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SPINAL NEURONAL ACTIVITY DURING THE PECTORAL FIN REFLEX OF THE DOGFISH: PATHWAYS FOR REFLEX GENERATION AND CEREBELLAR CONTROL

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Summary

Single units were recorded from the spinal cord of decerebrate dogfish (*Scyliorhinus canicula*) during pectoral fin reflexes (PFR) evoked by electrical pulse trains to the fin. The units were classified as primary afferent neurones, motoneurones or interneurones. Motoneurones discharged for limited (and various) periods during the reflex at latencies of 20 ms or more. There was no evidence for monosynaptic activation by primary afferents. Short-latency (S) units received monosynaptic input from fast-conducting afferents at latencies (<20 ms) appropriate for pre-motor interneurones. However, excitation of individual S-units by intracellular current injection never evoked motoneurone discharges, suggesting that convergence is necessary for motoneurone activation. Intracellular recordings from S-units which discharged for periods longer than the duration of the afferent volley generated by the fin stimulus showed that they receive other inputs in addition to those from primary afferent fibres. Intermediate-latency (I) units had similar properties to S-units except for a longer latency (>30 ms), which ruled out monosynaptic excitation by fast-conducting afferents. Antidromic activation of S- and I-units by high spinal stimulation was rarely seen and orthodromic driving was also uncommon. A significant number of interneurones with latencies greater than 60 ms (L-units) were antidromically activated by high spinal stimulation. Their discharges were often long-lasting (>1 s) and we suggest that they may provide input to the cerebellum during the PFR.

Introduction

When the pectoral fin of a dogfish is touched, the fin is elevated. This 'pectoral fin reflex' (PFR) comprises an initial rapid elevation with a latency of 30–40 ms (phasic component) which is then sustained for more than 1 s (tonic component)

* Experimental work performed at the Laboratory of the Marine Biological Association, Plymouth, UK.

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(Paul and Roberts, 1979). Experimentally, this reflex can be reproduced in full by stimulating the fin with a short burst of electrical pulses. Although the complete pattern is seen in spinal dogfish and thus can be generated by spinal circuits, the reflex is clearly affected by brain centres such as the cerebellum for, after the cerebellum has been ablated, the tonic component is virtually absent whereas the phasic component survives relatively unchanged (Paul and Roberts, 1979). This cerebellar action is mediated *via* the brainstem because in elasmobranchs, as in other vertebrates, Purkinje cells project to deep cerebellar nuclei (Ebbesson and Campbell, 1973; Paul and Roberts, 1984; Fiebig, 1988) which then synapse on brainstem neurones. Although Purkinje cells (Paul and Roberts, 1981), cerebellar nuclear neurones (Paul and Roberts, 1983) and certain reticular neurones (Paul and Roberts, 1978) are all active during the PFR, only the reticular neurones discharge early enough to influence the phasic component. Cerebellar neuronal activity, in contrast, has a longer latency and occurs only when there is sufficient spinal interneuronal activity to drive motoneurones (Paul and Roberts, 1981, 1984). It has therefore been suggested (Paul and Roberts, 1979, 1984) that the PFR-related cerebellar activity depends upon inputs from spinal interneurones which are associated with motor pattern generation.

We had two goals in the present study. The first was to obtain information about the spinal cord circuitry underlying the PFR and to identify appropriate targets for brainstem-mediated cerebellar action. The second was to determine, by antidromic activation from the rostral spinal cord, which of the spinal neurones that discharge during the PFR project to the brain and thus could be afferent to the cerebellum.

Materials and methods

Animals and operating techniques

These experiments were carried out at the Laboratory of the Marine Biological Association, Plymouth, using 22 dogfish (*Scyliorhinus canicula*). Details of the general preparatory techniques and the maintenance of a fish in an experimental tank with its gills perfused by sea water have been described previously (Paul and Roberts, 1979, 1981, 1984). After an initial decerebration under MS222 anaesthesia (Sandoz, 166 mg l^{-1}), a laminectomy was performed to expose two or three segments of the spinal cord between segments 5 and 9, where most of the motoneurones supplying the pectoral fin muscles are located (Timerick, 1983). The caudal hindbrain and the first and second spinal segments were also exposed for stimulation. The cord was sectioned at the level of the pelvic fins and the caudal part destroyed. Water temperature was 17–19°C.

Stimulating and recording techniques

The pectoral fin reflex

The PFR was evoked by short trains of pulses (hereafter referred to as the PFR stimulus) applied to the fin and recorded with wire electrodes inserted in the sing

elevator muscle. With constant stimulus-pulse parameters and a stimulus-train repetition rate of 0.2 Hz or less, the PFR could be evoked consistently for many hours. Full details of the technique and the characteristics of the reflex have been described previously (Paul and Roberts, 1979). The parameters of stimulation required to evoke the reflex were established at the beginning of the experiment and the latency and duration of the reflex were measured from electromyogram (EMG) records taken from the muscle. When sufficient data about the reflex had been obtained, curare (*d*-tubocurarine, Wellcome Laboratories) was given intravenously through the suborbital sinus in a dose of approximately 7 mg kg^{-1}. The stimulus parameters were then not changed during the course of the experiment.

Spinal nerves and spinal cord

Electrode wires used to record the EMG from the fin muscle in the initial part of the experiment were later used in the paralysed fish to stimulate motor nerve endings in the muscle, so that elevator motoneurones could be identified by antidromic activation. A concentric bipolar electrode, placed in the junctional region between hindbrain and spinal cord, was used to stimulate ascending and descending spinal pathways.

In some experiments, spinal nerves were dissected close to the spinal cord and separated into their motor and sensory rami for stimulation and recording, using bipolar platinum hook electrodes. Motoneurone discharges were monitored by recording from the motor ramus and stimulation of the ramus was used for antidromic identification of motoneurones in the cord. The incoming sensory volley generated by the PFR-stimulus was recorded from the sensory ramus.

Single units

Extracellular and intracellular recordings of single-unit activity in the exposed spinal cord ipsilateral to the stimulated fin were made with glass micropipettes filled with 3 mol l^{-1} KCl. The responses were stored on magnetic tape (Racal Store 4, passband d.c.–5 kHz) for subsequent analysis using either a DEC 11/34 computer (DEC Inc.) or a NOVA computer (Data General Inc.) with custom-written software, as described previously (Paul and Roberts, 1981). Each unit was tested by fin stimulation to determine whether it discharged in relation to the PFR; by high spinal stimulation to determine whether it projected to or received inputs from the brain; and by spinal motor nerve stimulation to determine whether it was a motoneurone.

The classification of a response to high spinal stimulation as either antidromic or orthodromic depended, in the majority of cases, on latency – its duration and constancy. The collision test for antidromic excitation was of little utility since the estimated time of collision usually fell within the refractory period of the cell. Latency measurements were made using a digital oscilloscope (Nicolet). There was a clear division between those units classed as being antidromically activated, where the latency was less than 3 ms and varied by tens of microseconds only, and

those units classed as being orthodromically activated, with latencies of more than 3 ms, which varied by hundreds of microseconds or even milliseconds.

Results

Responses of spinal neurones

Discharges were recorded from 256 spinal neurones which responded to at least one of the stimuli used (fin, high spinal, motor nerve). Of these, 80 responded to the fin stimulus and have been analysed in detail. Numerous motoneurone axon spikes were also recorded from motor rami.

Sensory neurones

Recordings taken from sensory rami show that the PFR-stimulus produced a barrage in afferent fibres lasting some 50 ms (Fig. 1A), but the last 20–25 ms contained only very small-amplitude responses, presumably indicating small,

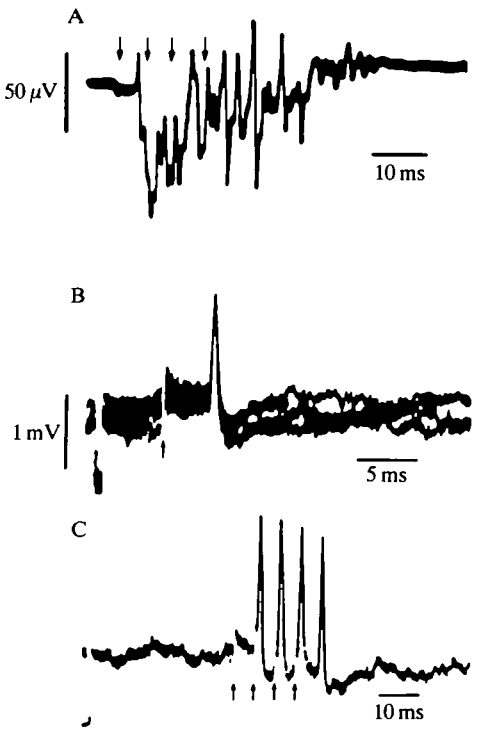

Fig. 1. The afferent input from the fin evoked by the PFR-stimulus. (A) An afferent volley recorded from the whole sensory ramus. Note the duration of the volley is almost 60 ms but the last 15 ms has only very small-amplitude units. (B) A single unit recorded from the spinal cord near the entrance of the dorsal root; five consecutive responses superimposed. Note the very consistent latency. (C) The same unit, responding to each pulse of the PFR-stimulus. Individual stimulus pulses are marked by arrows.

slowly conducting axons. Occasional fast-rising, small-amplitude (<2 mV) spikes with a short (around 4 ms) constant latency in response to fin stimulation were recorded in the spinal cord, near to the entry of the dorsal root, and these were the only units showing one-to-one following of the stimulus train (Fig. 1B,C). These characteristics are suggestive of large, fast-conducting sensory afferent fibres.

Motoneurones

Discharges of single motoneurones were recorded either as axon spikes in the motor ramus, or as soma spikes (identified by antidromic activation) in the cord. The activity of three units recorded simultaneously from the motor ramus of a pectoral fin nerve during a PFR is shown in Fig. 2A. One unit discharged once only; the post-stimulus-time histograms (PSTHs) of the other two are shown in Fig. 2B,C. Note that one discharged at a higher rate but only during the early part of the reflex (Fig. 2B); the other discharged at a lower rate but for a longer period (Fig. 2C).

Intracellular recording of soma spikes of a fin elevator motoneurone and its simultaneously recorded axon spikes, which were evoked by the PFR-stimulus, are shown in Fig. 2D,E with the corresponding PSTH shown in Fig. 2F. The response consisted of an early spike at a latency of 23.7±0.7 ms (N=26, range 18.2–30.0 ms) followed by a burst of spikes starting at 113.5±4.1 ms (N=30, range 66.9–151.5 ms). None of the motoneurones recorded discharged throughout the reflex, in agreement with a previous study in which single motor units were recorded from the active fin muscle (Paul and Roberts, 1979).

Intramuscular stimulation of the muscle nerve never evoked a short-latency spike or EPSP in a motoneurone, although responses at long latencies (i.e. >5 ms) were sometimes obtained. High spinal stimulation evoked a single monosynaptic spike at a latency of 2 ms (Roberts and Williamson, 1983).

Interneurones

Interneurones were located at depths of 400–2000 μm from the dorsal cord surface, but most (>90 %), including all those responding to the PFR-stimulus, were between 900 and 1600 μm deep, corresponding to the depth of the intermediate and ventral horn grey matter. Fifty-seven of the interneurones responding to the PFR-stimulus were excited, with latencies (to the first spike) falling into three non-overlapping groups of less than 25 ms (short-latency S-units), 30–55 ms (intermediate-latency I-units) and more than 60 ms (long-latency L-units). The spontaneous activity of 10 interneurones was slowed or stopped by the PFR-stimulus.

Short-latency (S) units. Of the 22 S-units studied, 15 responded to the PFR-stimulus with a monophasic discharge pattern which varied, in different units, from a single spike in otherwise silent units to a prolonged burst (often >100 ms) in units with a significant, if slow, spontaneous discharge (Figs 3A, 4A). In some units, the earliest response was an EPSP only, whilst in others it was an EPSP together with a spike. In the recordings shown in Fig. 3A–C, the latency of the

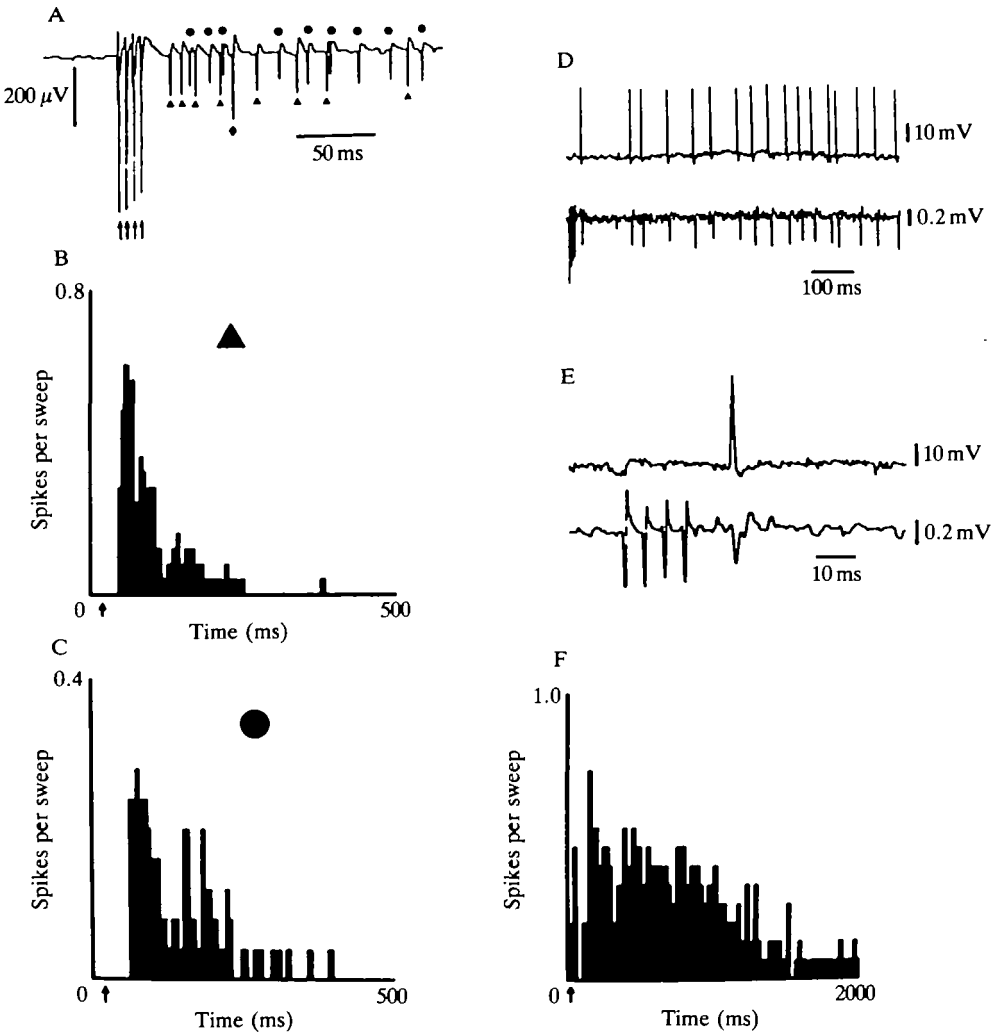

Fig. 2. Motoneurone discharges during the pectoral fin reflex (PFR). (A) Top trace, multi-unit recording from a motor ramus during the PFR (PFR-stimulus pulses marked by arrows). Three distinct axon spikes marked by ●, ▲, ◆ can be seen. (B,C) Post-stimulus-time histograms (PSTHs) of the axon spikes ● and ▲ in A. The beginning of the PFR-stimulus is marked by an arrow on the abscissa. Note the difference in the discharge patterns. (D) Response of a single neurone during the PFR. Top trace, intracellular record from the cell soma; lower trace, simultaneously recorded axon spike in the motor ramus. (E) As in D but at a faster sweep speed. Note the delay between soma and axon spike is just over 1 ms. (F) PSTH of motoneurone discharge during the PFR; the beginning of the PFR-stimulus is marked by the arrow. In this and all subsequent records positivity is upwards.

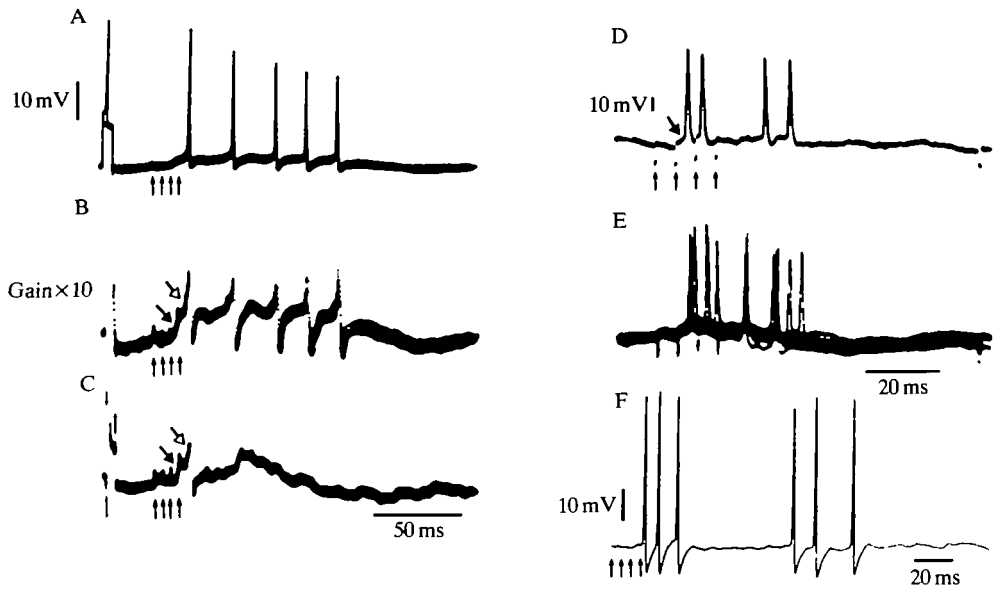

Fig. 3. Responses of short-latency (S) interneurones to the PFR-stimulus recorded intracellularly. (A,B,C) Recording from a unit responding with a burst of spikes. (A) Low gain. At the start of the sweep the unit was fired by intracellular current injection (bridge off balance), followed by responses to the PFR-stimulus (stimulus artefacts too small to register above noise level). Note progressive decline in spike amplitude during the burst. (B,C) High gain. Stimulus pulse artefacts marked by arrows are just visible as brief positive-going deflections. (B) Note that the first EPSP, marked by an oblique solid arrow, fails to fire a spike; summation of a second EPSP, marked by an oblique open arrow, leads to spike generation. Later spikes are preceded by distinct EPSPs superimposed on a persistent depolarization. (C) As in B, but only the initial spike fired. Note the late depolarization with small unitary EPSPs superimposed. (D,E) Another unit firing a short burst only during the PFR-stimulus (pulses marked by arrows). (D) Single response. Note the first EPSP at a latency of about 9 ms (solid oblique arrow) fires a spike. (E) Five consecutive traces superimposed. Note the long-lasting depolarization and marked latency jitter of the spikes. (F) A unit discharging with a biphasic pattern in response to the PFR-stimulus (EPSPs not discernible). The PSTH of this unit is shown in Fig. 4A. The pulses of the PFR-stimulus are marked by arrows.

initial EPSP was only 6 ms and comparing this with the latency of the afferent fibre responses (Fig. 1) suggests that the unit was monosynaptically driven by the afferent input. Even the unit whose activity is shown in Fig. 3D, where the latency of the first EPSP and spike was 9 ms, may well have been monosynaptically driven. These units discharge sufficiently early to be presumptive pre-motor interneurones.

Seven S-units gave a biphasic response to the PFR-stimulus consisting of an initial burst of up to three spikes at a latency of 10–20 ms in the different units, a silent period lasting for 20–70 ms and then a second burst of spikes (Figs 3F, 4A).

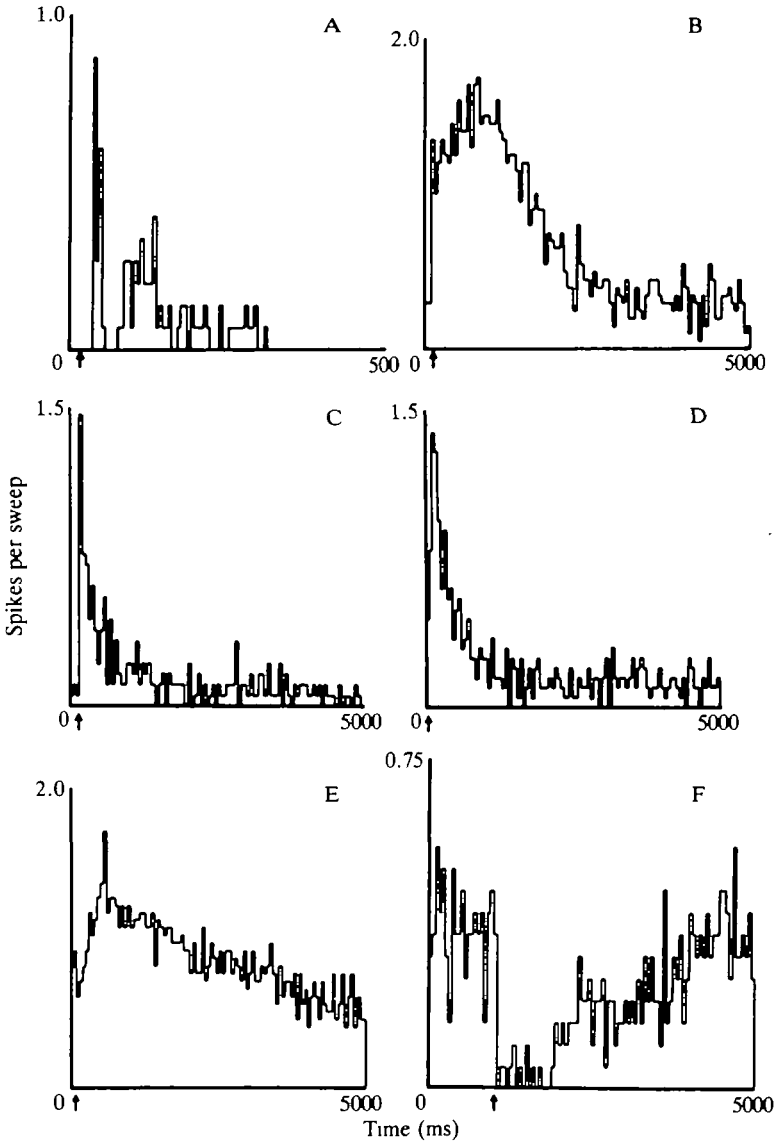

Fig. 4. Post-stimulus-time histograms (PSTH) of interneurones responding to the PFR-stimulus (the stimulus begins at the arrow on the abscissa). (A) An S-unit (illustrated in Fig. 3A) with a biphasic discharge. (B) A spontaneously discharging I-unit showing a long-lasting response of a duration similar to that of the PFR. (C,D) PSTHs of the same I-unit in response to the PFR-stimulus (C) and to a single, high spinal cord stimulus (D). Note the similarity of the two responses. (E) Long-duration burst in a spontaneously active L-unit, in response to the PFR-stimulus. This cell was antidromically driven by a high spinal cord stimulation. (F) PSTH of a unit with its discharge attenuated by the PFR-stimulus.

The records of Fig. 3A–C give some indication of the synaptic events which occur during the response to the PFR-stimulus. The decreasing amplitude of successive spikes (Fig. 3A) is suggestive of a long-lasting increase in membrane conductance, which significantly short-circuited the action potentials. In the record of Fig. 3C, the late burst of spikes did not occur, revealing a substantial and slowly declining depolarization upon which are superimposed EPSPs. This suggests that the late discharge may normally be generated by summation of unitary EPSPs with a long-lasting depolarization (Fig. 3B).

In a number of experiments S-units were fired by intracellular current injection while recordings were made from motor rami. Up to five spikes at a frequency of 100–200 Hz were produced in the unit but no responses were ever evoked in the motor rami, suggesting that a single interneurone was unable to drive a motoneurone, even allowing for temporal summation.

High spinal cord stimulation antidromically excited only one of the S-units; three were orthodromically excited but none was inhibited. Thus, they seem to be a population of interneurones with very few, mainly descending, central connections but which respond at a short, and probably monosynaptic, latency to peripheral stimulation and at longer latency to a second source of input which may utilize a polysynaptic pathway.

Intermediate-latency (I) units. There were 16 units in this group and, apart from the longer latency, the discharge patterns evoked by the PFR-stimulus resembled those of the S-units. Only four were not spontaneously active and had responses limited to 1–3 spikes, whereas spontaneously active units had responses often lasting for hundreds of milliseconds (Fig. 4B).

Eleven of the units were tested for responses to spinal cord stimulation; seven gave an orthodromic response but only one an antidromic response and none was inhibited. Occasional units responded to spinal stimulation with a discharge resembling that produced by the PFR-stimulus (Fig. 4C,D). As a group, I-units have a higher degree of connectivity with the brain than have S-units, but this is still almost entirely of a descending, orthodromic nature.

Long-latency (L) units. Nineteen units responded to the PFR-stimulus at a latency greater than 60 ms. They were presumably small cells, as intracellular penetration was never achieved and the extracellular spikes were always of small amplitude. In six cells which were either otherwise silent or had slow intermittent spontaneous activity, the response was a short burst of 1–3 spikes, occasionally followed by one or two spikes at latencies of at least 500 ms. The remaining 13 active units discharged spontaneously at rates ranging from 1 to 20 impulses s^{-1}. They responded to the PFR-stimulus with a long burst of spikes, which frequently lasted for over 1 s (Fig. 4E), but in most of them there were few or no spikes during the first 30–100 ms following the PFR-stimulus.

Of the 15 units tested with spinal cord stimulation, six gave an antidromic response and six were excited orthodromically. Thus, this group of units has both ascending and descending connections with the brain.

'Inhibited' units. The response to the PFR stimulus in 10 spontaneously active

units was a period of decreased or suppressed discharge (Fig. 4F). In six units the discharge ceased abruptly some 20 ms after the stimulus and, in the other four units, after 45, 50, 150 and 675 ms. The shortest period of inhibition was 70 ms and the longest was over 2 s, but in six of the 10 units inhibition lasted for more than 500 ms (Fig. 4F). It is also evident from Fig. 4F that the attenuation of the discharge was not always complete, but no spike was ever seen within the first 30 ms of the inhibitory period.

Discussion

The PFR exhibits a phenomenon commonly encountered in neurophysiological studies of behavioural responses, namely that a prolonged output is evoked by a relatively brief input and, as pointed out by Jankowska *et al.* (1967), 'reflex actions, which can be sustained because of a long duration of intraspinal processes seem well suited to subserve natural movements'. It is clear that the PFR does not depend on a 'long loop' circuit involving the brain because the reflex has the same pattern in spinal preparations, but brain centres such as the cerebellum can affect the expression of the reflex (Paul and Roberts, 1979).

Although spinal interneuronal activity has been recorded in elasmobranchs (Leonard *et al.* 1978), in lampreys (Grillner *et al.* 1986) and in tadpoles (Roberts *et al.* 1986) related to swimming movement production, and has been extensively studied in mammals, particularly cats (reviewed by Willis and Coggeshall, 1978), there have been no previous reports of long-lasting spinal interneurone discharges following a brief afferent stimulus under experimental conditions similar to those used in this present study. Unlike the cat, the dogfish, in common with other fishes, has no muscle spindles in its body musculature (Bone, 1978) and the closest mammalian analogy to the PFR circuit may be that involving cutaneously activated flexor reflex afferents (FRAs) which exert, through the mediation of an interneurone, a disynaptic action on motoneurones (Eccles and Lundberg, 1959). This analogy is further sustained by the suggestion that, in cats, ascending FRA-derived activity supplies the cerebellum with information about interneuronal actions on motoneurones (Lundberg and Oscarsson, 1962; Lundberg *et al.* 1987). In dogfish, interneurones associated with motor circuits appear to be the major source of input to the cerebellum (Paul and Roberts, 1984).

Activity of spinal neurones during the PFR

Since individual motoneurones were seen to discharge with different patterns during the PFR, its full expression must depend on the combined activity of the pectoral fin motoneurones, which began to discharge only some 20 ms after fin stimulation. The afferent volley reached the dorsal horn in about 4 ms and direct stimulation of afferent fibres did not monosynaptically excite motoneurones: thus, the PFR must involve interneurones. We propose that the phasic part of the PFR depends principally upon the activity of S-units, which we have shown to discharge at the appropriate time. However, motoneurones were never activated when a

single S-unit was multiply fired by intracellular current injection, implying that convergence from several interneurones is needed to drive motoneurones, a view supported by the observation that, whilst some interneurones discharged at a latency of 9 ms, motoneurone latency was at least 20 ms. Presumably too few S-units discharged at this very short latency to drive motoneurones.

The latencies of I-units (35–55 ms) are just short enough for them to contribute to the phasic component of the PFR, whilst those exhibiting prolonged discharges could also contribute to the later, tonic component. However, L-units, with latencies in excess of 60 ms, can contribute to the tonic part of the reflex only.

Neither S-units nor I-units had significant projections to the brain, in contrast to spontaneously active L-units, many of which had both ascending and descending connections. Moreover, their discharges strikingly resembled those of cerebellar cortical and nuclear neurones (Paul and Roberts, 1981, 1983) and L-units could be the major source of afferent input to the cerebellum during the PFR.

Interaction between cerebellum, brainstem and spinal cord

The effects of spinal section and cerebellar ablation on the PFR can now be partially interpreted. Because the reflex remains intact after spinal section (Paul and Roberts, 1979) and high spinal stimulation almost always excites spinal neurones, attenuation of the reflex after cerebellectomy must be mediated *via* a descending pathway that drives spinal inhibitory interneurones. A similar descending inhibitory system has been reported in the stingray and compared to circuits in mammals (Livingston and Leonard, 1985). Presumably cerebellectomy either attenuates activity in a descending excitatory pathway or enhances descending activity driving spinal inhibitory interneurones.

We have suggested that the phasic part of the PFR, which is virtually unaffected by cerebellar lesions, depends principally upon the activity of S-units and, appropriately, these receive very few descending inputs. The marked attenuation of the tonic part of the PFR that follows cerebellar ablation may then depend on modulation of the activity of those I- and L-units which both exhibit long-lasting discharges and receive a descending input. In some I-units, single-shock stimulation of descending pathways led to prolonged discharges which mirrored those seen during the PFR. Clearly such pathways could provide a route for central modulation of the PFR. However, more information about the organization of descending systems is required before we can interpret further the role of the cerebellum.

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