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HINDBRAIN CIRCUITRY MEDIATING COMMON MODE SUPPRESSION OF VENTILATORY REAFFERENCE IN THE ELECTROSENSORY SYSTEM OF THE LITTLE SKATE *RAJA ERINACEA*

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Summary

Elasmobranch fish have an electrosensory system which they use for prey detection and for orientation. Sensory inputs to this system are corrupted by a form of reafference generated by the animal's own ventilation, but this noise is reduced by sensory processing within the medullary nucleus of the electrosensory system. This noise cancellation is achieved, at least in part, by a common mode rejection mechanism. In this study we have examined characteristics of neurones within the medullary nucleus in an attempt to understand the neural circuitry responsible for common mode suppression. Our results are in accord with previous indications that ascending efferent neurones of the medullary nucleus are monosynaptically activated from the ipsilateral electrosensory nerves and project to the midbrain. We demonstrate that in *Raja erinacea*, as has been previously shown in one other species (*Cephaloscyllium isabella*), ascending efferent neurones typically have a discrete focal excitatory receptive field and an inhibitory receptive field which may be discrete or diffuse and which often includes a contralateral component. We identify a group of interneurones within the medullary nucleus which are driven monosynaptically from the electrosensory nerves, have simple discrete excitatory receptive fields and respond vigorously to imposed common mode signals. The simplest model of the circuitry underlying common mode rejection that is consistent with the evidence is that direct afferent input impinges onto the basal dendrites of the ascending efferent neurones and onto interneurones within the nucleus, and the interneurones in turn inhibit the ascending efferents. The pattern of this projection, including commissural inputs, determines the nature and extent of ascending efferents' inhibitory surrounds and mediates the suppression of common mode signals.

Introduction

Elasmobranch fish have an electrosensory system which they use for the detection of bioelectric fields produced by their prey and for orientation (Kalmijn, 1988).

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Electrosensory afferent fibres in elasmobranchs are strongly driven by electrical potentials produced by the animal's own ventilation (Montgomery, 1984; New and Bodznick, 1990). This reafference can be considered as a form of noise which ought to be removed at an early stage of sensory processing. The dorsal octavolateralis nucleus (DON) is the electrosensory centre in the medulla, and the ascending efferent neurones (AENs) from this nucleus do indeed show a significant reduction in ventilatory modulation without sacrificing their response to extrinsic electric fields. The key to understanding the suppression of ventilatory noise is the observation that the ventilatory modulation of afferent activity is the same in all afferents, making it a 'common mode' signal, which could be reduced by subtraction of inputs from different parts of the body. Extrinsic signals are differentially represented within the afferent population and would not be suppressed by such a mechanism. The further observation that an artificial common mode signal (produced by an electrode inserted into the gut) is also suppressed provided good evidence for the existence of a common mode suppression mechanism within the DON (Bodznick *et al.* 1992). Recently it has also been shown that secondary neurones within the medullary nucleus have both excitatory and inhibitory components to their receptive fields, as would be predicted from the common mode rejection hypothesis (Bodznick and Montgomery, 1992). The suppression of common mode signals within the electrosensory system is now a well-established phenomenon in three different species of elasmobranch (Montgomery, 1984; New and Bodznick, 1990; Bodznick and Montgomery, 1992). However, the central pathways subserving the phenomenon are unknown. This study was designed to examine aspects of the circuitry of the DON and is consistent with the hypothesis that common mode suppression is mediated by inhibitory interneurones in the ventral neuropile of the DON rather than by alternative pathways.

Materials and methods

Little skates, *Raja erinacea* Mitchill, were caught during short-duration trawl tows in Vineyard Sound, MA. They were held in chilled sea water until the return to the Marine Biological Laboratory, Woods Hole, where they were transferred into seawater tanks held at 12–15°C. Animals were anaesthetised for surgery by immersion in a 0.007% solution of tricaine methane sulphonate (MS222) in sea water. The cranium was opened, the animal was decerebrated by diencephalic section, and the spinal cord was pithed. The animal was paralysed by intravenous injection of tubocurarine chloride ($3 mg kg^{-1}$). A saltwater bridge electrode made from PE 90 tubing filled with 1.5% agar in sea water was inserted into the gut through the anus. An additional Ag/AgCl electrode made from 0.2mm diameter silver wire, Teflon-coated except near the tip, was implanted through a small skin incision into the interior of the animal in the region between the hyoid and buccal clusters on the head. The skin incision was then sealed with tissue adhesive (Histoacryl, Trihawk). Bipolar cuff electrodes were fixed bilaterally around the hyomandibular ramus of the anterior lateral-line nerve in the region immediately behind the spiracle, and the skin incisions were sealed with tissue adhesive. The animal was then positioned on an acrylic head holder to stabilize the brain for microelectrode recording. A stream of oxygenated sea water directed into the mouth provided for respiration. An

acrylic dam was cemented around the cranial opening, allowing the skate to be fully immersed in sea water, and the temperature in the experimental bath was regulated at 8–10°C. A concentric bipolar stimulating electrode was placed in the lateral mesencephalic nucleus (LMN) on one side, and its depth was adjusted to maximise the evoked antidromic field potential in the contralateral dorsal octavolateralis nucleus (DON). In some animals, concentric bipolar stimulating electrodes were placed in the contralateral DON. These procedures followed NIH guidelines for the care and use of experimental animals and were approved by the Institutional Animal Care and Use Committees of the Marine Biological Laboratory and Wesleyan University.

Electrosensory afferent activity was recorded with glass micropipettes (4mol l^{-1} NaCl; 25 MΩ) inserted into the anterior lateral-line nerve within the cranium. Platinum-black-tipped indium electrodes (2–5 μm tip diameter, 2–7 MΩ) were used to make recordings from neurones within the dorsal octavolateralis nucleus, where ascending efferent neurones (AENs; the principal output neurones) were identified by antidromic stimulation from the contralateral LMN.

The electrosensory system was stimulated with uniform longitudinal and transverse fields and with local dipoles. Uniform fields typically had a maximal voltage gradient of 2 μV cm^{-1} and were modulated sinusoidally at 2Hz. The responses of afferents and central neurones were measured as the peak-to-peak changes in discharge rates from peristimulus time histograms (e.g. Fig. 6). The response of the neurone, or signal level, was taken as the square root of the sum-of-squares of the individual responses to the longitudinal and transverse fields. In effect, this provides a characterisation of the response to uniform field stimulation that should be independent of the orientation of the receptor canal. Dipole electrodes were made from seawater/agar-filled glass tubes and were positioned normal to the skin surface so that the poles were 10 and 15mm from the surface of the skin. Dipole field intensities relative to a distant reference electrode were measured along the dipole axis at a distance of 10mm from the electrode and thus approximate intensities at the skin surface. For experiments on receptive fields of central neurones, up to four dipoles were placed around the fish. Dipole 1 was located in the excitatory receptive field, and was activated by a 5 μV stimulus modulated sinusoidally at 1 Hz. Dipoles at other locations were activated singly, or in concert, by a 2 μV, 200ms square pulse with the cathode (excitatory for the electroreceptors) near the skin of the fish. These pulses were timed to coincide with the excitatory response evoked by the first dipole. Control experiments were carried out during recordings from electrosensory primary afferents to show that these stimulus strengths applied outside the excitatory receptive field did not directly inhibit afferent firing by cathodal stimulation of the capsular region through the skin.

The stimulus delivered through the gut electrode was a 1 or 2Hz sine wave centred about 0V,and applied between the gut electrode and the salt bridges located along all four sides of the experimental bath. This has previously been shown to create an artificial ‘common mode’ stimulus that, like the animal’s normal ventilatory potentials, modulates the activity of nearly all the electroreceptors to the same degree and in common phase (Bodznick *et al.* 1992). The amplitude of the gut stimulus current was adjusted to give a 20 μV peak-to-peak signal measured between the interior of the animal and an indifferent

electrode placed in the seawater bath. These amplitude and frequency values were chosen to approximate normal ventilatory potential modulations recorded from skates (Bodznick and Montgomery, 1992).

Results

Ascending efferent neurones could be identified by antidromic stimulation of the contralateral midbrain (Fig. 1A), as has been described by New (1990). In addition to the antidromic activation, many AENs exhibited a period of inhibition which typically had a latency of approximately 15ms and persisted in most cases even when the stimulus was reduced to a level below threshold for the antidromic activation.

In control experiments, midbrain stimulation had no effect on electrosensory afferents until stimulus levels reached an order of magnitude greater than those used for AEN identification.

AENs with receptive fields including the hyoid group of electroreceptors were monosynaptically activated by electrical stimulation of the ipsilateral hyomandibular nerve (Figs 1B, 2). In almost all cases, excitation was followed by a period of reduced activity (Fig. 1B). This reduced activity cannot unequivocally be attributed to inhibition, because in control experiments investigating the effects of electrical stimulation on electrosensory afferents two effects were observed which could contribute to this period of suppression. First, electrical stimulation of the afferent nerves can result in a resetting phenomenon in which the antidromic spike that invades the peripheral terminal resets the spike-generating mechanism. Subsequent spikes then occur at intervals corresponding to the normal interspike interval of spontaneous activity (mean spontaneous activity in primary afferents is 14impulses s^{-1}, which correspondes to an interspike interval of 71.4ms). The second observed effect was that electrical nerve stimulation could have direct effects on the electroreceptor cells in the periphery. Shocks (10V, 0.5ms) applied to the hyomandibular nerve of (about 2.5 times threshold for the field potential in the DON) produced weak direct effects on buccal receptors, which are innervated by the buccal ramus of the anterior lateral-line nerve. These effects could be either excitatory or inhibitory, depending on stimulus polarity, and had latencies of 20–25ms, typical of electrical activation of electroreceptors.

AENs were typically (17/20) inhibited by a single shock applied to the contralateral hyomandibular nerve (Fig. 1C). The latency between the stimulus and the onset of the inhibition was commonly 10–20ms. Inhibition could also be elicited by stimulation of the contralateral DON. This inhibition is almost certainly mediated through the direct commissural projection between dorsal nuclei because the latencies are too short to involve less direct paths.

The receptive field characteristics of AENs were complex. In almost all cases there was a discrete well-defined excitatory receptive field. Most AENs also received inhibitory inputs, but in different neurones these formed receptive fields which ranged from being discrete to being diffuse, ill-defined and including contralateral input. Fig. 3 illustrates two examples of discrete antagonistic fields. For one unit (a47) the excitatory and inhibitory receptive fields were on the dorsal fin edge (Fig. 3A). For the other unit, the

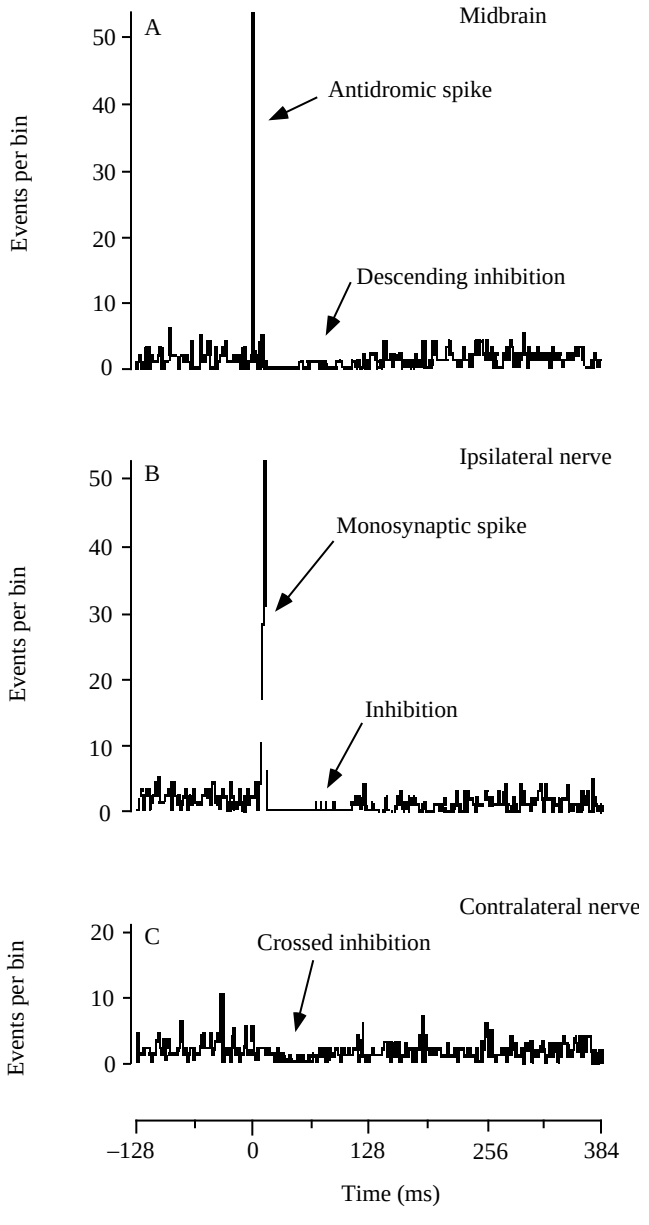

Fig. 1. Peristimulus time histograms of an ascending efferent neurone (AEN) response to electrical stimulation (200 trials; 2ms bin width). The horizontal axis applies to all traces. (A) Response to single shocks applied to the contralateral mesencephalic nucleus (midbrain). The bin with a large number of events corresponds to the time of the antidromic spike. This is followed by a period of descending inhibition. (B) Response to single shocks applied to the ipsilateral hyomandibular nerve. The monosynaptic spike occurs at a latency of 8–10ms and is followed by a period of suppressed firing. (C) Single shocks applied to the contralateral hyomandibular nerve result in a relatively short-latency crossed inhibition.

receptive fields were on the ventral hyoid cluster. When a47 was stimulated with a 1Hz sinusoid presented in the excitatory receptive field (Fig. 3B) its peak excitatory response occurred after approximately 500ms. A 200ms square-wave input applied to the inhibitory field and timed to coincide with the peak excitatory response clearly inhibited the response of a47 and was followed by a post-inhibitory rebound (Fig. 3B).

An AEN with a diffuse inhibitory surround is illustrated in Fig. 4. Its excitatory receptive field was located among the dorso-medial hyoid pore group. Activation of dipole 2 on the fin edge produced a partial inhibition of activity. This inhibition could be augmented by simultaneous activation of dipoles 3 and 4 on the posterior fin margin and on the contralateral fin edge.

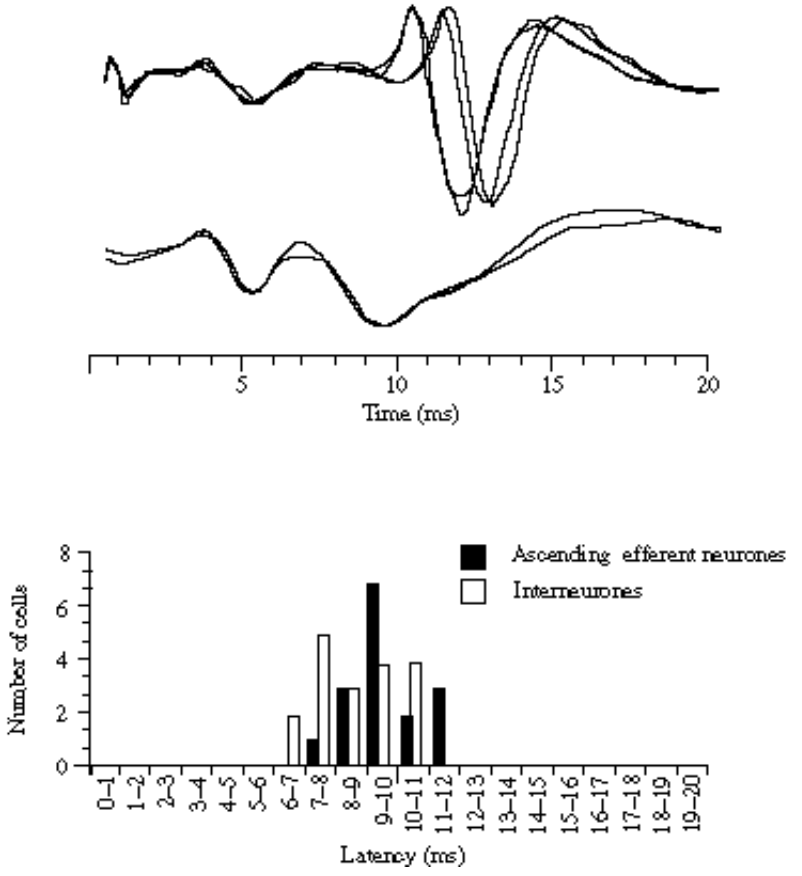

Fig. 2. Monosynaptic responses of interneurones (INs) and ascending efferent neurones (AENs) to stimulation of the ipsilateral hyomandibular nerve. Top trace: four superimposed sweeps of an IN responding with a latency of about 9–10ms to electrical stimulation of the hyomandibular ramus of the anterior lateral-line nerve. Lower trace: a field potential generated by hyomandibular nerve stimulation. The afferent fibre potential (onset latency 3.6ms) is followed by a slow negative synaptic potential (onset latency 7.5ms). The histogram presents AEN and IN response latencies to nerve stimulation.

Intensity response functions for dipole and uniform fields for AENs reflected the arrangement of their antagonistic receptive fields. One example (unit a47) is given in Table 1. The dipole field response increased approximately linearly with voltage increase and showed no sign of saturation at 20 μV. The response to uniform fields declined at stimulus strengths greater than 5–10 $\mu V\,cm^{-1}$; this was particularly evident for transverse fields, where uniform field strengths of 10, 20 and 50 $\mu V\,cm^{-1}$ elicited no response.

The distribution of signal/noise ratios (vector addition of the responses to uniform field stimulation divided by the response to the gut stimulus, see Materials and methods) in AENs is shown in Fig. 5. Most showed a modest improvement in comparison with the primary afferent fibres, and several showed a virtually complete suppression of the imposed common mode signal.

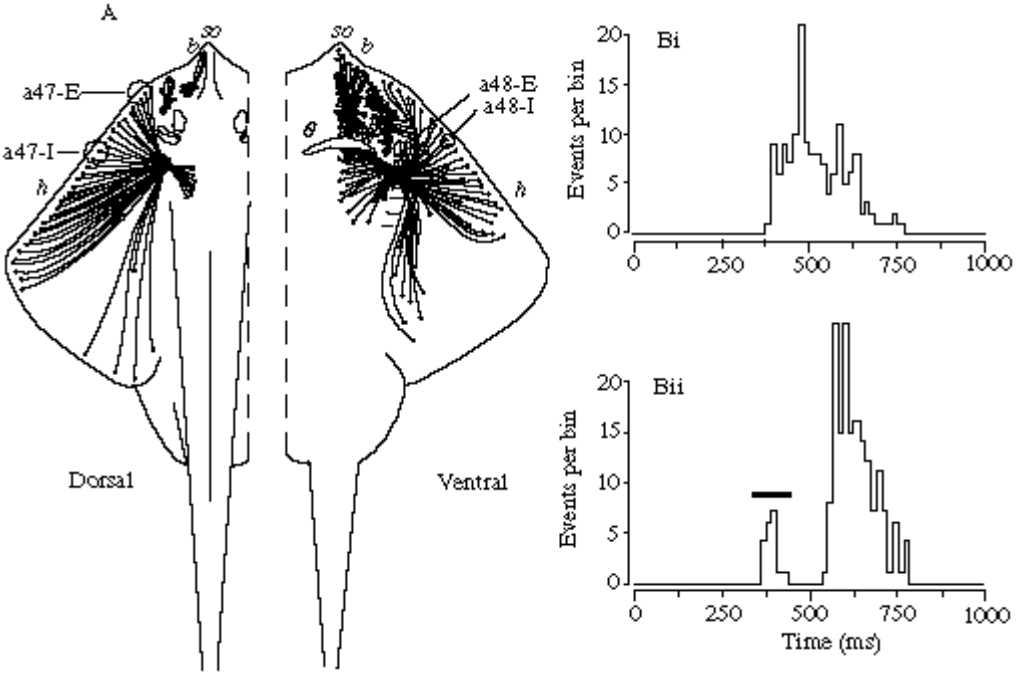

Fig. 3. Examples of ascending efferent neurones (AENs) with discrete antagonistic receptive fields. (A) Drawings of left dorsal and left ventral halves of the little skate showing the distribution of ampullary clusters (dots) (*b*, buccal; *h*, hyoid; *so*, superficial ophthalmic) and electrosensory canals (lines). Unit a47 had two discrete receptive fields on the anterior dorsal fin margin, one (a47-E) being excitatory and the other (a47-I) inhibitory. Unit a48 had two discrete receptive fields on the ventral hyoid cluster (a48-E, excitatory; a48-I, inhibitory). (B) Peristimulus time histograms (50 trials) illustrating the antagonistic receptive fields of unit a47. Bi shows the response to a 1Hz, 5 μV sinusoidal stimulus presented in the excitatory receptive field. Bii shows the effect of simultaneously presenting a 100ms square-wave pulse of 4 μV (indicated by the solid bar) timed to coincide with the excitatory response to the sinusoid. The inhibitory input completely suppressed firing for a duration equivalent to the square-wave stimulus duration. A post-inhibitory rebound also followed the square wave.

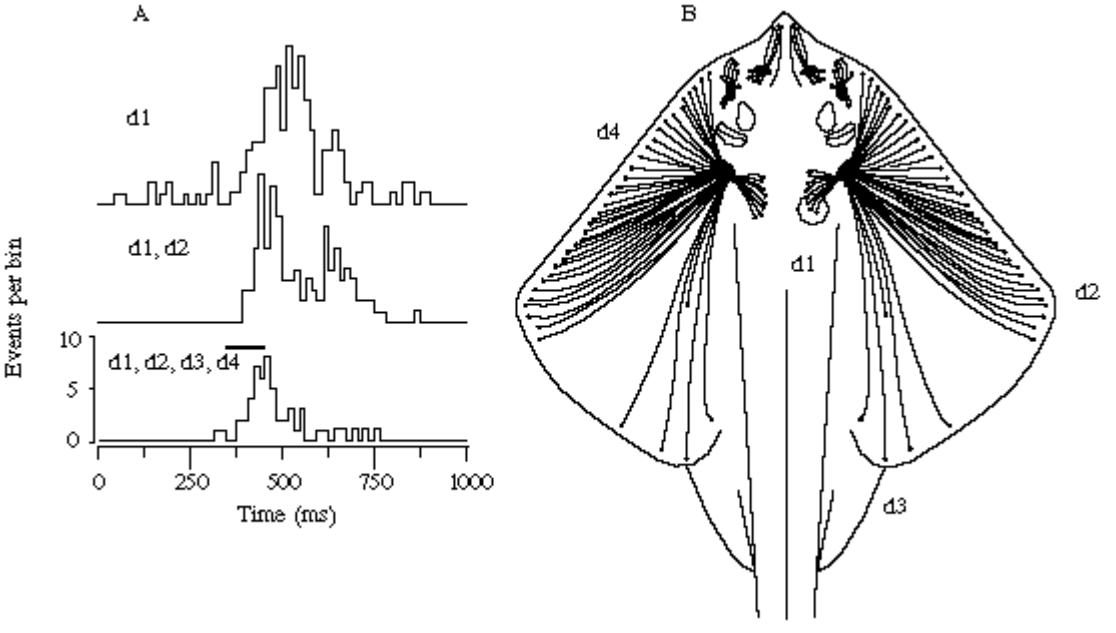

Fig. 4. Example of an ascending efferent neurone (AEN) with a discrete excitatory field (circled) and a diffuse inhibitory field. (A) The top panel shows its response to a 1Hz, 5 μV stimulus presented through dipole 1 (d1) located in the excitatory receptive field on the dorso-medial hyoid pore group. The middle panel shows the suppressive effect of adding a 100ms, 2 μV square-wave pulse (bar) through dipole 2 (d2) located near the lateral fin edge. Simultaneously activation of additional dipoles on the caudal and contralateral fin edges (d3, d4) increased the degree of inhibition (lower panel in A).

Units recorded within the DON which were not antidromically activated by midbrain stimulation could be any one of a variety of types. Primary afferent fibres could be distinguished by their very short latency response (3–5ms) to ipsilateral hyomandibular nerve stimulation. Other units could be AENs which project ipsilaterally (although this projection is very sparse), commissural neurones, interneurones or descending fibres afferent to the DON. One commonly encountered type (20/28 cells) is a class of neurones which in this study have been termed interneurones (INs). INs have the following

Table 1. *Intensity response functions for one ascending efferent neurone (RL, response to a longitudinal uniform field; RT, response to a transverse uniform field)*

	Dipole field (μV)				
	1	2	5	10	20
Response (impulses s^{-1})	7	15	53	58	105

	Uniform field (μV cm^{-1})						
	0.5	1	2	5	10	20	50
RL	8.5	16	16	20	34	32	30
RT	0	10	15	42	0	0	0

characteristics. They are activated from the ipsilateral hyomandibular nerve at latencies comparable to the latencies of the AENs (6–11ms; Fig. 2). They are not inhibited by stimulation of the contralateral nerve or activated at short latency (neither antidromically nor synaptically) by stimulation of the midbrain. They have a relatively high level of spontaneous activity for DON cells. AENs had a spontaneous activity close to zero and

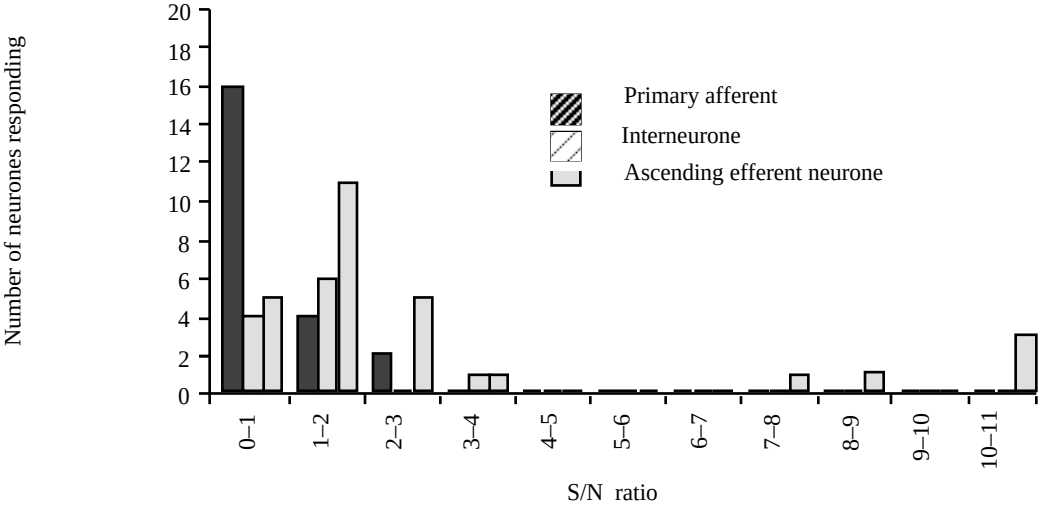

Fig. 5. Histogram of the distribution of signal/noise (S/N) ratios in primary afferents, interneurones and ascending efferent neurones.

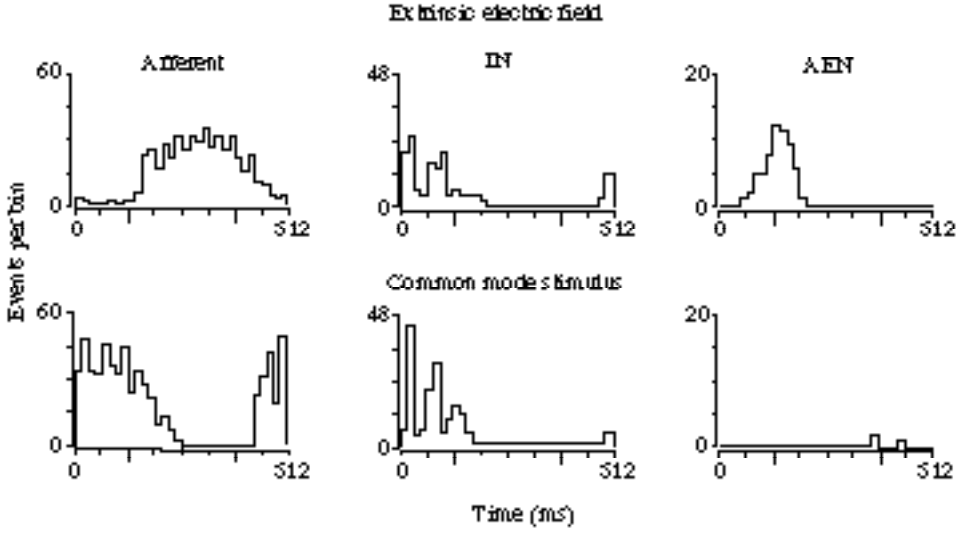

Fig. 6. Illustration of the responses of a primary afferent, interneurone (IN) and ascending efferent neurone (AEN) to extrinsic electrical field stimulation and to a common mode stimulus applied through the gut electrode. Afferent neurones and INs respond vigorously to both stimuli, whereas this particular AEN shows a strong suppression of the common mode stimulus (50 stimulus presentations).

primary afferents had a spontaneous activity of around 14 impulses s^{-1} under these conditions whereas INs averaged 5 impulses s^{-1}. Cells included in this group have simple ipsilateral excitatory receptive fields and respond vigorously to an artificial common mode signal (Figs 5 and 6). The group of cells exhibiting this set of characteristics was designated as interneurones (INs) in this study, but it may also include some commissural neurones.

Other dorsal nucleus neurones showed a variety of responses to electrical stimulation. Two units (out of 29) were synaptically activated by stimulation of the contralateral lateral mesencephalic nucleus (LMN) (latencies 6 and 7.5ms). Longer-latency effects of nerve stimulation are not reported here because of the potential confounding effect of direct electrical stimulation on the electrosensory receptors. However, LMN stimulation (which did not produce direct effects on the afferents) commonly produced a late (latency 30–70ms) weak excitation of DON neurones including INs. The pathway mediating these longer-latency effects is unknown.

Discussion

Ascending efferent neurones (AENs) of the dorsal octavolateralis nucleus (DON) are large multipolar cells with cell bodies in the peripheral zone of the nucleus that extend dendrites out into the molecular layer cap overlying the DON and a set of ventral dendrites into the neuropile or central zone of the nucleus (Bodznick and Boord, 1986; Paul and Roberts, 1977*a*). These ventral dendrites receive input from electrosensory afferents (Paul and Roberts, 1977*b*; Paul *et al.* 1977). The AENs project to the contralateral LMN, thus providing a monosynaptic relay of electrosensory afferent information up to the level of the midbrain. However, we have demonstrated that the responses of AENs differ from primary afferent responses in at least the following two ways. AENs have complex receptive fields, including inhibitory components, and many AENs show a reduced sensitivity to common mode fields, such as ventilatory potentials or the gut stimulus. The evidence of this study is that these signal-conditioning effects can be attributed to the neural circuitry of the ventral neuropile of the DON. Interneurones (INs) within the ventral neuropile of the DON have the characteristics appropriate to mediate the complex receptive fields and common mode suppression seen in AENs. In particular, they receive simple short-latency excitatory inputs from the primary afferents, respond well to common mode stimuli and have relatively high spontaneous firing rates that should permit them to signal both inhibitory and excitatory inputs. Furthermore, the simple receptive fields of INs and their lack of contralateral input indicate that the convergence of information from different INs and from commissural neurones occurs on the AENs themselves. The direct inhibitory connection from INs onto AENs is still conjectural. However, the proposed model (that common mode suppression is mediated by inhibitory interneurones that are activated directly by afferent input) is the simplest model consistent with the evidence.

One alternative model, which has been considered, is that common mode signals could be suppressed by an inhibitory collateral feedback pathway between AENs (Montgomery and Bodznick, 1991). This alternative model provides a number of predictions which are not supported by the evidence presented in the present study. The INs are not activated by

LMN stimulation (*via* the axon collaterals of the AENs proposed in the collateral feedback model); they respond well to common mode signals (the collateral feedback model includes suppression of common mode signals in both INs and AENs); and the evidence points to monosynaptic activation rather than to disynaptic activation (*via* AENs) from the ipsilateral nerve. The synaptic delay in elasmobranch fishes at 15˚C is around 2ms (Montgomery and Roberts, 1979; Montgomery, 1984). The coincidence of the latency histograms of AENs and INs is good evidence that the INs, like the AENs, are monosynaptically activated by the electrosensory afferents.

It is also unlikely that the molecular layer system of the DON mediates the common mode rejection mechanism because the major inputs to the molecular layer are proprioceptive and descending electroreceptive information (Conley and Bodznick, 1989) of a form that would not be appropriate to mediate common mode suppression.

The precise location of the INs has not been determined in this study. It may be in the central or peripheral zones of the ventral neuropile of the DON. An anatomically distinct cell type can be observed in the central zone of the nucleus (Collins and Montgomery, 1989). Commissural neurones, which could share similar properties and hence have been classified as INs in this study, are found in both the central and the peripheral zones. Many of the central zone commissural neurones exhibit GABA-immunoreactivity (Duman and Bodznick, 1991).

The INs characterised in the present study probably correspond to the type I interneurones identified physiologically by New (1990). His type I cells have the same response latency to electric fields as AENs, consistent with our finding that both INs and AENs respond at short latency to afferent nerve stimulation, and his type I cells have focal excitatory receptive fields comparable to the AEN excitatory receptive fields. It is of interest that 2 of 71 type I cells he recorded had their excitatory receptive fields on the contralateral body surface and could have been commissural fibres; they may constitute a special subgroup of our INs.

The receptive fields of AENs in the skate, as shown here, are comparable to those reported in the carpet shark (Bodznick and Montgomery, 1992). They grade from an apparently simple pairing of focal excitatory and inhibitory inputs to focal excitatory inputs with diffuse inhibitory surrounds. However, from our small sample, there appears to be a greater occurrence of paired antagonistic fields in *Raja erinacea* compared with the carpet shark. It is difficult to demonstrate inhibitory inputs exhaustively (Bodznick and Montgomery, 1992), so AENs which appear to have focal excitatory and inhibitory fields may also receive other weak inhibitory inputs that are not revealed in the current circumstances. The nature of the receptive fields has considerable implications for the central processing of electrosensory information. Focal antagonistic fields widely spaced on the animal's surface, and on opposite sides of their ampullary clusters, would be maximally sensitive to uniform voltage gradients; focal antagonistic fields located close together would respond best to dipoles of equivalent size; and diffuse inhibitory inputs could effectively eliminate the response to uniform fields while preserving sensitivity to local dipoles. A reduced response to uniform fields is seen in the results presented in Table 1. The response to a local dipole field increases up to stimulus intensities of 20 μV

and reaches 105impulses s^{-1}, whereas the uniform field response reaches a maximum of 42impulses s^{-1} and decreases at stimulus intensities above 5 $\mu V\,cm^{-1}$.

The degree of noise cancellation exhibited by AENs varies considerably, with most only showing a modest improvement over that shown by primary afferents. It is uncertain whether this degree of variability is real, in the sense that it exists in the animal under normal circumstances, or is an artefact of the experimental situation. In the current experiments, the cranial opening was surrounded by an acrylic dam to allow complete submergence of the fish, particularly the dorsal hyoid group of receptors. However, this procedure did not dramatically improve the signal/noise ratio of AENs. The low signal/noise ratio of some AENs could be due to a number of other factors. Using uniform fields as the standard comparison for sensitivity between primary afferents and AENs will underestimate the S/N ratio for AENs, which are not particularly sensitive to uniform fields because of their inhibitory surround organization. Other contributing factors could be that the gut electrode did not produce a precise common mode stimulus, or that there was a change in skin resistance or central processing parameters as a result of the experimental situation. Despite the fact that common mode suppression, as demonstrated in these experiments, is relatively modest in *Raja erinacea*, the effect has now been clearly shown in three unrelated species of elasmobranch (*Platyrhinoidis triseriata*, Montgomery, 1984; *Raja erinacea*, New and Bodznick, 1990; *Cephaloscyllium isabella*, Bodznick and Montgomery, 1992). Common mode suppression is likely to be a general phenomenon of elasmobranch electroreception, serving to suppress electrosensory reafference generated by the animal's own bioelectric fields that might otherwise interfere with the detection and central processing of biologically important extrinsic electric fields.

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