evoked i.p.s.p.s appeared with latencies compatible with disynaptic and trisynaptic linkage. Since these latencies were too short to allow their mediation by group II afferents the i.p.s.p.s are attributed to a selective action of I a afferents. The i.p.s.p.s did not appear after the nerves to triceps surae and plantaris had been cut.

3. Electrical stimulation of quadriceps and hamstring nerves which was near threshold for I a afferents and well below threshold for either the I b component of the incoming volley or group II afferents, similarly evoked non-reciprocal i.p.s.p.s. They were found in those motoneurones in which inhibition was evoked by stimulation maximal for group I afferents. Such I a i.p.s.p.s were evoked both in homonymous motoneurones and in motoneurones of four other hind-limb muscles. Their latencies corresponded to di- and trisynaptic coupling.

4. In some motoneurones of the pretibial flexors (anterior tibial, extensor digitorum longus and peroneus longus), disynaptic i.p.s.p.s evoked from triceps surae and/or plantaris which were depressed by a conditioning ventral root stimulation (i.e. I a reciprocal i.p.s.p.s) were followed by trisynaptic i.p.s.p.s which were not depressed in this way (I a 'non-reciprocal' i.p.s.p.s). It thus appears that the same motoneurones may be inhibited by impulses in group I a afferents via different spinal pathways.

5. The study leads to the conclusion that the non-reciprocal inhibition from group I a muscle spindle afferents operates in parallel with the inhibition from group I b tendon organ afferents in all motoneurone species tested.

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INTRODUCTION

Inhibition of spinal motoneurones by group I afferents of synergistic and homonymous muscles has long been attributed to the tendon organ (Ib) afferents (Granit, 1950; Laporte & Lloyd, 1952; Hunt, 1952; Eccles, Eccles & Lundberg, 1957*a*, *b*). Observations compatible with some contribution of muscle spindle (Ia) afferents to the effects of Ib afferents were only occasionally reported (see e.g. Eccles *et al.* 1957*a*. and Lundberg, Malmgren & Schomburg, 1977*b*), and the Ia origin of the lowest-threshold inhibitory post-synaptic potentials (i.p.s.p.s) seen in these studies could not be conclusively established. Using selective activation of Ia muscle spindle afferents of triceps surae and plantaris by adequate stimuli, a recent study by Fetz, Jankowska, Johannisson & Lipski (1979) presented evidence for inhibition evoked from Ia afferents in motoneurones of homonymous muscles and their close synergists (hereafter referred to as Ia non-reciprocal inhibition). However, the latter study left unanswered the question of whether the Ia contribution to Ib effects is a general phenomenon or only a feature of the autogenetic inhibition of motoneurones of ankle and toe extensors; this question is taken up in this and in the following paper. Results now reported extend the observations of Fetz *et al.* (1979) to the autogenetic inhibition of three other motoneurone species and to inhibition of all motoneurones in which inhibitory effects of group Ib afferents of triceps surae and plantaris have been established. The paper to follow (Jankowska, McCrea & Mackel 1981*b*) further extends these observations to excitatory actions of Ib tendon organ afferents and shows that the Ia muscle spindle afferents contribute to them as well. Some of the preliminary results of this study have been reported briefly (Jankowska, Mackel & McCrea, 1980; Jankowska & McCrea, 1981).

METHODS

Preparation. The experiments were performed on twenty-eight cats, using the same preparation and general experimental procedures as described by Fetz *et al.* (1979). In order to maximize the possibility of detection of the effects to be analysed the experiments were performed on spinalized cats, since both group Ib actions and Ia autogenetic inhibition are stronger in such preparations than in those with intact spinal cord (Holmqvist & Lundberg, 1961; Fetz *et al.* 1979). Chloralose anaesthesia (50–60 mg/kg I.V. initial dose) was chosen as being the least depressant for polysynaptic actions and 4-aminopyridine (0·5–1·0 mg/kg I.V.) was administered in seventeen experiments (while recording from about 60 % of motoneurones) to enhance synaptic transmission in the pathways that were being investigated (cf. Jankowska, Lundberg, Rudomin & Sykova, 1977). The cats were paralysed with gallamine triethiodide and artificially ventilated.

All nerve branches of the femoral nerve and of the sciatic nerve from the hamstring nerve distally (except those to triceps surae and plantaris) were always sectioned, resulting in denervation of a great part of the hind-limb. In addition both adductor and gracilis or only gracilis branches of the obturator nerve were sectioned in thirteen experiments; glutei nerves were sectioned in one experiment.

Stimulation of Ia afferents and criteria for attributing the synaptic actions to them. Group Ia muscle spindle afferents of triceps surae and plantaris were activated by longitudinal stretches, of 30–35 μm or less, of the calcaneal tendon at initial tension of 5·0–5·5 N, as described by Fetz *et al.* (1979). Under these conditions activation of Ib afferents has been found both by Fetz *et al.* (1979) and by us in an additional sample of Ib fibres (total $n = 64$) to require more than 40 μm stretch. Lower thresholds of two Ib fibres reported by Ellaway & Trott (1978) are thus interpreted as due to different parameters of muscle stretches or some particular experimental conditions.

The most sensitive group II muscle spindle afferents are excited by muscle stretches < 35 μm,

as reported previously (Lundberg & Winsbury, 1960; Stuart, Mosher, Gerlach & Reinking, 1970) and as found in the case of six out of fifty-eight group II afferents of triceps surae and plantaris analysed in this study. Since such afferents might also inhibit motoneurones, the group Ia i.p.s.p.s were differentiated from group II i.p.s.p.s in each individual motoneurone. To this end it was first ascertained that electrical stimulation of the nerves to triceps surae and plantaris below threshold for group II afferents resulted in the appearance of responses similar to those evoked by muscle stretches (Fig. 2*C*, *D* and *G*, *H*). The thresholds for group II afferents to electrical stimuli may be as low as about 1·5 times the thresholds for Ia afferents (cf. Jack, 1978, and Coppin, Jack & MacLennan, 1970; confirmed for thresholds of group II afferents of both gastrocnemii and plantaris). Any stretch-evoked i.p.s.p.s which were not matched by i.p.s.p.s evoked by weaker electrical stimuli were consequently eliminated, unless the electrically evoked i.p.s.p.s occurred with a latency $\leqslant$ 1·6 msec. The latter were considered to be too early to be mediated by group II afferents and were classified as being due to group I afferents independently of their threshold. The shortest reported latencies of monosynaptic excitatory post-synaptic potentials (e.p.s.p.s) evoked in motoneurones by group II afferents stimulated electrically are about 1 msec with respect to group I incoming volleys (Stauffer, Watt, Taylor, Reinking & Stuart, 1976; Lundberg, Malmgren & Schomburg, 1977*a*), only about 0·6 msec longer than the minimal latencies of Ia e.p.s.p.s. No disynaptic i.p.s.p.s of group II origin have been reported in the literature but if present, their minimum latencies should similarly be only 0·6 msec longer than those of group I disynaptic, i.p.s.p.s, or about 1·7 msec. For trisynaptic group II i.p.s.p.s the shortest latencies of the electrically evoked effects would be about 2·4 msec (cf. Lundberg, Malmgren & Schomburg, 1975).

The effectiveness in evoking i.p.s.p.s of electrical stimuli below threshold for group II muscle spindle afferents did not exclude the contribution of these afferents, if they had been excited by stretch, to the stretch-evoked i.p.s.p. in the same motoneurones. However, we assumed that any such contribution could be possible only in the case of i.p.s.p.s evoked with longer latencies measured in relation to Ia incoming volleys. Stuart *et al.* (1970) have reported that impulses set up in spindle secondaries by muscle stretches are seen at the dorsal root entry zone 3 msec after impulses in the fastest-conducting group Ia afferents. When testing whether similar delays would occur under our experimental conditions we found that nerve impulses in two of the six group II afferents activated by stretches $\leqslant$ 35 μm reached the spinal cord only 1·0 and 1·7 msec after the earliest responses of group Ia afferents in the same preparation. It turned out, however, that if the stretch of the muscle is preceded by relaxation (as in Fig. 1*B*) instead of being followed by it (as in Fig. 1*A*), the relative increase in the latency of activation of group II afferents is larger than that of group Ia afferents. The earliest impulses in group II afferents arrived then 2·6–2·8 msec after the earliest impulses in group Ia afferents. Muscle stretches preceded by relaxation had the disadvantages that the resulting excitation of group Ia afferents was somewhat less synchronous (cf. Fig. 1*A* and *B*) and that about one third of these afferents showed increased thresholds. However, only one out of twenty group Ia afferents tested increased its threshold from below to above 30 μm, and both the threshold stretches and stretches for evoking maximal e.p.s.p.s in motoneurones were within the same ranges as described previously (Fetz *et al.* 1979). These disadvantages were therefore considered as outweighed by the advantage of increasing the safety margins for effects attributable to a selective activation of group Ia afferents: to all those evoked with segmental latencies up to 3·7 msec (allowing 2·6 msec for the later arrival of group II volleys and 1·1 msec for two synaptic delays of the hypothetical disynaptic group II i.p.s.p.s). Consequently, stretches preceded by relaxation were used in the main part of this study.

To test for a possible contribution of pacinian and paciniform receptors to post-synaptic potentials attributed to Ia afferents, we compared the effects of long-duration (10–20 msec) increases and decreases in muscle length. As did Ellaway & Trott (1978', we assumed that these receptors would respond to both rapid stretch and relaxation of the muscle while spindle primaries would respond only to stretch. In all eight motoneurones tested, post-synaptic potentials caused by changes in muscle length were seen only following muscle stretch and not following muscle relaxation (Fig. 1*C*, *D*). Therefore, we found no evidence for a contribution of pacinian and paciniform receptors to the reported effects.

The comparison between the effects evoked by stretching triceps surae and plantaris muscles and the effects of electrical stimulation of their nerves was also used to eliminate effects of inadvertently activated afferents of other muscles. A great part of the hind-limb was denervated but some hip muscles and back muscles had their innervation intact. If the muscle spindles of these muscles were sufficiently sensitive, stretches of triceps surae might secondarily excite them; if they had any effects

on the motoneurones that were being tested these effects could erroneously be attributed to the triceps surae and plantaris afferents. A discrepancy between effects of stretches of triceps surae and plantaris and of electrical stimulation of their nerves would then be apparent. Such a discrepancy was observed only in the case of some motoneurones of sartorius and adductor, and all motoneurones of these species were consequently eliminated from the analysis. Additional control records (from eleven quadriceps, five anterior biceps–semimembranosus, six posterior biceps–semitendinosus, five flexor digitorum longus, five tibial and six peronei) did not show any p.s.p.s after section of the nerves to the stretched muscles, in agreement with control experiments by Fetz *et al.* (1979).

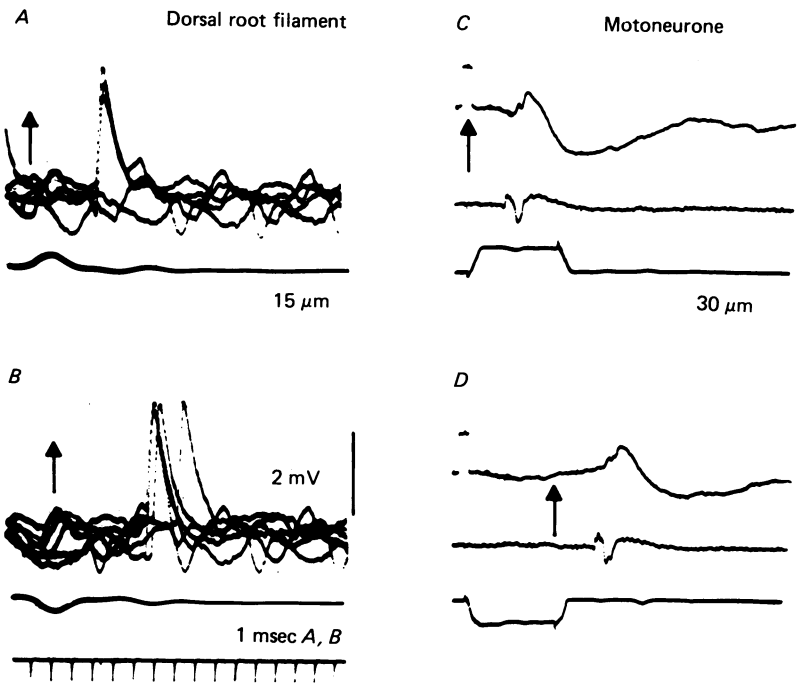

Fig. 1. Comparison of effects of changes in muscle length (bottom traces) in a sequence: stretch–relaxation (*A*, *C*) and relaxation–stretch (*B*, *D*). *A* and *B*, records of impulses in a Ia afferent of medial gastrocnemius (upper traces) following muscle stretches (arrows) which were followed (*A*) or preceded (*B*) by relaxation. Note that the latency of activation of the afferent increased but without increase in threshold. For twenty Ia afferents the ranges of minimal latencies for stretches as in *A* were 3·2–5·0 msec and for stretches as in *B* 4·2–6·5 msec. *C* and *D*, averaged records of e.p.s.p.s and i.p.s.p.s (uppermost traces) evoked in a flexor digitorum longus motoneurone. Note that they followed muscle stretch (Arrows) and that muscle relaxation (*D*) did not evoke any intracellular effects or afferent volleys (middle records). Calibration pulse: 200 μV, 1 msec.

Recording. Intracellular records from motoneurones were obtained mainly using potassium-citrate-filled micro-electrodes. Depending on the motoneurone species, the presence of i.p.s.p.s of Ia origin was tested for while the neurones were depolarized (usually by 20–50 nA) and using potassium citrate electrodes, or both depolarized and hyperpolarized (usually by 10–30 nA) and using potassium chloride electrodes. The latter procedure was applied primarily while recording from quadriceps and hamstring motoneurones and testing for the occurrence of Ia autogenetic inhibition combined with monosynaptic excitation, as described by Fetz *et al.* (1979).

Records of both single and average (64–256) responses were taken from each neurone that was investigated. The latencies of the responses were measured from the averaged records, while the

amplitudes were estimated from either averaged or single-sweep records. The minimal amplitudes of the i.p.s.p.s were $\geqslant 100\ \mu V$; in the case of the smallest i.p.s.p.s at least two or three series of records were taken in order to ensure that the responses were repeatable. With few exceptions, motoneurones selected for analysis had membrane potentials $\geqslant 50$ mV. The experiments were carried out only in preparations in which 30–35 μm stretches of triceps surae and plantaris evoked near-maximal e.p.s.p.s in their motoneurones (cf. Fig. 1 of Fetz *et al.* 1979) and in which i.p.s.p.s were seen following these e.p.s.p.s. Such tests ensured that the muscle spindle afferents were effectively activated and that the interneuronal systems responsible for the transneuronally mediated effects were functioning.

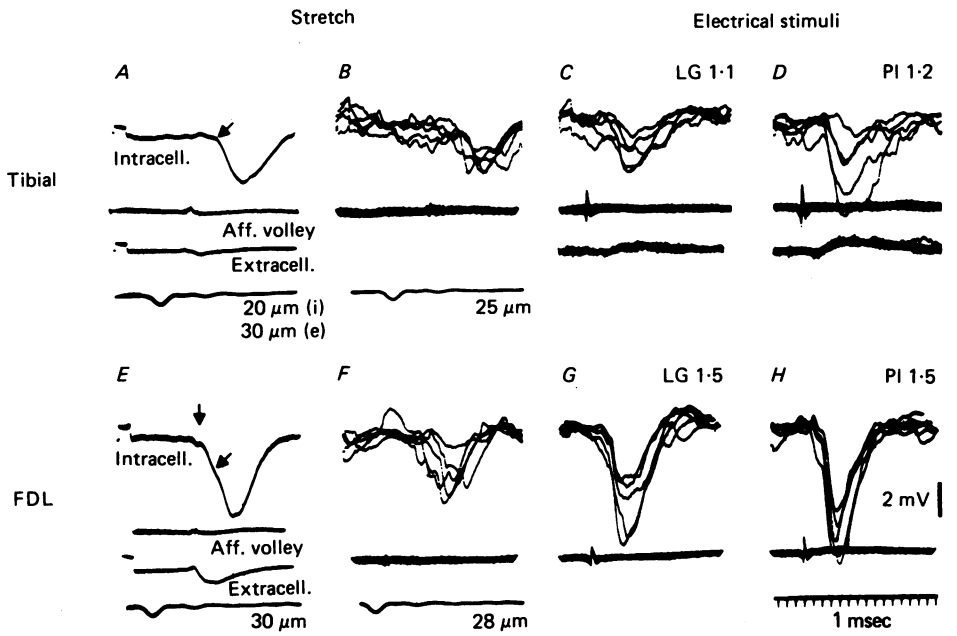

Fig. 2. Examples of stretch and electrically evoked i.p.s.p.s from group I afferents. The uppermost records in *A–D* and *E–H* are from motoneurones with axons in tibial and flexor digitorum longus (FDL) nerves, respectively. Other records are as indicated: of afferent volleys taken from the surface of the spinal cord, of extracellular field potentials just outside the recorded neurones and of changes in length of medial and lateral gastrocnemius, soleus and plantaris muscles (stretch indicated by upward deflexion). In *A* and *E* are sixty-four averaged records with 200 μV calibration pulses at the beginning of the micro-electrode records; the diagonal arrows indicate onset of the i.p.s.p.s, while the vertical arrow indicates a monosynaptic e.p.s.p. preceding the i.p.s.p. Electrical stimulation of lateral gatrocnemius (LG) and plantaris (PL) nerves was with stimulus intensities expressed in multiples of threshold for Ia afferents; stimulus intensities are indicated above records in both this and the following Figures.

RESULTS

Inhibition of various hip, knee, ankle and toe muscles from group Ia muscle spindle afferents of triceps surae and plantaris

According to previously reported observations, Ib afferents of extensors are more potent in evoking inhibition of motoneurones than those of flexors (see Tables 1–3 in Eccles *et al.* 1957*b*), and it was considered that if Ia non-reciprocal inhibition is evoked in parallel with inhibition of Ib origin it might likewise be most effectively

evoked from extensors. The occurrence of Ia non-reciprocal inhibition was therefore tested while activating Ia afferents of extensor muscles: medial and lateral gastrocnemii, soleus and plantaris. The selectivity of effects evoked by stimulation of these afferents by muscle stretches was ensured as described in Methods.

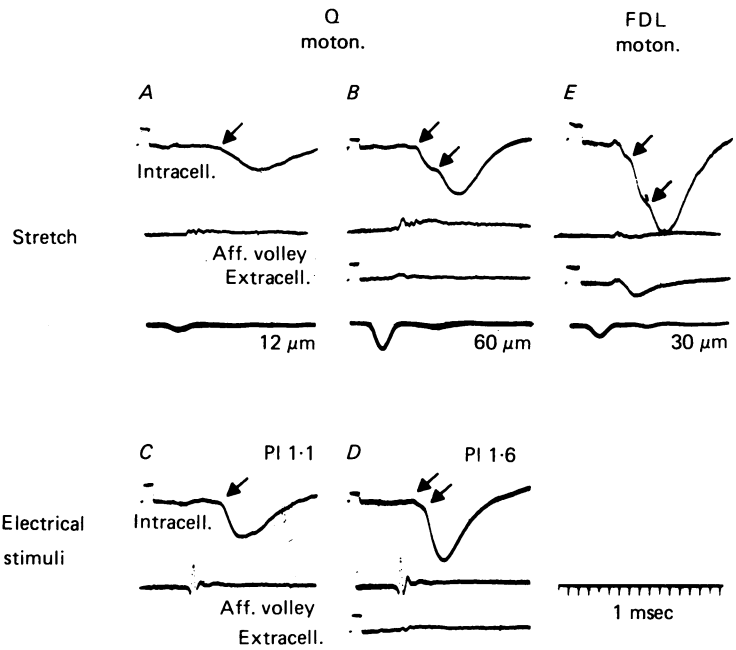

Fig. 3. Short and long-latency components of stretch and electrically evoked i.p.s.p.s. The uppermost records in *A–D* and *E* are from quadriceps (Q) and flexor digitorum or hallucis longus (FDL) motoneurones, respectively. Arrows indicate onset of i.p.s.p.s. Note shorter latency components appearing with stronger stimuli in *B* and *D;* whether they were of Ia or Ib origin could not be defined. Other abbreviations as in Fig. 1. Calibration pulse: 200 μV.

Distribution of Ia non-reciprocal i.p.s.p.s. Examples of stretch-evoked i.p.s.p.s recorded in motoneurones of intrinsic foot muscles and of flexor digitorum or hallucis longus are shown in Fig. 2*A*, *B* and *E;* those in quadriceps and anterior biceps–semimembranosus motoneurones are shown in Fig. 3*A* and *B* and Fig. 7 E, respectively. Note that the first components of intracellular records (top traces) in Fig. 2*A* and *E* and in Fig. 3*E* are field potentials (cf. extracellular records), and that the i.p.s.p.s (indicated by diagonal arrows) start somewhat later. Records in Fig. 2*B–D* and *F–H* show a similar time course of i.p.s.p.s evoked by muscle stretches and by electrical stimulation of group I afferents, which would include the I b afferents. The amplitudes of the stretch-evoked i.p.s.p.s (*B*, *F*) are, on the other hand, smaller. It will be noted that the stretches were submaximal for Ia afferents but could activate up to 80% of Ia afferents of the four muscles (Fetz *et al.* 1979). Nevertheless, stretch effects are smaller than the effects of electrical stimulation of individual muscle nerves. The amplitudes of stretch-evoked Ia i.p.s.p.s in non-depolarized motoneurones only rarely exceeded 1 mV.

When a distinct i.p.s.p. was evoked in any of the motoneurones with 30–35 μm

stretches, lowering the amplitude of the stretch to < 20 μm still allowed detection of the i.p.s.p. whenever this was attempted. Low stretch thresholds (10–20 μm) were established in ten quadriceps, six anterior biceps–semimembranosus, six flexor digitorum longus and one tibial motoneurone.

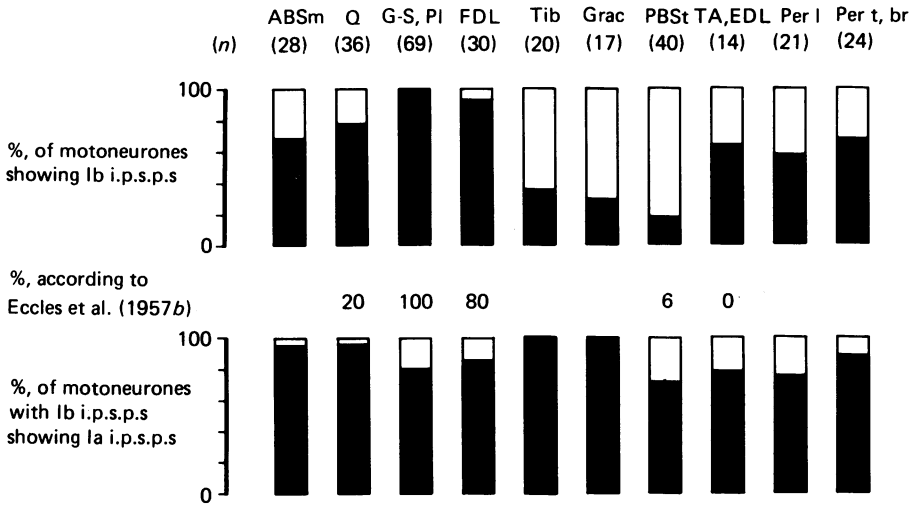

Fig. 4. Proportions of motoneurones inhibited by group Ib and group Ia afferents of gastrocnemius–soleus and plantaris. Upper diagrams: the proportions of motoneurones of different species with i.p.s.p.s evoked by Ib, or entire group I, afferents stimulated electrically; numbers of motoneurones are given in parentheses. The proportions found by Eccles *et al.* (1957*b*) are indicated below. Lower diagrams: the proportions of motoneurones with i.p.s.p.s evoked by muscle stretches ($\leqslant 35$ μm) among those motoneurones with Ib or entire group I i.p.s.p.s. Data for triceps surae and plantaris motoneurones are from Fetz *et al.* (1979). Pooled data for i.p.s.p.s evoked di- and trisynaptically in all the motoneurones except those of anterior tibial, extensor digitorum longus and peronei; only trisynaptically evoked i.p.s.p.s were considered for the latter. For further explanations see text and Table 1 in Jankowska *et al.* (1981*b*).

Abbreviations: ABSm, anterior biceps–semimembranosus; EDL, extensor digitorum longus; FDL, nerve branches to flexor digitorum and hallucis longus, popliteal and posterior tibial muscles; G–S, gastrocnemius and soleus; Grac, gracilis; PBSt, posterior biceps–semitendinosus; Per l, peroneus longus; Per t, br, peroneus tertius and brevis; Pl, plantaris; TA, anterior tibial; Tib, tibial nerve branches innervating intrinsic foot muscles; Q, quadriceps.

Fig. 4 shows the distribution of Ia non-reciprocal i.p.s.p.s, as compared with the distribution of i.p.s.p.s of predominantly Ib or entire group I origin, in various motoneurone species. The top diagrams give proportions of triceps surae and plantaris motoneurones with Ib i.p.s.p.s according to Fetz *et al.* (1979) and the proportions found in the present series of experiments for other motoneurone species. The bottom diagrams of Fig. 4 show the percentage of motoneurones with Ib i.p.s.p.s in which Ia i.p.s.p.s were evoked by muscle stretches. In four motoneurone species more than 90 % of these motoneurones were stretch-inhibited. In six other motoneurone species the proportion was 75–90 % (see also Table 1 in Jankowska *et al.* 1981*b*). The diagrams of Fig. 4 take into account any i.p.s.p.s of group I origin whether they were evoked di- or trisynaptically, except for motoneurones of pretibial flexors: anterior

tibial, extensor digitorum longus and peronei. For these motoneurones only the trisynaptically evoked i.p.s.p.s were counted (cf. pp. 402–405).

Latencies of Ia non-reciprocal i.p.s.p.s. The segmental latencies of the stretch-evoked Ia i.p.s.p.s measured in relation to the incoming volleys (Fig. 5*A*) were often longer than those of electrically evoked ones (Fig. 5*B*). They were distributed within similar ranges to those of triceps surae and plantaris motoneurones (*C; Fetz et al.* 1979). It will be noted that although the borders (arrows) between latencies corresponding to di- and trisynaptic coupling were less distinct, some i.p.s.p.s in all of the motoneurone species clearly fell among those with disynaptic latencies.

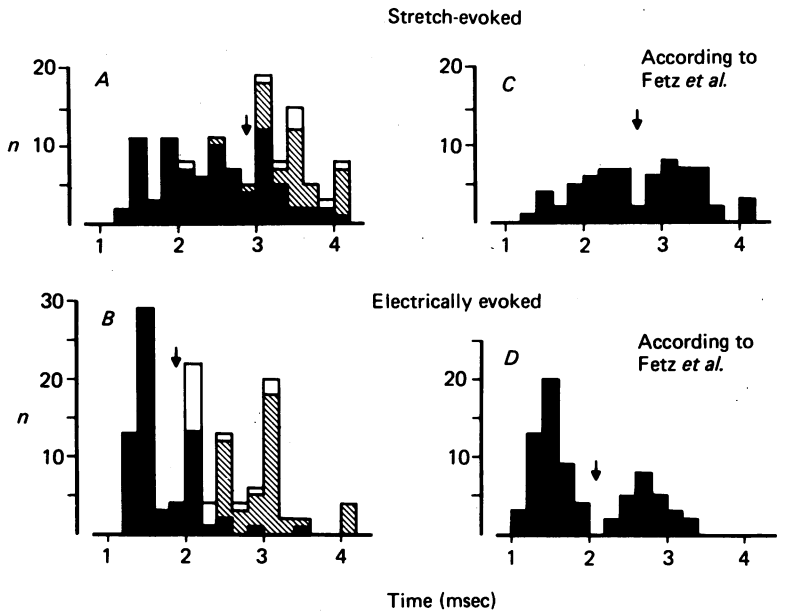

Fig. 5. Distribution of latencies of stretch-evoked and electrically evoked i.p.s.p.s recorded in motoneurones in the present study (*A* and *B*) as compared with latencies of stretch- and electrically evoked i.p.s.p.s in triceps surae and plantaris motoneurones as described by Fetz *et al.* (1979) (*C* and *D*). Arrows indicate likely borderlines between the latencies of di- and trisynaptically evoked i.p.s.p.s. Filled columns, earliest evoked i.p.s.p.s except for pretibial flexors; hatched columns, trisynaptic i.p.s.p.s evoked in pretibial flexors; open columns, trisynaptic i.p.s.p.s following disynaptic i.p.s.p.s in other motoneurone species.

As seen in records of muscle length (e.g. in the lowest traces in Fig. 2*A*, *B*, *E*, *F* or Fig. 3*A–F*), a single muscle stretch was often followed by small length oscillations. The amplitudes of these oscillations were usually below threshold for reactivating the muscle spindles and only exceptionally evoked a second afferent volley about 3–4 msec after the first one. Such a volley might give rise to some i.p.s.p.s with a further delay of 1·0 msec, but since we did not take into account any responses appearing with total segmental delays > 4·0 msec they would not be included in our material.

The records of Fig. 3 show that individual motoneurones may be inhibited via a disynaptic as well as a trisynaptic pathway of the non-reciprocal inhibition from group 1 afferents, the different components of the i.p.s.p.s being indicated by diagonal arrows. Ia afferents were found to contribute to both the earlier and the later components of the i.p.s.p.s (Fig. 3*E*), only to the earlier (not illustrated), or only to

the later as indicated by the records of Fig. 3*A–D*. The latter records show earlier components of the i.p.s.p.s (first arrows) appearing after a larger stretch and stronger electrical stimuli to group Ib afferents.

When it was possible clearly to measure the latency of more than one component of Ia i.p.s.p.s in a motoneurone both are included in the histograms of Fig. 5; the later i.p.s.p.s are indicated by the open columns. Trisynaptic i.p.s.p.s in pretibial flexors are indicated by hatching. The occurrence of late components is probably underestimated since only ones with distinct onset have been included in the histograms.

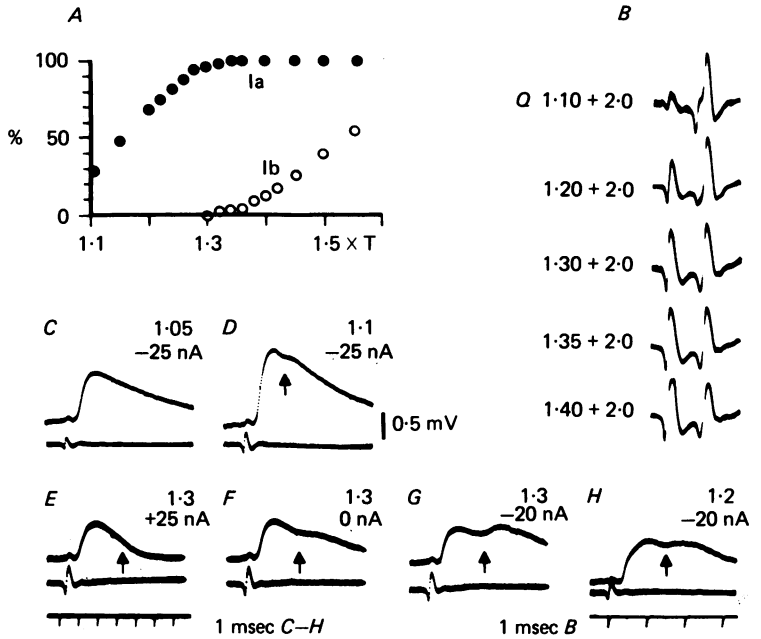

Fig. 6. Autogenetic inhibition of quadriceps motoneurones, evoked by electrical stimulation of Ia afferents. *A*, amplitudes of the first (●, Ia) and of the second (○, Ib) components of the incoming volleys as a function of the intensity of the stimuli (expressed in multiples of threshold, T) applied to the quadriceps nerve. *B*, examples of afferent volleys evoked by two stimuli at an interval shorter than the refractory period of the fibres being tested. The second stimulus was maximal for group I afferents (2 times threshold), while the intensity of the first one was increased in successive records. The first stimulus reached the maximum for Ia afferents at 1·3–1·35 and threshold for Ib afferents at 1·35, as judged by the amplitude of the volleys evoked by this stimulus and by the reduction in amplitude of the second component of the volleys evoked by the second stimulus. These amplitudes are plotted in *A*. Upper records in *C–D* and *E–H* are averaged post-synaptic potentials evoked in two quadriceps motoneurones in the same preparation. The records were taken during a hyperpolarization (−25 nA in *C* and *D*, −20 nA in *G* and *H*), depolarization (+25 nA in *E*) and without polarization (*F*) of the motoneurones. Arrows indicate onset of the i.p.s.p.s. Strengths of the electrical stimuli are indicated above, with corresponding records of afferent volleys below.

Contribution of group Ia muscle spindle afferents of quadriceps and hamstring to autogenetic inhibition of motoneurones

Electrical stimulation of quadriceps and hamstring Ia afferents was used to disclose any Ia autogenetic inhibition from these afferents. The motoneurones were penetrated with potassium-chloride filled electrodes and the responses to nerve stimulation were recorded while the motoneurones were hyperpolarized and depolarized. The occurrence of i.p.s.p.s following monosynaptic e.p.s.p.s was evidenced by the appearance of a hump during the decay phase of the e.p.s.p.s due to reversal of the i.p.s.p.s following chloride injection, as illustrated in Figs. 6*D*, *F–H* and 7*M* (see also Fetz *et al.* 1979). The onset of the i.p.s.p. was defined as the point of deviation of records taken during hyperpolarization and during depolarization; the depolarization hastened the decay of the e.p.s.p.s by enhancing the i.p.s.p.s (Figs. 6*E* and 7*K*).

The electrical stimuli were kept close to the threshold of the first (Ia) component of the incoming volleys and clearly below the threshold of the second (Ib) component of these volleys, which usually appeared with stimuli about 1·3–1·5 times nerve threshold (Fig. 6*A* and *B*).

I.p.s.p.s appearing with the first component of the incoming volleys have been found in eleven quadriceps motoneurones, as illustrated in Fig. 6 and in one anterior biceps–semimembranosus motoneurone, as illustrated in Fig. 7*A–E*, in addition to four posterior biceps–semitendinosus motoneurones previously analysed by E. Fetz, E. Jankowska & J. Lipski (see Fig. 5 in Jankowska, 1979). Autogenetic i.p.s.p.s were seen in about half of the motoneurones tested.

Inhibition of other motoneurone species from group Ia muscle spindle afferents of quadriceps and hamstring

The first reported observations of possible non-reciprocal inhibitory actions of group Ia afferents concerned effects of low-threshold group I quadriceps afferents on gastrocnemius (Eccles *et al.* 1957*b*) and tibial motoneurones (Lundberg *et al.* 1977*b*). We can now extend these observations to effects of low-threshold group I afferents of quadriceps on flexor digitorum longus motoneurones and of similarly low-threshold group I afferents of posterior biceps–semitendinosus on anterior biceps or semimembranosus (Fig. 7*F–G* and *K–M*), on flexor digitorum longus, and on gastrocnemius or soleus (Fig. 8*A*, *B*) motoneurones.

The latencies of non-reciprocal Ia i.p.s.p.s from quadriceps and hamstring were within the same ranges as those of the Ia i.p.s.p.s evoked by stretches of triceps surae and plantaris. Fig. 8*D* shows the distribution of latencies of the i.p.s.p.s described in this section as well as of the autogenetic i.p.s.p.s described in the preceding section.

Ia i.p.s.p.s evoked by two different neuronal systems in antagonist motoneurones

In a number of motoneurones of pretibial flexors, stretches of triceps surae and plantaris evoked both disynaptic and later-appearing i.p.s.p.s; the latencies of the later i.p.s.p.s were 2·6–4·0 msec, which would define them as trisynaptic. Such double i.p.s.p.s were seen in nine out of twenty-one peroneus longus, in ten out of twenty-three peroneus tertius or brevis and in eight out of fourteen extensor digitorum longus or anterior tibial motoneurones following 20–35 μm stretches. The sample included only motoneurones in which electrical stimuli ($\leqslant$ 1·5 times threshold) evoked late i.p.s.p.s.

The disynaptic stretch-evoked i.p.s.p.s were depressed by a preceding stimulation of the ventral roots (Fig. 9*B*, *I*). The depression was found in all anterior tibial and extensor digitorum longus motoneurones ($n = 11$), in all peroneus longus motoneurones ($n = 21$) and in six out of eight peroneus tertius and brevis motoneurones. Disynaptic i.p.s.p.s evoked by electrical stimulation of triceps surae, plantaris or flexor digitorum longus were likewise depressed in twelve out of fifteen peroneus tertius and brevis motoneurones. The effect was found independently of the occurrence of any recurrent i.p.s.p.s in the motoneurones tested and thus could be attributed to the inhibition of the interposed interneurones. According to previous studies (Hultborn, Jankowska & Lindström, 1971*b*; Jankowska & Roberts, 1972) these disynaptic i.p.s.p.s would be mediated by Ia-excited and recurrently inhibited laminae VII interneurones.

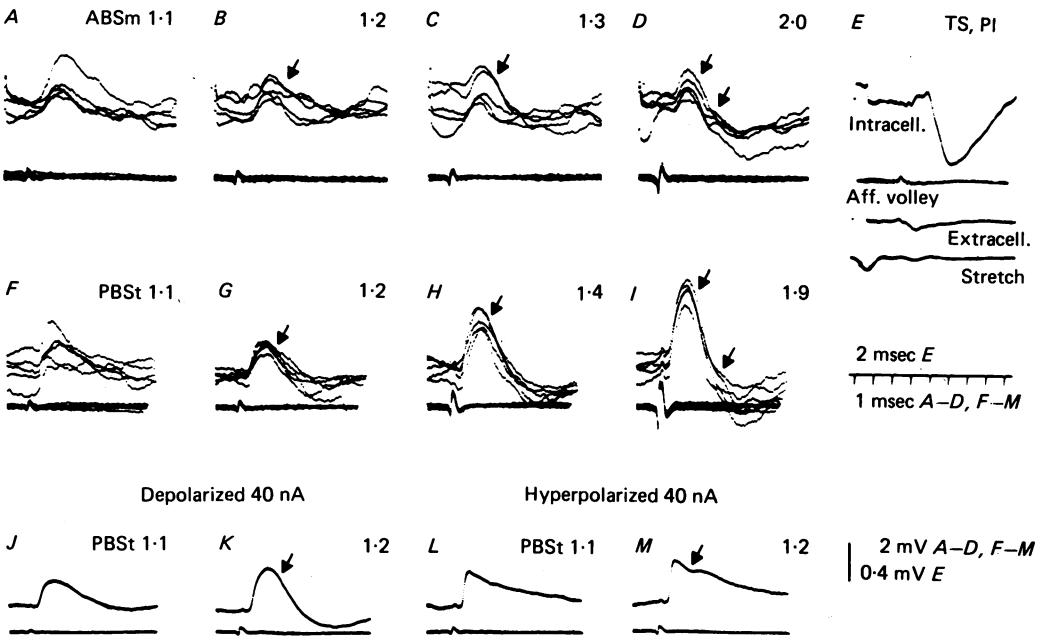

Fig. 7. Ia non-reciprocal i.p.s.p.s evoked by electrical stimulation of afferents in hamstring nerve and by muscle stretches in anterior biceps–semimembranosus motoneurones. *A–E* and *F–M*, intracellular records (upper traces) from two anterior biceps or semimembranosus (ABSm) motoneurones. *A*, monosynaptic e.p.s.p. *B*, responses at threshold for appearance of a disynaptic autogenetic i.p.s.p. (in two of the five traces, arrow). *C* and *D*, autogenetic i.p.s.p.s regularly following monosynaptic e.p.s.p.s; those in *C* were evoked probably mainly by Ia afferents and those in *D* by both Ia and Ib and possibly group II afferents. Note later component of the i.p.s.p.s indicated by the second arrow. *E*, i.p.s.p. evoked by stretch of triceps surae and plantaris (TS, Pl). Note small e.p.s.p. preceding the i.p.s.p. (cf. Jankowska *et al.* 1981*b*). *F*, *J*. and *L*, monosynaptic e.p.s.p.s evoked from posterior biceps–semitendinosus (PBSt) in the second anterior biceps–semimembranosus motoneurone. *G–I*, *K* and *M*, disynaptic i.p.s.p.s (arrows) following monosynaptic e.p.s.p.s, enhanced after depolarization and reversed after hyperpolarization. *H* and *I*, responses evoked just below threshold for Ib afferents and by stimuli maximal for group I afferents, respectively. Note a second component of the incoming volley in *I* but not in *H* and the second component of i.p.s.p.s (second arrow, *I*) attributable to oligosynaptic Ib or group II actions.

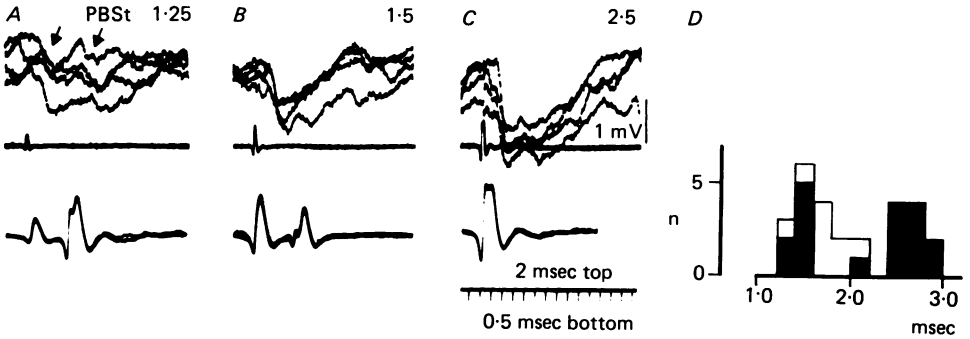

Fig. 8. Ia non-reciprocal i.p.s.p.s evoked from posterior biceps–semitendinosus in a gastrocnemius motoneurone. Top records in *A–C* are i.p.s.p.s evoked with increasing stimulus intensities; they appeared below the maximum for the first component and below the threshold, which was at 1·7, for the second component of the incoming volleys (middle traces). Bottom traces show incoming volleys evoked by two stimuli; the first is that used to evoke the i.p.s.p.s and the second is maximal for the entire group I afferents. Stimuli that were 1·25 or 1·5 times threshold made only a fraction of group Ia afferents contributing to the first component of the maximal group I volleys (cf. *A*, *B* with *C*) refractory. Note that bottom records were taken with a higher amplification and a faster time base. *D*, histogram of latencies of Ia i.p.s.p.s ($n = 27$) evoked from quadriceps (filled columns) and hamstring (open columns) afferents in homonymous and other motoneurone species.

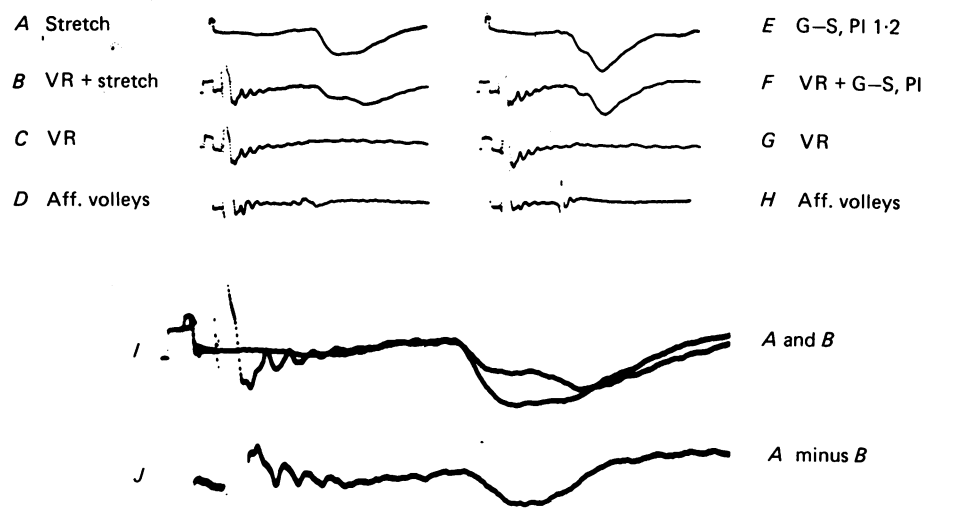

Fig. 9. Reciprocal and non-reciprocal Ia i.p.s.p.s evoked in a peroneus longus motoneurone. In *A–C* are averaged intracellular records of effects of 25 μm stretch of triceps surae and plantaris (*A*), of stimulation of L7–S1 ventral roots (VR) (*C*), and the two together (*B*). In *E–G* is a similar series of records showing the effects of eletrical stimulation of gastrocnemius–soleus and plantaris (G–S, Pl) nerves which was above threshold for Ib afferents and of stimulation of the ventral roots, alone and together. In *D* and *H* are afferent volleys recorded from the surface of the spinal cord. Below on a larger scale are the records of *A* and *B* superimposed, and the computer-derived difference between them. Note the recurrent depression of the first but not of the second component of the i.p.s.p.s elicited by stretch. Note also a similar reduction of the earlier but not the later component of the i.p.s.p.s which were evoked by electrical stimuli (*G*) and the larger amplitude of the second component of the i.p.s.p.s evoked by nerve stimulation. Calibration: 200 μV, 1 msec.

The later i.p.s.p.s were practically unaffected by the ventral root stimulation, as illustrated in Fig. 9*B*, *F* and *I*. When their peak amplitude looked to be somewhat decreased, the decrease would be more apparent than genuine; it would be due to the fact that the later i.p.s.p.s were superimposed on the disynaptic i.p.s.p.s and the reduction of these i.p.s.p.s would tend to make the trisynaptic i.p.s.p.s appear smaller. The difference between the conditioned and the unconditioned responses (Fig. 9*J*) shows the time course of the disynaptic i.p.s.p.s. The amplitude of the later i.p.s.p.s was increased considerably when the Ib afferents were excited by either increasing the amplitude of muscle stretches or by electrical stimulation of the nerves (cf. Fig. 9*A* and *E*). Lack of effect of ventral root stimulation and the facilitation by Ib input show that these late i.p.s.p.s are due to a neuronal system different from that mediating the disynaptic i.p.s.p.s. They are attributed to the same interneuronal system which would be responsible for the non-reciprocal Ia i.p.s.p.s evoked in other motoneurone species.

When ventral root stimulation evoked some recurrent i.p.s.p.s in the pretibial flexor motoneurones the lack of depression of the late components supplied further support for the conclusion that the disynaptic i.p.s.p.s were depressed as a consequence of inhibition of the interposed interneurones and not secondarily to changes in the motoneurone membrane conductance. The effects of ventral root stimulation were tested on the late components with the same conditioning–testing intervals (in relation to the onset of the i.p.s.p.s) as for the disynaptic i.p.s.p.s.

DISCUSSION

The results of this study lead to a generalization of the observations of Fetz *et al.* (1979) and to the conclusion that group Ia muscle spindle afferents from a given muscle may contribute to inhibition of a great variety of motoneurone species.

The wide distribution of the Ia inhibitory effects, and their appearance in motoneurones of homonymous and synergistic as well as antagonistic muscles, makes the distinction between Ia reciprocal and non-reciprocal inhibition rather awkward. We propose, nevertheless, to retain this terminology using the term '*Ia reciprocal inhibition*' to denote inhibition from antagonists evoked via Rexed's lamina VII interneurones (Hultborn *et al.* 1971*b*; Jankowska & Roberts, 1972), the only interneurones which are under the control of Renshaw cells. The term '*Ia non-reciprocal inhibition*' could then be applied to inhibition evoked from Ia afferents via other neuronal pathways. If the conclusions from previous experiments (Hultborn *et al.* 1971*b*; Jankowska & McCrea, 1980) find final experimental confirmation, such non-reciprocal inhibition would be *mediated by Rexed's laminae V–VI interneurones*. We will also propose the use of the term 'Ia-like-Ib inhibition' as a synonym for 'Ia non-reciprocal inhibition' when stressing that it is evoked in parallel from Ia and Ib afferents.

In evaluating the present evidence for distribution of Ia inhibitory effects among the same motoneurone species in which inhibition is evoked from Ib afferents, we have used similar criteria for Ia effects to Fetz *et al.* (1979). As previously, we have considered stretches of 10–35 μm as being subthreshold for group Ib tendon organ afferents, since none of those tested under the same conditions were excited by stretches < 45 μm. However, in view of the observation of Ellaway & Trott (1978, and personal communication), who found two of their Ib afferents excited by 25 and 30 μm stretches, we verified that inhibition of at least some motoneurones is evoked

by stretches of 10–20 μm as well as of 30–35 μm. The contribution of group II muscle spindle afferents was considered possible only for i.p.s.p.s with segmental latencies > 3·7 msec (see Methods) and that of pacinian and paciniform corpuscles was estimated as negligible.

The more distinct contribution of Ia afferents of quadriceps and hamstring to the synergistic and autogenetic inhibition seen in this study than that seen in previous ones (that of ankle and toe extensor afferents not having been analysed before) may be related to two factors: the use of chloralose instead of pentobarbital anaesthesia and the potentiation of synaptic transmission by 4-aminopyridine (Jankowska *et al.* 1977). The laminae V–VI interneurones (Jankowska, Johannisson & Lipski, 1981*a*) expected to mediate common actions of Ia and Ib afferents (Jankowska & McCrea, 1980) are not very effectively excited by Ia afferents alone. They are not in fact very effectively excited by the Ib afferents either, and their activation often requires facilitation by descending or other segmental actions (Hongo, Jankowska & Lundberg, 1969; Lundberg *et al.* 1977*b*; Lundberg, Malmgren & Schomburg, 1978). Lack of the latter, however, might be compensated by an increase in the effectiveness of group I volleys by 4-aminopyridine. It should be stressed in this context that the differences between 4-aminopyridine-treated and non-treated preparations were only quantitative. All the types of response found in the former were also seen in the latter.

The observations of Fetz *et al.* (1979) demonstrated that Ia non-reciprocal inhibition is evoked not only in homonymous motoneurones but also in motoneurones of close synergists. The present study extends these observations and shows that non-reciprocal inhibition from Ia afferents of triceps surae and plantaris is evoked in all motoneurone species in which inhibition from group I afferents of the same muscles has been found. As shown in Fig. 4, the Ia-like-Ib inhibition occurs both in motoneurones of flexor and extensor muscles, and in motoneurones of muscles which act at all of the hind-limb joints. Such inhibition together with Ib inhibition could thus be useful during movements involving the entire limb.

It has been previously considered that Renshaw cells might to some extent contribute to Ia autogenetic inhibition and to Ia inhibition between close synergists (Fetz *et al.* 1979). These cells may be activated secondarily to the monosynaptic excitation of motoneurones and add to their trisynaptic, though not disynaptic, Ia inhibition. Such a contribution could also be expected in some of the motoneurone species now analysed. In other species (quadriceps, gracilis, extensor digitorum longus and anterior tibial) it would be unlikely, since recurrent inhibition from gastrocnemius, soleus and plantaris appears to be missing in them (H. Hultborn, R. Katz & R. Mackel, unpublished data). In any case, Renshaw cells would equally contribute to the effects of electrical stimulation of the nerves; thus they would not endanger the comparison between the distribution of inhibitory actions of the whole group I afferents (previously attributed to Ib afferents) and of Ia afferents.

Comparing the distribution of i.p.s.p.s shown here with the distribution of Ib i.p.s.p.s reported previously (Eccles *et al.* 1957, see Fig. 4), we similarly find that the inhibitory effects from the ankle extensors are stronger to other extensors than to flexors. The occurrence of Ib inhibition of antagonists has previously been seen only occasionally (Eccles *et al.* 1957*b*) and our observations extend the pattern of its distribution from triceps surae and plantaris to motoneurones of the pretibial flexors. As in other motoneurone species, the Ib and the Ia-like-Ib inhibition of these motoneurones has been found to appear in parallel.

Since the pretibial flexors receive Ia reciprocal inhibition from the ankle and toe extensors, the demonstration of Ia-like-Ib inhibition in the same motoneurones implies that volleys in Ia afferents may inhibit them via two neuronal pathways: one under control of Renshaw cells and the other linked with Ib actions. Differential control of transmission via these pathways (Hultborn, 1976; Lundberg *et al.* 1977*b*, 1978) would allow either a summation of their effects or their selective use.

Using lack of recurrent depression as the criterion of Ia-like-Ib inhibition we could not decide whether such inhibition was evoked only trisynaptically or whether it contributed to the disynaptic i.p.s.p.s as well. As previously described (Hultborn, Jankowska & Lindström, 1971*a*), the Ia disynaptic i.p.s.p.s evoked by near-threshold stimuli are very effectively depressed by a preceding conditioning stimulation of ventral roots, while i.p.s.p.s evoked by stronger stimuli are only reduced to about one half. The weaker recurrent effect on the maximal Ia i.p.s.p.s has been explained by less effective inhibition of strongly excited interneurones (Hultborn *et al.* 1971*b*). In view of the present observations it might be also considered that the non-depressed components of the disynaptic Ia i.p.s.p.s could in part be due to interneurones mediating the Ia-like-Ib inhibition.

Although the main observations of this study have been on effects of Ia afferents of ankle and toe extensors, similar effects were seen upon stimulation of Ia afferents in hamstring and quadriceps nerves. We would thus generalize the conclusion that Ia muscle spindle afferents may evoke both reciprocal and non-reciprocal inhibition to afferents of other muscles as well.

In view of great similarities in the input to α- and γ- motoneurones, except for monosynaptic connexions from Ia afferents, it is of interest to note that the Ia actions found in the present study have likewise been seen in γ-motoneurones (Ellaway & Trott, 1978). Even if excitation of γ-motoneurones has been reported as a predominant effect of Ia afferents excited by muscle vibration or brief muscle stretches (Trott, 1976; Fromm & Noth, 1976; Ellaway & Trott, 1978) and inhibition as a main effect of Ib afferents excited by muscle contractions (Ellaway & Trott, 1979), it is clear that both excitation and inhibition may be evoked from each of these two subgroups of group I afferents.

The Ia autogenetic inhibition of γ-motoneurones following small muscle stretches below threshold for Ib afferents (Ellaway & Trott, 1978) appeared with similar minimal segmental latencies (1·7 msec) to those in α-motoneurones; a small difference might be accounted for by the use of extracellular instead of intracellular records from γ-motoneurones. These minimal latencies would be clearly within ranges for stretch-evoked disynaptic i.p.s.p.s (see Fig. 4 in this paper and Fig. 6 of Fetz *et al.* 1979). Therefore, it would be more likely that the early autogenetic inhibition of γ-motoneurones is evoked by the same interneurones which mediate the non-reciprocal Ia inhibition of α-motoneurones than via Renshaw cells (Fromm & Noth, 1976). Renshaw cells activated secondarily to excitation of α-motoneurones could contribute only to later responses. Longer latencies (5–14 msec) of Ia excitatory actions on γ-than on α-motoneurones make the comparison between them more difficult and leave open the question as to whether they are mediated by the same neuronal systems.

The functional consequences of the similar reflex actions of Ia muscle spindle afferents on α- and γ-motoneurones and of the parallelism between actions of these afferents and of Ib Golgi tendon organ afferents will be discussed in a forthcoming paper (E. Jankowska & D. McCrea, in preparation), together with the evidence of shared reflex pathways from Ia and Ib afferents.

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