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Pyramidal-cell plasticity in weakly electric fish: a mechanism for attenuating responses to reafferent electrosensory inputs

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Abstract Recordings within the posterior eminentia granularis of the weakly electric fish, *Apteronotus leptorhynchus*, revealed multiple types of proprioceptive units responsive to changes in the position of the animal's trunk and tail. Intracellular labelling showed that the proprioceptor recordings were made from axons that ramify extensively within the EGp. The location of the somata giving rise to these axons is presently unknown. Electroreceptor afferent responses to electric organ discharge amplitude modulations caused by movement of the animal's tail were compared to responses caused by electronically generated AMs of similar amplitude and time course. These did not differ. Electrosensory lateral line lobe pyramidal cells responded significantly less to electric organ discharge amplitude modulations caused by changing the animal's posture as compared to electronically produced AMs, suggesting that central mechanisms attenuate pyramidal cell responses to reafferent electrosensory inputs. Experiments in which the pattern of reafferent input associated with changes in posture was altered revealed that the pyramidal cells learn, over a time course of several minutes, to reject new patterns of input. Both proprioceptive input and descending electrosensory input to the posterior eminentia granularis are involved in generating the observed plastic changes in pyramidal cell responsiveness.

Key words Pyramidal cells · Neuronal plasticity
Long-term depression · Reafferent input
Weakly electric fish

Abbreviations *AM* amplitude modulation
EGp posterior eminentia granularis
ELL electrosensory lateral line lobe
EOD electric organ discharge
HRP horseradish peroxidase
LTD long-term depression · *LTP* long-term potentiation

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Introduction

Weakly electric fish possess an active sensory system composed of an electric organ located in the animal's trunk and tail, and electroreceptors scattered over the surface of the body. Distortions of the electric organ discharge (EOD) field result from the presence of nearby objects differing in conductivity from the surrounding water or from the discharges of other electric fish. These distortions are encoded by the electroreceptors and subsequently analyzed by the central nervous system to extract behaviorally relevant information. The motor activities of weakly electric fish are also expected to alter the EOD. These animals commonly bend their trunk and tail adopting an arc-like posture during exploratory movements and both EOD simulation studies (Heiligenberg 1975; Hoshimiya et al. 1980) and empirical field measurements (Assad et al. 1990) show that these postural changes produce significant changes in EOD amplitude. Proprioceptive pathways, particularly those providing data about the position of the tail and trunk, could supply information needed for the correct interpretation of self-imposed or reafferent patterns of electrosensory input.

The electrosensory lateral line lobe (ELL) is the primary electrosensory processing area within the brain of weakly electric fish. In addition to the electroreceptor afferent projection, the ELL receives electrosensory inputs descending from higher centers (Maler et al. 1981; Sas and Maler 1983; Bastian 1986a,b; Bastian and Bratton 1990; Bratton and Bastian 1990) and inputs from other sensory systems, possibly including proprioceptive information as well as corollary discharges of motor commands (Sas and Maler 1987). A simplified diagram summarizing the circuitry of the ELL and its connections with other electrosensory processing regions of the brain is shown in Fig 1. The ELL is a multilaminar structure; the receptor afferents terminate within the most ventral laminae, the deep fiber layer (DFL), while the descending electrosensory inputs and those from other systems ultimately project to the ELL's dorsal (DML) and ventral (VML) molecular layers. The principal ELL efferent

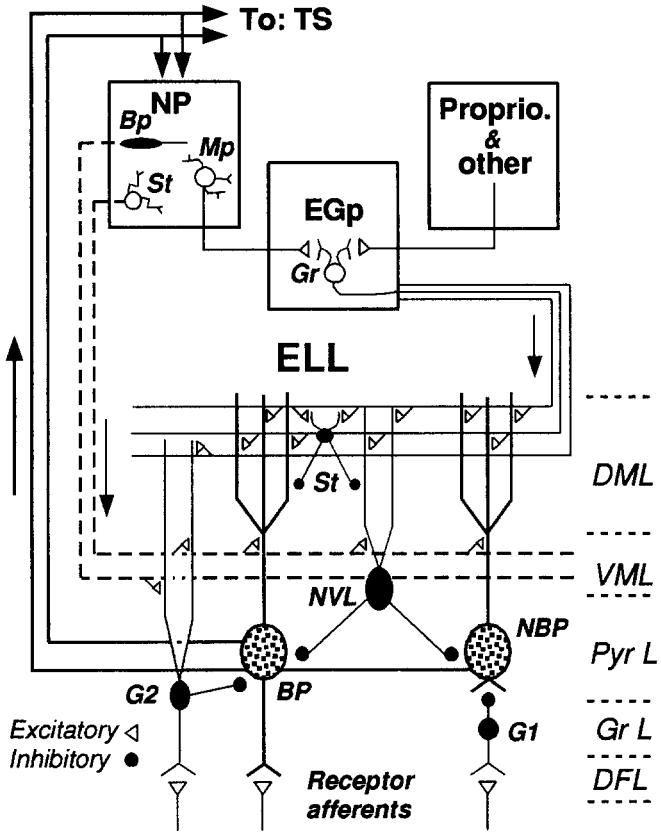

Fig. 1 Summary of electrosensory lateral line lobe circuitry. *BP*, Basilar pyramidal cell; *Bp*, bipolar cell; *DFL*, deep fiber layer; *DML*, dorsal molecular layer; *EGp*, posterior eminentia granularis; *G1*, type 1 granule neuron; *G2*, type 2 granule neuron; *Gr*, granule cell; *Gr L*, granule neuron layer; *Mp*, multipolar cell; *NBP*, nonbasilar pyramidal cell; *NP*, n. praeemintialis; *NVL*, neuron of the ventral molecular layer; *Proprio.*, proprioceptive; *Pyr L*, pyramidal cell layer; *St*, stellate cell; *VML*, ventral molecular layer

neurons receive receptor afferent input either directly (basilar pyramidal cells, BP) or indirectly via an inhibitory interneuron (nonbasilar pyramidal cells, NBP). The pyramidal cells, as well as several categories of inhibitory interneurons including the type 2 granule cells (G2) and neurons of the ventral molecular layer (NVL), extend large apical dendrites into the superficial molecular layers where they receive descending electrosensory and other inputs mentioned above. These various inputs to the pyramidal cell apical dendrites, and to dendrites of inhibitory neurons, are thought to control pyramidal cell gain and receptive field properties (Bastian 1993; Bastian and Bratton 1990; Bratton and Bastian 1990; Shumway and Maler 1989; Maler and Mugnaini 1993).

The ELL pyramidal cells project to the n. praeeminentialis (NP) and to the torus semicircularis (Maler et al. 1982). The NP, which also receives an input from the torus, projects back to the ELL closing an electrosensory feedback loop. Axons of NP bipolar and stellate cells project directly to the ELL (dashed lines) forming its ventral molecular layer while the axons of the NP multipolar, fusiform, and polymorphic cells project to the posterior eminentia granularis (EGp), a mass of cerebellar granule cells overlying the ELL (Sas and Maler 1983). The axons of the EGp granule cells (typical parallel fibers) project to the ELL forming the larger dorsal molecular layer. The EGp may also receive proprioceptive inputs and corollary discharges of motor commands (Sas and Maler 1987), hence the EGp to ELL projection may be involved in compensating for the electrosensory consequences of motor activity. Recent studies of electroreception in elasmobranchs and of mechanoreception in teleosts revealed mechanisms for adaptively filtering the electro- and mechanosensory consequences of the animals own movements (Bodznick and Montgomery 1994; Montgomery and Bodznick 1994a,b).

This study describes the physiological and anatomical characteristics of proprioceptive neurons that project to the EGp. Also, electroreceptor responses and responses of ELL pyramidal cells to the electrosensory stimuli resulting from changes in the animal's posture were compared with responses to similar patterns of electrosensory stimuli generated without changes in the animals' posture. The relative insensitivity of pyramidal cells to electrosensory stimuli resulting from changes in posture indicate the presence of mechanisms that enable pyramidal cells to reject the electrosensory inputs associated with body movements (reafferent inputs). Lastly, experiments are described which demonstrate that pyramidal cell responses are highly plastic. Their ability to reject a given pattern of reafferent electrosensory input changes with the recent stimulation history of the system. The pyramidal cells can "learn" to adaptively filter (reject) different patterns of electrosensory input if these are presented repetitively. The characteristics of ELL pyramidal-cell plasticity are strikingly similar to the plastic effects described for other electrosensory and the mechanoreceptive lateral line systems (Bell 1981, 1982, 1984, 1986, 1989; Bell and Grant 1989, 1992; Bell et al. 1992, 1993; Bodznick and Montgomery 1994; Montgomery and Bodznick 1994a,b). Both the descending electrosensory and proprioceptive inputs provide information sufficient for pyramidal cell rejection of reafferent inputs and the expression of pyramidal-cell plasticity.

Methods

The weakly electric fish *Apteronotus leptorhynchus* and *Eigenmannia* sp. were used in this study. Surgical procedures and methods used for intracellular labelling with HRP have been previously described (Bastian and Courtright 1991; Bastian et al. 1993).

Physiological studies

Animals were suspended in a plexiglas tank 30×30×7 cm deep and artificially respirated with a continuous flow of aerated water. Water temperature ranged from 25 to 27°C and conductivity was maintained at approximately 10 kΩ·cm.

Intracellular recordings were made with borosilicate pipettes filled with either 3 *M* KCl or with 3 *M* potassium acetate; these had resistances ranging from 80 to 150 MΩ. For labelling experiments, borosilicate glass pipettes were filled with 10% HRP in 1 *M* KCl plus 0.01 *M* Tris adjusted to pH 7.4. The HRP-filled elec-

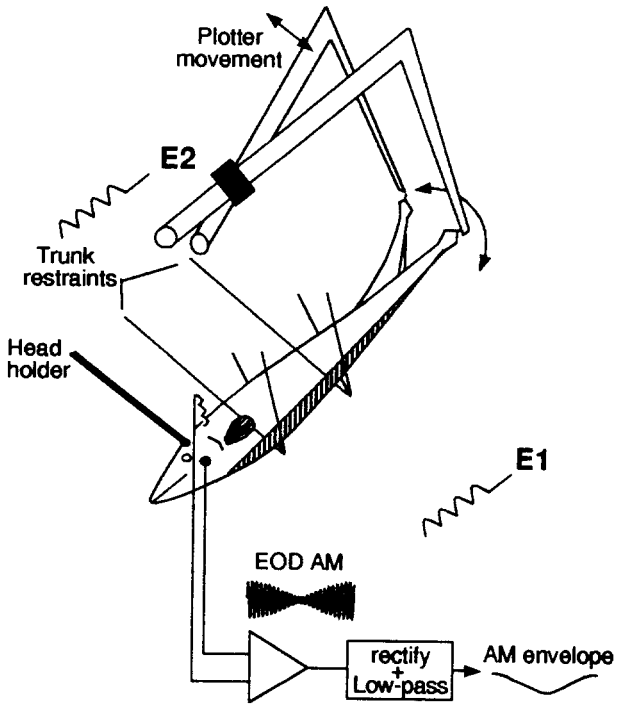

Fig. 2 Diagram of apparatus used to apply proprioceptive and electrosensory stimuli, see Methods for details

trodes were beveled in a jet-stream containing 0.05 μm gamma alumina particles until resistance fell to less than 100 MΩ. Extracellular recordings were made with metal filled glass pipettes described by Frank and Becker (1964).

Tail and trunk displacements similar to those produced by freely moving animals were generated as shown in Fig. 2. The animal's skull was glued to an immobile head-holder and the trunk was supported by two trunk restraints made from glass capillary tubing. The tip of the tail was attached to a movable arm which was linked to the drive mechanism of an x-y plotter. Movement of the arm resulted in displacement of the tail through an arc as illustrated by the curved arrow in Fig. 2. The amplitude and period of the approximately sinusoidal pattern of tail movement was determined by the settings of the function generator that provided a sinusoidal signal to the plotter. The arc through which the tail was moved was measured via a protractor mounted over the tail-movement arm. The caudal trunk support was typically placed about midway along the length of the body, directly beneath the pivot point of the arm, so that only the caudal 50% of the trunk participated in the tail displacement.

The EOD amplitude modulations that result from tail displacements were recorded via an electrode placed on the surface of the skin, near-to or within the receptive field of the cell under study, and a second implanted in the dorsal musculature. The small EOD AMs that result from tail movements are most easily quantified by measuring the envelope of the EOD waveform recorded across the skin. This was produced by rectifying and low-pass filtering the EOD ('precision absolute value circuit', circuit 2.42d. 1968 Philbrick/Nexus applications manual). Electrosensory stimuli designed to mimic the EOD AMs that result from tail displacements were produced electronically by multiplying the EOD waveform recorded via electrodes at the animal's head and tail by a suitable modulation waveform (e.g. the sinusoidal waveform applied to the plotter that displaced the tail). The resulting modulated version of the EOD was attenuated and applied to the fish via electrodes on either side of the animal (Fig. 2, E1 and E2). This technique produced electronic mimics that evoked receptor afferent responses similar to the responses resulting from tail-displacement AMs.

Only receptor afferents and ELL pyramidal cells having receptive fields within the rostral 30% of an animal's body were selected for study. Extracellular data were processed on-line and displayed as period histograms or raster displays that relate the cell's activity to the tail movement cycle, or to the AM cycle of electronically produced stimuli. Intracellular data were recorded on magnetic tape (Hewlett Packard model 3960, 3.75 ips, FM mode) along with synchronization pulses indicating the timing of tail movements or electronically produced AMs, and later processed as above. In all experiments the EOD waveform measured at the receptive field of the cell under study was recorded for later measurement of AM amplitudes.

The phase of each cell's peak response to cyclic tail displacement or electronically produced AMs was determined by calculating the direction of the mean vector for each period histogram (Batschelet 1981). The phase of the cell's minimum firing frequency was taken to be 180° away from that of the response peak. Maximum and minimum spike frequencies were computed from 13 bins centered on the times or phases of the peak and minimum responses, respectively. Means are given ±1 standard error unless indicated otherwise.

Results

Proprioceptive input to the EGp

Single units were recorded within the posterior eminentia granularis which responded in a nonadapting manner to displacements of the animal's trunk and tail. These cells were spontaneously active at high and regular rates, averaging 77±7.2 spikes/s, and were not sensitive to increases or decreases in the amplitude of the animal's ongoing EOD. Nor were they responsive to low-frequency electrosensory stimuli which activate ampullary electroreceptors. These proprioceptive units were subdivided into three response types. Type I cells increased firing rate as the tail was displaced toward the side of the body ipsilateral to the EGp recorded from and firing decreased with contralateral displacements (Fig. 3A). Type II cells behaved oppositely, decreasing firing with ipsilateral, and increasing firing frequency with contralateral displacements (Fig. 3B). The majority of proprioceptive cells encountered (92%) fell into these two categories. Type III cells (8%) produced maximum firing rates when the animal's trunk and tail were approximately straight (Fig. 3C); these typically gave smaller changes in activity for a given degree of tail displacement.

The tail positions at which the cells responded maximally, expressed as the percent of the total displacement (typically an arc subtending ±45°), are summarized in Fig. 3D. The type I and II cells' peak responses occurred near the limits of the imposed tail movement. Individual cells were rarely found that were 'tuned' to intermediate amounts of displacement.

Figure 4A summarizes the responses of two type I cells (circles and triangles) and one type II cell (squares) to continuous sinusoidal tail movements of various amplitudes. Clear changes in firing were seen with displacements of less than ±5°, and the average slopes of the best-fit lines to these data indicate a gain of approximately 4 spikes/s per degree of tail displacement. Since the increasing tail displacement amplitudes of Fig. 4A

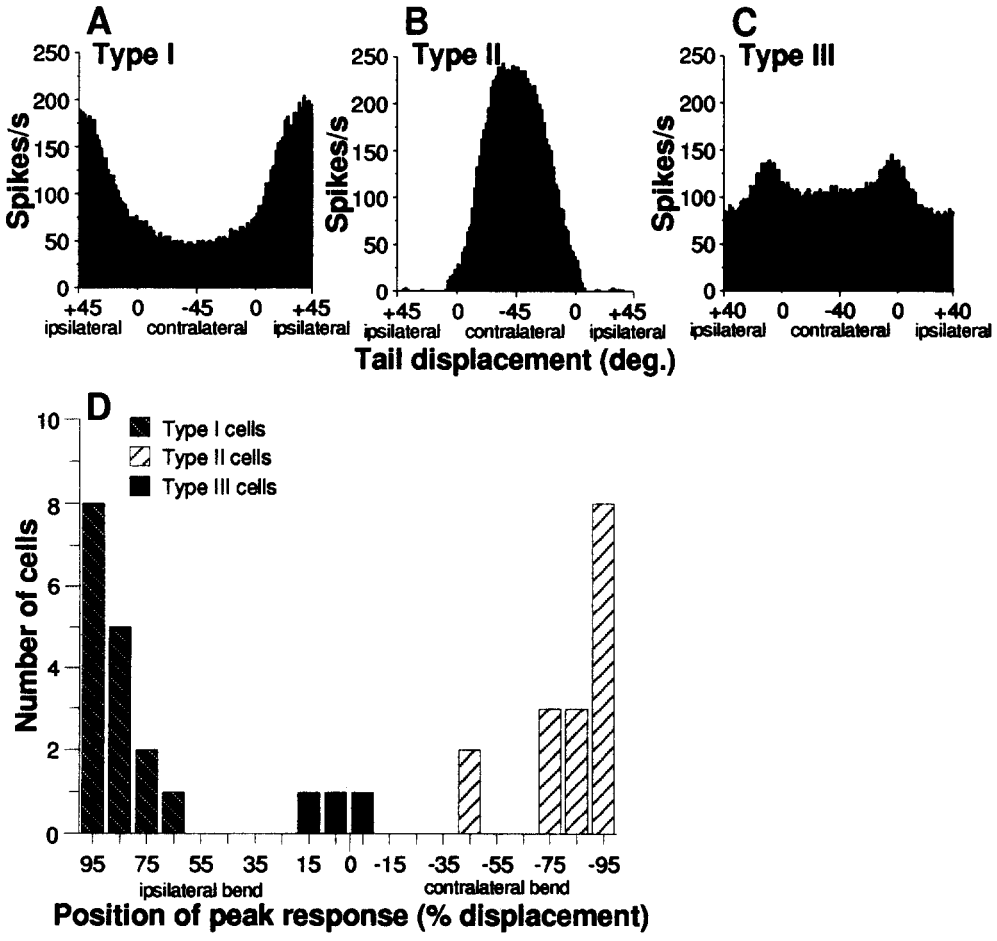

Fig. 3A–C Period histograms of proprioceptive unit activity during continuous 0.1 Hz sinusoidal displacement of the tail (average of 5 cycles). **D** Histogram of proprioceptive units phase of peak response expressed as percentage of maximum displacement

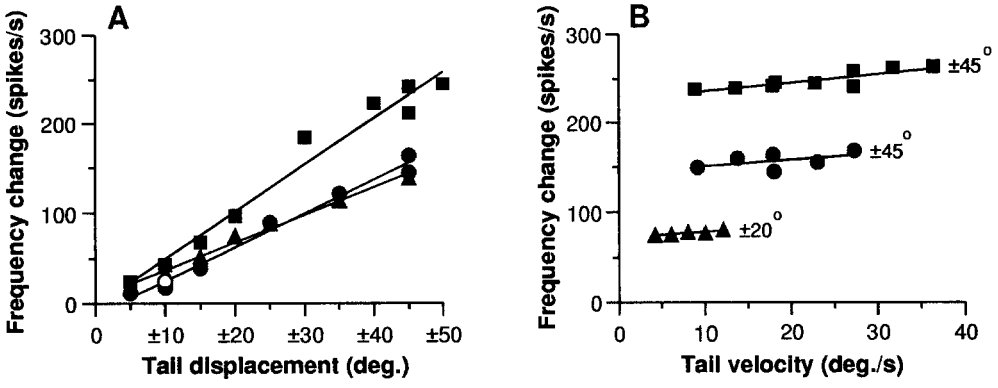

Fig. 4A Plots relating change in firing frequency (maximum minus minimum firing frequency within the displacement cycle) to the amplitude of the tail displacement for two type I cells (*circles* and *triangles*) and one type II cell (*squares*). The *open circle* at ±10° shows change in firing frequency determined from interval histograms of the type II cell's activity during static ±10° displacements. **B** Plots relating change in firing frequency of the same type I and II cells to constant amplitude tail displacements (indicated to the left of each line) presented with different velocities

were produced with a constant (0.2 Hz) oscillation frequency, tail movement velocity increased with increased movement amplitude. The effect of velocity alone was assessed by moving the tail through a constant amplitude arc at various oscillation frequencies. The effect of velocity is small as shown by Fig. 4B; these cells increase firing rate by an average of 0.8 spikes/s per degree/s increase in tail velocity. The proprioceptive neurons are approximately five-fold more sensitive to position changes as compared to changes in velocity.

These proprioceptive units encode tail position in a predominantly nonadapting manner; static displacements result in only slightly smaller changes in firing frequency as compared to when the tail is dynamically displaced by the same amount. The open circle of Fig. 4A indicates the difference in firing frequency seen with the type II cell as a result of changing the static position of the tail from + to −10°.

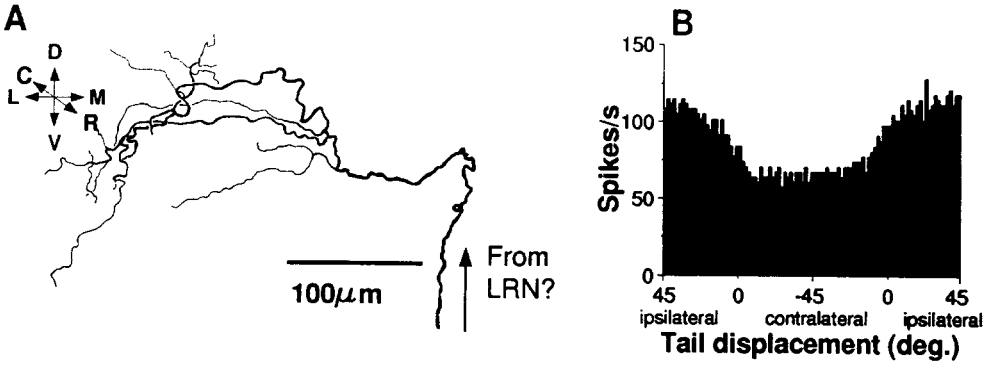

Fig. 5A Reconstruction of HRP filled proprioceptive axon recorded within the EGp. **B** Period histogram of this cell's responses to 5 replicates of a 0.1 Hz ±45° tail displacement

Anatomy of proprioceptive units

The identity of the EGp units giving proprioceptive responses was determined by recording intracellularly and filling the cells with HRP or Lucifer yellow. In all cases the fills showed that the type I and type II responses were recorded from axonal plexi similar to that shown by the reconstruction of Fig. 5A. An example of this reconstructed type I cell's response to tail displacement is summarized in Fig. 5B. The parent axons of these arborizations course ventro-dorsally entering the EGp at its rostro-medial pole. The axons then branched profusely, ramifying throughout the majority of the rostro-caudal extent of the EGp, a distance of about 960 μm in this case. The morphology of these proprioceptive afferents is quite similar to that of the previously described electroreceptive axons projecting from the NP to the EGp (Bastian and Bratton 1990). No fills were sufficiently complete to identify the cell bodies associated with these axons, however extracellular injections of HRP or wheat germ agglutinin HRP into the EGp retrogradely label cells in the lateral reticular nucleus (Sas and Maler 1987 and this study). These lateral reticular nucleus cells may relay spinal proprioceptive information to the EGp, but additional studies are needed to verify this prediction. No fills of type III cells have been made thus far.

Electroreceptors respond similarly to EOD AMs caused by tail displacement and to electronic mimics of these

Sinusoidal displacement of the fish's trunk and tail causes amplitude modulations of the EOD, the magnitude of which varies with the size of an individual's EOD, with the site on the body surface at which the AM is measured, and with the amplitude of the tail displacement. Modulation amplitude generally ranged between 50 and 150 μV peak-to-peak for displacements ranging between ±20 to 50°. These modulations, roughly 1 to 5% of the normal EOD amplitude, are well above threshold for the electroreceptors.

An example of the effects of EOD AMs due to tail displacement on a receptor afferent's firing is shown in the period histogram of Fig. 6A. The envelope of the EOD modulation resulting from the tail displacement is

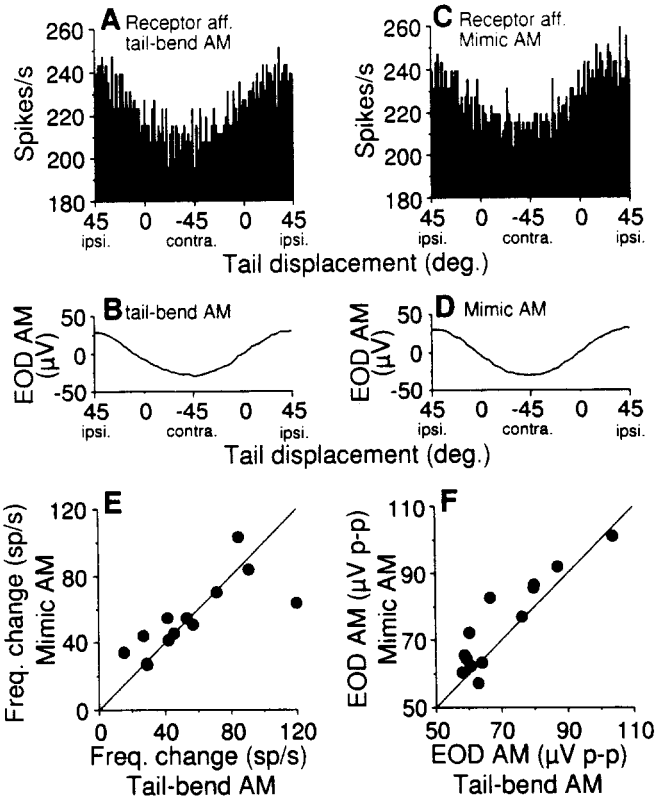

Fig. 6A,C Period histograms summarizing a single electroreceptor afferent's response to AMs due to tail displacement and to the mimic AM, respectively (5 cycles at 0.2 Hz). **B** Envelope of the EOD AM due to tail displacement and **D** the envelope of the electronic mimic. **E** Scatter plot relating each receptor afferent's response to the mimic AM to that resulting from the tail-bend. **F** Scatter plot showing the amplitudes of the mimic AMs and the tail-bend AMs used to stimulate the receptor afferents

shown in Fig. 6B. In this case the peak-to-peak EOD modulation, measured across the skin near the receptor's location, was 58 μV and this resulted in a change in firing frequency averaging 29 spikes/s. The response of the same receptor afferent to electronically generated AMs of similar amplitude and time course (mimic AM) is shown Fig. 6C and the envelope of the mimic AM is shown in Fig. 6D. The mimic AM's amplitude was 64 μV p-p and this caused a change in receptor afferent firing averaging 28 spikes/s. Figure 6E compares the re-

sponses of 13 receptor afferents to AMs resulting from tail displacement and to similarly sized mimic AMs. The response of each afferent to the mimic AM is plotted against the same cell's response to that caused by tail displacement. Most points fall close to the solid diagonal line indicating that receptors typically respond similarly to these two stimuli. The average change in afferent firing frequency evoked by the mimic AMs, 53.9±6.1 spikes/s, was not significantly different from the average of responses to the tail displacement AMs, 54.3±8.3 spikes/s (P=0.98, t-test). The amplitude of the mimic AM used for each afferent is plotted against AM magnitude due to tail displacement in Fig. 6F. The average mimic amplitude, 74.7±3.8 μV, was not significantly different than that due to tail displacement which averaged 70.5±3.9 μV (P=0.45, t-test).

ELL pyramidal cells respond differently to EOD AMs caused by tail displacement and to electronic mimics of these

Six basilar pyramidal cells (E-cells) and three nonbasilar pyramidal cells (I-cells) were studied using both tail-displacement AMs and electronic mimics of these as stimuli. Typically, several different oscillation frequencies, ranging from 0.1 to 0.4 Hz were used for each cell. The period histogram of Fig. 7A summarizes the responses of a basilar pyramidal cell to 50 cycles of a 0.1 Hz tail displacement. Despite the fact that the EOD AM measured within the cell's receptive field, Fig. 7B, was well above the threshold for electroreception, this pyramidal cell's firing was not significantly modulated by the stimulus. The period histogram is not different from that expected for a cell firing randomly (nonsignificant Raleigh statistic, Batschelet 1981). The cell's firing was clearly modulated by the mimic AM (AM without tail displacement) as is shown in Fig. 7C (P<<0.05, Raleigh statistic). The significant modulation of the pyramidal cell's firing was not due to larger mimic AM amplitude since the latter (98 μV p-p) was slightly smaller than that of the AM due to tail displacement (103 μV p-p).

The responses of the population of cells studied are summarized in Fig. 7E where each cell's responses to the electronically generated AMs are plotted against responses resulting from tail movement. The pyramidal cells usually responded more strongly to the mimic AMs; the points cluster above the diagonal line that indicates equivalent responses. The average pyramidal cell change in firing frequency in response to the mimic AM, 9.8±1.1 spikes/s, is significantly greater (P=0.005, t-test) than the change of 5.9±0.8 spikes/s caused by tail-movement AMs. The amplitudes of the mimic AMs and those due to tail movement are compared in Fig. 7F and in all cases the mimic AMs were smaller, averaging 59.9±2.9 compared to 65.7±3.0 μV p-p for the tail-displacement AMs.

Although the electroreceptor afferents respond similarly to tail-displacement AMs and to mimics of these,

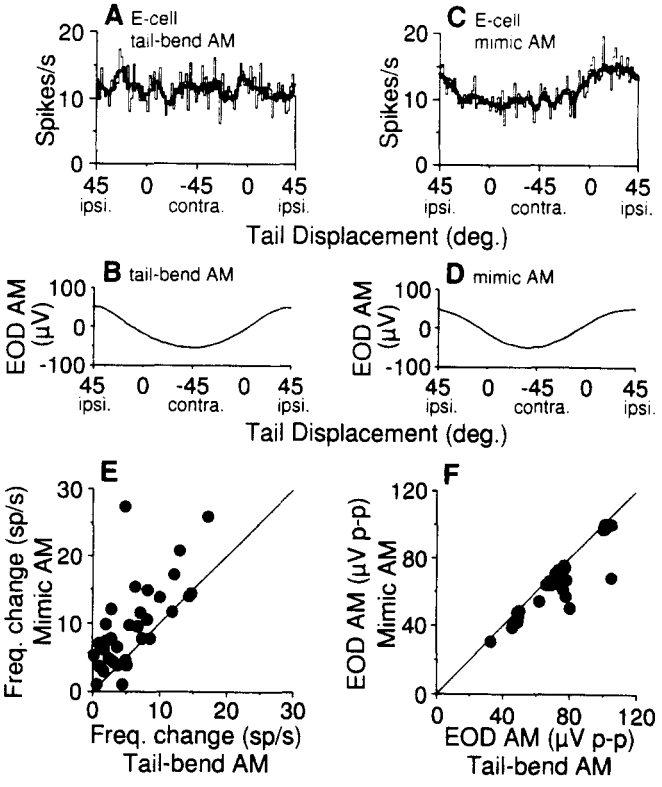

Fig. 7A,C Period histograms summarizing a basilar pyramidal cell's responses to AMs resulting from tail displacement and to mimic AMs, respectively (50 cycles at 0.1 Hz). The fine lines indicate raw data and the heavy lines indicate data smoothed by a four bin moving average filter. **B,D** EOD envelopes of the tail-displacement and mimic AMs, respectively. **E** Scatterplot relating each pyramidal cell's responses to mimic AMs and to AMs due to tail displacement. **F** Scatter plot showing the amplitudes of the mimic AMs and the tail-bend AMs used to stimulate the pyramidal cells

the ELL pyramidal cells are significantly less responsive to the AMs associated with changes in posture. In many cases pyramidal cells show virtually no response to AMs that are clearly above threshold for the electroreceptor afferents as long as these AMs are coupled to movements of the trunk and tail. Since these posture changes also strongly activate proprioceptive inputs to the EGp, these results suggest that the proprioceptive inputs may participate in mechanisms that attenuate the electrosensory consequences of the animal's own movements.

Synaptic plasticity is involved in pyramidal cell insensitivity to the electrosensory consequences of changes in posture

Free-swimming animals generate more complex and variable patterns of body movements than those used in these experiments, hence the pyramidal cells will normally be exposed to a range of EOD modulation amplitudes and waveforms. Therefore considerable flexibility is expected in whatever mechanisms are responsible for attenuating pyramidal cell responses to these reafferent patterns of electrosensory stimuli. Recent studies of sen-

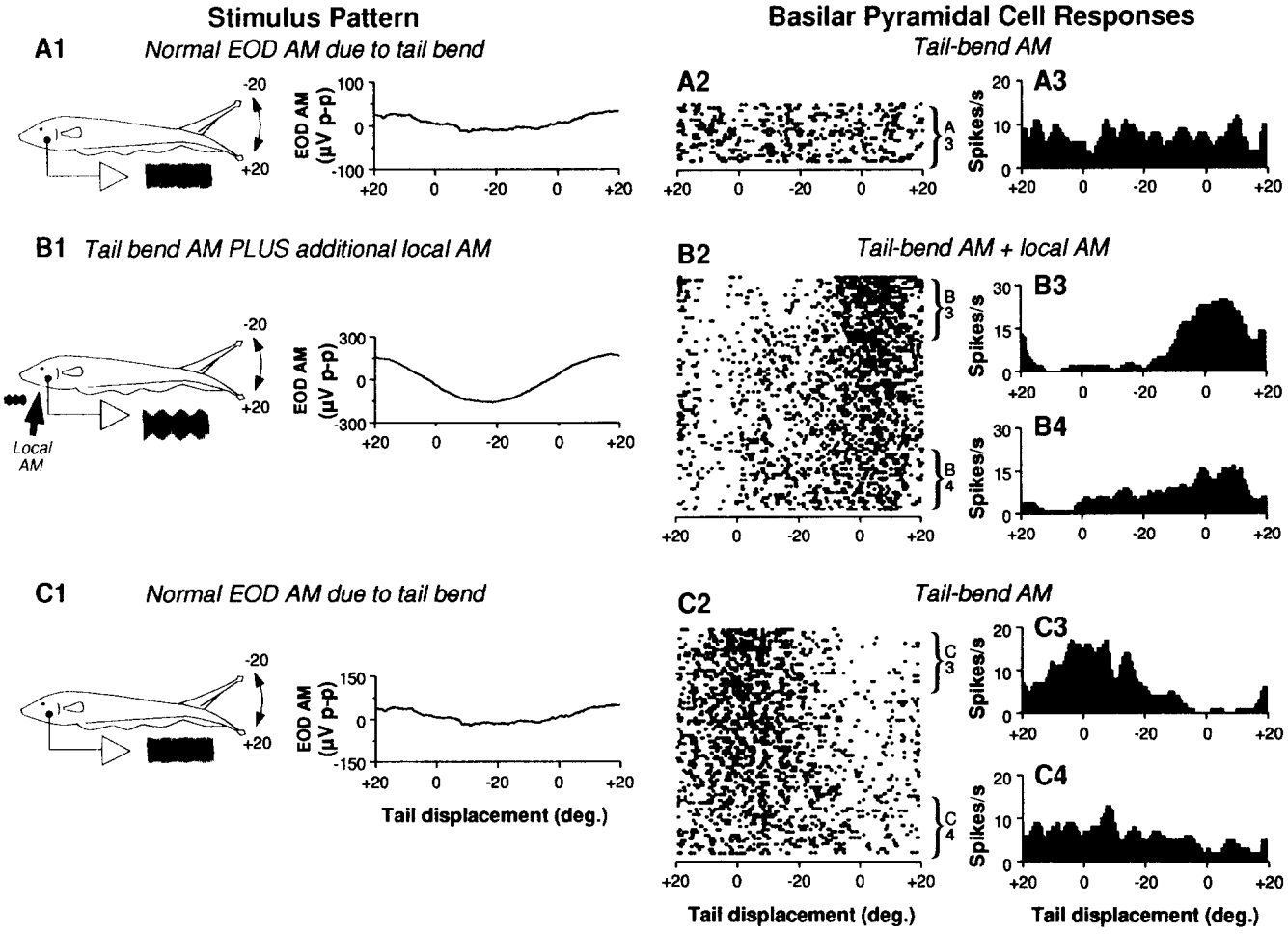

Fig. 8A1 Diagram illustrating the tail displacement stimulus and the measurement of the EOD waveform along with a plot of envelope of EOD AM. **A2** raster display of a basilar pyramidal cell's response to 20 cycles of a ±20° 0.4 Hz continuous sinusoidal tail displacement. **A3** Period histogram of the data shown in **A2**. **B1** Diagram of the tail displacement stimulus plus the local stimulus (*arrow*) along with a plot of the EOD AM envelope resulting from the combined stimuli. **B2** Raster display of the cell's response to 80 cycles of the 0.4 Hz tail displacement plus the 0.4 Hz local EOD AM. **B3,B4** Period histograms of data from the first and last 20 stimulus cycles of **B2**. **C1** Diagram of the tail-bend stimulus and plot of the envelope of the resulting EOD AM. **C2** Raster display of responses to 80 cycles of the tail-bend stimulus alone, **C3,C4** period histograms of responses to the first and last 20 stimulus cycles of **C2**

sory processing in related systems (Bell 1981, 1982, 1984, 1986, 1989; Bell and Grant 1989, 1992; Bell et al. 1992, 1993; Bodznick and Montgomery 1994; Montgomery and Bodznick 1994a,b) have shown that neurons, analogous to the pyramidal cells studied here, have plastic properties that allow them to adaptively filter sensory inputs resulting from the animal's own activity. To test the idea that ELL pyramidal cells can "learn" or adaptively reject varied patterns of electrosensory input, experiments were done in which a cell's responses were monitored while altering the pattern of electrosensory input related to tail movement.

The envelope of the AM measured within a basilar pyramidal cell's receptive field, resulting from ±20° tail displacement (45 μV p-p), is shown in Fig. 8A1 and a raster display of the cell's activity during tail movement is shown in Fig. 8A2. Figure 8A3 is a period histogram summarizing this raster data. As described above, the basilar pyramidal cell's activity is not modulated by this stimulus. After completion of these first 20 tail displacement cycles, an additional electronically produced sinusoidal AM, synchronized to the tail-movement cycle, was locally applied to the fish at the site of the pyramidal cell's receptive field (arrow in the diagram of Fig. 8B1). This additional AM increased the amplitude of the stimulus measured within the cell's receptive field to approximately 300 μV p-p (Fig. 8B1). The cell initially responded strongly to this new pattern of electrosensory input, but the response grew weaker with continuous presentation of the tail-bend plus the local AM (Fig. 8B2). The response decayed to less than 50% of its initial value over the time (200 s) of the 80 paired presentations of the tail-bend plus local AM stimuli. The period histograms of Fig. 8B3,B4 summarize the data from the first and last 20 presentations, respectively, of these paired stimuli.

Following the 80 presentations of the paired stimuli, the local AM was removed restoring the electrosensory input due to tail displacement to its original pattern (Fig. 8C1). Although at the start of the experiment the cell

was unresponsive to this stimulus (Fig. 8A2,A3), it responded vigorously to the tail-bend AM alone following the period of paired stimulation (Fig. 8C2,C3). The phases of the excitatory and inhibitory portions of the response were, however, opposite those seen when the local AM was paired with the tail displacement (compare Fig. 8B3,C3). The firing pattern seen after the paired stimulation is very surprising since basilar pyramidal cells normally only increase firing frequency in response to *increasing* EOD amplitude. The newly acquired response to the tail-bend AM shows strongly increased pyramidal cell activity apparently in response to *decreasing* EOD amplitude. This response also decayed with continued stimulus presentation (compare the period histograms of Fig. 8C3,C4).

This pyramidal cell response plasticity is similar to that described for ELL neurons of mormyrid electric fish (Bell 1981, 1982), for cells within the electrosensory system of elasmobranchs and within the normal lateral line system of teleosts (Bodznick and Montgomery 1994; Montgomery and Bodznick 1994a,b). In all cases the neurons involved are able to reject patterns of sensory input related to the animal's own activity patterns. The mechanism by which this is accomplished involves the central generation of what has been described as a "negative image" of the expected reafferent input (Bell 1984) or a "cancellation signal" (Montgomery and Bodznick 1994b). The increased responsiveness of the pyramidal cell of Fig. 8 to the tail displacement alone, seen after the local stimulus was paired for a time then removed, is an example of this negative image response. The development of this negative image input to the pyramidal cell is responsible for the progressive attenuation of the cell's response to the paired stimuli shown in Fig. 8B2.

Coincidence of tail-bend and local stimuli are necessary

The attenuation of pyramidal cell responses to the locally applied AM only occurs if this stimulus is applied simultaneously and in synchrony with the tail displacement as shown in Fig. 9. Again, this cell was initially unresponsive to the AM due to tail bending; the EOD AM due to tail bending, a raster display of the cell's responses and a period histogram of these data are shown in Fig. 9A1–A3. The local AM used in this experiment was in antiphase relative to that produced by the tail displacement (Fig. 9A4) and the basilar pyramidal cell initially responded strongly to the rising phase of the local stimulus paired with the tail-bend stimulus (Fig. 9A2,A5). The response diminished with repetitive presentations as shown by the raster and period histograms of data taken during the first and last 20 cycles of the paired stimulation (compare Fig. 9A5,A6). Following the removal of the local AM the cell responded strongly to the tail-bend AM alone, and this response was a negative image of the response to the previously applied paired stimuli (Fig. 9A8).

When the cell was stimulated with the local AM in the absence of simultaneous tail displacement, neither attenuation of the response to the local AM, nor the development of a negative image response was seen. Following the initial presentation of the tail displacement stimulus (Fig. 9B1-B3), tail motion was stopped and the local AM was presented alone (Fig. 9B4). The cell responded with a somewhat different firing pattern to the local AM alone and responses remained constant throughout this period of stimulation (Fig. 9B5,B6). Following the cessation of stimulation with the local AM, tail displacement was restored, but the cell remained unresponsive to this stimulus (Fig. 9B7,B8). Significant attenuation of pyramidal cell responses to the local AM and the appearance of negative images of expected inputs only occurred when the local AM was presented in synchrony with tail displacement.

Proprioceptive and electrosensory information can independently mediate plastic changes in pyramidal cell responsiveness

The inputs to the pyramidal cells giving rise to the negative images of expected inputs are likely to be received via the cell's apical dendrites. As described above, proprioceptive inputs are present within the EGp, and this information will be transmitted to the pyramidal cell apical dendrites via the ELL dorsal molecular layer parallel fibers. The pyramidal cells also receive information about amplitude modulations of the EOD field via DML parallel fibers since the EGp receives nonadapting electroreceptive inputs that descend from the nucleus praeeminentialis (Bastian and Bratton 1991). Additionally, axons of the NP stellate and bipolar cells project directly to the ELL ventral molecular layer providing another source of descending electrosensory information to the ELL Pyramidal cells (Sas and Maler 1983; Bratton and Bastian 1990; Maler and Mugnaini 1994). Therefore, displacing the tail simultaneously provides both proprioceptive and electroreceptive inputs to the ELL pyramidal cells' apical dendrites along with the ascending electroreceptor afferent input.

Experiments were done in which localized electrosensory stimuli were paired with whole-body patterns of electrosensory input which mimic the AMs generated by tail displacement. Since these mimic AMs were generated in the absence of tail displacement, this experiment can determine if proprioceptive input is *necessary* for pyramidal-cell plasticity. Likewise, in order to determine if whole-body electrosensory stimuli are necessary for plasticity, experiments were done in which local electrosensory stimuli were paired with tail displacements in animals whose electric organ discharge was abolished (proprioceptive input without the associated whole-body EOD AM).

Figure 10 summarizes an experiment in which a local 0.4 Hz AM was paired with a weaker tail-bend mimic AM of the same frequency applied transversely to the whole animal via electrodes E1,E2 of Fig. 2. Both stimu-

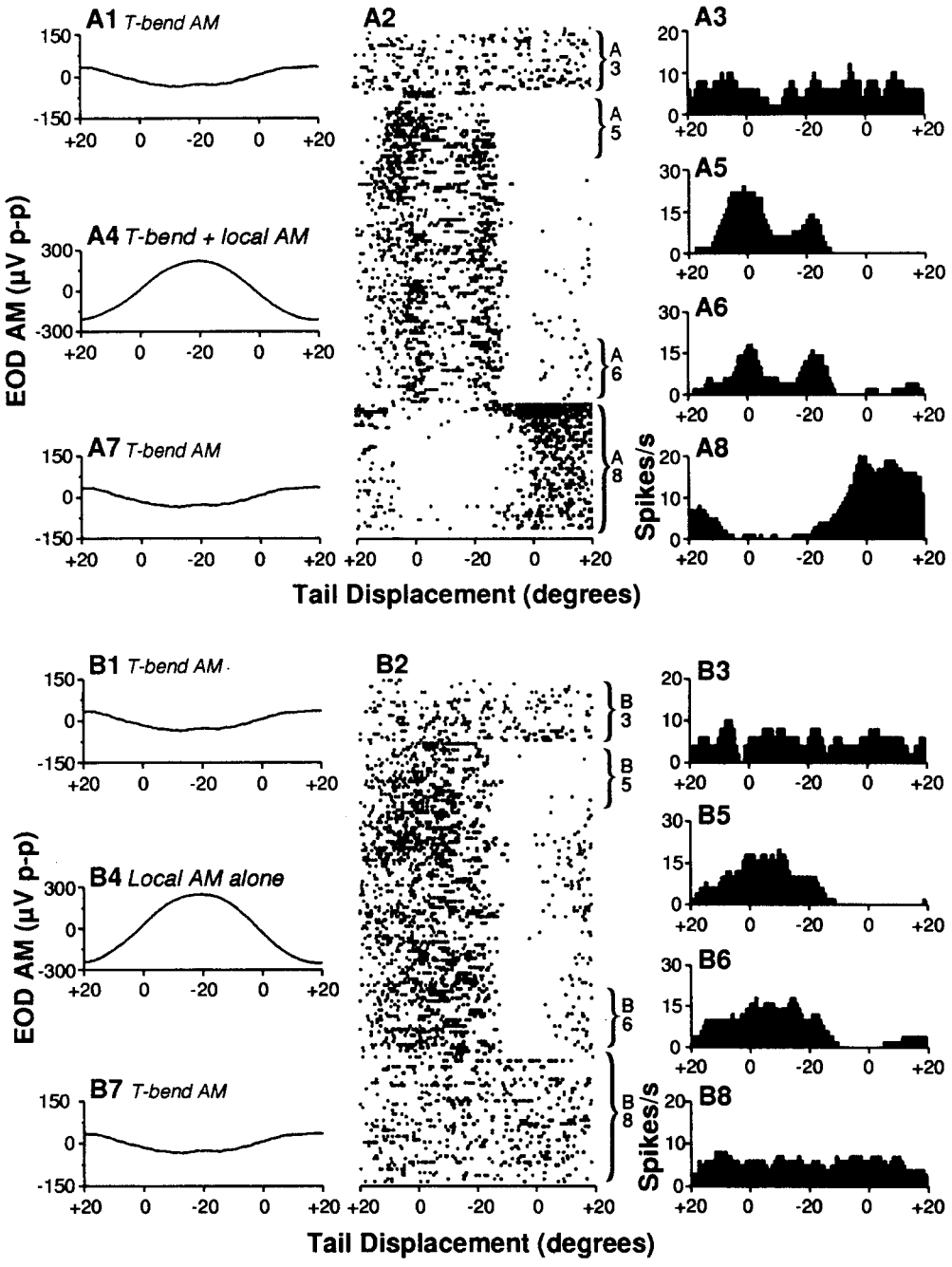

Fig. 9A1, A4, A7 EOD AM envelopes resulting from tail displacement (**A1**,**A7**) and tail displacement plus a locally applied AM (**A4**). **A2** Raster display of a basilar pyramidal cell's responses to 20 cycles of ±20°, 0.4 Hz tail displacement followed by 100 cycles of the paired local AM plus tail-bend stimulus followed by 40 cycles of the tail-bend stimulus alone. **A3, A5, A6, A8** Period histograms of the data indicated by the *labeled brackets* to the right of the raster display. **B1, B4, B7** EOD AM envelopes due to tail-bend stimuli (**B1**, **B7**) and due to the local stimulus presented alone (**B4**). **B2** Raster display of the cell's responses to 20 cycles of ±20°, 0.4 Hz tail displacement followed by 100 cycles of the local AM alone followed by 40 cycles of the tail-bend stimulus alone. **B3, B5, B6, B8** Period histograms of the data indicated by the *labeled brackets* to the right of the raster display

li were generated electronically and the animal's posture remained unchanged. This basilar pyramidal cell responded weakly to the whole-body AM presented alone as shown by the initial portion of the raster display (Fig. 10A) and the period histogram of these data (Fig. 10B1). The magnitude of the whole-body AM was 9% p-p of the animal's normal EOD measured within the cell's receptive field. Addition of the local AM increased the modulation at the cell's receptive field to 24% p-p, and, initially, the cell responded vigorously. The local AM was first applied at the point indicated by the top of the bracket labeled B2 of Fig. 10A and a period histogram of the cell's responses to the first 20 paired local and whole-body stimuli is shown in Fig. 10B2. This response decayed over the 200 s that the paired stimuli were applied (compare Fig. 10B2,B3) and upon removal of the local AM, the cell showed the typical negative image response which also decayed over time (Fig. 10B4,B5). This experiment shows that proprioceptive inputs are not necessary for the pyramidal-cell plasticity; electrosensory inputs alone are sufficient. The system can learn to reject local patterns of electrosensory stimuli that differ in amplitude from stimuli received over larger regions of the body. Control experiments verified that the local AM must occur simultaneously and in synchrony with the AM received over the rest of the animal's body if the cancellation is to occur.

In order to test whether the proprioceptive inputs

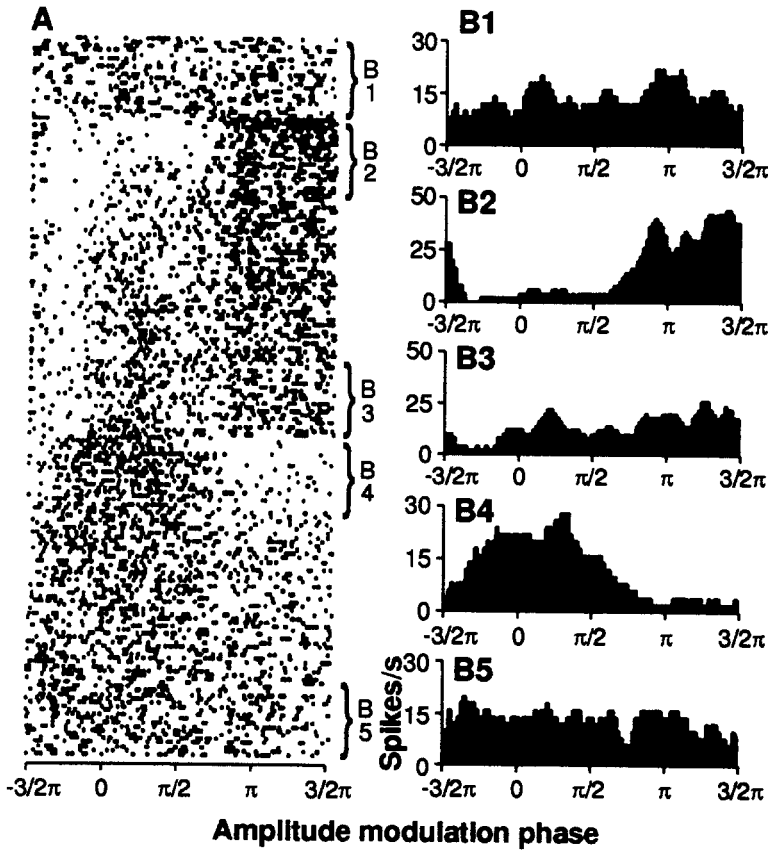

Fig. 10 Raster display (**A**) of a basilar pyramidal cell's responses to an electronically produced 0.4 Hz AM applied to the whole body (section **B1**), to this AM plus a local synchronous AM applied to the cell's receptive field (sections **B2** through **B3**), and to the whole-body AM presented alone following the paired stimuli (sections **B4** through **B5**). **B1–B5** Period histograms produced from data of the raster display indicated by the *labeled brackets*

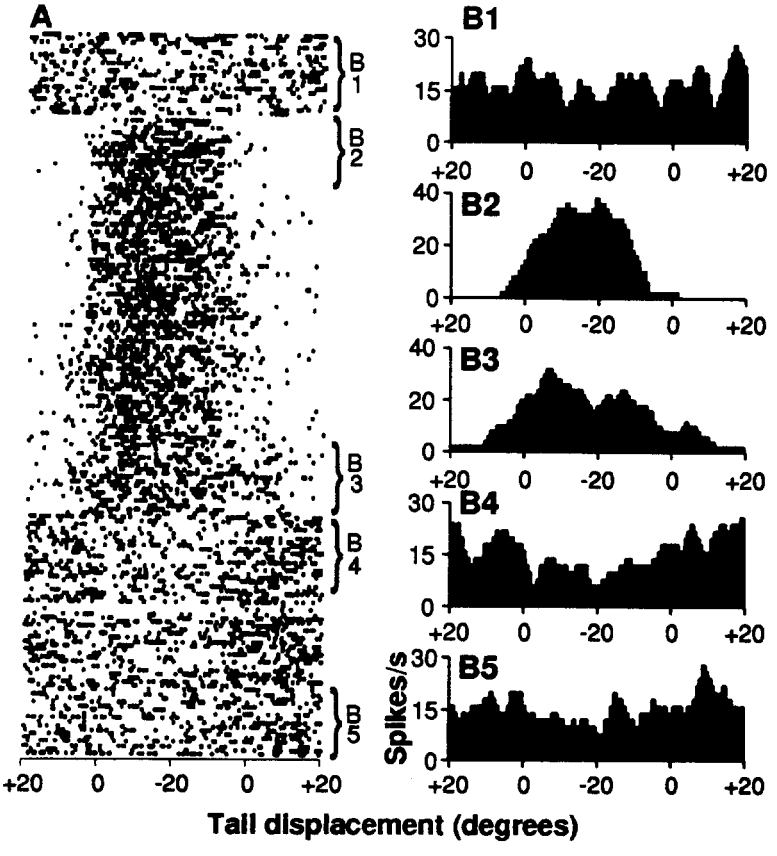

Fig. 11 Raster display (**A**) of an ELL pyramidal cell's responses to ±20°, 0.4 Hz tail displacements recorded from *Eigenmannia* in the absence of the animal's EOD (section **B1**). Section **B2** through **B3** show the cell's responses to the tail displacement plus a local 0.4 Hz AM applied to the cell's receptive field and section **B4** through **B5** shows the cell's response to the tail displacement alone following the paired stimulation. **B1–B5** period histograms produced from the data of the raster display indicated by the *labeled brackets*

alone are also sufficient to support the pyramidal-cell plasticity, the animal's electric organ discharge field must be removed. Then local patterns of electrosensory stimuli can be paired with tail displacements in the absence of the whole-body AMs normally resulting from changes in posture. It is difficult to completely remove the EOD of *Apteronotus* since this animal's electric organ is composed of modified spinal motor neurons and therefore insensitive to curare-like drugs. The related fish *Eigenmannia* was used instead, since its electric organ discharge can be completely abolished via curarization. The first segment of the raster of Fig. 11A, indicated by the bracket labeled B1, and the period histogram of these data (Fig. 11B1) show that this pyramidal cell was initially unresponsive to ±20° 0.4 Hz tail displacements. During the second phase of this experiment (Fig. 11A, sections B2 through B3) a local electrosensory stimulus was applied to the cell's receptive field (±25% AM of a 400 μV, 300 Hz sinusoidal waveform). The cell's initial response gradually became weaker over the 250 s that the paired stimuli were applied (compare Fig. 11B2,B3). Upon removal of the local AM, tail displacement alone evoked a modulation of the cell's firing rate (negative image) which decayed with continuous stimulation (Fig. 11B4,B5). This experiment shows that proprioceptive information alone, when paired with a local electrosensory stimulus, also provides sufficient information for the generation of pyramidal-cell plasticity.

Discussion

Proprioceptive inputs to the EGp

The presence of proprioceptive information within the EGp, a structure intimately associated with the electrosensory system, is not surprising given that changes in the animal's posture result in modifications in electrosensory input. The proprioceptive fibers recorded and labeled within the EGp of *A. leptorhynchus* show properties similar to fibers recorded and labeled within the EGp of the mormyrid weakly electric fish, *Gnathonemus petersii*, (Serrier et al. 1991), and proprioceptive responses have also been recorded in dorsal granular ridge of elasmobranchs (Conley and Bodznick 1994). This structure is analogous to the EGp of gymnotiform and mormyriform fishes. High and regular firing rates and tonic responses to changes in posture are typical of the proprioceptive fibers seen in each of these animals, and in all cases the axons of the granule cells receiving the proprioceptive inputs, parallel fibers, project to the apical dendrites of efferent neurons of the first-order sensory processing area. These proprioceptive units have been predicted to participate in mechanisms which somehow identify, or reduce the consequences of, posturally generated electrosensory inputs (Bell et al. 1992; Szabo 1993; Conley and Bodznick 1994).

The origin of the proprioceptive fibers in the gymnotiforms has not been determined. The intracellular labeling of fibers in this study was never sufficiently complete to identify the parent somata. Retrograde labeling experiments of Sas and Maler (1987), and of this study, identified the lateral reticular nucleus as a major source of input to the EGp, raising the possibility that proprioceptive information may be relayed from the spinal cord via this structure. Thus far, no studies have indicated that a direct spinal projection to the EGp exists in gymnotiforms although such a projection has been reported for the mormyrids along with projections via the anterior and posterior lateral funicular nuclei and the lateral reticular nucleus (Szabo et al. 1979; Szabo 1993). Additional anatomical and physiological studies are needed to further explore additional possible routes by which proprioceptive information might reach the EGp in gymnotiforms.

Receptor afferent and ELL pyramidal cell responses

The technique used in these studies to determine if proprioceptive information influences ELL pyramidal cell responses involved comparing a cell's responses to electrosensory stimuli arising as a consequence of changes in posture (tail-bend AMs) with responses to similar electrosensory stimuli generated electronically without changing the animal's posture (mimic AMs). The validity of this comparison requires that the electronically produced stimulus is an accurate copy of the tail-bend AM. The results summarized in Fig. 6 show that mimic amplitudes and time courses are quite similar to those of tail-bend AMs, and, as expected for equivalent stimuli, receptor afferents show no statistical difference in their responses to these. However, the agreement is not perfect; some individual receptors responded differently to these stimuli. This might result from using mimics imperfectly matched in amplitude due to inaccurate measurement of the voltage developed across the skin at the receptor site.

Pyramidal cell responses to AMs resulting from tail displacement were significantly less than responses to the mimics of these. In some cases a given cell was virtually unresponsive to the tail-bend AM but clearly sensitive to the mimic as shown in Fig. 7A,C. The average pyramidal cell responses were about 70% stronger to mimics as compared to tail-bend AMs even though average mimic amplitude was 10% smaller. This suggests that central nervous system mechanisms exist which selectively attenuate pyramidal cell responses to AMs that result from changes in posture.

Only receptor afferents and ELL neurons with receptive fields localized to the anterior 30% of the fish were studied. Rostrocaudal variations in local EOD amplitude are minimal over this region (Bastian 1981; Hoshimiya et al. 1980; Rasnow et al. 1993), and EOD AMs due to tail displacement are, therefore, expected to be homogeneous within this region. However, since EOD amplitude was only measured at one site within a cell's receptive field, I cannot eliminate the possibility that some spatial variations in AM amplitude occurred. Such variations, between the spatial distribution of the mimic AM and

that resulting from tail displacement could possibly contribute to the differences seen in pyramidal cell responses.

The signal used to produce the mimic AM was presented transversely via electrodes on either side of the fish (Fig. 2,E1,E2). With this geometry, current flow will be perpendicular to the skin over the broad lateral surface of the animal and uniform changes in EOD amplitude are expected. Smaller voltages will be developed across the animal's dorsal and ventral surfaces since current flow is tangential to the skin in these regions. Therefore, the EOD AMs resulting from the transversely applied field are expected to be smaller in these dorsal and ventral regions. The amplitude of the discharge generated by these animals shows only a slight decrease in amplitude at more dorsal and ventral sites as compared to sites on the body wall (Rasnow et al. (1993). Hence, the principal spatial variation expected in these experiments is a reduced mimic AM amplitude restricted to the dorsal and ventral regions of the animal's body as compared to the AM generated by actual changes in posture. It is unlikely that this reduced mimic amplitude could account for the stronger pyramidal cell responses to this stimulus.

Pyramidal-cell plasticity

As proposed for other related systems, pyramidal cell insensitivity to the AMs resulting from repetitive tail displacements probably results from the summation of a centrally generated "negative image" (Bell et al. 1993) or "cancellation signal" (Bodznick and Montgomery 1994; Montgomery and Bodznick 1994a,b) and the receptor afferent input from the periphery. Experiments in which the pattern of electrosensory input associated with the tail displacement is suddenly changed by adding a spatially localized AM (e.g. Fig. 8) show a gradual decay of the cell's initial response to the new stimulus pattern. The gradual reduction in the cell's response is a result of the development of the negative image input which decreases pyramidal cell excitability at times when increased input is expected from the periphery and increases excitability at times decreased input is expected. Once developed, the negative image input persists for a time after the stimulus is returned to its original pattern. Hence, pyramidal cells show strong responses to an originally ineffective stimulus pattern. These responses are mirror images of the pattern that the cell had recently learned to reject. This cancellation signal also decays with continued stimulation as the cell "relearns" to cancel the current pattern of afferent input. This plastic characteristic enables pyramidal cells to dynamically alter their sensitivity to repetitive stimuli. Predictable inputs are thereby canceled, enhancing the cells' ability to respond to novel stimuli.

The plastic development of negative images or cancellation signals in gymnotiform ELL pyramidal cells shares many similarities to effects seen in other systems. In mormyrid electric fish a corollary of the command signal which ultimately initiates the animal's electric organ discharge projects to the EGp, and Bell (1981, 1982, 1984, 1986, 1989), Bell and Grant (1989, 1992), and Bell et al. (1992, 1993) have shown that the effects of this corollary discharge signal on ELL cell activity are plastic. The corollary discharge evokes patterns of activity in ELL cells that depend on the system's recent stimulation history and the effects of this input oppose the effects of repetitive stimuli received from the periphery. Hence, the corollary discharge signal cancels repetitive peripheral inputs.

More recently Bodznick and Montgomery (1994) and Montgomery and Bodznick (1994a,b) have shown that responses to electrosensory stimuli in skates and to normal lateral line stimuli in scorpion fish are adaptively filtered or rejected if these are coupled to the animal's own ventilatory movements. The cells studied were found in the first order electrosensory and mechanosensory nuclei, respectively, which, as in the case of the gymnotiform and mormyrid ELL, receive parallel fiber inputs from associated populations of granule cells. Both proprioceptive inputs responsive to ventilatory movements as well as corollary discharges of ventilatory motor commands project to these granule cells (Conley and Bodznick 1994; Montgomery and Bodznick 1994b), and these inputs are the most likely source of the information used to generate the cancellation signals.

All of these structures showing sensory plasticity involving the generation of negative images of expected inputs share important anatomical features and all are cerebellar-like in their organization (Coombs et al. 1993, 1994; Maler and Mugnaini 1993). Bell et al. (1993) have proposed a model in which the negative image of a cell's expected receptor afferent input is sculpted from the parallel fiber inputs to its apical dendrites, and long-term depression (LTD) is proposed as the cellular mechanism underlying this plasticity. Montgomery and Bodznick (1994a,b) propose a model, expressed as a set of learning rules, which shares important features with the Bell et al. model. The mechanisms proposed by these authors can readily account for the plasticity seen in this study and Fig. 12 summarizes features of these models incorporating the properties of the ELL of *Apteronotus*.

As a consequence of tail displacements, the pyramidal cells receive ascending electrosensory inputs as well as inputs to the apical dendrites via the proprioceptive fibers that project to the EGp. Additionally, the EGp receives a descending electrosensory input from the n. praeeminentialis multipolar cells. These neurons have firing properties similar to those described for the proprioceptive fibers and their tonic response characteristic allows them to accurately encode the low-frequency EOD AM resulting from tail displacement. These multipolar cells also have large bilateral receptive fields and are well suited to encoding spatially extensive changes in EOD amplitude (Bastian and Bratton 1990). The axons of both the descending electrosensory and proprioceptive fibers ramify extensively within the EGp providing input to many individual granule cells. The granule cell axons,

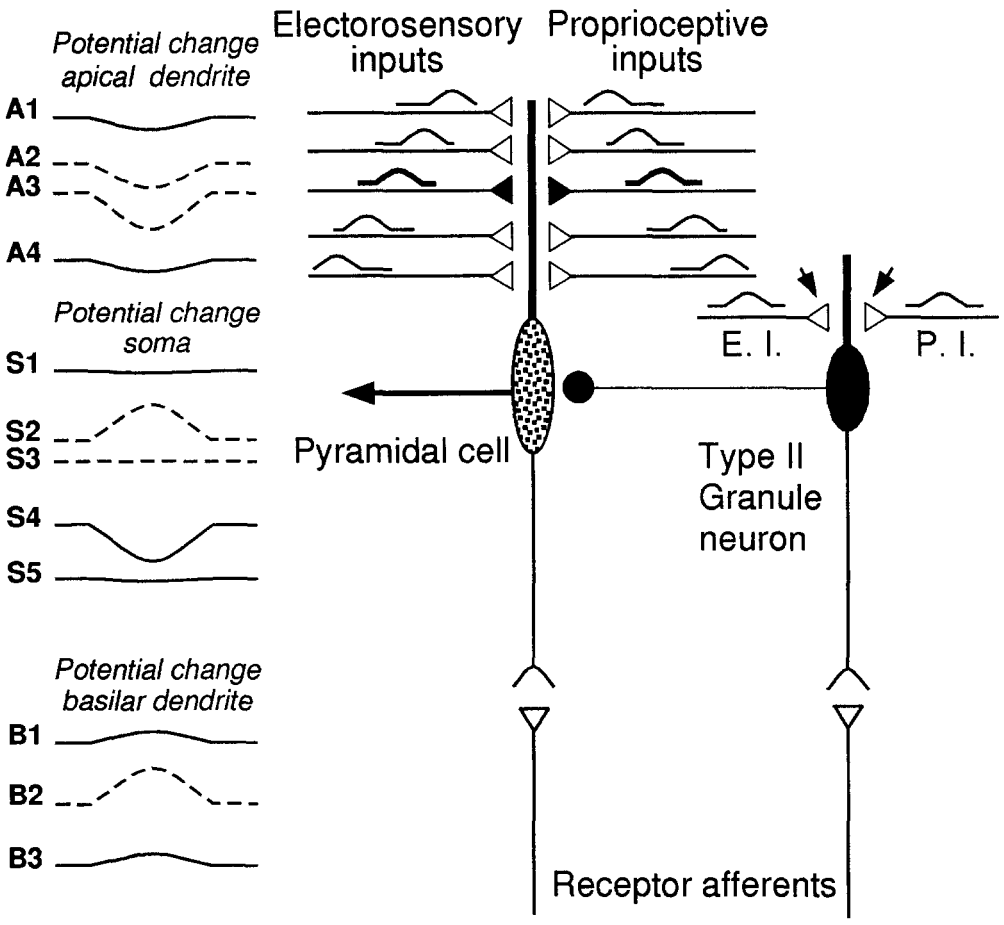

Fig. 12 Diagram summarizing mechanisms proposed to account for ELL pyramidal cell insensitivity to repetitive electrosensory inputs occurring alone or together with changes in the animal's posture. *Triangles* and *circles* indicate excitatory and inhibitory synapses, respectively. See text for explanation

dorsal molecular layer parallel fibers, enter the DML over most of its rostro-caudal extent and run for varying distances within the DML (Maler et al. 1974). Very large numbers of parallel fibers probably convey proprioceptive and descending electrosensory signals to the pyramidal cell apical dendrite as illustrated in Fig. 12. The proprioceptive and electrosensory signals carried by individual parallel fibers are presumed to have heterogeneous phase relationships. The type I and II proprioceptive fibers respond to cyclic stimuli roughly in antiphase (Fig. 3). Likewise, two categories of NP fibers project to the EGp and these are excited by increased and decreased EOD amplitude, respectively (Bastian and Bratton 1990). Differential delays due to varying conduction distances among parallel fibers synapsing on a given pyramidal cell should further separate individual inputs in time. These out-of-phase proprioceptive and electroreceptive signals are symbolized by the waveforms above each parallel fiber input to the apical dendrite of Fig. 12. These waveforms can be thought of as measures of parallel fiber spike rate which continuously fluctuate with periodic proprioceptive or electrosensory stimulation.

Assuming equivalent parallel fiber to pyramidal cell synaptic strengths, the summation of the enormous number of parallel fiber inputs carrying these out-of-phase signals will result in a steady (DC) or unpatterned input to the cell. Alteration of the synaptic strengths of specific subsets of these parallel fiber inputs can result in the generation of inputs to the apical dendrite capable of cyclically modulating pyramidal cell excitability. Specifically, reduction of the synaptic efficacy of parallel fiber inputs showing increased activity coincident with increased pyramidal cell firing (long-term depression or LTD), will result in the generation of an input that is the negative image of the current pattern of receptor afferent input received by a cell. Subtracting a periodic signal from a DC signal results in a signal of the same period, but in antiphase.

A pyramidal cell's initial lack of responsiveness to the EOD AMs due to tail displacement can be explained as a result of the cell's receipt of a negative image input via the apical dendrite (Fig. 12 A1) simultaneous with the receptor afferent input (Fig. 12B1); these cancel, hence the potential change at the soma is minimal (Fig. 12S1) and the cell does not respond. The negative image arises as a summation of signals from parallel fiber inputs to the apical dendrite, a subset of which show depressed synaptic strength. The depressed synaptic strength arises only at inputs active coincident with the increased receptor afferent input (e.g. the inputs indicated by thick lines and filled terminals are depressed). Prior to the depression of these specific synapses the increased receptor afferent activity is expected to increase pyramidal cell firing allowing LTD to develop as suggested by Bell et al.

(1993). Since, in these experiments, pyramidal cells were insensitive to the tail displacement AMs from the start, it is assumed that this initial negative image is normally present due to the animal's activity prior to the experiments or results from use of tail-displacements as a search stimulus.

When the local AM is introduced, increased receptor afferent input (Fig. 12B2) causes the pyramidal cell to initially respond strongly (Fig. 12S2) because the negative image generated at the apical dendrite (Fig. 12A2) is insufficient to completely cancel the receptor afferent input. The increased pyramidal cell activity further depresses the synaptic strength of simultaneously active parallel fiber inputs (possibly also increasing the number of depressed inputs) thereby increasing the amplitude of the negative image input. Over time, as the negative image grows and approaches the magnitude of the receptor afferent input (Fig. 12A2,A3) pyramidal cell responses are abolished (Fig. 12S2,S3).

Removal of the local AM restores receptor afferent input to its original value (Fig. 12B3). Since immediately after removal of the local stimulus, the negative image input is still large (Fig. 12A3) the cell's response is principally due to this input and the negative image response is observed (Fig 12S4). This response to the negative image input decays (Fig. 12S5) as the synaptic strengths of parallel fiber inputs are readjusted (Fig. 12A4) to cancel the current pattern of receptor afferent input. The experiments described in conjunction with figures 10 and 11 show that either electrosensory information or proprioceptive information alone is sufficient to allow the development of negative image inputs capable of at least partially canceling local electrosensory stimuli.

The lack of pyramidal cell responses to the electrosensory inputs associated with tail displacements probably results from the generation of negative image signals composed of both descending electrosensory and proprioceptive inputs. The pyramidal cells respond to the electronic AMs that mimic the tail-bend AMs because in the absence of tail movement all cyclic proprioceptive input to the system is removed. This is expected to reduce the size of the negative image signal below that needed to completely cancel the effects of the receptor afferent input due to the mimic AM, hence the cells respond as shown in Fig. 7C. It remains to be determined if, given enough time, the negative image generated solely from descending electrosensory information can completely cancel mimic AMs.

The role of inhibitory interneurons and LTD in pyramidal cells

The adaptive cancellation of repetitive inputs to pyramidal cells may also involve changes in the characteristics of ELL inhibitory interneurons. Several categories of these exist; some receive only inputs from molecular layer fibers, some receive primary afferent input, while others, such as the type I granule neuron shown in Fig. 12, receive both. Bell et al. (1993) suggest that the strength of the inhibitory synapses (Fig. 12, filled circle) on the ELL purkinje-like neuron of mormyrids (analogous to pyramidal cells studied here) is strengthened during depolarization, thereby augmenting the cancellation of peripheral inputs. Potentiation of inhibitory inputs resulting from increased depolarization of cerebellar purkinje cells has been shown (Kano et al. 1992). Additionally, the parallel fiber inputs to the inhibitory neurons (Fig. 12, arrows) may also be plastic, however, as indicated by Montgomery and Bodznick (1994b) the rules governing plasticity at this site must be opposite those governing the synaptic modifications of pyramidal cell apical dendritic synapses. Increased receptor afferent input coincident with apical dendritic inputs in inhibitory interneurons should strengthen the latter synapses. The enhanced inhibitory interneuronal activity would then help cancel the patterns of receptor afferent input received by the pyramidal cell. Field potential recordings demonstrate long-term potentiation of parallel fiber EPSP's within the ELL dorsal molecular layer of *Apteronotus* (Turner and Maler 1989), but whether inputs to inhibitory interneurons are selectively potentiated is not known.

The site of the plastic changes need not be at the synapses discussed thus far; changes could occur at synapses between proprioceptive and electroreceptive fibers and granule cells within the EGp or even in antecedent brain areas. However, preliminary experiments in which tail-bend stimuli or whole-body electrosensory stimuli are paired with pyramidal cell intracellular current injection indicate that the site of plasticity is at pyramidal cell synapses (data not shown). As seen by Bell et al. (1993) in mormyrids, intracellular current injection effectively substitutes for the local AM used in the experiments described here. Pyramidal cell responses to depolarizing current injection gradually diminish when paired with the tail-bend stimulus or whole body AMs, and upon removal of the synchronized current injection, strong negative image responses are seen.

Does plasticity play a role in the rejection of jamming stimuli?

In addition to roles in rejecting the electrosensory stimuli associated with changes in posture, the pyramidal-cell plasticity may also provide some measure of immunity to cyclic electrosensory stimuli from other sources. When individual weakly electric fish approach one another each animal is exposed to the sum of the EOD fields produced. In the case of animals like *Apteronotus*, which produce a continuous EOD, the summed fields produce a beat pattern. The AM frequency (beat frequency) is determined by the difference of the two individual's discharge frequencies and when the beat frequency is low, e.g. less than about 10 Hz, their ability to localize objects via electroreception deteriorates (Heiligenberg 1973, 1977). Most species of weakly electric fish can recover from this electrosensory jamming by executing the

"jamming avoidance response" (JAR); a behavior by which animals alter their EOD frequencies to enlarge the beat frequencies to higher values which are less deleterious (see Heiligenberg 1991 for review of JAR). Behavioral studies of *Apteronotus* suggest that central nervous system mechanisms may also contribute to an animal's ability to selectively reject the repetitive AMs during these jamming situations (Bastian 1987a,b). The ELL pyramidal cells are clearly capable of undergoing large changes in sensitivity to repetitive electrosensory inputs even in the absence of associated proprioceptive inputs as shown in Fig. 10 and preliminary experiments verify that this plasticity also occurs when the AM frequencies are higher, in excess of 4 Hz. Therefore, the pyramidal-cell plasticity could contribute to the ability to detect novel stimuli when the animal is simultaneously encountering repetitive AMs due to the presence of the discharges of other fish. This mechanism could be particularly important for the weakly electric fish *Sternopygus*; these animals do not produce the jamming avoidance behavior yet their ability to electrolocate is very insensitive to jamming stimuli that simulate the discharges of conspecifics (Matsubara and Heiligenberg 1978).

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