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A SHORT-LATENCY CROSSED PATHWAY FROM CUTANEOUS AFFERENTS TO RAT HINDLIMB MOTONEURONES

BY S. A. EDGLEY AND N. A. WALLACE
From the Department of Anatomy, Downing Street, Cambridge CB2 3DY

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SUMMARY

1. A novel pathway is described which mediates excitation of hindlimb motoneurones from contralateral afferents. Stimulation of contralateral limb nerves evoked short-latency (less than 5 ms) EPSPs in 55% of motoneurones tested.

2. The EPSPs were evoked by fast-conducting contralateral afferents activated by electrical stimuli of 1·04–2·5 times nerve threshold. Stimulation of contralateral muscle nerves did not evoke short-latency EPSPs, whereas nerves which contain afferents from distal skin territories (sural, superficial peroneal, tibial and saphenous nerves) did.

3. Central latencies were determined for the EPSPs from the arrival of the earliest components of the afferent volleys at the spinal cord. The earliest latencies were very brief (1·5 ms). These are comparable to the latencies of disynaptic inhibition in rat motoneurones from ipsilateral group I muscle afferents (1·5–1·7 ms). We conclude that a disynaptic relay is likely to be responsible for the earliest of the crossed EPSPs.

4. Short-latency crossed EPSPs were found in all types of motoneurone tested (including both flexor and extensor motoneurones) except in six cells innervating intrinsic foot muscles. In view of the origin and distribution of the crossed EPSPs, their possible functional role is discussed.

INTRODUCTION

A variety of crossed reflexes have been described, the best understood being the crossed extension reflex (see e.g. Sherrington, 1910; Holmquist, 1961). More recently crossed reflexes have been demonstrated in walking animals, and these reflexes are modulated in a phase-dependent manner during the step cycle. These can be evoked by noxious stimuli (Gauthier & Rossignol, 1981) or from large, fast-conducting cutaneous afferents (e.g. Forssberg, 1979; Duysens & Loeb, 1980; Duysens, Loeb & Weston, 1980). Analysis of the central pathways which mediate crossed reflexes of the latter type is still at an early stage. At least in the limb enlargements of the spinal cord, few large primary afferents cross the mid-line (see Light & Perl, 1979*a*, *b*), so commissural interneurones are likely to be involved in any crossed reflexes from large cutaneous afferents. One such group of interneurones can be found in the medial part of the ventral horn, in Rexed's lamina VIII, and there is evidence that some of them

make direct contacts with contralateral motoneurones (Harrison, Jankowska & Zytnicki, 1986). The most prominent input to these neurones was from group II or higher threshold muscle afferents, although some also had input from group I muscle afferents. Input from large cutaneous afferents was not reported.

In this report we describe a pathway which might be responsible for some of the reflexes described during locomotion. The pathway mediates short-latency EPSPs in motoneurones from contralateral cutaneous afferents in the hindlimb of the rat.

A short report of some of this work has been published (Edgley & Wallace, 1988).

METHODS

Experiments were performed on adult male rats (300–550 g) anaesthetized with urethane (1·3 g/kg initially, supplemented as necessary to maintain deep anaesthesia). Arterial and venous cannulae were inserted to allow monitoring of blood pressure and intravenous delivery of drugs or fluid respectively. The trachea was cannulated to allow artificial ventilation.

The lumbo-sacral segments of the spinal cord were exposed for recording and a variety of nerves from both hindlimbs were dissected free for stimulation. These were mounted on silver wire electrodes either in a paraffin oil pool made from skin flaps or in tunnel electrodes buried under the skin. Single-pulse stimuli (0·1 or 0·2 ms) with a repetition rate of 0·25–0·5 Hz were used. Throughout the stimulus strength will be expressed in terms of threshold for the most excitable afferents in the nerve (T). In one experiment contralateral nerve fibres were activated by stimulation of dorsal or ventral spinal roots. In this experiment a long laminectomy which extended caudally to the sacrum was made, to allow separation of long lengths of dorsal and ventral roots for stimulation individually. The roots to the L4 and L5 segments were used for this purpose and were stimulated via pairs of silver wire electrodes.

Intracellular recordings were made from hindlimb motoneurones using glass microelectrodes filled with 1 M-potassium citrate (impedance 1·5–20 MΩ). These were inserted into the spinal cord through the lateral funiculus which was exposed by reflecting the dorsal roots. To prevent drying and maintain temperature (at 35–37 °C) the spinal cord was immersed in warm mineral oil. The vertebral column was stabilized using clamps on thoracic and low-lumbar vertebrae. Just before the onset of recording the animals were paralysed with a single dose of gallamine triethiodide (6–8 mg) and artificially ventilated. Paralysis was maintained with one or more supplementary doses (2 mg) during the next 1–2 h but was then discontinued. During the period of paralysis the animals were over-ventilated so that they did not resume large breathing movements as the paralysis waned. Adequacy of anaesthesia was ensured by verifying that the animals were deeply anaesthetized prior to the induction of paralysis. During the period of paralysis we routinely monitored the blood pressure to ensure that there were no cardiovascular responses to noxious stimuli; none were seen.

In parallel with intracellular recordings, the arrival of volleys in peripheral afferents at the spinal cord was monitored via a silver ball electrode placed on the dorsal roots. All signals were recorded on magnetic tape for off-line analysis. Data were reproduced either as single sweeps or averages of eight or sixteen consecutive traces using a digital storage oscilloscope and plotter (Gould, 1425). The latencies of intracellular potentials were measured by from the onset of the dorsal root volleys.

RESULTS

General

Our observations are based on intracellular recordings from 225 motoneurones, 205 of which were identified by antidromic invasion from muscle nerves. The remaining twenty neurones were unidentified, but are included as motoneurones since they were easily penetrated cells located within the motoneurone pool. Most of

the latter were probably hamstring motoneurones which were unidentified because the peripheral nerve branch in which they projected was not dissected: most had monosynaptic EPSPs on stimulation of the hamstring nerve. Only cells with resting membrane potentials more negative than −40 mV were tested.

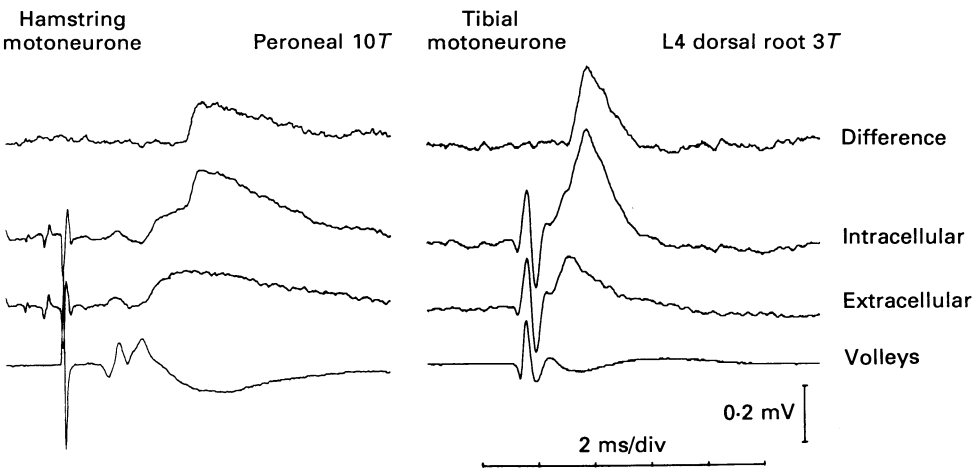


Fig. 1. Subtraction of extracellular field potentials from intracellular potentials. Recordings from a hamstring and a tibial motoneurone with EPSPs from the contralateral peroneal nerve and the L4 dorsal root, respectively, are illustrated. From bottom to top the traces are: volleys recorded from the dorsum of the spinal cord at the recording location, extracellular field potentials recorded immediately outside the motoneurones, intracellular potentials and in the uppermost traces are the result of subtracting the extracellular potentials from the intracellular ones (difference). All records are averages compiled from eight consecutive stimulus presentations. Field potentials with onset latencies of 1·0–1·2 ms were regularly encountered, so the subtraction procedure was used before determining latencies for the crossed EPSPs. Stimulus strengths in this and the following figures are expressed in terms of threshold (*T*) for the most excitable fibres.

Clear short-latency EPSPs following single stimuli to contralateral nerves were found in 123 (55%) of the motoneurones tested. In some cases estimation of the onset latency and size of the EPSPs was complicated by the presence of extracellular field potentials, which were evoked by contralateral nerves throughout the ventral horn. These had onset latencies of about 1·5 ms from the onset of the afferent volleys and could be as large as 0·2 mV (Fig. 1). In cells in which EPSPs were evoked by stimuli of less than 2·5 *T* this was not a problem, since the field potentials only appeared to stimuli above this strength. In others they obscured the early phases of the EPSPs. To identify genuine postsynaptic events we recorded extracellular field potentials in the proximity of each of the cells studied. These were subtracted from the intracellularly recorded potentials to reveal the true synaptic potentials (Fig. 1). This procedure was used to obtain latencies for all of the EPSPs included in this report.

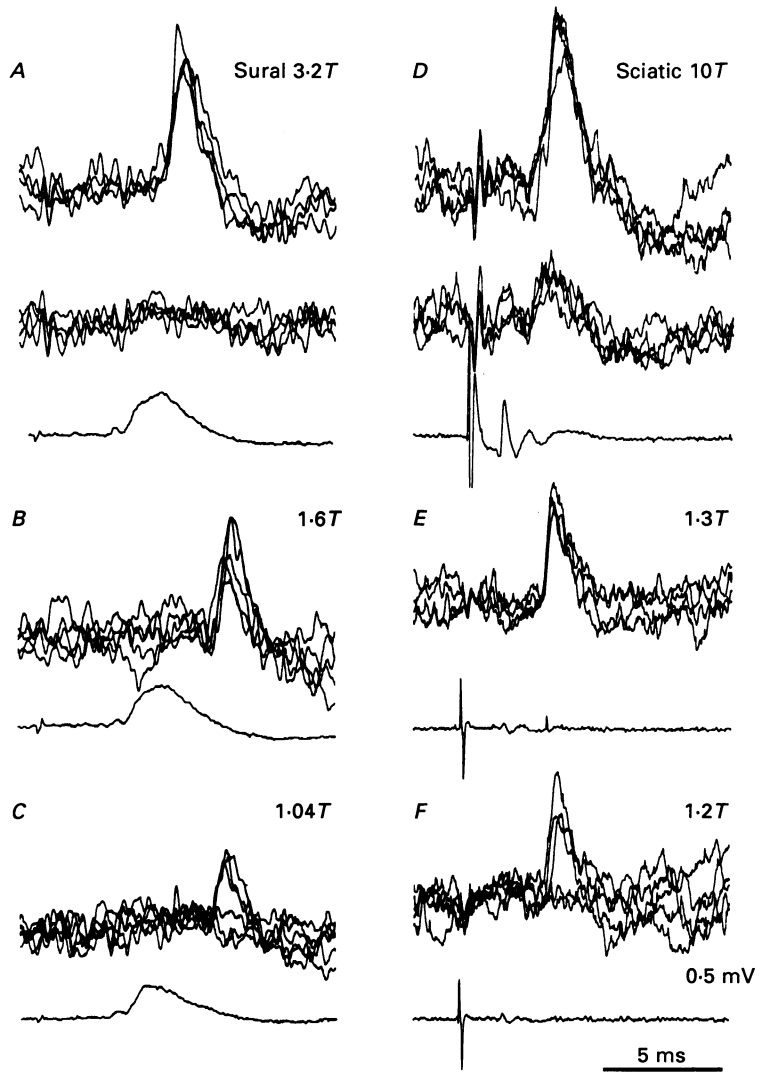


Fig. 2. Responses of two tibial motoneurones (*A–C* and *D–F*) to graded stimulation of contralateral afferents. Upper traces are intracellular potentials (single sweeps), lower traces are volleys recorded from the cord dorsum. The middle traces included in *A* and *D* show extracellular field potentials recorded just outside the motoneurones. In *A* the sural nerve did not evoke a field potential, but in *D* stimulation of the sciatic nerve at 10*T* can be seen to elicit a field potential which is also present in the intracellular (upper) trace. In both neurones stimuli close to threshold (*C* and *F*, note the small size of the volleys) evoked small unitary EPSPs, indicating that large myelinated fibres are involved. Note also the shift in latency between *A* and *B*.

Origin of the crossed EPSPs

Whenever possible the threshold for evoking EPSPs was investigated using finely graded electrical stimuli (Fig. 2). In most cases the EPSPs were made up of a few unitary components, as can be seen from the single-sweep records in Fig. 2*C* and *F*.

This made precise determination of threshold difficult since responses occasionally failed, even with stimuli well above the strength at which EPSPs first appeared. With graded stimulation the EPSPs generally appeared with stimuli between 1·1 and 2·5 times the threshold of the most excitable afferents in the nerve. As Fig. 2*C* shows, the most excitable fibres were responsible for the EPSPs in some cells: the weakest

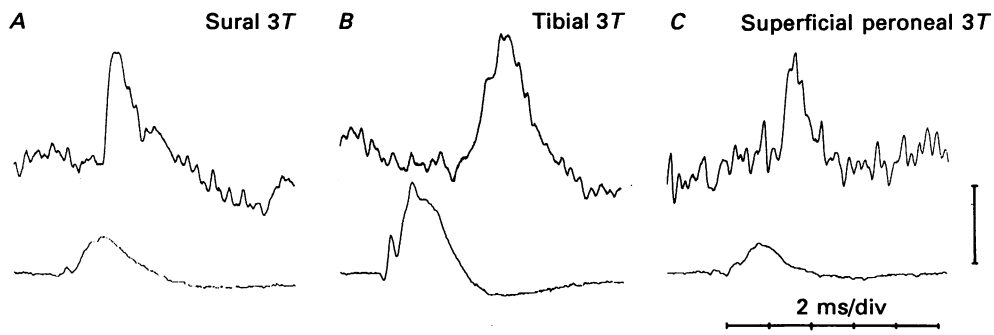


Fig. 3. Averaged intracellular records (extracellular fields subtracted) from a tibial motoneurone in which the contralateral sural nerve (*A*), tibial nerve below its branches to the calf muscles (*B*) and the superficial branch of the peroneal nerve (*C*) all evoked EPSPs. As in Fig. 2 the lower traces are volleys recorded from the cord dorsum. The voltage calibration bar is 0·4 mV for *A* and *B*, 0·2 mV for *C*.

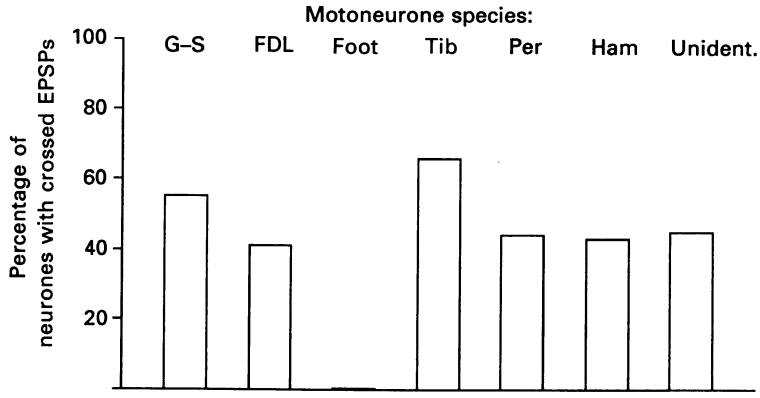
stimuli used evoked unitary EPSPs of about 0·2 mV in this cell. A common observation was that the central latencies of the crossed EPSPs often shortened on increasing the stimulus intensity; this is dramatically illustrated in Fig. 2*A* and *B* where the latency decreases by 2 ms on increasing the stimulus.

In view of the origin of the crossed EPSPs from nerve fibres recruited by stimuli close to threshold, the identity of the fibres responsible could immediately be narrowed down to a few types. Although few data are available for rat nerves, in cats group I muscle afferents (both Ia muscle spindle and Ib Golgi tendon organ afferents), low-threshold cutaneous (Aβ) fibres and α-motoneurone axons are recruited by stimuli in this range. Rat nerves are unlikely to differ grossly from this pattern.

In order to eliminate some of these possibilities we stimulated the different types of fibre individually. As a first step we tested the possibility that a crossed pathway from recurrent collaterals from α-motoneurones could evoke EPSPs (Jankowska & Odutola, 1980). In one experiment we separated the spinal roots from the L4 and L5 segments and investigated the effects of fibres in either dorsal or ventral roots on contralateral motoneurones. In this experiment twenty-four motoneurones were tested: crossed EPSPs were identified in twenty of them from the dorsal roots (e.g. Fig. 2*D–F*), but in no case were EPSPs evoked from ventral roots. We can therefore eliminate neurones contacted by axon collateral of α-motoneurones as a source of the short-latency crossed EPSPs.

The remaining possibilities are that the EPSPs are evoked by group I muscle afferents or by Aβ cutaneous afferents. In the early experiments we stimulated the entire sciatic of the tibial and peroneal nerves in the popliteal fossa, which are all

mixed nerves. In order to separate the effects from muscle and skin afferents as much as possible we dissected individual nerve branches separately in later experiments. None of the muscle nerves evoked short-latency EPSPs in contralateral motoneurones; the nerves we tested were to quadriceps and sartorius (ten cells tested),



	G–S	FDL	Foot	Tib	Per	Ham	Unident.
All nerves	6/11	7/17	0/6	73/110	18/41	23/54	9/20
Sciatic	5/7	1/1	—	11/14	4/7	4/6	1/4
Tibial	1/4	3/16	0/6	25/65	7/19	7/29	7/11
Peroneal	1/4	5/16	0/6	15/49	9/20	9/29	8/11
Sural	—	—	0/6	23/38	5/11	7/15	2/2
Saphenous	—	—	—	2/18	1/3	1/10	2/2
Superficial peroneal	—	—	—	4/28	4/8	1/5	1/1

Fig. 4. Distribution of the crossed EPSPs. EPSPs recorded in different species of motoneurone are shown in the vertical columns. The bar graph gives the percentage of neurones with short-latency crossed EPSPs from any source. The figures below are the numbers of motoneurones of each species found to have EPSPs/numbers tested, from each source. Abbreviations are as follows: G–S, gastrocnemius and soleus; FDL, the deep calf muscles flexors hallucis and digitorum longus, plantaris, popliteus and tibialis posterior; Foot, motoneurones with axons in the plantar nerves. Tib, the tibial nerve in the popliteal fossa. Per, the peroneal nerve. Ham, hamstring muscles. Unident., unidentified cells.

hamstrings (forty-three cells tested), gastrocnemius–soleus and the nerve to the deep calf muscles (forty-three cells tested). On the other hand, all of the nerves containing skin afferents evoked crossed EPSPs in some motoneurones and in some neurones EPSPs could be evoked from more than one nerve (Fig. 3). These were the sural (Fig. 3*A*), the tibial (plantar nerves, Fig. 3*B*), the superficial branch of the peroneal nerve (Fig. 3*C*) and the saphenous nerve. The table in Fig. 4 shows the relative effectiveness of these nerves. One point which should be borne in mind when looking at the figures is that the larger nerves are effective in higher proportions of motoneurones. This is perhaps not surprising for an oligosynaptic pathway. With this in mind, the sural and tibial nerves were most effective, the saphenous and

superficial peroneal nerves less so. In some experiments EPSPs were evoked by stimulation of the nerves to calf muscles, but in all cases these required strong stimuli which spread to activate the main branch of the tibial nerve (as could be seen from the cord dorsum potentials).

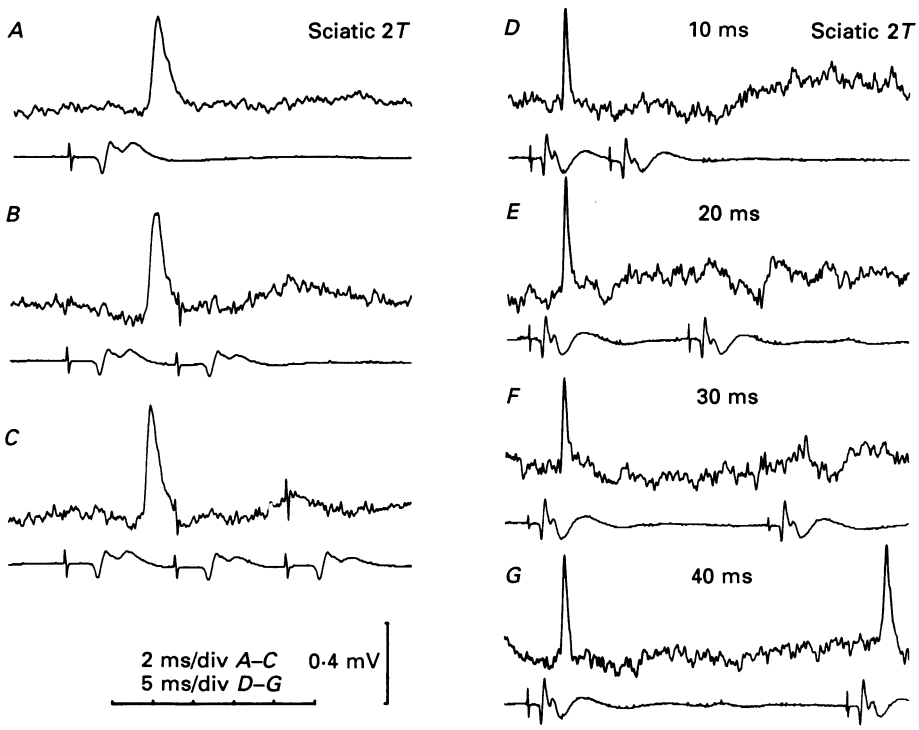


Fig. 5. Frequency-following characteristics of the crossed EPSPs. *A–C* show EPSPs evoked by stimulation of the contralateral sciatic nerve in a tibial motoneurone. Single stimuli at 2*T* (*A*) evoked a large EPSP. This EPSP first appeared with stimulation at 1·6*T*. Stimulation at 2*T* is used for the illustrations since at this strength the EPSP appeared reliably to each stimulus. Subsequent stimuli at intervals of 5 ms were ineffective (*B* and *C*). *D–G* show the effectiveness of a second stimulus given at different intervals. In this cell a second stimulus given at an interval of less than 30 ms from the first was ineffective (*D* and *E*). An interval of 40 ms was required before the EPSP reappeared (*G*). All records are averages of eight consecutive stimulus sequences, with extracellular fields subtracted.

Distribution and properties of the crossed reflexes

Short-latency crossed EPSPs were seen in all of the types of motoneurone tested, with the exception of six motoneurones which innervated intrinsic foot muscles (antidromic from the plantar nerves). However, they were larger and more frequent in some species than in others. Figure 4 summarizes the distribution of short-latency crossed EPSPs. As can be seen from the bar graph, the EPSPs were found in about 40% of the motoneurones of most species. From this distribution it is obvious that crossed cutaneous EPSPs from nerves innervating the foot can be found in muscles

which are functionally different, e.g. ankle and toe flexors (the peroneal muscles) and extensors of the same joints (calf muscles). The largest EPSPs were found in gastrocnemius–soleus motoneurones and had amplitudes of about 1·3 mV. In some cases the EPSPs were small and not evoked by each stimulus. The small EPSPs were identified using averaging, but this cannot provide a reliable estimate of EPSP size because of the occasional response failures. The smallest accepted EPSPs had amplitudes of about 80 μV.

Many of the EPSPs had components which appeared in an all-or-none fashion with near-threshold stimuli. Presumably there is a population of neurones which mediate the crossed EPSPs. In order to obtain an idea of the maximum size of the EPSPs we used trains of stimuli in the hope that the pathway would show temporal summation. This was unsuccessful: trains of stimuli were not more effective (Fig. 5). Where single stimuli reliably evoked responses, trains of stimuli did not cause an increase in the size of the EPSPs or in their frequency of occurrence. In fact the first stimulus always evoked the largest EPSPs (Fig. 5*A–C*). Evidence for temporal summation was also sought using trains of stimuli sub-threshold for evoking EPSPs when given singly, but was not found. The frequency-following properties of the pathway were also examined using two stimuli separated with different intervals. In all of the cells tested the second stimulus only became effective when it followed the first with an interval greater than 20 ms. This property is illustrated in Fig. 5*D–G*, where the response recovered after 40 ms.

Latency and synaptic linkage

One of the most striking features of the crossed EPSPs was their short central latencies. These were measured from the arrival of afferent volleys at the dorsal root entry zone. Two examples are shown in Fig. 6*A* and *B*. As was illustrated in Fig. 2, the latencies of some of the EPSPs shortened as stimulus strength increased. For this reason we always used responses to stimuli of 2·0–3·5 *T* in order to calculate latencies, and extracellular field potentials were always subtracted from the intracellular potentials. A latency distribution histogram is shown in Fig. 6. An apparently bimodal distribution is found with one peak 1·5–2·8 ms (peak 2·0 ms) and a second at 3·0–5·0 ms.

The values obtained from peripheral nerve stimulation may not be true measures of central latency, since the thresholds for evoking the EPSPs were sometimes more than double the threshold for the most excitable fibres. In these cases the fibres responsible for the EPSPs could have conduction velocities considerably slower than those of the lowest threshold fibres in the nerve. This difference would lead to a delay between the onset of the dorsal root volleys from the fastest conducting fibres and the arrival of volleys in the afferents responsible for the EPSPs. This delay would be greater as the conduction distance becomes greater. It seems possible that this could be the case since the EPSPs evoked by direct stimulation of the dorsal roots, where the arrival of volleys at the spinal cord would be almost synchronous, had central latencies which were generally shorter than those of EPSPs evoked by contralateral nerves (compare the shaded and unshaded blocks in Fig. 6*C*). The EPSPs from the dorsal roots had latencies between 1·5 and 2·4 ms (mean 1·87 ms).

The bimodal distribution of latencies might represent the effects of activation of

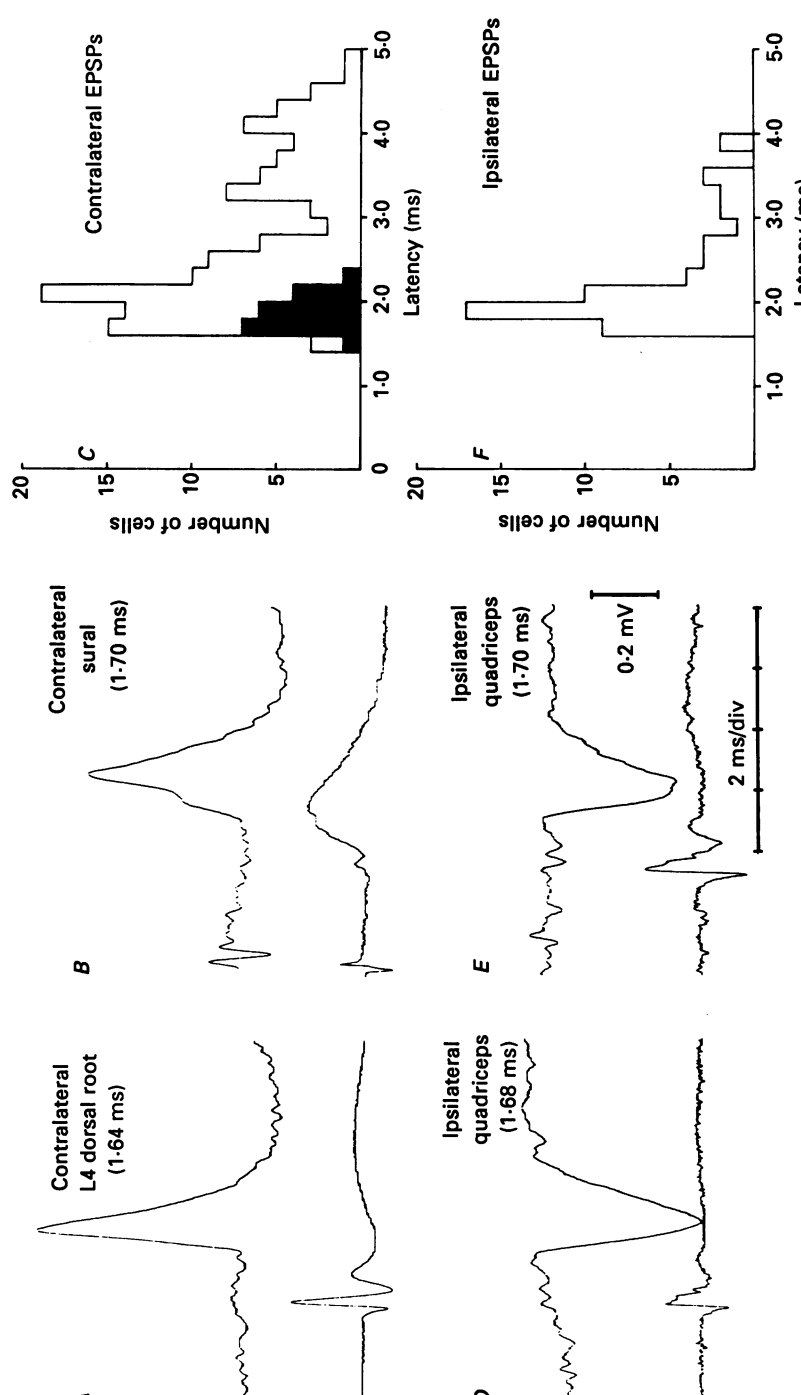


Fig. 6. Central latencies of the EPSPs. *A–C* show the central latencies of crossed EPSPs. Examples from two different motoneurones are shown in *A* and *B* and their central latencies are given. *C* is a latency distribution histogram for the crossed EPSPs. Open blocks represent EPSPs from contralateral nerves; filled blocks represent EPSPs from contralateral dorsal roots. Stimulus strengths were between 2·0 and 3·5*T* in all cases and extracellular field potentials were always excluded. *D–F* show the latencies of IPSPs evoked from ipsilateral nerves. *D* and *E* are recordings from hamstring motoneurones and show reciprocal IPSPs evoked from the quadriceps nerve. *F* is a latency distribution histogram of ipsilateral IPSPs. Note that the earliest IPSPs had latencies comparable to those of the earliest crossed EPSPs.

different pathways. It does not, however, arise from pathways to different species of motoneurone since motoneurones of all species tested had EPSPs which fell into both groups. Also note that in the cell of Fig. 2*A–C* stimulation at 1·6*T* evoked EPSPs which would fall into the later peak (4 ms), whereas stimulation at 3·2*T* evoked EPSPs of much shorter latency (2 ms) which would fall into the earlier peak.

The latencies of the earliest crossed EPSPs were very short (1·5 ms). Such short latencies are indicative of a rather direct pathway. As a comparison we have investigated the latencies of IPSPs evoked via ipsilateral pathways from group I afferents. These pathways correspond with those of reciprocal inhibition (e.g. from quadriceps Ia afferents to hamstring motoneurones) and non-reciprocal inhibition (e.g. from deep calf muscle group I afferents in gastrocnemius–soleus motoneurones). Both of these pathways have been extensively studied in the cat and are minimally disynaptic (i.e. they involve a single interneurone, see Baldissera, Hultborn & Illert, 1981). Two examples of reciprocal IPSPs are shown (Fig. 6*D* and *E*) and a latency distribution histogram is plotted in Fig. 6*F*. The minimal latencies of these IPSPs are directly comparable to those of the earliest crossed EPSPs.

DISCUSSION

Short-latency crossed EPSPs of the type we have described have not been reported in previous studies of other species. Our conclusion that the EPSPs were evoked by cutaneous afferents is based on stimulation of nerves which contain primarily cutaneous afferents. However, both the sural and plantar nerves also contain some muscle and joint afferents. We cannot exclude that these afferents could contribute to the EPSPs but we have no evidence that they do: stimulation of nerves without cutaneous territories never produced any short-latency effects. On the other hand, stimulation of the superficial peroneal nerve (which is very small in the rat) or of the saphenous nerve, did evoke crossed EPSPs. All of these nerves were dissected to mid-calf and therefore contained afferents from the distal skin.

In view of their short central latencies, a rather direct pathway should be responsible for the crossed EPSPs we have described. The earliest EPSPs had latencies almost identical to those of the shortest latency IPSPs evoked via ipsilateral pathways (1·5 ms). These earliest IPSPs are likely to have been evoked via disynaptic reciprocal or non-reciprocal inhibitory pathways from group I afferents which have been investigated extensively in the cat (see Baldissera *et al.* 1981). Since the crossed EPSPs have similar central latencies, it seems probable that the earliest of these were also evoked disynaptically.

In the lumbo-sacral spinal cord of the cat, some ipsilateral pathways from cutaneous afferents to hindlimb motoneurones have recently been shown to have central latencies indicative of a disynaptic linkage (Fleshman, Lev-Tov & Burke, 1984; Fleshman, Rudomin & Burke, 1988). Initially the short-latency pathway was thought to affect only a single motoneurone pool, but when facilitated by activity in descending pathways EPSPs with latencies of 1·5 ms could be evoked in several species of motoneurone.

A direct comparison of the latencies of the EPSPs we have described and those in ipsilateral pathways in cats is difficult: in rats the spinal cord dimensions are smaller and fibre conduction velocities differ. In addition, the pathway we have described is

crossed and includes intraspinal conduction across the mid-line. The earliest EPSPs we found had central latencies of 1·5 ms, which is comparable to those described in ipsilateral pathways by Fleshman *et al.* (1988). In agreement with their conclusions, we feel it rather unlikely that three synaptic delays could occur within such a short period. Our conclusion is therefore that the earliest EPSPs were evoked disynaptically.

As to the likely locations of the interneurones responsible for the short-latency EPSPs, primary afferents in some parts of the spinal cord have collaterals which cross the mid-line, but these are rare in the cervical and lumbo-sacral enlargements (see Light & Perl, 1979*a*, *b*) and those which do cross tend to have small diameters. Commissural interneurones are therefore likely to be responsible for the EPSPs. These are found primarily in the lateral part of the base of the dorsal horn and in the medial part of the ventral horn in lamina VIII (see e.g. Molenaar & Kuypers, 1978). However, only those located in lamina VIII are likely to contact motoneurones (see Harrison et al. 1986); the dorsal horn neurones have long ascending and/or descending projections. The properties of some lamina VIII interneurones have been investigated but powerful, short-latency excitation from low-threshold cutaneous afferents has not been described (see Harrison *et al.* 1986).

Functional considerations

In view of the distribution of effects (Fig. 4) it is not straightforward to determine what the functional role of this pathway might be. In many motoneurones the crossed EPSPs were rather small, but it should be borne in mind that the single stimuli routinely used may not have optimally activated the pool of interneurones responsible for these effects. On the other hand, our attempts to demonstrate temporal summation in the pathway were unsuccessful. Since the first stimulus of a train was always the most effective and subthreshold stimuli did not summate, it would appear likely that the population of interneurones involved in the pathway have a small subliminal fringe.

A sizeable proportion of all of the species of motoneurone tested had short-latency crossed EPSPs (Fig. 4), with the exception of the small sample of intrinsic foot muscle motoneurones. In the absence of some external control, this suggests that this pathway might act on the motoneurones closest to firing threshold at the time of delivery of the stimulus. Since the pathway is activated from cutaneous nerves, it is likely to be active when the contralateral foot makes skin contact. This would normally occur during standing, when extensor muscles are active; thus during this time the pathway would act on extensors. During locomotion with an alternating gait the pathway might be activated during the stance phase of the contralateral limb step cycle. Should this occur during the time when active flexion occurs, the pathway would facilitate flexor muscle activity. Such a pattern of crossed reflexes from large, fast-conducting cutaneous afferents has been described in the cat (Duysens & Loeb, 1980; Duysens *et al.* 1980) and is functionally appropriate.

Finally, in our experiments the spinal cord was intact so that possibility of descending control of the pathway remains and is an obvious direction for further investigation, especially in view of the findings of Fleshman *et al.* (1988). Both vestibulo- and reticulospinal pathways terminate heavily in lamina VIII which is a possible location for the interneurones responsible for the short-latency EPSPs.

Supraspinal control of the interneurones could facilitate or depress the pathway, to allow its operation during specific types or phases of movement.

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