Journal of Physiology (1992), **455**, *pp.* 205–217
With 6 figures
Printed in Great Britain

CROSSED ACTIONS ON GROUP II-ACTIVATED INTERNEURONES IN THE MIDLUMBAR SEGMENTS OF THE CAT SPINAL CORD

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(*Received 17 September 1991*)

SUMMARY

1. Evidence has been sought for crossed actions on midlumbar propriospinal neurones activated by ipsilateral group II muscle afferents, with particular emphasis on those neurones with projections to the ipsilateral hindlimb motor nuclei.

2. A large majority of group II-activated midlumbar neurones were influenced by stimulation of contralateral group II afferents. The most frequent and most powerful actions were from those nerves which most effectively influenced ipsilateral midlumbar neurones. Crossed actions from group I afferents were rare.

3. In the great majority of neurones the pattern of actions was similar from both limbs, the neurones being bilaterally excited, bilaterally inhibited or had both EPSPs and IPSPs from both sides.

4. The latencies of crossed actions suggest that the earliest crossed EPSPs from group II afferents were evoked disynaptically (i.e. via a single commissural neurone) and that the crossed IPSPs were evoked trisynaptically.

5. The pattern of crossed actions suggests a strong bilateral interaction between midlumbar neurones. The possible role of these neurones in postural control and the production of co-ordinated movements of the hindlimbs is discussed.

INTRODUCTION

A system of premotor interneurones which are powerfully activated by group II afferents and which make synaptic contact with hindlimb motoneurones has been described in the midlumbar segments of the spinal cord (Edgley & Jankowska, 1987*b*). The description of these neurones has renewed interest in the central processing of information from group II afferents: these afferents are known to have monosynaptic connections with homonymous motoneurones (see e.g. Munson, Sypert, Zengel, Lofton & Fleshman, 1982) and also produce large EPSPs or IPSPs in hindlimb motoneurones via interneuronal pathways (Lundberg, Malmgren & Schomburg, 1987*a*). Individual group II afferents have widespread axonal arborizations in the caudal segments of the lumbosacral enlargement (Fyffe, 1979; Hoheisel, Lehmann-Willenbrock & Mense, 1989), but few interneurones which might mediate the indirect actions on motoneurones have been described (Fu & Schomburg,

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1974; Lundberg, Malmgren & Schomburg; 1987*b*; Harrison & Riddell, 1989). The midlumbar segments have been found to contain many neurones influenced by group II afferents, including ascending tract and locally projecting neurones, as well as last-order propriospinal neurones (Edgley & Jankowska, 1987*a*, *b*; Bras, Cavallari, Jankowska & Kubin, 1989). These neurones may be influenced by group II afferents from many muscle groups, but the most potent and most frequent actions are from quadriceps, sartorius, tibialis anterior and extensor digitorum longus, and flexor digitorum and hallucis longus (Edgley & Jankowska, 1987*a*, *b*).

On the basis of their inputs, it has been proposed that midlumbar propriospinal neurones might play a role in determining the timing of the switch from the stance to swing phase of the step cycle during locomotion (Edgley & Jankowska, 1987*b*). This switch occurs when the hindlimb becomes fully extended and is partly mediated by muscle stretch receptors (see Grillner, 1981; Edgley & Jankowska, 1987*b*). Other evidence also indicates that group II-activated midlumbar neurones might play a role in locomotion (Edgley, Jankowska & Shefchyk, 1988). Furthermore some group II-activated midlumbar neurones are phasically active during fictive locomotion evoked by brainstem stimulation in decerebrate cats (Shefchyk, McCrea, Kriellars, Fortier & Jordan, 1990), implying that they are involved in generating the locomotor drive potentials in hindlimb motoneurones in this preparation. One prediction arising from this hypothesis, which has not been tested, is that the midlumbar neurones should be influenced by receptors signalling the position of the contralateral hindlimb (see Grillner & Rossignol, 1978; Edgley & Jankowska, 1987*b*). In the present experiments we have sought evidence for an influence on midlumbar propriospinal neurones with group II input from contralateral afferents. Some of these results have been presented in an abstract (Bajwa, Edgley & Harrison, 1991).

METHODS

The experiments were performed on five adult cats (2·5–4·5 kg). Anaesthesia was induced with ketamine (28–30 mg/kg, I.M.) and maintained with halothane (1·5–2·5%, in a 40%:60% oxygen and nitrous oxide mixture) during surgery. A tracheal cannula was introduced to allow artificial ventilation. In-dwelling cannulae were inserted into the external jugular or cephalic veins for fluid administration and a carotid artery was cannulated to permit the monitoring of blood pressure. Nerves of both hindlimbs were dissected free for stimulation. In the left (ipsilateral) limb these included: the nerves to the hamstring muscles (biceps femoris, semitendinosus and semimembranosus; iHAM), the nerves to quadriceps (iQ), either the common tibial nerve (iTIB) or the nerve to gastrocnemius and soleus (iGS), and the common peroneal nerve (iCP) or the nerve to tibialis anterior and extensor digitorum longus (iTA–EDL). In the right (contralateral) limb the nerves to quadriceps (cQ), the hamstring muscles (cHAM), the tibialis anterior and extensor digitorum longus (cTA–EDL), the gastrocnemius and soleus (cGS), the two heads of flexor digitorum longus (including branches to popliteus, tibialis posterior and the interosseous nerve; cFDHL) were dissected free. In some experiments the right sartorius nerve (cSART) was also stimulated. Spinal cord segments from L3 to the cauda equina were exposed by laminectomy. Paraffin oil pools made from skin flaps were used to prevent drying of the nerves and spinal cord and to maintain temperature at 37 °C–38 °C.

When surgery was complete halothane anaesthesia was discontinued and α-chloralose given intravenously to maintain deep anaesthesia (60–80 mg, initially, supplemented with 10–15 mg doses as necessary to maintain deep anaesthesia). During recording the animals were paralysed with gallamine triethiodide (16 mg initial dose, subsequently 8 mg single doses were given when paralysis waned) and artificially ventilated. A bilateral pneumothorax was made to prevent movements of the thorax. During paralysis deep anaesthesia was ensured by checking that blood

pressure and heart rate were stable and did not alter in response to noxious stimulation. The pupils were also checked to ensure complete constriction and that there was no dilation in response to noxious stimulation. These tests were made at regular intervals.

Intracellular recordings were made using glass microelectrodes filled with 1 M-potassium citrate (3–15 MΩ resistance). Neurones with projections to the motor nuclei in the L7 or S1 segments were identified by antidromic activation following stimulation (0·2 ms square pulses, 0–100 μA) via a varnish-insulated tungsten microelectrode. This electrode was placed in the motor nuclei using the antidromic field potentials evoked from the iGS and iHAM nerves for guidance. Nerves were stimulated with 0·2 ms square pulses, delivered singly or in trains of 2–3 stimuli, 2–3 ms apart. These stimuli were delivered at a frequency of 0·75–1·2 Hz. To allow central latencies and nerve thresholds to be determined in relation to the group I volley, recordings from the cord dorsum were made via a silver ball electrode placed at the L6–L7 junction. Signals were recorded on digital audio tape for off-line analysis. At the end of each experiment the animal was killed with a large dose of barbiturate.

RESULTS

This report is based on intracellular recordings obtained from 102 midlumbar interneurones all of which were powerfully influenced by group II afferents in ipsilateral nerves (Figs 1–3). The neurones were distributed widely in the spinal grey matter; their depths (determined from the microelectrode drive unit) ranged from 1·6 to 4·3 mm from the surface of the spinal cord. As has been described previously (Edgley & Jankowska, 1987*a*), neurones in the dorsal horn (less than 2·0 mm deep) had input from group II, but not group I, afferents, and were not antidromically activated from the motor nuclei whereas more deeply located neurones generally had convergent group I and group II inputs and some were antidromically activated from the caudal motor nuclei. Seven of the cells were located in the superficial area and were activated by group II afferents alone; five of these were influenced by contralateral afferents.

Antidromic activation from the hindlimb motor nuclei in the L7 segment was demonstrated for forty-four of the remaining ninety-five neurones. Some of the neurones which were located in the intermediate zone but were not antidromically identified from the motor nuclei had similar inputs from group I and group II afferents to those which were antidromically activated. It is likely that some of these may have had projections to the motor nuclei, since a single stimulating electrode was placed in the motor nuclei.

In relation to ipsilateral inputs, most neurones (70/102) were monosynaptically excited by group II afferents, in some cases (26/70) in combination with disynaptic IPSPs. (Fig. 5*A* and *B*). Other neurones (32/102) were not excited but were inhibited by group II afferents (Fig. 5*C*). In many cases earlier EPSPs and/or IPSPs from group I afferents were also present. These patterns of ipsilateral input have been described previously in midlumbar neurones (Edgley & Jankowska, 1987*a*).

Examples of intracellular recordings from interneurones are shown in Figs 1–3. Actions from contralateral group I afferents were rare; small EPSPs or IPSPs were seen in 4/102 neurones. In contrast, crossed actions were evoked in all but eighteen of the neurones with stimuli sufficient to activate group II afferents (2–5T where T = threshold for the most excitable fibres). Thus, 84/102 (82%) midlumbar neurones with input from ipsilateral group II afferents were also influenced by contralateral group II afferents. These included 5/7 dorsal horn neurones and 79/95

intermediate zone neurones. In sixty-three of these neurones crossed group II actions were evoked by single stimuli (e.g. Figs 2 and 3), indicating secure transmission through the intercalated neurones. The crossed actions in the remaining twenty-one neurones only appeared to a second or third stimulus.

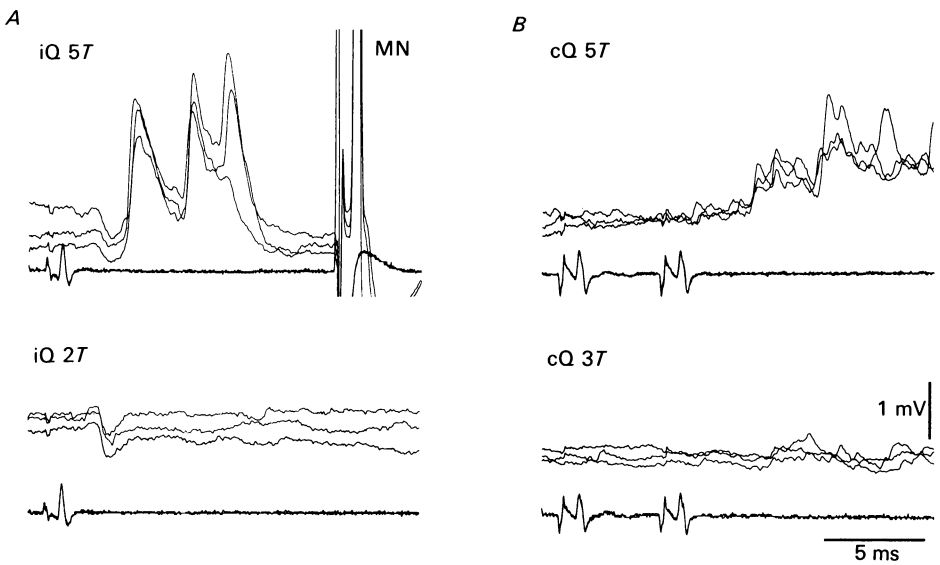

Fig. 1. Ipsilateral and contralateral actions of quadriceps group II afferents in a midlumbar propriospinal neurone. In each set of traces the upper records are intracellular potentials and the lower traces are recordings from the cord dorsum. *A* shows actions evoked by the ipsilateral quadriceps nerve (iQ). Stimulation at twice the threshold for the most excitable fibres (2 *T*) which activates most group I but few group II afferents evokes a small IPSP. Stimulation at 5 *T* which activates most group II afferents evokes in addition large EPSPs. *B* shows that longer-latency EPSPs are evoked by group II afferents in the contralateral quadriceps nerve (cQ) since they are elicited by stimulation at 5 *T*, but not at 3 *T*. Antidromic activation of the neurone from the motor nuclei (MN) is shown in *A* (stimulus intensity 70 μA).

Pattern of crossed group II actions

The contralateral nerves were not equally effective in producing PSPs in midlumbar neurones: the nerves which had ipsilateral group II actions on midlumbar neurones also had actions on contralateral midlumbar neurones. Most effective was the quadriceps nerve, but SART, TA–EDL and FDHL nerves were also effective (Figs 2 and 3). Group II afferents from gastrocnemius and soleus were either ineffective or evoked small crossed PSPs. For crossed actions to be evoked the intervening neurones would have to be discharged, and this may account for the absence of crossed effects in some neurones. In order to minimize this possibility short trains of two or three stimuli (0·2 ms pulses at 150–300/s) were tested routinely. Figure 4 shows, in histogram form, the proportions of midlumbar neurones in which crossed group II actions were evoked from different nerves. For comparison the proportions of neurones with inputs from group II afferents of ipsilateral nerves are

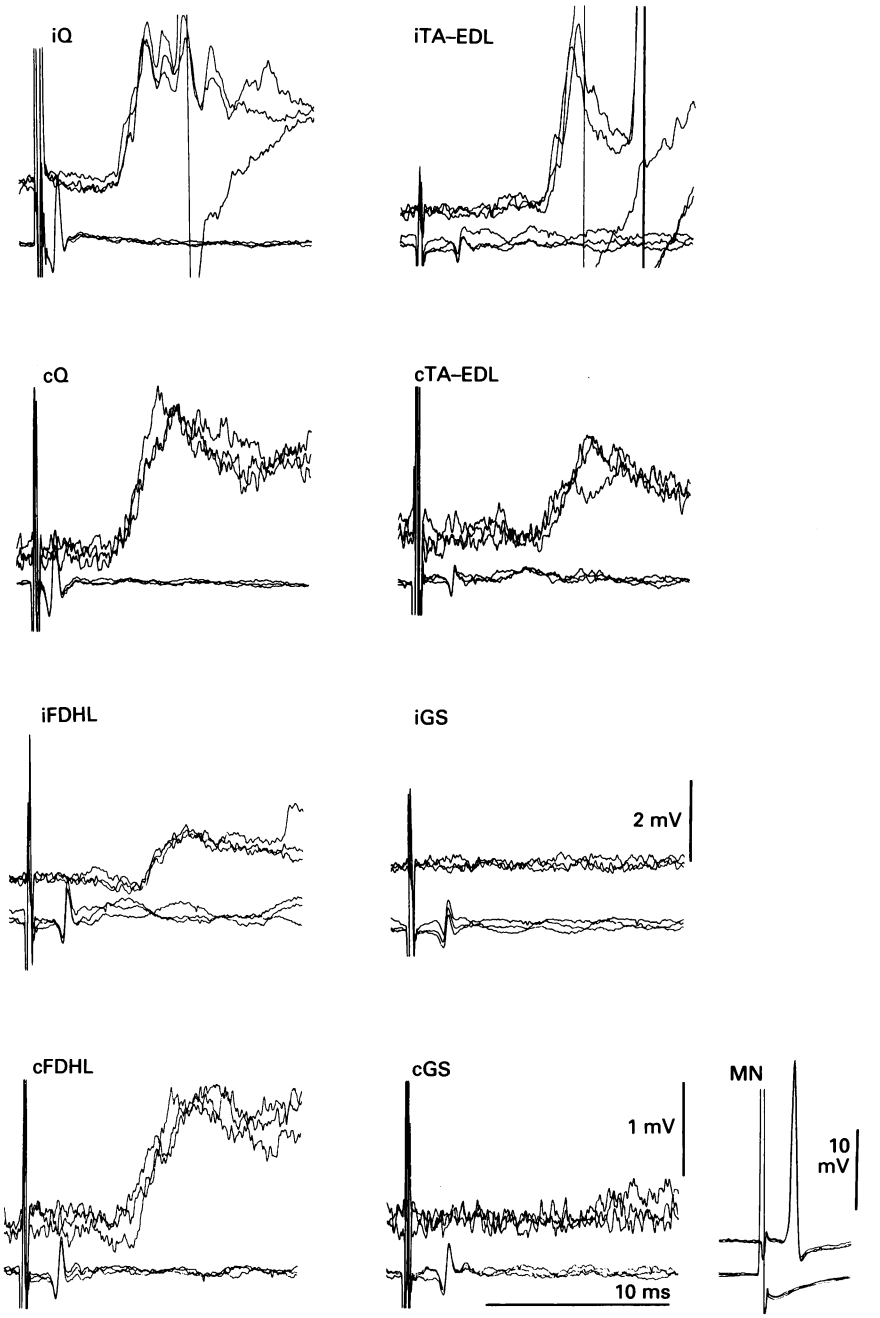

Fig. 2. Convergence of inputs from different nerves in a midlumbar propriospinal neurone. The effects of ipsilateral and contralateral nerves are shown. Stimulus intensity is $5T$ in all records. In each case where an ipsilateral nerve evokes EPSPs its contralateral counterpart also does so. No effects were evoked from either of the gastrocnemius-soleus nerves. Antidromic activation from the motor nuclei (stimulus intensity 10 μA) is shown at the bottom right (MN). Note the different voltage calibrations for effects evoked from ipsilateral and contralateral nerves.

included. As is evident from Fig. 4, there is a close correspondence between the frequency of actions of contralateral nerves and their ipsilateral counterparts.

Crossed actions evoked from group II afferents included EPSPs, IPSPs and a combination of both (Fig. 5). In addition to the similarity in frequency of actions of specific ipsilateral and contralateral nerves, a notable feature of the crossed actions

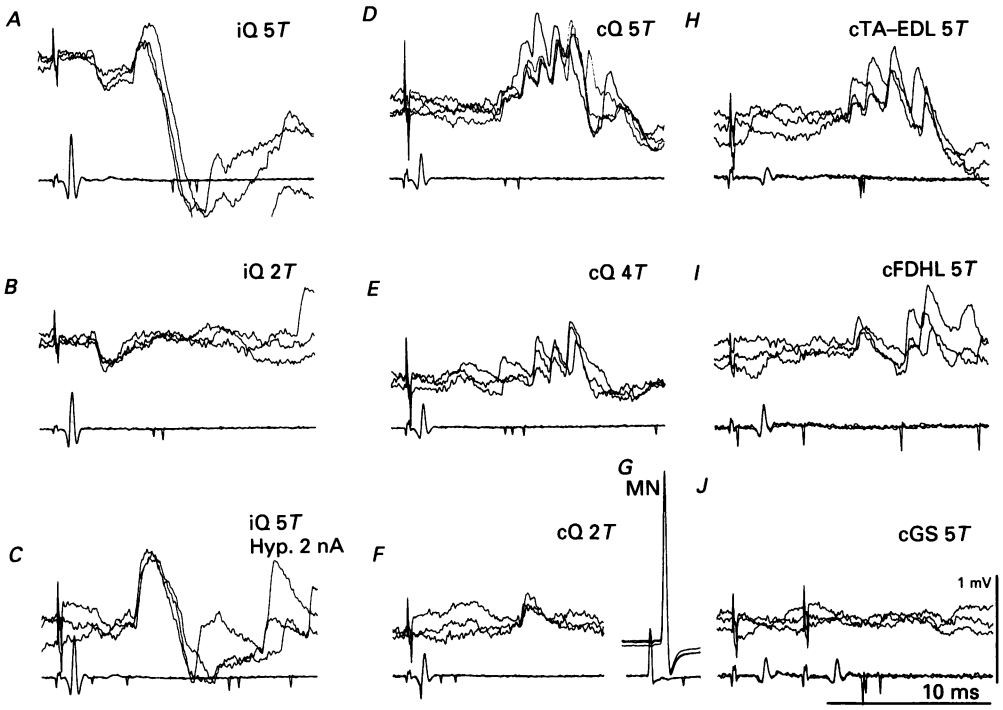

Fig. 3. Intracellular recordings from a midlumbar propriospinal neurone with both EPSPs and IPSPs from group II afferents. Responses to stimulation of ipsilateral group II afferents are shown in *A–C*. Stimulation of the iQ nerve at 5*T* (*A*) evokes an early IPSP which can be attributed to group I afferents (since it is also evoked by stimuli at 2*T*; *B*), followed by EPSPs and IPSPs evoked by group II afferents. The EPSP is made more obvious following hyperpolarization of the neurone (Hyp.) by 3 nA (*C*). Group II afferents from the cQ nerve evoke clear EPSPs in response to a single stimulus (*D–F*) as do group II afferents of cTA–EDL and cFDHL (*H* and *I* respectively). The cGS nerve is ineffective even with double stimuli (*J*). Antidromic activation of the neurone from the motor nuclei (MN, stimulus intensity 20 μA) is shown in *G*. The sharply falling edges of the EPSPs in *D* and *H* suggest that group II afferents from cQ and cTA–EDL also evoked IPSPs.

was that the pattern of crossed actions generally paralleled the pattern of actions from ipsilateral group II afferents. Thus of seventy neurones in which EPSPs were evoked from the ipsilateral quadriceps nerve (sometimes in combination with IPSPs), fifty-six (80%) also had EPSPs from the contralateral quadriceps nerve (e.g. Figs 1, 2, 3 and 5*A*). In thirty-two neurones the ipsilateral quadriceps nerve evoked IPSPs alone and twenty-four of these (75%) also had crossed IPSPs (e.g. Fig. 5*C*). The ipsilateral nerve evoked an EPSP/IPSP sequence in twenty-six neurones and a similar sequence was evoked from the contralateral nerve in fourteen (54%) cases

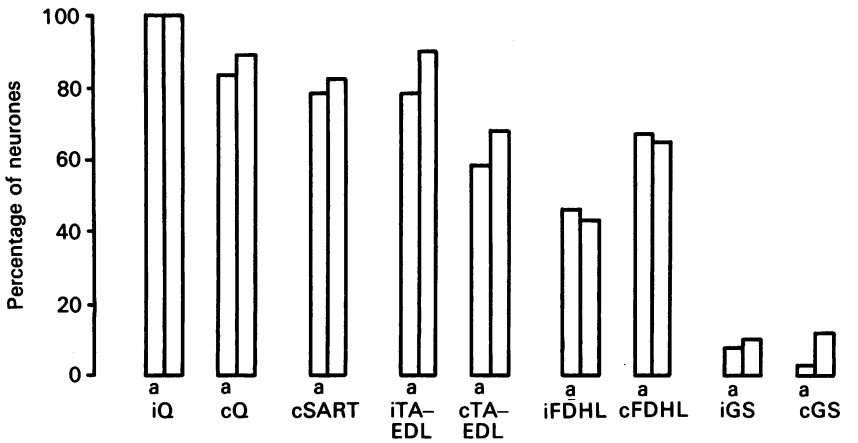

Fig. 4. Frequencies of occurrence of postsynaptic potentials from group II afferents of different ipsilateral and contralateral nerves. In each case the proportions of neurones antidromically activated from the motornuclei (a) and of those not antidromically activated are given for each nerve. As can be seen, ipsilateral nerves and their contralateral counterparts influenced similar proportions of neurones. Group II afferents of both iGS and cGS had effects only infrequently, but in similar proportions of neurones.

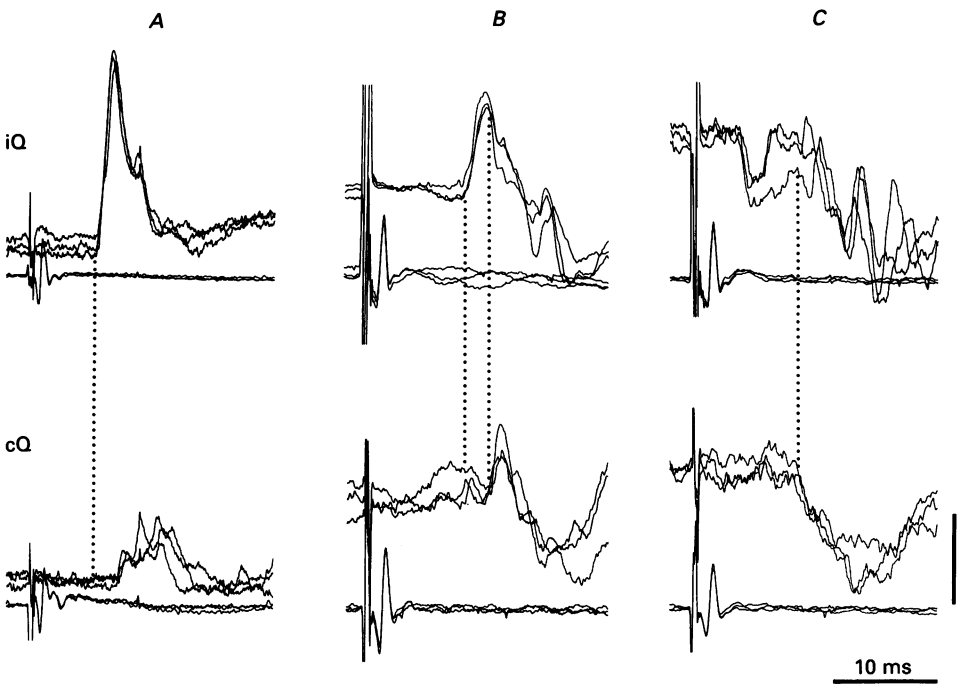

Fig. 5. *A–C* show intracellular recordings from three different midlumbar neurones, in each the upper and lower records show PSPs evoked by stimulation of iQ and cQ, respectively, at 5*T*. In *A* EPSPs are evoked bilaterally, in *B* an EPSP–IPSP sequence is evoked and in *C* only IPSPs were evoked (from both group I and group II afferents from iQ). The dotted lines mark the onset latencies of PSPs from iQ. The voltage calibration is 1 mV in *A*, the lower part of *B* and *C*; 2 mV in the upper part of *B*.

(e.g. Fig. 5*B*). Only in one neurone were opposite actions from the two sides observed, although in some cases the actions from the two sides were not identical. For example in some neurones only EPSPs were evoked from the ipsilateral nerves but both EPSPs and IPSPs were evoked from the contralateral nerves, or vice versa.

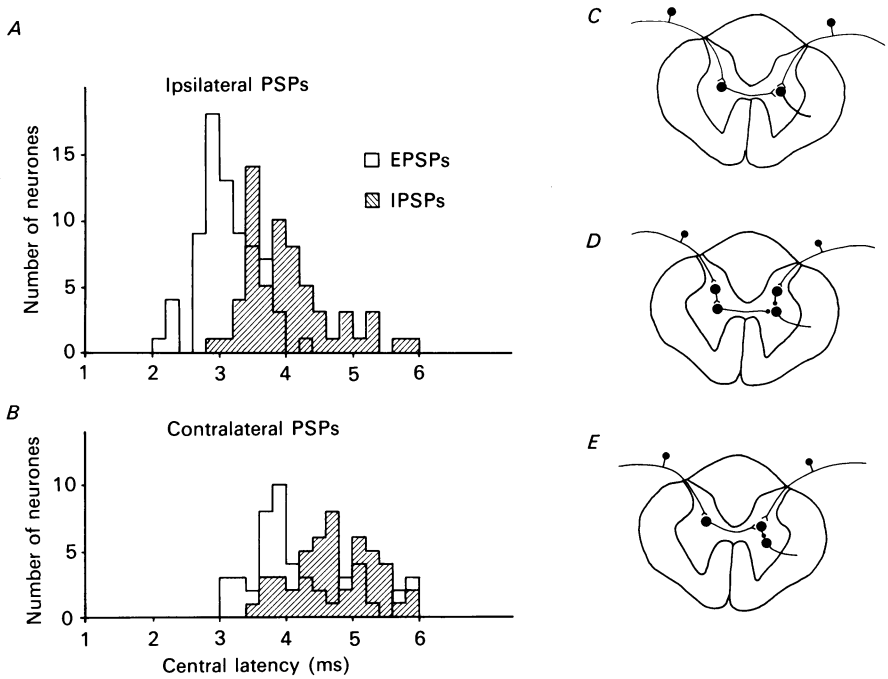

Fig. 6. Latencies and synaptic linkage of crossed group II PSPs. The histogram in *A* shows the distribution of latencies of EPSPs and IPSPs evoked from iQ in the current sample of neurones. *B* shows a similar distribution histogram for EPSPs and IPSPs evoked from the cQ nerve, albeit shifted to the right by about 1·0 ms. All latencies were measured from the onset of the group I volleys recorded at the L6–L7 border. *C* shows schematically a circuit which could be responsible for the crossed (disynaptic) EPSPs in midlumbar neurones. *D* and *E* show alternative circuitry which might be responsible for trisynaptic crossed IPSPs in midlumbar neurones.

One such case is illustrated in Fig. 3; ipsilateral group II afferents evoke both EPSPs and IPSPs but contralateral group II afferents evoke only EPSPs. Note, however, that the steep falling phases of the EPSPs, especially in Fig. 3*D* and *H*, suggest that IPSPs may have been present. The large size of the EPSPs, especially those from the ipsilateral quadriceps nerve, makes the presence of subsequent IPSPs difficult to detect. Whenever possible polarizing currents were used to demonstrate mixed effects. Nevertheless, some neurones with a combination of EPSPs and IPSPs may have been missed.

Latencies and synaptic linkage of crossed group II actions

Latencies of the EPSPs and IPSPs evoked in midlumbar neurones by contralateral group II afferents were measured from the arrival of the group I volley at the dorsal

root entry zone at the L7–L6 border. This was done because of the difficulty of measuring volleys in group II afferents directly. Since there is some difficulty in determining the timing of activity in group II afferents, the synaptic linkage of the crossed actions has been examined by comparison with EPSPs and IPSPs evoked from ipsilateral nerves. It has been reported previously that the minimal linkage of the EPSPs is probably monosynaptic and for the IPSPs is probably disynaptic (Edgley & Jankowska, 1987*a*, *b*). In order to obtain minimal values, latencies were measured from the second or third stimulus of a train, wherever this was possible.

Figure 6*A* shows the latency distributions of EPSPs and IPSPs evoked from the ipsilateral quadriceps nerve. As reported previously (Edgley & Jankowska, 1987*b*) the minimal latency of EPSPs evoked from ipsilateral group II afferents in mid-lumbar short-propriospinal neurones is approximately 2·0 ms and the IPSPs in these neurones have latencies minimally approximately 1·0 ms longer than the EPSPs (see Fig. 5*A* and *B*). Latency distributions for EPSPs and IPSPs evoked from group II afferents in the contralateral quadriceps nerve are illustrated in Fig. 6*B*. The minimal latencies of the contralaterally evoked EPSPs are approximately 3·0 ms, which are comparable to those of the ipsilaterally evoked IPSPs and about 1·0 ms longer than the monosynaptic ipsilateral EPSPs suggesting that, if they are mediated via intermediate zone neurones, they were evoked disynaptically (via a circuit like that illustrated in Fig. 6*C*). Contralaterally evoked IPSPs had longer latencies, lagging about 1·0 ms behind the contralateral EPSPs and the ipsilaterally evoked IPSPS. The minimal linkage for crossed group II IPSPs might therefore be trisynaptic, via circuitry illustrated in Fig. 6*D* and *E*. These arguments can only be applied to the neurones which have the shortest latencies. The EPSPs and IPSPs evoked from ipsilateral nerves in some neurones had longer latencies than the crossed PSPs in others. One reason behind this spread of latencies in different neurones is the different location of the neurones; some were recorded in the caudal part of the L3 segment while others were recorded in mid-L5 (a separation of about 15 mm), some were superficial (1·9 mm from the cord dorsum) while others were deep (3·9 mm from the surface). It is known that dorsal column collaterals and intraspinal branches of group II afferents have a very low conduction velocity (Fu & Schomburg, 1974; Edgley & Jankowska, 1987*a*; Fern, Harrison & Riddell, 1988; Harrison, Jami & Jankowska, 1988).

DISCUSSION

The data presented in this paper reveal that a large proportion of midlumbar neurones with group II input are also influenced by contralateral group II afferents. Furthermore, in a large majority of cases the pattern of actions evoked from either side is similar.

The actions evoked from contralateral group II afferents were often strong, sometimes sufficient to discharge the neurones. The latencies imply a simple linkage, involving one or two interposed neurones. Comparison of the latencies of contralaterally evoked EPSPs and IPSPs with those evoked from ipsilateral nerves suggests that a simple pathway involving a single commissural neurone may be involved for the crossed EPSPs, and that an additional neurone may be interposed

in the pathway for crossed IPSPs. In the latter case we cannot draw any conclusions from these data as to whether the additional interneurone is located ipsilaterally or contralaterally (Fig. 6*C*), but the EPSPs should have been evoked via commissural neurones with monosynaptic input from group II afferents since afferent fibres of that size do not cross the midline of the spinal cord (Light & Perl, 1979). These arguments are based on the assumption that the interposed neurones are located in the intermediate zone of the spinal grey matter.

It might be possible that the interposed neurones are located superficially in the dorsal horn; many group II-activated neurones are found in the dorsal horn and these are activated at latencies 0·5–1·0 ms shorter than those of intermediate zone neurones (Edgley & Jankowska, 1987*a*). If superficial interneurones are involved then it is conceivable that the pathways involve an additional interneuronal relay (i.e. two interneurones in the pathways for crossed EPSPs and three interneurones for the crossed IPSPs). If this is the case then the synaptic linkages in the pathway must be strong to permit reliable responses to single stimuli over three synapses.

Obvious and likely candidates to mediate these actions are contralaterally located midlumbar neurones; the nerves which evoked crossed actions were those which also activate ipsilateral midlumbar neurones. Neurones with the appropriate convergence have not been identified elsewhere, but there is evidence from axonal recordings that neurones with similar convergence do exist caudal to the L5 segment (Harrison & Riddell, 1989). The actions exerted by ipsilateral group II afferents are notable for their strength, readily discharging many midlumbar neurones. Many of the neurones also have group I input but the EPSPs are usually too small to discharge the neurones. Crossed actions from group I afferents were rare, but it is possible that interneurones with group I and group II convergence mediated the crossed actions and that group I inputs were not sufficiently powerful to discharge the neurones in response to single stimuli or the short trains used in these experiments. Many midlumbar neurones have this type of input (Edgley & Jankowska, 1987*b*). Commissural premotor interneurones are generally located in the ventral and medial parts of the spinal grey (mainly lamina VIII, see Harrison, Jankowska & Zytnicki, 1986). Neurones which might mediate crossed group II actions are found in the midlumbar segments as short-propriospinal neurones which project to contralateral motor nuclei (Jankowska & Noga, 1990). If these neurones do have collateral projections to contralateral midlumbar neurones then this would imply a tight interlimb coupling since the crossed actions on midlumbar neurones would be collateral to direct actions on motoneurones. Other cells which might mediate crossed group II actions in the midlumbar segments are contralaterally projecting group II activated neurones in the lateral part of the dorsal horn or in the intermediate zone (Bras *et al.* 1989). Whatever the source of the crossed actions in midlumbar neurones, the close similarity to the ipsilateral actions implies a tight coupling between midlumbar neurones on either side. Since many of these neurones project to motoneurones, the consequences of this for movement would be to produce a tight interlimb coupling.

The hypothesis which provided the impetus for these experiments was that the short-propriospinal neurones in the L4 region might be involved in co-ordination of limb movement during locomotion by mediating the timing of the switch from extensor to flexor muscle activity which occurs at the transition from the stance to

the swing phase of the step cycle. The reasoning behind this proposal was that, due to their inputs from stretch receptors in parts of quadriceps and sartorius, midlumbar neurones should be activated when the hip becomes fully extended and that the neurones excite or inhibit motoneurones (see Edgley & Jankowska, 1987*b*), hip extension being the key event in the transition between stance and swing. This hypothesis does not account for the excitation of the neurones from parts of quadriceps other than rectus femoris. It would be predicted that during most gaits (in which the hindlimb movements alternate) the neurones responsible for the switch in either hindlimb would be reciprocally active, such that when flexion of one limb occurs, the contralateral (supporting) limb should be prevented from flexing. Our results reveal an organization for the parallel activation of midlumbar neurones on both sides of the spinal cord. Thus a specific event which activates midlumbar neurones may, under our experimental conditions, activate the systems of group II-activated midlumbar neurones on both sides. Many of the neurones were shown to have projections to the hindlimb motor nuclei and previous data would imply that many are likely to have contacted motoneurones. We might therefore expect that an event in one hindlimb which led to the activation of midlumbar neurones would promote bilaterally symmetrical responses. The strength and frequency of the crossed actions in midlumbar neurones strongly suggests that they play a role in the bilateral co-ordination of limb movements. This organization might be appropriate to an in-phase gait (such as galloping) or for the co-ordination of postural reactions. In relation to postural actions, it has been shown that some group II-activated midlumbar neurones are powerfully excited on rotation of the neck (Yates, Kasper & Wilson, 1989) and by descending vestibulospinal pathways (Suzuki, Timerick & Wilson, 1985; Davies & Edgley, 1991). Muscle responses to these stimuli may be bilaterally symmetrical; for example, actions evoked from the vestibulospinal pathways are similar in corresponding motor nuclei of both hindlimbs, but with one additional interneurone in the crossed pathway (Hongo, Kudo & Tanaka, 1975). The crossed actions we have described in these experiments might be more appropriate to these actions.

All of the recordings described here were made with the spinal cord intact. This may be relevant since in similar experiments crossed group II actions in motoneurones show marked differences dependent on the integrity of the spinal cord; with the cord intact stimulation of group II afferents in those nerves which influence midlumbar neurones produces inhibition in a very high proportion of motoneurones in the contralateral hindlimb, including both flexors and extensors (Arya, Bajwa & Edgley, 1991). Midlumbar neurones are the most likely candidates to produce these reflex actions. After spinal section, group II afferents evoke actions which conform to the crossed extension reflex. It is possible, therefore, that spinalization could also have a profound effect on the pathways which mediate crossed group II actions on midlumbar interneurones.

We wish to thank the Wellcome Trust for funding this work, and the Dale and Rushton funds of the Physiological Society for their support to S.B. We also wish to thank Helen Davies for permission to include some results obtained in experiments done with her.

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