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ADAPTIVE MECHANISMS IN THE ELASMOBRANCH HINDBRAIN

DAVID BODZNICK[1,*], JOHN C. MONTGOMERY[2] AND MEGAN CAREY[1]

[1]*Department of Biology, Wesleyan University, Middletown, CT 06459-0170, USA* and [2]*School of Biological Sciences, University of Auckland, Private Bag 92019, Auckland, New Zealand*

*e-mail: dbodznick@wesleyan.edu

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Summary

The suppression of self-generated electrosensory noise (reafference) and other predictable signals in the elasmobranch medulla is accomplished in part by an adaptive filter mechanism, which now appears to represent a more universal form of the modifiable efference copy mechanism discovered by Bell. It also exists in the gymnotid electrosensory lateral lobe and mechanosensory lateral line nucleus in other teleosts. In the skate dorsal nucleus, motor corollary discharge, proprioceptive and descending electrosensory signals all contribute in an independent and additive fashion to a cancellation input to the projection neurons that suppresses their response to reafference. The form of the cancellation signal is quite stable and apparently well-preserved between bouts of a particular behavior, but it can also be modified within minutes to match changes in the form of the reafference associated with that behavior. Motor corollary discharge, proprioceptive and electrosensory inputs are each relayed to the dorsal nucleus from granule cells of the vestibulolateral cerebellum. Direct evidence from intracellular studies and direct electrical stimulation of the parallel fiber projection support an adaptive filter model that places a principal site of the filter's plasticity at the synapses between parallel fibers and projection neurons.

Key words: elasmobranch, electrosensory, parallel fiber, sensory reafference, synaptic plasticity, hindbrain.

Introduction

The ampullae of Lorenzini of sharks, skates and rays are the most sensitive electroreceptors known, and this is in keeping with their function as detectors of the very weak natural electric fields that exist in the sea (Murray, 1962; Kalmijn, 1971, 1974). But extremely sensitive receptors are of no use without equally well-developed mechanisms for separating the important weak signals from the meaningless background noise that is often much stronger than the signals themselves. A very common source of noise in sensory systems is self-stimulation created by an animal's own behavior. For the elasmobranch electrosense, ventilatory movements cause potent self-stimulation; the fish literally cannot breath without creating electrosensory inputs that can mask relevant external fields. However, work on the skate electrosense over the last 10 years has demonstrated that selectively eliminating such self-generated noise and other predictable patterns of sensory inflow is a principal function of the very first electrosensory nucleus in the brain. Our efforts to understand how this is accomplished together with related work on electric fishes have provided perhaps the best examples of synaptic plasticity for which the functional role is well understood. In addition, they have allowed us to begin making sense of a striking anatomical organization that has long puzzled researchers, the parallel fiber systems that are a hallmark feature of the hindbrain nuclei of the electrosense, other octavolateralis sensory systems and the cerebellum.

Extracting weak signals from noise

In skates and other elasmobranchs, the weak modulations in electroreceptor firing frequency caused by behaviorally important external fields ride on top of nearly continuous and strong modulations due to electric fields generated by the fish's own breathing movements. However, the principal neurons of the dorsal nucleus (termed ascending efferent neurons, AENs) quite effectively ignore the ventilatory reafference and respond well only to the environmental fields (Fig. 1). This is despite the fact that they receive their electrosensory input monosynaptically from the receptors. The ventilatory noise is nearly continuous, so it cannot be eliminated simply by shutting off the electrosensory neurons during breathing movements. Rather, the cancellation mechanisms must selectively null the reafference while keeping the neurons near their firing threshold and responsive to external fields.

The potentials created by the fish's breathing are nearly uniform stimuli for the entire receptor array, and so some of the noise can be discarded through lateral inhibitory interactions which permit the AENs to select for local contrast in the electrosensory surround (Montgomery, 1984; New and

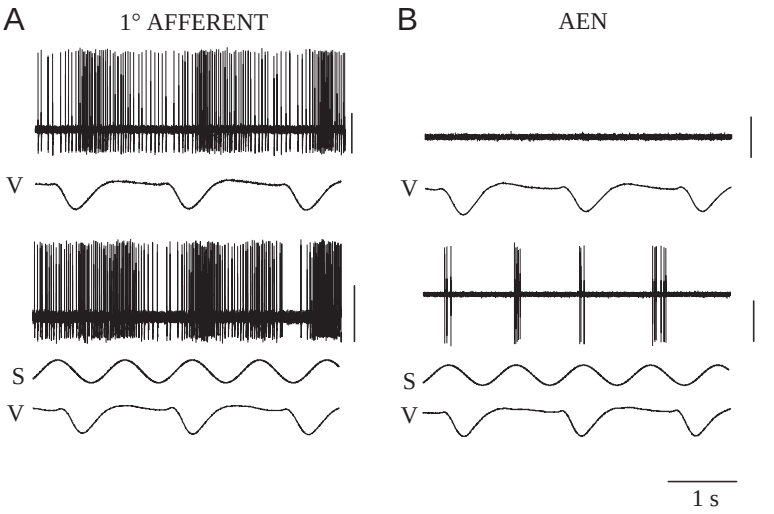

Fig. 1. In the skate electrosense, ventilatory self-stimulation is selectively eliminated between the primary electroreceptor afferent fibers and the second-order electrosensory neurons in the brainstem (ascending efferent neurons, AENs). (A) Responses of an electrosensory afferent to the fish's own ventilatory movements (top) and when a dipole electric field stimulus is superimposed on the activity driven by ventilatory movements (bottom). (B) Responses of an AEN taken just minutes after the afferent recording. The bottom trace in each set of recordings is the output of a force transducer signaling ventilatory movements (V). The dipole stimulus (S), indicated by the sinusoid in the lower traces, is 1 Hz, 2 μV peak-to-peak amplitude at the skin surface relative to a distant reference. Vertical scale bars, 200 μV.

Bodznick, 1990; Bodznick et al., 1992). Lateral inhibition is a familiar feature of the initial processing in many senses. More of a surprise was the recent discovery of a more versatile adaptive filter mechanism in the dorsal nucleus that eliminates any residual ventilatory noise as well as reafference created by swimming and other movements that is not uniform across the receptor array (Bodznick, 1993; Montgomery and Bodznick, 1994). This discovery was presaged by much earlier work on a modifiable efference copy mechanism for reafference reduction in the mormyrid electrosense (Bell, 1982), and it is now apparent that the same basic mechanism also exists in the gymnotid medulla (Bastian, 1995) and the first-order lateral line nucleus of at least one teleost (Montgomery and Bodznick, 1994). Here, we review the properties of the electrosensory adaptive filter in the elasmobranchs and discuss the specific adaptive filter model of Montgomery and Bodznick (1994). Evidence for the model and for a central role of the parallel fiber projection in the mechanism is then considered.

Adaptive filter: properties and model

The term adaptive filter has been used to describe what appears to be an additive mechanism for selectively eliminating a wide range of different patterns of reafference within the principal neurons of the dorsal nucleus without affecting their responsiveness to external stimuli (Montgomery and Bodznick, 1994). The adaptive feature of the filter led to its discovery. The basic observation is that, when an electrosensory stimulus is presented consistently time-linked to a skate's breathing movements, the principal neuron (AEN) 'learns' within minutes to ignore the stimulus selectively (Fig. 2A). The link to the ventilatory movements is essential. The response to the same stimulus presented repeatedly at a similar rate but free-running with respect to ventilation does not decline (Fig. 2B). The filter mechanism is remarkably specific in several respects (Bodznick, 1993). It is specific to (1) the time within the ventilatory cycle when a stimulus is presented; after the period of coupling, the AEN will still respond vigorously to the same stimulus offered at other times within the cycle; (2) the spatial location of a stimulus; the same stimulus similarly linked to breathing but presented outside the particular AEN's receptive field has no effect on that neuron's response when the stimulus is subsequently offered within the receptive field; and (3) the polarity of a stimulus, excitatory *versus* inhibitory.

The mechanism appears to be additive. When the coupled extra stimulus is suddenly removed, an inverted replica of the initial response to that stimulus is apparent in the AEN's firing pattern during ventilation (Fig. 2A). Bell (1982) termed this a negative image when he saw it after coupling sensory stimuli to the electric organ discharge (EOD) command in mormyrids, and it is the characteristic feature of the adaptive filters in each of the other systems noted earlier. The negative image fades over several minutes as the AEN relearns the original pattern of reafference. This negative image is thought to reflect the existence of a cancellation signal input that is specific to each AEN and is the negative of the anticipated reafference reaching that cell. This input can apparently be altered within minutes to match new patterns of reafference. In the simplest model, the cancellation signal simply adds to the reafferent input to remove it, leaving the neuron ready to respond to other stimuli.

The distinctive anatomical organization of the medullary electrosensory and lateral line nuclei is a key to understanding the adaptive filter mechanisms. The AENs, like the principal neurons in each of the other nuclei, have separate basal and apical dendritic arborizations extending, respectively, into a ventral neuropil and a broad overlying molecular layer. The electrosensory afferents synapse directly onto the basal dendrites, while the apical dendrites receive synaptic input from tens of thousands of parallel fibers and the stellate interneurons in the molecular layer (Paul and Roberts, 1977; Paul et al., 1977). The parallel fiber projection originates in the dorsal granular ridge (DGR) adjacent to the nucleus (Boord, 1977; Schmidt and Bodznick, 1987). Electrophysiological studies of the DGR indicate that the parallel fibers carry central copies of motor commands, including ventilatory commands

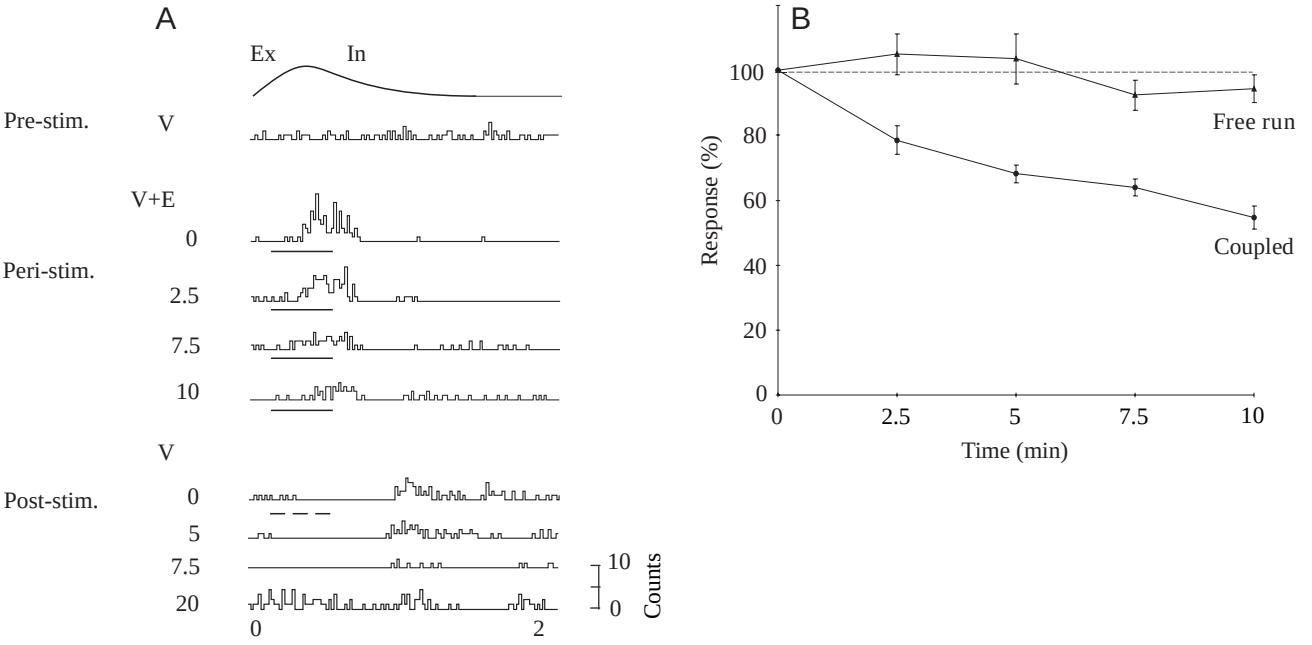

Fig. 2. Ascending efferent neurons (AENs) learn to ignore selectively external sensory stimuli presented time-linked to the skate's own ventilatory movements. (A) Each graph is a peristimulus time histogram (30 trials) of AEN spiking. Histograms were collected from top to bottom at the times (min) indicated from the onset of the stimulus and post-stimulus periods. The onset of the histogram in each case is triggered by the start of the ventilatory cycle depicted at the top. Ex, exhalation; In, inhalation; Pre-stim. V, ventilation alone; Peri-stim. V+E, 2 μV local dipole stimulus presented as a direct current step phase-locked to ventilation, as indicated by the solid line beneath the histograms. Note the presence of the negative image of the initial response in the AEN's activity immediately after the coupling (Post-stim. V, lowest four traces). Dashed line below the histogram indicates the period of the previous dipole stimulus. (B) The response declines when a stimulus is presented time-linked to the skate's ventilatory movements but not when the same stimulus is delivered free-running with respect to ventilation. The graphs show the mean relative response amplitudes (± S.E.M.) for 24 AENs tested with the dipole stimulus coupled to ventilation and with the dipole presented free-running at a rate slightly higher or lower than ventilation.

(Hjelmstad et al., 1996), propriosensory signals related to movements and descending electrosensory feedback (New and Bodznick, 1990; Conley and Bodznick, 1994). The striking feature of this anatomy is that the AENs, as the second-order electrosensory neurons, actually receive thousands of times as many inputs from parallel fibers, carrying mostly non-electrosensory information, as they do from the electroreceptors. The parallel fiber projection is the proposed source of the cancellation signals.

In the model of Montgomery and Bodznick (1994) (Fig. 3), the parallel fiber projection represents a rich matrix of reference signals (sensory and motor corollary discharge) with a wide range of specific temporal relationships to the fish's behaviors. To construct the cancellation signal, the AEN need only select from this matrix the appropriate subset of reference signals whose activity is reliably correlated, positively or negatively, with the reafference. This selection of inputs can be accomplished through adjustments in the strength of the parallel fiber synapses onto the AEN following two simple learning rules. (1) Reduce the gain of synapses from parallel fibers that are consistently active when the AEN is active. This has the effect of reducing molecular layer excitatory input at those times during the behavior when the AEN is activated by the reafference. (2) Increase the gain

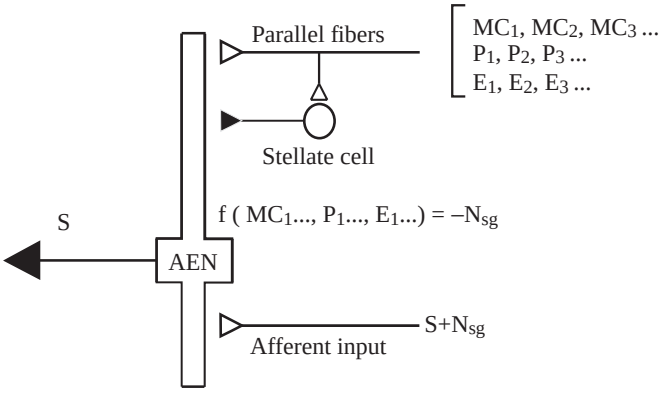

Fig. 3. Model for the adaptive filter. From a matrix of molecular layer inputs that includes motor corollary discharge (MC_1..), propriosensory (P_1..) and descending electrosensory (E_1..) signals, the ascending efferent neurons (AENs) extracts a cancellation input (f) equal to the inverse of the self-generated noise ($-N_{sg}$). The selection of the cancellation inputs is accomplished by adjustments in the weightings of the molecular layer synapses following two learning rules described in the text. Signals (S) only escape the filter because they cannot be predicted by any of the molecular layer inputs (after Montgomery and Bodznick, 1994). Open triangles, excitatory synapses; filled triangle, inhibitory synapse.

of synapses from parallel fibers that are consistently active when the AEN is not. This adds excitation when the AEN is otherwise inhibited by the reafference. The inhibitory stellate cell synapses onto the AEN may also be plastic following the inverse learning rules, or they may simply provide background inhibition against which excitatory parallel fiber input is added or removed. Like cerebellar long-term depression (Ito, 1989), the proposed parallel fiber synaptic gain changes are anti-Hebbian, i.e. they act to reduce correlated activity of the pre- and postsynaptic neurons. In addition to nulling the reafference, the same synaptic plasticity mechanisms should also act in negative feedback fashion to prevent the molecular layer inputs themselves from driving the principal neurons or burying them with inhibition. This normalization function should keep the neuron near its firing threshold.

According to this model, the memory of the specific cancellation signal input to each AEN is stored in the relative strengths of its synaptic inputs from the subset of parallel fibers that are consistently active during the behavior. It is presumed that these synaptic weightings, once established, are relatively stable through time. The plasticity, which is the most striking feature of the filter, would only be needed to update the filter to accommodate changes in the reafference associated with a behavior. Different behaviors will generate different patterns of reafference, but they will also be accompanied by activity in different subsets of parallel fibers that provide the required cancellation inputs.

Tests of the model

Computer modeling

Nelson and Paulin (1995) tested the feasibility of these ideas using a computational model of the proposed network interactions and anti-Hebbian synaptic plasticity mechanisms. They incorporated the known anatomical circuitry and used mathematically accurate descriptions of neuron response properties from physiological data. The parallel fibers carrying signals related to ventilation were modeled as a set of half-wave sinusoids with various durations and phase relationships to the ventilatory cycle. The model easily reproduced the results of the physiological coupling experiments described above and, through a combination of common-mode rejection and the hypothesized molecular layer plasticity, it was able to suppress all reasonable patterns of ventilatory reafference. The model worked well when either just the parallel fiber or just the stellate cell synapses onto the AEN were adjustable, but it was most robust when both exhibited plasticity. Finally, the model provided an illustration of the usefulness of the second-stage adaptive filter for dealing with even common-mode noise such as ventilatory reafference. The presence of an interneuron in the inhibitory path of the common-mode rejection circuits, unless compensated in some way, will lead to mismatches in the timing of the excitatory and inhibitory inputs to an AEN and thus to an incomplete subtraction of the ventilatory reafference. This residual noise can then be effectively cancelled by the adaptive filter.

Independent contributions of motor commands, propriosensory and electrosensory signals to the cancellation signal

The adaptive filter model proposes that the cancellation signal comprises independent contributions from proprioceptive signals, motor commands and descending electrosensory feedback, i.e. each type of reference signal represented in the parallel fiber array. To test this aspect of the model, we have performed experiments on paralyzed skates, recording AEN responses to local sensory stimuli consistently paired with passive fin movements, ventilatory motor commands or whole-body electrosensory stimuli.

Conley and Bodznick (1994) reported that many propriosensory units in the DGR demonstrate activity related to the positions and movements of the ipsilateral pectoral fin that forms the expanded body disk of skates. Undulations along the fin provide the propulsive force for swimming. This arrangement supports the idea that the parallel fiber system also contributes to cancellation signals for suppressing reafference created by swimming and other fin movements. When we coupled local electric field stimuli to passive fin displacements in paralyzed skates, the responses of many AENs diminished significantly with time, as predicted. A more robust effect was the negative image of the initial response to the coupled stimulus, which was evident in the AEN's activity during the fin movement immediately after the pairing ended (Fig. 4B). The absence of a significant decline in the response to the coupled stimulus in many cells that, nevertheless, show a significant negative image after the coupling, has been a common finding in all the coupling experiments, including those with natural ventilatory movements. This is presumed to be because the suppression of the response to the local electrosensory stimulus is beyond the working range of the mechanism, at least under these test conditions.

Using the ventilatory motor discharge recorded from the seventh cranial nerve in paralyzed skates, we have also recorded AEN responses to local electrosensory stimuli presented phase-locked to the fictive ventilatory cycle with just the same outcome. The responses to the coupled sensory stimulus declined significantly in some cases, and in these and other cells the offset of the coupled stimulus revealed a negative image of the response in just the same phase of the fictive ventilatory cycle. As the responses of the neuron depicted in Fig. 4A illustrate, the filter mechanism appears to be able to supply cancellation input specific to virtually any phase of the cycle. The same result is seen with unparalyzed animals and when sensory stimuli are paired with normal ventilation.

Finally, the DGR also receives descending electrosensory inputs, and many of the same units recorded there respond well to the ventilatory reafference and other common-mode stimuli (Conley and Bodznick, 1994; Hjelmstad et al., 1996).

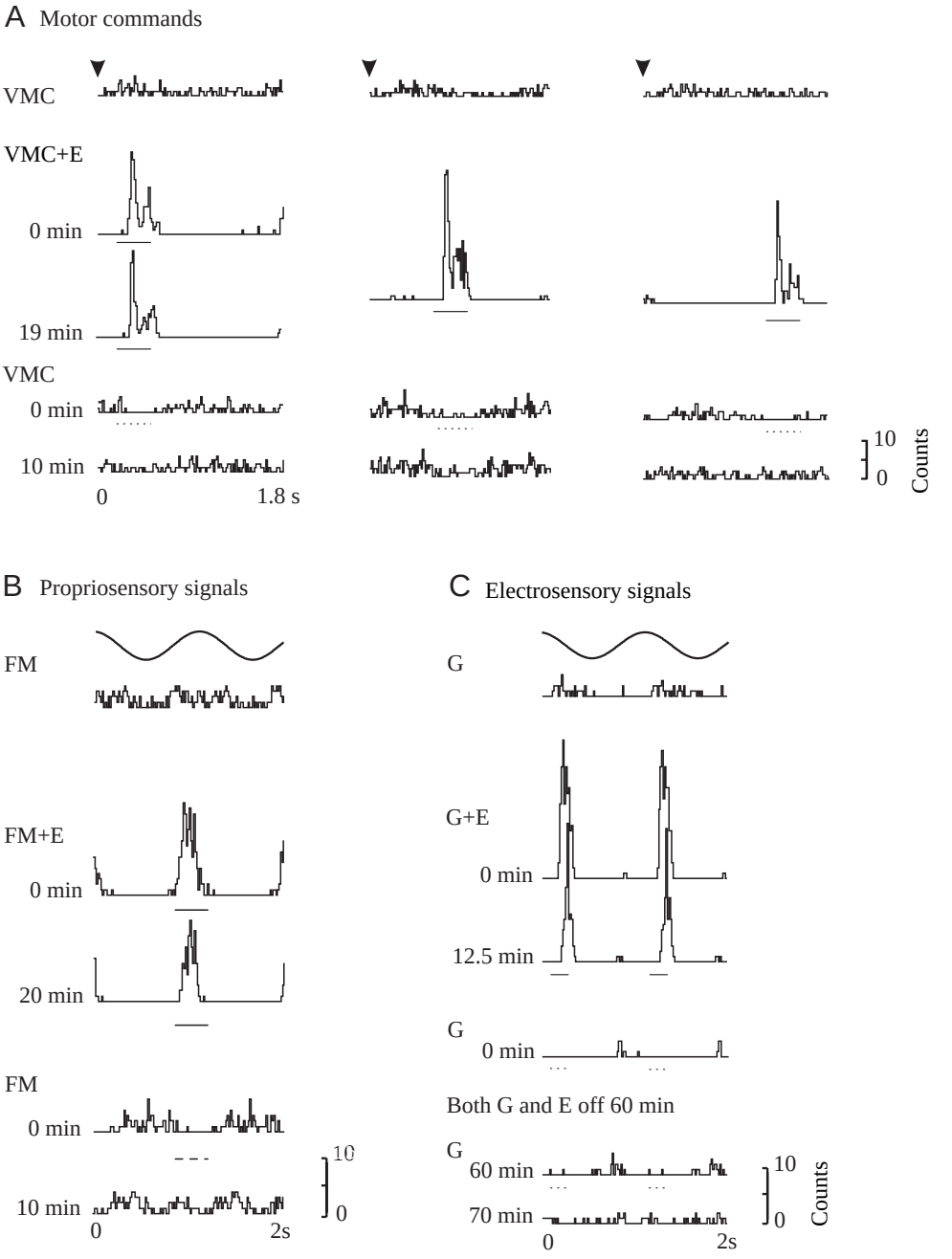

Fig. 4. Central copies of motor commands, propriosensory and electrosensory signals each contribute to the cancellation signal inputs to the ascending efferent neurons (AENs). In paralyzed skates, coupling a local electrosensory stimulus to ventilatory motor commands, passive fin movements or whole-body electrosensory stimuli results in the development of cancellation inputs and the negative images characteristic of the adaptive filter mechanism. (A) A 2 μV direct current step dipole electric field (E; indicated by the line beneath histograms) was presented time-linked to the ventilatory motor command (VMC) recorded in the seventh cranial nerve. Each column represents a separate test series with the dipole coupled at different delays relative to motor command onset (arrowheads). Note the negative images at dipole offset. Dashed lines indicate the period of the now-missing dipole. (B) A 2 μV dipole stimulus is coupled to passive movement of the ipsilateral pectoral fin (FM, sinusoid at top). The responses of the AEN to the dipole decline during the coupling period and are replaced by a negative image of the response when the dipole is discontinued (bottom two traces). (C) A dipole stimulus is coupled to a whole-body 10 μV sinusoidal electric field stimulus (indicated by sinusoid at top) delivered through the fish's gut (G). After the period of coupling, the cancellation signal, or negative image, associated with the gut stimulus persists for at least an hour. It then fades as the gut stimulus is resumed in the absence of the dipole. Each panel is arranged and labeled as in Fig. 2A.

We assume that these units receive their inputs indirectly relayed from the subset of AENs that do not suppress ventilatory reafference effectively. It is easy to see that parallel fibers whose activity is driven by the ventilatory reafference would be well suited to contribute to the cancellation inputs needed to suppress activity in the AENs that is also correlated with ventilation. In a paralyzed skate, we paired local dipole electrosensory stimuli with common-mode electric field stimuli delivered through an electrode inserted into the fish's gut. The result was as expected: the response of the AENs to the coupled dipole stimulus gradually declined in several cases, and in more cases the characteristic negative image was seen at stimulus offset (Fig. 4C). Pairing with the gut stimulus also allowed us to test one other assumption of the model, that the cancellation signals and their underlying synaptic weightings are relatively stable and are only altered during updating. If, at the end of the period of pairing, the dipole sensory stimulus and the gut stimulus were both turned off simultaneously, then after periods of an hour or more when the gut stimulus was restarted the negative image was still present. It then faded in the usual way over the next several minutes as the gut stimulus continued without the coupled dipole (Fig. 4C). Testing in this way, we have found that the cancellation signals persist for at least 3 h, which is the longest time tested.

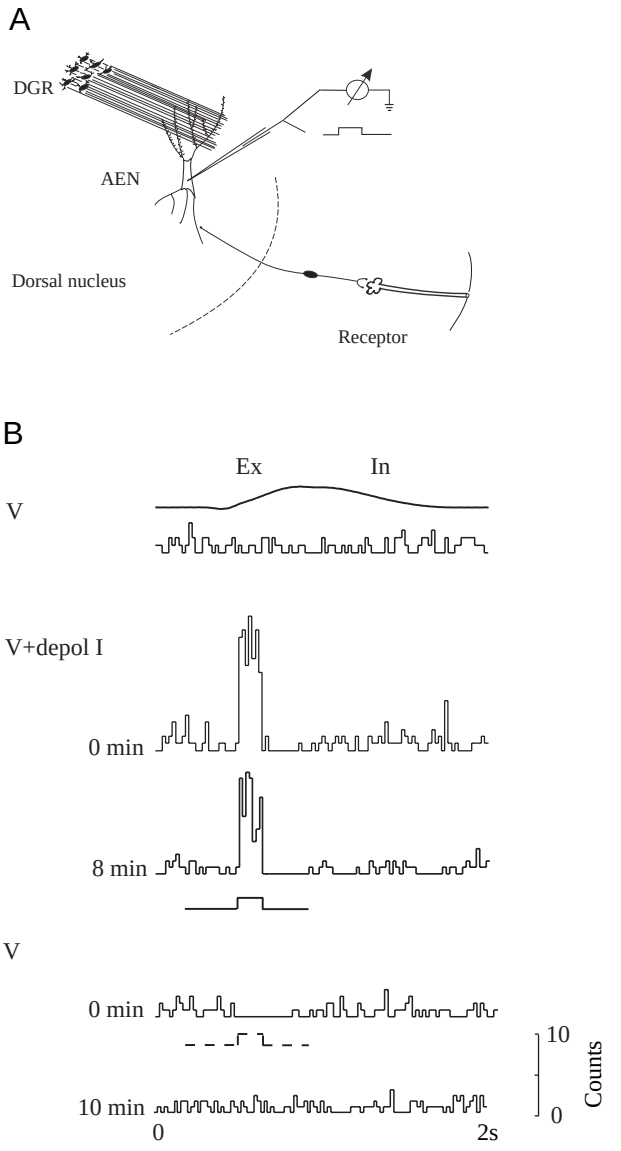

Fig. 5. In freely breathing skates, the responses of an ascending efferent neuron (AEN) to intracellular depolarizing current injection decline when coupled to the fish's ventilatory movements. (A) The experimental arrangement (DGR, dorsal granular ridge). (B) The histograms are arranged and labeled as in Fig. 2A. A depolarizing current step (0.4 nA, 150 ms) was injected into the AEN at the times indicated by the line under the histograms (V + depol I). Note the negative image of the response at stimulus offset (bottom traces). The dashed line indicates the period of the previous current injection. Ex, exhalation; In, inhalation.

Intracellular current stimuli

In an important experiment for understanding the adaptive filter mechanisms, Bell et al. (1993) showed that, in mormyrids, coupling intracellular current stimulation of the principal neurons to the EOD command elicited changes in the effects of the EOD command on the principal neurons similar to those after coupling electrosensory stimuli. By minimizing network effects in this way, the experiments provided evidence that the plasticity of the efference copy inputs was local and probably at synapses directly onto each principal neuron. Bastian (1996) provided a similar demonstration in gymnotid electrosensory lateral lobe (ELL) pyramidal neurons. Carey (1997) and Bodznick et al. (1996) recently recorded intracellularly from AENs in freely ventilating skates for extended periods to test the efficacy of intracellular stimulation for inducing changes to the cancellation signal inputs. In skates, stimulating an AEN with intracellular current pulses repeatedly coupled to the animal's ventilatory movements for 5 or 10 min led to distinct and temporally specific negative images in the firing pattern of the AEN after the coupling (Fig. 5). Both hyperpolarizing and depolarizing currents induced compensatory changes, although depolarizing currents were much more robust. Finally, it was found that blocking AEN responses to a coupled local excitatory sensory stimulus with simultaneous hyperpolarizing current pulses blocked the development of the cancellation signal otherwise induced by the sensory stimulus. The results are consistent with the model, and the proposed learning rules in which the changes in the efficacy of the synaptic inputs to each AEN are based on the activity of that same AEN.

Direct evidence of plasticity at molecular layer synapses

By directly activating a beam of parallel fiber inputs onto the AENs while presenting electrosensory stimuli, direct evidence for the hypothesized plasticity of molecular layer synaptic inputs has been obtained (D. Bodznick and B. W. Larner, unpublished observations). Activation of a parallel fiber beam with either single shocks or short trains delivered locally in the DGR typically results in brief excitation followed by prolonged inhibition of firing in the AENs, presumably reflecting parallel fiber and then stellate interneuron synaptic inputs as seen in the cerebellum. As Fig. 6 illustrates, coupling sensory stimuli to a train of threshold shocks to the DGR produced an effect nearly identical to coupling the sensory stimulus to ventilation. The response to the coupled sensory stimulus declined gradually, and the compensatory molecular layer input, or cancellation signal, was plainly evident in the effect of the parallel fiber stimulation on the AEN after the coupled stimulus had been removed. Both excitatory and inhibitory sensory stimuli predictably altered the effects of the molecular layer stimulation. The experiments also provided evidence for plasticity in the inhibitory synapses. An excitatory sensory stimulus coupled to the period just after the offset of the parallel fiber stimulus train resulted in a strong inhibitory cancellation signal input to the AEN at this period when there should presumably be no excitatory synaptic drive. The results clearly indicate that normal afferent input on the ventral dendrites of the AENs can alter the effectiveness of the molecular layer inputs on the apical dendrites in a distant part of the cell.

DGR lesions and plasticity

One additional test of the model was to determine whether

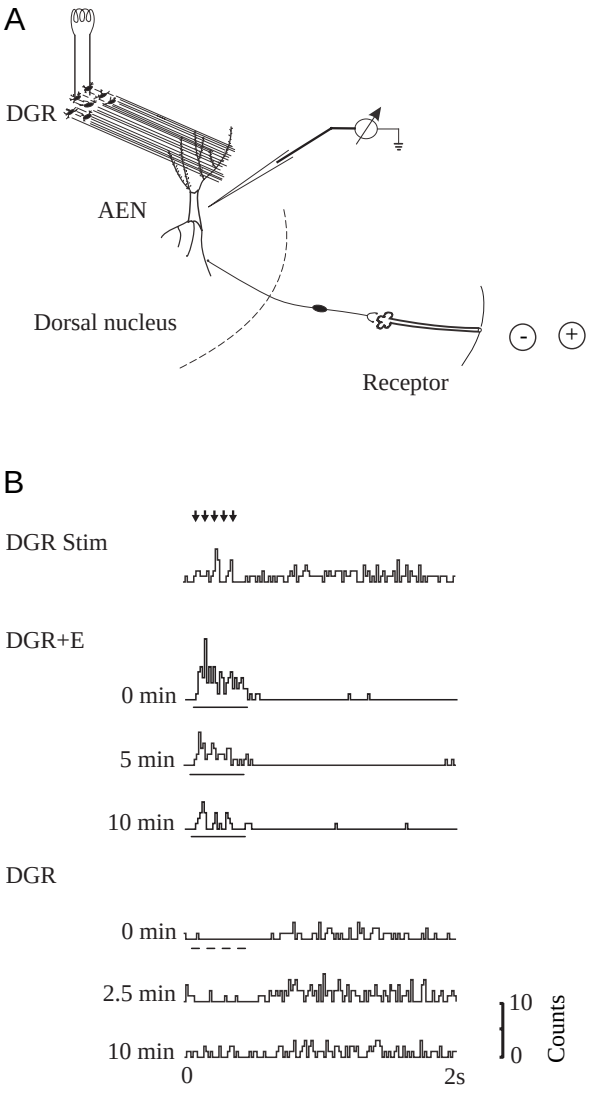

Fig. 6. The responses of an ascending efferent neuron (AEN) to a dipole electrosensory stimulus gradually declined when the stimulus was presented time-coupled to direct electrical activation of the parallel fiber inputs (DGR). As in Fig. 5, A illustrates the experimental arrangement. In each of the histograms of AEN firing in B, the parallel fibers were activated by just suprathreshold stimulation of the DGR (DGR Stim) with a 300 ms, 50 Hz train of 3.5 V pulses (as indicated by arrows above the top histogram). The coupled 2 μV dipole stimulus (E) was presented at the times indicated by the line beneath the histograms (DGR+E). Each histogram represents the results of 30 trials. As a result of the coupling, the effect of the parallel fiber activation on the AEN is changed from weak excitation to strong inhibition (compare the top histogram before coupling with the histogram immediately after the coupling, 0 min).

an intact parallel fiber projection was necessary for the adaptive filter mechanism (Bodznick et al., 1996). Large unilateral lesions predictably eliminated all traces of the plasticity in most AENs as measured by the absence of both a significant decline in the responses to stimuli coupled to ventilatory movements and in the negative image following the coupling. However, in a few cells, including one from the animal with the largest DGR lesion (approximately 98 %), clear evidence of the plasticity persisted. The results are consistent with an important role of the parallel fiber projection in the adaptive filter mechanism, but also suggest that there may be contributions from other inputs yet to be identified.

Discussion

It is now apparent that the adaptive mechanism for removing unwanted reafference discovered in mormyrid electric fishes and well studied in gymnotids is much more than a specialization of the unusual active sensing of electric fishes. We have shown in elasmobranchs that an array of central copies of motor commands along with propriosensory and electrosensory feedback signals can all contribute to the construction of a modifiable cancellation input that effectively removes the reafference associated with ventilation, fin movements and undoubtedly other behaviors from electrosensory signals as they enter the brainstem. The parallel fiber projection onto the medullary principal neurons is the common feature in the three independently evolved electrosensory systems and the mechanosensory lateral line in which the adaptive filters are known, and it appears to be the principal source, although not the only source (see Bastian, 1996; Wang and Maler, 1997), of the cancellation signals. Good direct evidence now exists in each of the separately evolved electrosensory systems (Bell et al., 1997a,b; Bastian, 1998) that the molecular layer synapses onto the principal neurons exhibit the necessary plasticity for the adaptive filtering.

The anatomical organization of parallel fiber projections in the hindbrain octavolateralis nuclei and in the cerebellum in all vertebrates has been both intriguing and enigmatic. Each principal neuron (or Purkinje cell) receives seemingly indiscriminate input from tens of thousands of fine-caliber parallel fibers in a densely packed array. Each granule cell in turn makes *en passant* synapses with a very large number of the principal neurons. This pattern of convergent and divergent connectivity seems hopelessly non-specific. But the difficulty of understanding such an organization seems to disappear when it is viewed in a well-understood functional and behavioral context. In the electrosensory and lateral line systems, the parallel fiber organization is a space-efficient way of presenting the principal neurons with a matrix of all available internal reference signals that includes all the information the animal has about its own behaviors. The useful signals are recognized and selected through well-established synaptic plasticity mechanisms that are gauged by their effect on the output of the principal neurons. The parallel pattern of connectivity is only non-specific if viewed in the absence of such output-directed synaptic plasticity. This is yet another good example of the importance of a behavioral context for understanding the basic principles of nervous system organization.

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