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The Mormyromast Region of the Mormyrid Electrosensory Lobe. I. Responses to Corollary Discharge and Electrosensory Stimuli

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Mohr, Claudia, Patrick D. Roberts, and Curtis C. Bell. The mormyromast region of the mormyrid electrosensory lobe. I. Responses to corollary discharge and electrosensory stimuli. *J Neurophysiol* 90: 1193–1210, 2003; 10.1152/jn.00211.2003. This is the first of two papers on the electrosensory lobe (ELL) of mormyrid electric fish. The ELL is the first stage in the central processing of electrosensory information from electroreceptors. Cells of the mormyrid ELL are affected at the time of the electric organ discharge (EOD) by two different inputs, EOD-evoked reafferent input from electroreceptors and corollary discharge input associated with the motor command that elicits the EOD. This first paper examines the intracellular responses of ELL cells to these two different inputs in the region of ELL that receives primary afferent fibers from mormyromast electroreceptors. Mormyromast electroreceptors are responsible for active electrolocation. The paper extends previous studies of the mormyrid ELL by describing the physiological responses of cell types, which had been previously identified only morphologically, including: the two types of Purkinje-like medium ganglionic cells, MG1 and MG2; the thick smooth dendrite cells; and the medium fusiform cells. In addition, two previously unrecognized cell types, the large thick smooth dendrite cell and the interzonal cell, are described both morphologically and physiologically for the first time. Finally, new information is provided on the two types of ELL efferent cells, the large ganglionic and large fusiform cells. All cell types, except for the medium fusiform cell, show nonlinear interactions between electrosensory and corollary discharge inputs. All cell types, except for the medium fusiform cell and the interzonal cell, also show plasticity of the corollary discharge response after pairing with electrosensory stimuli.

INTRODUCTION

Most sensory processing regions in the brain receive descending input from other central structures in addition to ascending input from the periphery, but the roles of this descending input and the precise information that it conveys are poorly understood. The present study is part of a long-term project aimed at understanding the integration of peripherally originating sensory information with centrally originating descending information in a structure that is particularly suited for examining this issue, the electrosensory lobe (ELL) of mormyrid electric fish.

Various types of descending inputs have been identified in sensory systems, including: input from higher levels of the same sensory modality as the structure under study (Bastian 1986; Brandt and Apkarian 1992; Sillito et al. 1994; Singer 1995); input from central structures that convey information about other sensory modalities (Jay and Sparks 1984; Meredith and Stein 1983); and corollary discharge input associated with motor commands (Bell et al. 1992; Suga and Schlegel 1972; Toyama et al. 1984; Zipser and Bennett 1976). The last type, corollary discharge input, prepares a sensory region for the reafferent (von Holst and Mittelstaedt 1950) sensory responses that will arrive as a consequence of the motor action. All three types of descending input are present in the mormyrid ELL, but corollary discharge effects are particularly prominent and particularly accessible in ELL. The present study focuses on the interaction in ELL between peripherally originating electrosensory information and centrally originating corollary discharge signals associated with the motor command that drives the electric organ discharge (EOD). The goal is to understand how the information provided by these two types of inputs is integrated within the cells and circuitry of ELL.

The mormyrid ELL is a cerebellum-like structure and the first stage in the central processing of information from electroreceptors. Primary afferent fibers from electroreceptors terminate in the deeper layers of ELL where they form a map of the body surface. Purkinje-like cells, known as medium ganglionic (MG) cells, and efferent cells of ELL are strongly affected by the afferent input from electroreceptors, the afferent input being relayed to the basilar dendrites of these cells via ELL granular cells that receive the afferent input directly. The apical dendrites of MG cells and efferent cells extend up into a molecular layer where they are contacted by parallel fibers that originate from an external granule cell mass known as the eminentia granularis posterior (EGp). As with true cerebellar Purkinje cells in all ray-finned fish, the MG cells of ELL are inhibitory interneurons that terminate locally on efferent neurons. Other types of interneurons, such as granular cells, stellate cells, thick smooth dendrite cells and medium fusiform cells are also present in ELL (see Han et al. 1999; Meek et al. 1996 for a description of the morphology of ELL cells).

Primary afferent fibers from two types of electroreceptors terminate in the cortex of ELL (Bell 1990b; Bell et al. 1989). Afferent fibers from mormyromast electroreceptors, the type of electroreceptors responsible for active electrolocation, terminate in the medial and dorsolateral zones of ELL. Afferent fibers from ampullary electroreceptors, the type responsible for low-frequency passive electrolocation, terminate in the ventrolateral zone. The mapping in all three zones is somatotopically

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organized. This study is concerned only with the mormyromast zones of ELL.

Some of the electric organ corollary discharge (EOCD) effects in ELL are plastic and depend on the sensory input that has followed the motor command in the previous few minutes (Bell and Grant 1992; Bell et al. 1997b). Other EOCD effects appear to be fixed and are not affected by previous pairing with a sensory stimulus (Bell and Grant 1992). The EOCD signals enter ELL via three different pathways: parallel fibers from EGp (Bell and Szabo 1986; Maler 1973); fibers from a higher-order electrosensory nucleus, the nucleus preeminentialis (Bell et al. 1981); and fibers from the juxtalobar nucleus (Bell et al. 1981).

A previous study of the mormyrid ELL examined the electrosensory and EOCD responses of MG cells and efferent cells (Bell et al. 1997b). This description was incomplete, however, because the responses of several morphologically distinct cell types were not determined. Most importantly, two types of MG cells (MG1 and MG2) can be distinguished morphologically (Han et al. 1999; Meek et al. 1996), but no corresponding difference was established physiologically in the previous study. The morphology suggested that MG1 cells might be inhibited by electrosensory stimuli (I cells), whereas MG2 cells might be excited (E cells), but the previous study found mostly inhibitory receptive fields for MG cells and the morphology was not good enough to distinguish the two types of MG cells (Bell et al. 1997b; Grant et al. 1998).

This first paper in the present series of two papers describes the electrosensory and EOCD responses of cell types, which had been previously described morphologically but not physiologically. These cells include the two types of MG cells, the thick smooth dendrite cells and the medium fusiform cells. New information is also provided concerning the common features of MG cells, the two types of efferent cells, and the properties of two previously undescribed cell types, the interzonal cell and the large thick smooth dendrite cell. The second paper in this series examines the origins of EOCD responses in ELL by recording the responses of morphologically identified ELL cells to electrical stimulation of two of the three sources of EOCD input to ELL, the juxtalobar and preeminential nuclei.

METHODS

Only a brief description of the methods is given here. A more complete description can be found in a previous publication (Bell et al. 1997b). All experiments that were performed in this study adhere to the American Physiological Society's *Guiding Principles in the Care and Use of Animals* and were approved by the Institutional Animal Care and Use Committee of Oregon Health and Sciences University.

Overview

Mormyrid fish of the species *Gnathonemus petersii* were used in these experiments. Surgery was done under anesthesia and curare was given after the surgery. Curare blocks the effect of electromotoneurons on the electric organ, preventing the EOD; but the motor command signal that would normally elicit an EOD continues to be emitted by the electromotoneurons at a variable rate of 2–5 Hz. Responses of ELL cells to the motor command alone are referred to as EOCD responses. The curare makes it possible to examine the EOCD responses in isolation from the EOD that normally follows the motor command and to control the electrosensory input that the cells receive. Responses to the motor command alone, to electrosensory stimuli alone, and to the motor command plus an electrosensory stimulus delivered at various delays were examined. EOCD plasticity was examined by delivering electrosensory stimuli at a fixed delay following the EOD command signal for 1–3 min and comparing the EOCD responses before and after such pairing.

ELL cells were recorded intracellularly from the medial and dorsolateral zones of ELL, the two zones that receive input from mormyromast electroreceptors (Bell and Grant 1989). The cell types are quite similar in the two zones and most of the recordings were taken from the medial zone, which is larger in size and more accessible than the dorsolateral zone.

The field potentials evoked by electrosensory stimuli and by the EOCD in ELL are prominent and change dramatically as a function of depth (Bell et al. 1992), allowing one to determine the ELL layer in which the recording electrode was positioned from the extracellular potentials recorded just outside the cell. The different layers of ELL are as follows (from the external to the internal surface of ELL): molecular, ganglionic, plexiform, superficial granular, deep granular, intermediate, and fiber (Grant et al. 1996; Meek et al. 1999). In most cases, the field potentials were recorded just outside a cell after intracellular recording, using the same electrode. In most cases, the field potentials were averaged and subtracted from averaged intracellular recordings to determine the true transmembrane potential changes evoked by electrosensory stimuli and the EOCD.

Surgery

A total of 42 fish with body lengths between 11.5 and 18 cm were used. The skull was exposed under anesthesia (MS 222, 1:25,000), and a plastic rod was cemented to the skull anteriorly to hold the head rigid. The posterior part of the skull was removed, and the underlying valvula cerebelli was reflected laterally to expose the molecular layer of the caudal lobe of the cerebellum and the EGp. The ELL is located just beneath these structures. Curare (d-tubocurarine, 10 μg/cm of body length) was given at the end of the surgery, the anesthetic was removed, and aerated water was passed over the fish's gills for respiration.

Recording, stimulation, and data analysis

The EOD command signal was recorded with an Ag-AgCl silver plate placed over the electric organ. The command signal lasts ~3 ms and consists of a small negative wave followed by three larger biphasic waves (Fig. 2, *B* and *D, bottom*). The latencies of synaptic and spike responses of ELL neurons to the EOCD were measured with respect to the negative peak of the first large biphasic wave in the command signal (*time 0* or t_0 in Bell et al. 1992). In the absence of curare, the EOD occurs 4.5 ms after t_0.

Intracellular recordings were made with sharp microelectrodes filled with 2% biocytin in 2 M potassium methyl sulfate (180–250 MΩ). Biocytin was injected into recorded cells by passing depolarizing intracellular current pulses at 1 Hz with a duty cycle of 50% and amplitudes of 1–1.2 nA for 5–12 min.

Electrosensory responses were evoked by means of a bipolar stimulating electrode consisting of two small Ag-AgCl balls 6 mm apart. The electrode was held with the axis of the dipole perpendicular to the skin. Individual electroreceptors can be easily distinguished on the skin surface with an operating microscope, and the stimulating electrode could be placed close to the individual receptors. Brief pulses of current (100 μs, 1.5–100 μA) were delivered through the electrode to activate electroreceptors. All cells were tested with sensory stimuli at the EOD delay of 4.5 ms. Cells were also examined either independently of the motor command or at long delays of 60–100 ms to examine the effect of a sensory stimulus alone.

The latency of the electrosensory response of mormyromast affer-

ent fibers decreases by 9–11 ms as intensity is increased from threshold intensity to the intensity that gives a maximum response (Bell 1989, 1990a). The latencies reported in this study for the electrosensory responses of different ELL cells are minimal latencies obtained by increasing stimulus intensity until no further reduction in latency was observed. The current intensity needed to obtain the minimal latency varied from cell to cell. Use of minimal onset latencies makes it possible to compare the timing of responses in different cells.

Data were recorded on tape and analyzed off-line with a Cambridge Electronic Design interface and with the same company's software. For statistical comparisons, we used the *t*-test.

Spikes were sometimes truncated by a linear extrapolation from the beginning of each spike to the end of the spike to estimate the size of the underlying excitatory postsynaptic potentials (EPSPs). For truncation purposes, we determined the beginning of each spike by first determining the time of a threshold crossing in the voltage record (for broad spikes) or in the derivative of the voltage record (for small spikes). Aligning and averaging the spikes about this threshold crossing allowed us to establish the time relative to the threshold crossing when the spike began and when the spike ended. The beginning of the spike was ~2 ms before the threshold crossing for broad spikes and ~1 ms before the threshold crossing for small spikes.

Histology

After the experiment, fish were anesthetized in concentrated MS 222 (1:10,000) and perfused through the heart with teleost Ringer solution, followed by a fixative, consisting of 2% paraformaldehyde and 2% glutaraldehyde in 0.1 M phosphate buffer. The brains were postfixed overnight and cryoprotected with 20% sucrose. Cryostat sections (50 μm) were reacted with avidin-biotin complex and diamino-benzidine to reveal the biocytin. The procedure was the same as that used by Han et al. (1999) except that $CoCl_2$ (0.02%) and ammonium nickel sulfate (0.02%) were added to the bath during development to enhance the reaction. Sections were mounted on slides and counterstained with Richardson's stain. Reconstructions of cells were made with a camera lucida attachment to the microscope.

RESULTS

MG cells

The Purkinje-like MG cells are a major cell type of ELL and are probably of central importance to its function. Synaptic plasticity has been demonstrated at the synapse between parallel fibers and the apical dendrites of MG cells (Bell et al. 1997c; Han et al. 2000), and this plasticity can explain the adaptive sensory processing that occurs in ELL (Bell et al. 1993, 1997b; Grant et al. 1998). Two morphologically distinct types of MG cells, MG1 and MG2, have been identified. The two cell types have similar apical dendrites, but their basal dendrites and axonal arbors terminate in different layers of ELL (Meek et al. 1996; Han et al. 1999) (Fig. 1). The dendritic morphology suggested the hypothesis that MG2 cells, with basal dendrites in the region of termination of primary afferent fibers, might be excited by electrosensory stimuli, whereas MG1 cells, with basal dendrites external to the region of afferent termination, might be inhibited by electrosensory stimuli (Han et al. 1999; Meek et al. 1996). We tested this hypothesis in the present experiments and also established some additional, previously undescribed, features of MG cells.

Seven MG1 and four MG2 cells were identified morphologically in this study. An additional four cells were morphologically identified as MG cells, but the basal dendrites and axons were not sufficiently stained to identify the cells further as MG1 or MG2 cells. We first describe the physiological properties that are shared by MG1 and MG2 cells and then describe the differences between the two cell types.

TWO TYPES OF SPIKES IN MG CELLS. Previous in vivo (Bell et al. 1997b) and in vitro studies (Grant et al. 1998) showed that MG cells have two types of spikes, a large broad spike and a small narrow spike (Fig. 2, *A*, *C*, and *D*). MG cells are the only

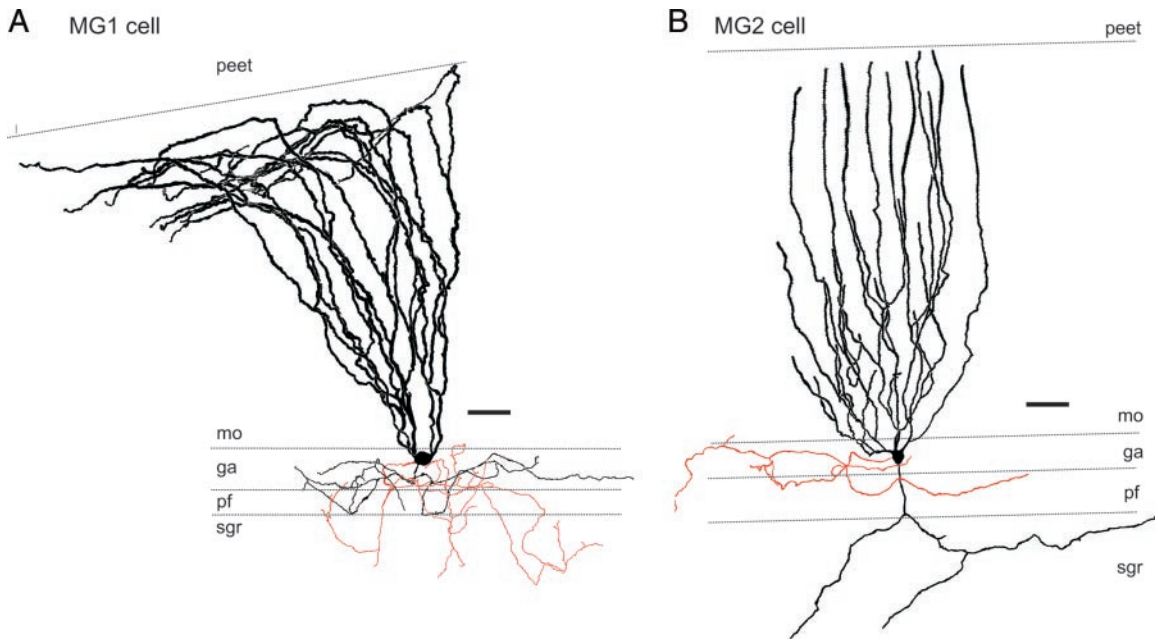

FIG. 1. Morphology of the 2 subtypes of medium ganglionic cells: MG1 (*A*) and MG2 (*B*). Dendrites are black and axons are red in this and subsequent figures. The thin lines indicate the different layers in electrosensory lobe (ELL). ga, ganglionic layer; mo, molecular layer; peet, preeminential-electrosensory tract; pf, plexiform layer; sgr, superficial granular layer. Bars: 50 μm. Both of these cells, as well as the other cells shown in subsequent figures, were recorded and studied intracellularly before being injected with biocytin for morphological identification.

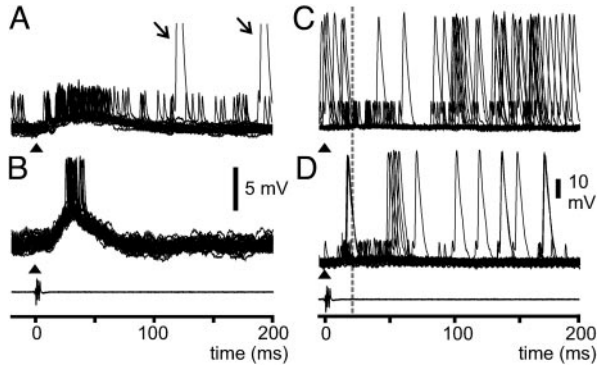

FIG. 2. Electric organ corollary discharge (EOCD) responses of medium ganglionic cells. *A*: EOCD evokes an EPSP and a burst of small spikes. The first small spike is more time-locked than the other spikes and is separated from them by a small gap. Arrows point to truncated broad spikes. *B*: the EOCD evokes an EPSP and 1 small spike. *C*: the EOCD response in a cell with a high spontaneous rate for both broad and small spikes. Note the decrease of broad spikes between 20 and 80 ms, and the increase of small spikes during part of this period. *D*: the EOCD frequently evokes a broad spike at a short fixed latency of 15 ms, followed by an apparent 30-ms pause in broad spike activity. *B* and *D, bottom*: the command signal as recorded over the electric organ. The first large negative wave of the command signal is defined as *time 0* (t_0) and is marked by black triangles in this and the following figures.

cells in ELL with large broad spikes (Bell et al. 1997b; Grant et al. 1998), and the presence of these spikes can therefore be used to identify MG cells electrophysiologically. We identified 62 cells as MG cells by this physiological feature, including the 15 MG cells identified morphologically. The large broad spikes ranged from 20 to 67 mV in amplitude (44.3 mV ± 9.7 mV (mean ± SD), $n = 48$) and 8–23 ms in duration (14 ± 3.4 ms, $n = 48$). The small narrow spikes ranged from 1.6 to 13.5 mV (6.6 ± 3.1 mV, $n = 49$) and were 2–6.2 ms (3.9 ± 0.9 ms, $n = 49$) in duration.

EOCD-evoked field potentials recorded extracellularly immediately after the intracellular recordings allowed us to estimate the ELL layer from which the recordings were taken. Broad spikes of similar amplitudes and durations were recorded in both the outer and inner halves of the molecular layer, suggesting that the broad spikes are propagated actively into the apical dendrites of MG cells. This result is consistent with other evidence for such propagation obtained in in vitro slices (Grant et al. 1998). In contrast, the small spikes of MG cells were either absent in the outer molecular layer or of lower amplitude than in the inner molecular layer or ganglionic cell layer (data not shown), consistent with the hypothesis that the small spikes are axon spikes that are not actively propagated into the soma or dendrites of MG cells (Grant et al. 1998).

EOCD RESPONSES. The EOCD evoked an EPSP in most MG cells (Fig. 2, *A* and *B*). The EPSPs had amplitudes of 0.6−6 mV (4.7 ± 4.9 mV, $n = 54$), durations of 45–100 ms ($n = 54$), and latencies following t_0 of 5.2–13.2 ms (8.5 ± 1.7 ms, $n = 35$). The EPSP usually evoked a burst of small spikes in which the first spike of the burst was more sharply time-locked to the command signal than later spikes (Fig. 2*A*). In some cells, the EOCD EPSP evoked only a single small spike (Fig. 2*B*) or no spike at all (*middle*, Fig. 4*A*).

The effect of the EOCD on broad spikes could not be easily determined in most MG cells because of the low probability of these spikes. Broad spikes occurred spontaneously in 17 MG cells, however, allowing the EOCD responses to be observed. In these cells, the EOCD usually evoked only an inhibition of broad spikes (11/17 cells; Fig. 2*C*). The inhibition, as indicated by a consistent pause in spontaneous broad spike activity, started at ~20 ms after t_0 and lasted between 10 and 80 ms. In some cells, the EOCD had an initial excitatory effect, evoking a time-locked broad spike between 12 and 18 ms after t_0 (6 of 17 cells; Fig. 2*D*). The time-locked broad spike of these cells was followed by a brief period in which no broad spikes occurred. This brief period without broad spikes was not due to refractoriness because it was also present in those sweeps in which the time-locked broad spike did not occur. Thus the brief initial excitation of broad spikes in some MG cells was followed by an inhibition.

The inhibition of broad spikes was particularly striking in that it occurred during the time of the EOCD-evoked EPSP and its accompanying burst of small spikes (Fig. 2, *C* and *D*). The presence of two such opposing EOCD effects in MG cells was further indicated by the finding that some cells showed an EOCD-evoked EPSP at one time during the recording and an EOCD-evoked inhbitory postsynaptic potential (IPSP) several minutes later. Transformation from an EPSP to an IPSP often occurred after several minutes of injecting current (1–1.2 nA) into a cell for morphological identification (Fig. 3*A*). The shape of the EPSP and the IPSP were not identical, usually the EPSP lasted longer (Fig. 3*A*, *2nd trace*). In some cases, the postinjection IPSP changed back into an EPSP (Fig. 3*A*, *3rd trace*) over several additional minutes of recording. The differences between depolarizing and hyperpolarizing EOCD responses were large and were not accompanied by any apparent changes in membrane potential. Thus, it is unlikely that the EOCD responses are due to an IPSP alone, that is, an IPSP which is

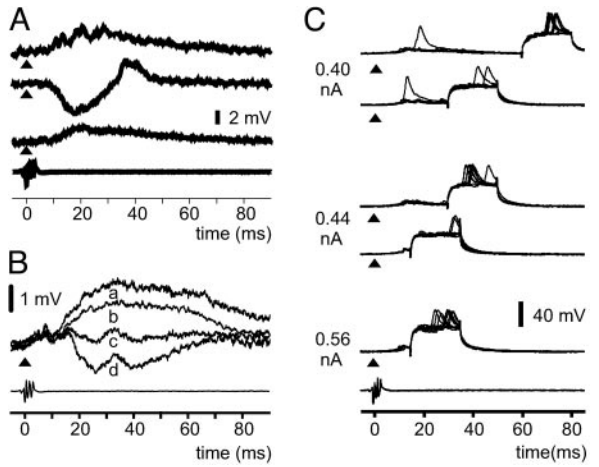

FIG. 3. EOCD evoked inhibitory effects in medium ganglionic (MG) cells. *A*: the EOCD-evoked excitatory postsynaptic potential (EPSP) observed before current injection in this cell (*top*) was replaced with an EOCD-evoked inhibitory postsynaptic potential (IPSP, *middle*) after several minutes of injecting 1-nA current pulses for 10 min to label the cell. The EOCD evoked response became an EPSP again (*bottom*) several minutes after the end of the current injection. *B*: depolarization of this MG cell with injected current reduced the EOCD-evoked EPSP and revealed the presence of an EOCD evoked IPSP that was roughly coincident in time with the EPSP. The traces recorded at different levels of depolarization have been overlaid for comparison purposes. Trace a is the least depolarized and d is the most depolarized. *C*: the EOCD evokes an increase in broad spike threshold. An intracellular current pulse was delivered at different delays after the EOD motor command. Intensity was adjusted until the pulse evoked a broad spike on 6 of 7 presentations, and the delay was then reduced. Note the higher intensity that is needed to evoke this number of spikes at a 15-ms delay (*bottom*) than at a 60-ms delay (*top*).

either depolarizing or hyperpolarizing depending on the membrane potential and the IPSP reversal potential. Moreover, our electrodes did not contain any chloride ions so that the current injection would not have altered the chloride concentration in the cell and the reversal potential for IPSPs.

Evidence for simultaneous EOCD excitation and EOCD inhibition was also obtained in one cell in which depolarizing the cell from resting potential with increasing amounts of injected current caused a progressive reduction of the EPSP and appearance of an IPSP which was shorter in duration (Fig. 3*B*, compare a and d). The different effects of the EOCD on broad spikes and small spikes indicates different initiation sites for the two types of spikes, with EOCD-driven inhibition having a stronger effect on broad spike initiation and EOCD-driven excitation having a stronger effect on small spike initiation.

The delivery of intracellular current pulses to evoke broad spikes at different delays after the command signal also revealed the EOCD-evoked inhibition of broad spikes. The current required to evoke broad spikes at a short delay of 20 ms was consistently greater than that required to evoke spikes at other delays (Fig. 3*C*). The effect was observed in four of the five cells tested.

RESPONSES TO ELECTROSENSORY STIMULI. Although the EOCD responses of MG1 and MG2 cells were the same, the responses to electrosensory stimuli were quite different. Our hypothesis that MG1 cells are inhibited by electrosensory stimuli and MG2 cells are excited was confirmed.

All seven of the morphologically identified MG1 cells responded with an IPSP to electrosensory stimuli delivered within a restricted skin region. The IPSP was often larger when the stimulus was given at the EOD delay of 4.5 ms after t_0 of the EOD motor command (Fig. 4*A*, *bottom*) than when it was given at long delays or independently (Fig. 4*A*, *top*). Thus the EOCD facilitates the inhibitory effect of electrosensory stimuli on MG1 cells, when such stimuli are given at the time of the EOD. The enhanced IPSP was sometimes followed by an increased excitation (Fig. 4*A*, *bottom*) although no EPSC was observed to the sensory stimulus alone (Fig. 4*A*, *top*). The excitation following the IPSP could be due to an alteration in the input to the cell through the circuitry of ELL or to an intrinsic postinhibitory rebound of the postsynaptic membrane, such as that which occurs when T type calcium channels are present (Carbone and Lux 1984). In some cells, delivery of the sensory stimulus at the EOD delay markedly depressed the EOCD evoked excitation without evoking an actual hyperpolarization (Fig. 4*B*). An additional 10 MG cells, identified as such by the occurrence of a broad spike, responded to electrosensory stimulus with an IPSP and showed similar EOCD facilitation of the inhibitory sensory effect or suppression of the EOCD EPSP, just like the seven morphologically identified MG1 cells. We classified all 17 cells as MG1 cells.

Low-intensity electrosensory stimuli close to threshold-evoked IPSPs from three to six neighboring electroreceptors in MG1 cells. Increases in stimulus intensity caused an increase in IPSP amplitude and a decrease in latency. Thresholds were between 2.5 and 6 μA (4.1 ± 1.1, $n = 10$). Minimum latencies at the highest stimulus intensities were between 2.8 and 5.1 ms (3.9 ± 0.8 ms; $n = 13$). No excitatory responses were observed when the stimulus electrode was placed just outside the cluster of receptors where stimulation caused an IPSP. Thus there was no evidence of an opponent, excitatory surround outside the region of inhibition.

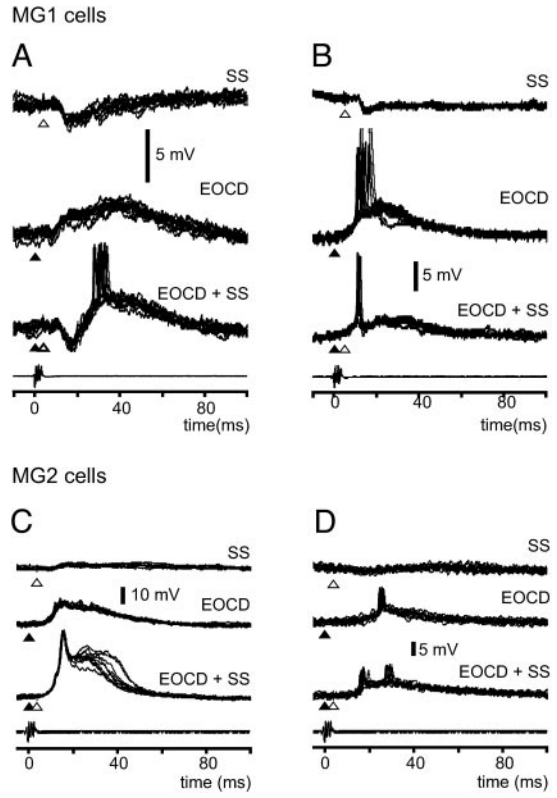

FIG. 4. Interactions between EOCD responses and sensory responses in MG cells. *A*: an MG1 cell showing an IPSP to the sensory stimulus alone (SS), an EPSP to the EOCD alone and an IPSP-EPSP to the EOCD plus a sensory stimulus given at the time of the EOD (EOCD + SS). The IPSP evoked by the sensory stimulus given at a short delay after the command signal (EOCD + SS) is significantly larger ($P < 0.001$) than the IPSP evoked by the sensory stimulus alone (SS). Similarly, the EPSP evoked by the sensory stimulus given at a short delay (which follows the IPSP) is significantly larger ($P < 0.001$) than the EPSP to the command signal alone (EOCD). The time of delivery of a sensory stimulus is indicated by a clear triangle in this and subsequent figures. *B*: another MG1 cell with an IPSP to a sensory stimulus alone (SS) and an EPSP to the EOCD alone (EOCD). The EPSP evokes small spikes and broad spikes. The 2 broad spikes are truncated. Delivery of the same sensory stimulus after the EOD command at the time of the EOD (EOCD + SS) reduces the EPSP significantly ($P < 0.001$). Note that the broad spikes are followed by afterhyperpolarizations. *C*: an MG2 cell with a small EPSP to the sensory stimulus alone and an EPSP to the EOCD. Delivery of the same sensory stimulus after the EOD command at the time of the EOD evokes a more rapidly rising EPSP that in turn evokes a broad spike followed by a variable plateau-like depolarization that is probably a combination of EPSP and intrinsic voltage response. The amplitude of this response is significantly larger than the response to sensory alone ($P < 0.001$). *D*: another MG2 cell with a small IPSP to the sensory stimulus alone, and an EPSP with a single spike to the EOCD. Delivery of the same sensory stimulus after the EOD command at the time of the EOD evokes a larger EPSP with a sharper rise time and 2 spikes.

Electrosensory responses of MG2 cells were quite different from those of MG1 cells. The difference was particularly striking when stimuli were given at the EOD delay. In all four of the morphologically identified MG2 cells, the excitatory response to the EOCD plus an electrosensory stimulus was considerably greater than the response to the EOCD alone (Fig. 4, *C* and *D*). This enhancing effect of an electrosensory stimulus on the EOCD response contrasted with the suppressive effect of such a stimulus in MG1 cells. An additional seven

MG cells showed the same enhancing effect of an electrosensory stimulus as observed in the four morphologically identified cells. We classified all 11 cells as MG2 cells. We conclude that electrosensory stimuli given at the time of the EOD have an inhibitory effect on MG1 cells (I cells) and an excitatory effect on MG2 cells (E cells).

The sensory responses of MG2 cells were more difficult to determine than those of MG1 cells. The thresholds were more variable and often much higher than those of MG1 cells (range: 6–50 μA, 24 $\pm$ 13 μA, $n = 10$). The responses to sensory stimuli given independently of the command were smaller and more varied than those of MG1 cells. Electrosensory stimuli given independently of the command evoked a small EPSP in six cells (Fig. 4*C, top*), a broad spike with no clear underlying EPSP in two cells, and a small IPSP-EPSP in three cells (Fig. 4*D, top*). The minimum latencies of sensory responses were between 4.9 and 12.9 ms (9.6 $\pm$ 2.9 ms). We expected that MG1 and MG2 cells would have similar sensitivities to electrosensory stimuli. The finding of higher thresholds and greater variability of electrosensory responses in MG2 cells than in MG1 cells was unexpected therefore and the reason for these differences is not known.

The occurrence of EPSPs in some MG2 cells and IPSPs in other MG2 cells in response to stimuli given independently of the command suggests the possibility of receptive fields with center-surround organization. But no evidence for such organization was obtained. Individual cells showed only EPSP responses or only IPSP-EPSP responses regardless of the location of the stimulus on the skin. The variations in electrosensory responses of MG2 cells point to the complexity of information transfer through the ELL granular layer from afferents to the basilar dendrites of MG2 cells.

PLASTICITY OF THE COROLLARY DISCHARGE RESPONSE. The responses of MG cells to the EOCD have been previously shown to be plastic. The EOCD responses could be altered by pairing the EOD motor command for a few seconds to a few minutes with intracellular current injections that evoke broad spikes (Bell et al. 1997b) or with electrosensory stimuli in the receptive field of the cell (Bell et al. 1997b). We tested the effects of pairing the EOD motor command with electrosensory stimuli to compare plasticity in MG1 and MG2 cells.

EOCD excitation of MG1 cells was plastic. EOCD excitation after 2–3 min of pairing with an inhibitory electrosensory stimulus given at the EOD delay was stronger than EOCD excitation before the pairing. After pairing, the EOCD evoked a larger EPSP, more spikes, or spikes at a shorter delay (Fig. 5). Both broad and small spikes were affected. The plasticity could also be observed during the pairing period as a gradual decrease in the inhibitory response to the combined effects of the EOCD and a sensory stimulus (note smaller IPSP in EOCD+SS end than in EOCD+SS start in Fig. 5*A*). Plasticity was observed in seven MG1 cells out of the eight tested.

EOCD excitation of MG2 cells was also plastic, but the plasticity was less marked than in MG1 cells. In seven of eight cells tested, the number of spikes in the EOCD response showed a slight increase after 2–3 min of pairing with an excitatory sensory stimulus (Fig. 6). In three cells, this increase in spike number was significant ($P < 0.001$). When the pairing with a sensory stimulus evoked a broad spike (3 of 7 cases), the amplitude of the underlying EOCD EPSP was reduced significantly ($P < 0.05$) (Fig. 6*A;* compare EPSP in EOCD before trace with EPSP in EOCD after trace). This apparently paradoxical result, of a decrease in EPSP size with an increase in the number of evoked spikes, was not accompanied by a change in the recorded membrane potential. The increased spike number could be due to a change in the intrinsic excitability of the cell, but this possibility was not tested directly. When no broad spike was evoked during the pairing, the EOCD EPSP after the pairing either remained the same or was slightly increased (4 of 7 cases; Fig. 6*C*). These latter amplitude changes were not significant.

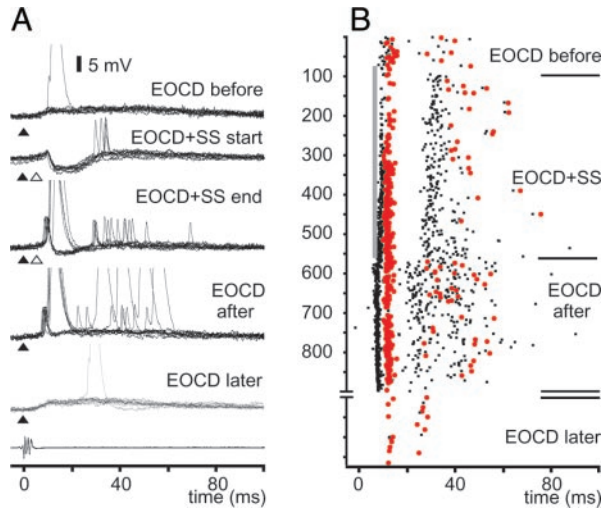

FIG. 5. Plasticity of the EOCD responses of an MG1 cell. *A*: intracellular recordings showing responses of an MG1 cell to the EOCD before pairing with an electrosensory stimulus (EOCD before), at the beginning of pairing (EOCD + SS start), at the end of 2 min of pairing (EOCD + SS end), immediately after the pairing (EOCD after), and several minutes later (EOCD later). Sensory stimuli were given at the EOD delay. Ten superimposed sweeps are shown in each of the 4 sets of traces. Note reduction of sensory-evoked IPSP and increased number of spikes during pairing. The number of small spikes after the pairing (EOCD after) is increased significantly ($P < 0.001$). *B*: raster display of responses to EOCD before, during, and after pairing with an electrosensory stimulus. Derived from the same cell and recordings as shown in *A*. The thick grey line indicates the occurrence and delay of the electrosensory stimulus, and the numbers on the ordinate indicate the sequential number of EOD command driven sweeps. Small spikes are shown as black dots and broad spikes are shown as red dots.

Thick smooth dendrite cells

Thick smooth dendrite (TSD) cells have been previously described morphologically (Han et al. 1999; Meek et al. 1996), and in vitro studies have shown that they respond to parallel fiber stimulation with an EPSP and to stimulation in the deep layers of ELL with an IPSP (Grant et al. 1998). However, the physiological responses of TSD cells to the EOCD or to electrosensory stimuli have not been determined. Seven cells were morphologically identified in this study after intracellular recording.

TSD cells are non-GABAergic interneurons with cell bodies in the ganglionic or plexiform layer (Han et al. 1999; Meek et al. 1996). The dendritic morphology is unusual for a vertebrate neuron in that one thin dendrite arises from the cell body and leads to one (Fig. 7*A*) or two (*C*) remarkably thick branches in the molecular layer that in turn give rise to additional thinner branches. The molecular layer dendrites are confined to the

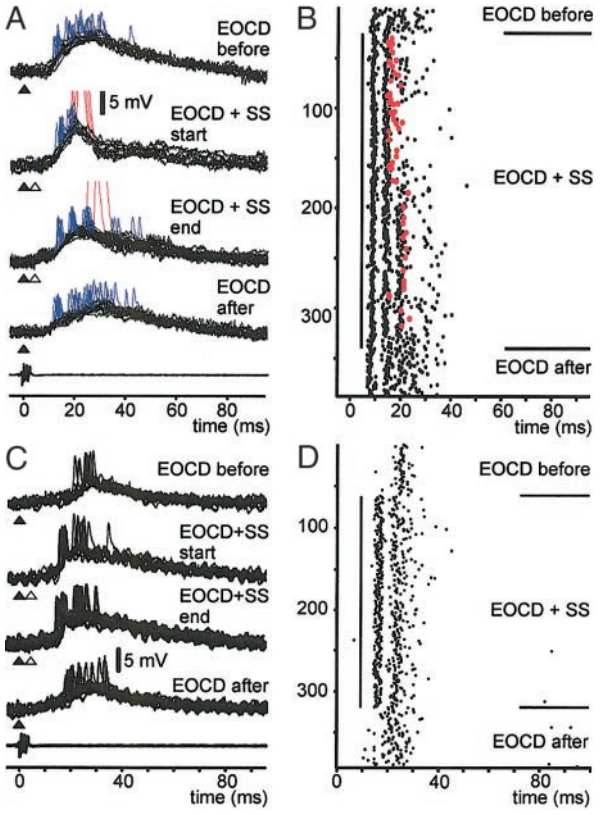

FIG. 6. Plasticity of the EOCD responses of two MG2 cells. *A*: intracellular recordings showing the responses of an MG2 cell before pairing with an electrosensory stimulus, at the start of pairing, at the end of 2 min of pairing, and after pairing. Ten superimposed sweeps are shown in each set of traces. Small spikes are shown in blue and truncated broad spikes are shown in red. Note that the EOCD-evoked EPSP after pairing (EOCD after) is reduced in comparison to the EOCD-evoked EPSP before pairing (EOCD before). *B*: raster display of EOCD responses of small spikes (black) and broad spikes (red) before, during, and after pairings with an electrosensory stimulus. Derived from the same cell and recordings shown in *A*. *C*: intracellular recordings showing the responses of a 2nd MG2 cell before, during, and after pairing with an electrosensory stimulus at the time of the EOD. The EOCD or the EOCD plus a sensory stimulus did not evoke broad spikes in this cell. Note the increase in the number of EOCD-evoked spikes after pairing, the increase was significant ($P < 0.001$). *D*: raster display of EOCD responses of small spikes before, during, and after pairing with an electrosensory stimulus. Derived from the same cell and recordings as shown in *C*.

lower half of this layer. Some of the finer dendritic branches are recurrent and descend back into the ganglionic and plexiform layers. The extent of the apical dendrite varied between 140 and 400 μm in the mediolateral direction and 150 and 500 μm in the rostrocaudal direction in the cells that we examined morphologically.

The axons of TSD cells descend from the soma to branch in the granular layers. The axonal arbors extend ~500 μm in the transverse direction and 400 μm in the rostrocaudal direction. Axonal swellings, which could be synaptic terminals, are present in both the deep and superficial granular layers and in the intermediate layer. Although the axonal arbor covers a large area, the branching is sparse and the number of swellings is <50. The extent of the axonal arbor of TSD cells found in our study was larger than that found in a previous in vitro study of ELL cells (Han et al. 1999) due most probably to the severing of axonal branches during preparation of the in vitro slices.

We supplemented our physiological findings from the seven morphologically identified TSD cells with recordings from an additional six cells that had the same physiological properties. The EOCD elicited a large, stereotyped EPSP (4–12 mV) in TSD cells that evoked a brief burst of one to six small spikes (2–9.5 mV; 6 ± 2 mV, $n = 13$; Fig. 7, *B* and *D*). The spike responses, especially the first spike, showed minimal temporal variability in relation to the time of the command signal. The latency of the EOCD EPSP following the command was very short, (2.5–4.2 ms in different cells; 3.5 ± 0.9 ms, $n = 13$), shorter than that in any other cell. EPSP durations were between 30 and 62 ms in TSD cells. The EOCD-evoked excitation, in form of synaptic responses and spikes, was the only activity that we observed in these cells in the absence of electrosensory stimuli.

Electrosensory stimuli given independently of the command evoked an EPSP-IPSP (Fig. 7*D*) in 9 of the 10 cells tested, of which 6 were morphologically identified. Only one cell responded with a pure IPSP (Fig. 7*B*). Minimal latencies (obtained at near maximal stimulus intensity) of the sensory responses were short, between 1.9 and 2.9 ms (2.4 ± 0.3 ms). Even high-stimulus amplitudes never evoked a spike in response to the sensory stimulus. Receptive fields were small, consisting of one to three electroreceptors. Stimuli outside this cluster of receptors did not elicit any responses, and thus there was no indication of an opponent surround to the receptive field.

Somewhat surprisingly, electrosensory stimuli given at the EOD delay caused a consistent and strong inhibition of the EOCD-evoked EPSP and spike burst (Fig. 7, *B* and *D, bottom*) even though stimuli given independently of the command usually evoked EPSP-IPSP sequences. We determined the net response of a sensory stimulus at the time of the EOCD by subtracting the response to the EOCD alone from the response to the EOCD plus a sensory stimulus. The net response of eight cells was a pure IPSP. Five cells showed an EPSP-IPSP sequence as a net response, but only the IPSP component in these cells was enhanced in comparison to the response evoked by an independent sensory stimulus. Thus the interaction between the EOCD and electrosensory inputs was markedly nonlinear for TSD cells. The electrosensory stimulus blocked later spikes of the EOCD-evoked burst but did not block the first spike, even at the strongest stimulus intensities.

EOCD excitation of TSD cells appeared to be plastic in the two cells tested. The EOCD EPSP was larger and evoked more spikes after 2 min of pairing with a sensory stimulus (Fig. 8).

A previous extracellular study of ELL described cells with a short-latency, stereotyped burst response to the EOCD that was inhibited by a well-localized sensory stimulus (Bell and Grant 1992). Such cells were referred to in the previous study as I_1 cells. The close similarities with TSD cells indicate that the previously described I_1 cells were almost certainly TSD cells.

Medium fusiform cells

Medium fusiform cells have been previously described morphologically, and an in vitro study have shown that they respond to parallel fiber stimulation with an EPSP and also to stimulation of the deep layers of ELL with an EPSP. These cells were previously referred to as "small fusiform cells" (Han et al. 1999; Meek 1993) but are now referred to as "medium

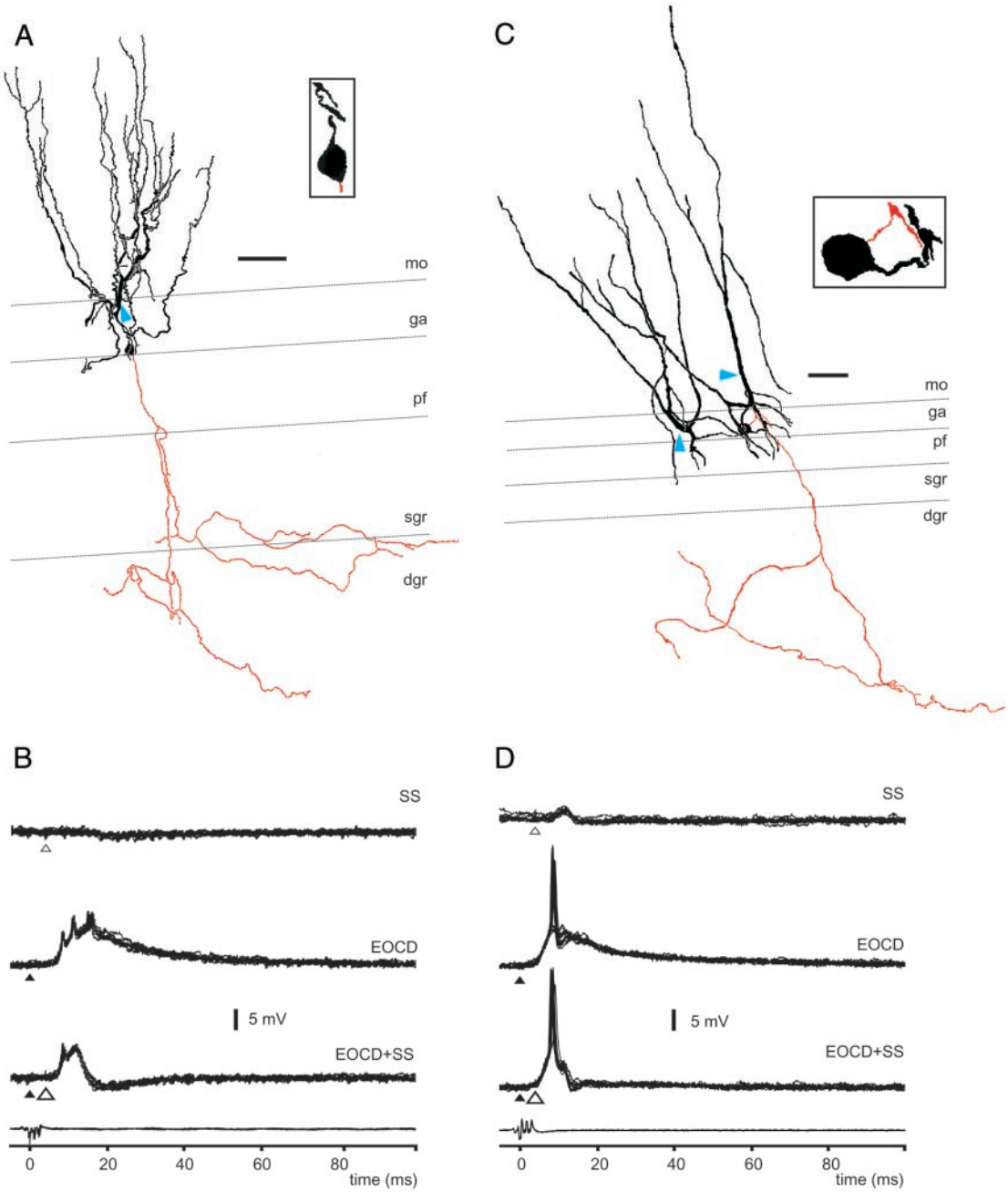

FIG. 7. Morphology and physiology of thick smooth dendrite (TSD) cells. *A*: TSD cell with 1 thick dendrite (black) and 1 axon (red). One thin dendrite leaves the soma (*inset*) and gives rise to a thick dendrite (blue arrow head) with both ascending and recurrent branches (note: 1 running across the cell body). The red line in the inset shows the start of the axon. dgr, deep granular layer. Bar: 50 μm. *B*: physiology of cell shown in *A*. An electrosensory stimulus alone elicits a small IPSP (SS), and the EOCD alone evokes a sharply rising EPSP with 3 small spikes (EOCD). Delivery of the same sensory stimulus at the time of the EOD blocks the 2nd and 3rd spike and evokes an IPSP (EOCD + SS) that is larger than that evoked by the sensory stimulus alone. *C*: TSD cell with 2 distinct regions of thick dendrites (black, blue arrow heads) and 1 axon (red). One thin dendrite leaves the soma (see *inset*) and gives rise to the 2 regions of thick dendrites with both ascending and recurrent dendrites (note: 2 running across the cell body). The red line in the *inset* shows the start of the axon. Bar: 50 μm. *D*: physiology of cell shown in *C*. An electrosensory stimulus alone elicits a brief EPSP followed by a small IPSP (SS), and the EOCD alone evokes a sharply rising EPSP with a single spike. Delivery of the same electrosensory stimulus at the EOD delay reduces the later portion of the EOCD EPSP (EOCD + SS).

fusiform cells" because a different and still smaller fusiform cell has been identified morphologically in ELL (J. Meek, personal communication). Four medium fusiform cells were morphologically identified in this study after intracellular recording.

Medium fusiform cells are GABAergic interneurons with cell bodies in the granular layer and a thick apical dendrite that extends up into the deep molecular layer where most of the dendritic tree is located (Fig. 9*A*) (Han et al. 1999; Meek 1993). The apical dendrite gives off some branches to the granular, plexiform and ganglionic layer as it ascends, and some thin basilar dendrites arise from the cell body in the

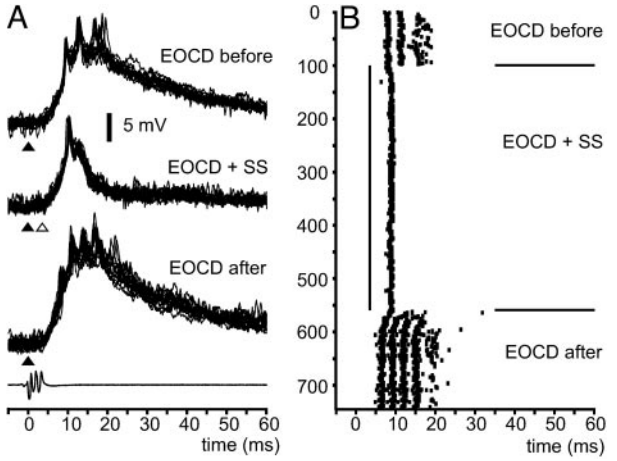

FIG. 8. Plasticity of the EOCD responses of a thick smooth dendrite cell. *A*: intracellular recordings of EOCD responses of TSD cell before (EOCD before), during pairing with an electrosensory stimulus (EOCD + SS), and after two minutes of pairing (EOCD after). Note the smaller number of spikes during pairing and the significantly larger EPSP ($P < 0.001$) after pairing. *B*: raster display of EOCD spike responses before, during, and after pairing with an electrosensory stimulus. Derived from the same cell and recordings as shown in *A*. Note the longer latency of the 1st spike and inhibition of later spikes during pairing. Note also the significantly larger number of spikes after pairing ($P < 0.001$).

granular layer. The apical dendritic arbor extends further in the rostrocaudal direction (150–300 μm) than in the transverse (80–140 μm). As with the TSD cells, our in vivo material yielded a more complete description of the axonal arbor of medium fusiform cells than was possible in previous morphological studies using the Golgi technique (Meek et al. 1996) or intracellular labeling in in vitro slices (Han et al. 1999). The axonal arbor of medium fusiform cells is similar to that of the TSD cell. The axon exits from the base of the soma and sends branches into the superficial granular, deep granular and intermediate layers. The arbor extends a large distance (250–650 μm) in both the transverse and rostrocaudal directions, but the branching is sparse, and the number of swellings or presumed terminals is small.

We supplemented our physiological findings from the four morphologically identified cells with recordings from an additional three cells that had the same physiological properties. The EOCD elicited a brief (20–37 ms) stereotyped EPSP at a latency of 4.6–6.4 (5.5 $\pm$ 0.7) ms after the command signal (Fig. 9*B; middle* in *C* and *D*). This EOCD response resembled the response recorded inside primary afferents that is presumed to be due to EOCD input to granular cells and that is observed inside the afferents because of the electrical synapses that the afferents make on granular cells (see following text) (Bell

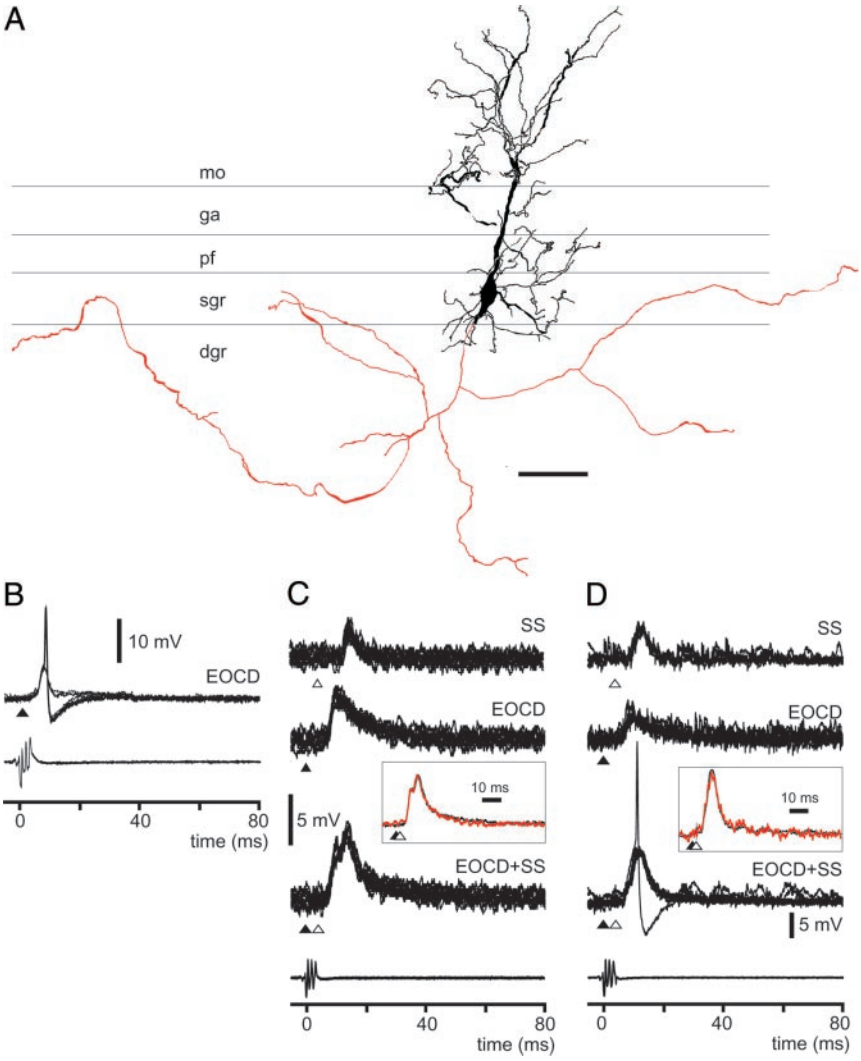

FIG. 9. Morphology and physiology of medium fusiform cells. *A*: morphology of a medium fusiform cell. Dendrites in black, axon in red. Bar: 50 μm. *B*: EOCD response of a medium fusiform cell, with a single spike rising from the EPSP. *C*: physiology of same cell shown in *A*. The cell gives a brief EPSP in response to both an electrosensory stimulus alone (SS) and the EOCD alone (EOCD). Delivery of the electrosensory stimulus at the time of the EOD evokes a response that is the simple sum of the 2 EPSPs (EOCD + SS). *Inset*: overlays of an average of the responses to the EOCD plus the electrosensory stimulus (black) and a sum of the independent responses to the EOCD and the sensory stimulus (red). The near perfect overlap shows that the 2 responses sum linearly. *D*: same as in *C* but for a different medium fusiform cell. Delivery of the electrosensory stimulus at the time of the EOD (EOCD + SS) sometimes evoked a spike in this cell

1990a; Bell et al. 1997b). The EPSP often gave rise to a single spike (Fig. 9*B*). The spike had amplitudes of 12.3–36 mV (22.7 ± 8.5 mV) and a prominent afterhyperpolarization of 4–10.2 ms (7.5 ± 2.2).

Electrosensory stimuli given independently of the command evoked a brief EPSP with a short minimal latency of 2–2.8 ms (2.4 ± 0.2 ms) in the medium fusiform cells (Fig. 9, *C* and *D, top*). For electrosensory stimulation, all cells with a spike in the EOCD response were hyperpolarized to reveal the underlying EPSP. When the electrosensory stimuli were given at the time of the EOD, the electrosensory EPSPs and EOCD EPSPs summed, and the summed excitation could elicit a spike (Fig. 9*D, bottom*). Increasing the stimulus amplitude never evoked more than one spike. The summation of the electrosensory and EOCD EPSPs was linear, in contrast to the nonlinear summation of these two signals in MG and TSD cells. The response to the two signals given together was the same as the sum of the two independent responses (Fig. 9, *C* and *D, inset*). No EOCD plasticity was observed in these cells. The EOCD response after pairing with an electrosensory stimulus was the same as the EOCD response before pairing.

Large fusiform cells

Large fusiform cells are one of the two types of efferent cells in ELL. These cells have been examined previously both in vivo (Bell et al. 1997b) and in vitro (Grant et al. 1998). The in vivo study showed that electrosensory stimuli delivered at the time of the EOD to the centers of the receptive fields of these cells are excitatory (E cells) and that the response to the EOCD is markedly plastic, being strongly affected by a period of pairing with an electrosensory stimulus. Our recordings from these cells confirm the previous findings and provide some additional information about these large fusiform cells.

The cell bodies of large fusiform cells are in the granular layer or at the border between the plexiform and granular layers (Grant et al. 1996) (Fig. 10*A*). Basilar dendrites are in the granular layers and apical dendrites extend throughout the molecular layer. Four large fusiform cells were intracellularly recorded and morphologically identified in this study. The axons could be followed all the way into the lateral lemniscus, confirming that they were efferent cells.

We supplemented the physiological findings from the four morphologically identified cells with recordings from seven additional cells that had similar physiological properties. Spikes ranged from 16 to 60 mV and were followed by an after-hyperpolarization. The EOCD evoked an IPSP in two of the morphologically identified cells and in four of the cells that were not identified (Fig. 10*B*). The onset of the IPSP varied between 6.9 and 17.3 ms (11.9 ± 3.6 ms). The IPSPs were sometimes preceded by a small EPSP that evoked a spike. The EOCD evoked EPSPs in two of the morphologically identified cells and three of the cells that were not identified (Fig. 10*C*). EPSP onsets were between 6.3 and 6.5 ms. A previous study found only EOCD-evoked IPSPs in these cells (Bell et al. 1997b). Large fusiform cells showed either EPSPs or IPSPs in response to the EOCD. None of the cells showed an EPSPs at one time and IPSPs at another. Thus although it is possible that the EOCD simultaneously evokes both inhibition and excitation in large fusiform cells as suggested for MG cells, with the relative strengths of these two inputs varying from cell to cell, we have as yet no evidence for such a possibility.

The EOCD alone could elicit EPSPs or IPSPs, but the response to electrosensory stimuli given at the time of the EOD was always excitatory. Electrosensory stimuli given independently of the command elicited EPSPs that could trigger spike trains at higher stimulus amplitudes. Minimal latencies of the EPSPs ranged from 3.7 to 4.4 ms (4 ± 0.3 ms). The excitatory response to a sensory stimulus was greatly facilitated when the stimulus was given at the EOD delay. Stimuli that were ineffective when given independently could elicit vigorous responses when given at the EOD delay (Fig. 10, *B* and *C*). Thus the interaction between EOCD and electrosensory inputs was markedly nonlinear in large fusiform cells. This nonlinear interaction is probably due to the local circuitry and the presence of an interneuron between the primary afferent input and the large fusiform cell, an interneuron that is excited by both primary afferent input and by the EOCD (see DISCUSSION).

EOCD responses were clearly plastic in large fusiform cells; both in cells with an EOCD-evoked EPSP and in cells with an EOCD-evoked IPSP. Pairing with an excitatory sensory stimulus for 2–3 min caused a reduction in the EOCD-evoked

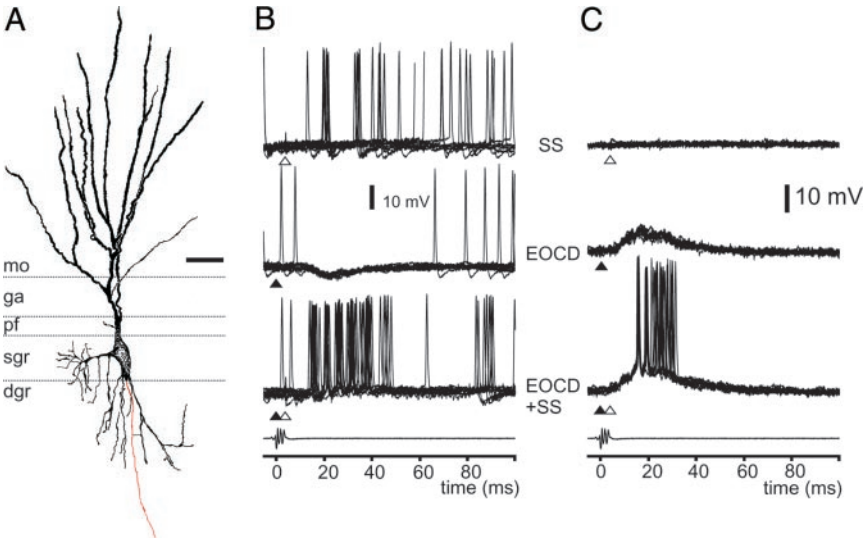

FIG. 10. Morphology and physiology of large fusiform cells. *A*: morphology of large fusiform cell. Dendrites in black, begin of axon in red. Bar: 50 μm. *B*: physiology of a large fusiform cell. A weak electrosensory stimulus alone evokes a slight acceleration in discharge rate (SS), and the EOCD alone evokes an IPSP and a pause in discharge rate (EOCD). Delivery of the same electrosensory stimulus at the time of the EOD evokes a strong burst of spike activity. *C*: physiology of same cell as shown in *A*. In this cell, the weak electrosensory stimulus alone does not evoke any response (SS), and the EOCD alone evokes an EPSP. Delivery of the same electrosensory stimulus at the EOD delay evokes a strong burst of spike activity.

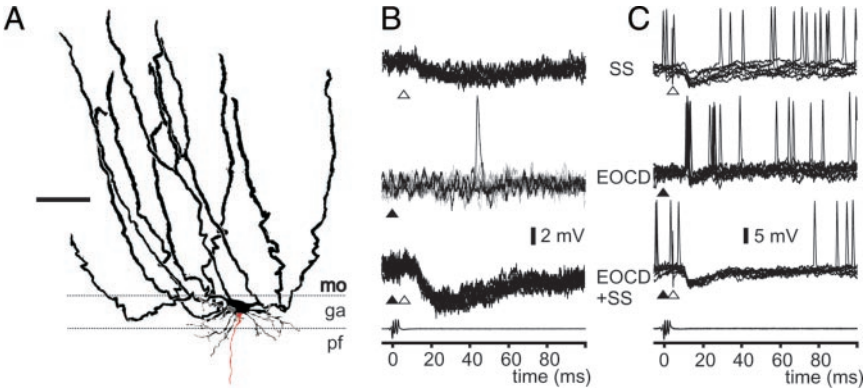

FIG. 11. Physiology and morphology of large ganglionic cell. *A*: morphology of large ganglion cell. Dendrites in black, begin of axon in red. Bar: 50 μm. *B*: physiology of a large ganglion cell. An electrosensory stimulus alone evokes a small IPSP (SS), but the EOCD alone has little effect. Delivery of the same electrosensory stimulus at the time of the EOD evokes a larger IPSP (EOCD + SS) than is evoked by the electrosensory stimulus alone. *C*: physiology of another large ganglionic cell. An electrosensory stimulus alone evokes a small, brief IPSP, and the EOCD alone evokes one or two spikes (EOCD). Delivery of the same electrosensory stimulus at the time of the EOD evokes a more prominent IPSP (EOCD + SS) than is evoked by the electrosensory stimulus alone.

EPSPs (in all 3 of the 3 cells tested; Fig. 12*A*) or an increase in the EOCD-evoked IPSPs (in 1 of the 2 cells tested; Fig. 12*B*).

Large ganglionic cells

Large ganglionic cells are the second type of efferent cell in ELL. These cells have also been examined previously both in vivo (Bell et al. 1997b) and in vitro (Grant et al. 1998). The in vivo study showed that electrosensory stimuli delivered at the time of the EOD to the centers of the receptive fields of these cells are inhibitory (I cells) and that the response to the EOCD is markedly plastic, being strongly affected by a period of pairing with an electrosensory stimulus. Our results concerning large ganglionic cells are described here for comparison with other cell types and as confirmation of previous findings. Four large ganglionic cells were intracellularly recorded and morphologically identified in this study.

The cell bodies of large ganglionic cells are in the ganglionic layer. Their basilar dendrites are in the plexiform layer and their apical dendrites extend into the molecular layer. (Grant et al. 1996) (Fig. 11*A*). The axons of large ganglionic cells, like those of large fusiform cells, could be followed into the lateral lemniscus, confirming that they were efferent cells.

We supplemented our physiological findings from the four morphologically identified cells with recordings from eight additional cells that had similar physiological properties. The EOCD evoked only minimal responses from large ganglionic cells, provided that no electrosensory stimulus had been paired with the EOCD during the preceding 4 or 5 min. A small EPSP was evoked in some cells at a latency of 7.8–11.6 ms, and this EPSP sometimes triggered spikes (Fig. 11*C*, *middle*). Spikes ranged in amplitude from 21 to 40 mV and were followed by an afterhyperpolarization.

Electrosensory stimuli delivered independently of the command evoked long-lasting IPSPs with minimal latencies between 2.4 and 3.6 ms (3.2 $\pm$ 0.5 ms). These IPSPs were markedly facilitated when delivered at the EOD delay (Fig. 11, *B* and *C*). Thus the interaction between these two signals was clearly nonlinear as in MG, TSD, and large fusiform cells.

EOCD responses were clearly plastic in large ganglionic cells. Pairing with an inhibitory sensory stimulus for 2–3 min resulted in a decrease in the IPSP amplitude during the pairing and an EOCD-evoked burst of spikes after the electrosensory stimulus was turned off (Fig. 12*C*). An increase in EOCD excitation was observed even after pairing periods as short as 10 s.

Interzonal cell

A previous anatomical study with tracer substances showed that the two mormyromast zones of ELL, the medial and dorsolateral zones, are mutually interconnected (Bell et al. 1981). Cells of the medial zone project to the dorsolateral zone and vice versa. But the cells of origin of these projections were not morphologically described, and their physiology was un-

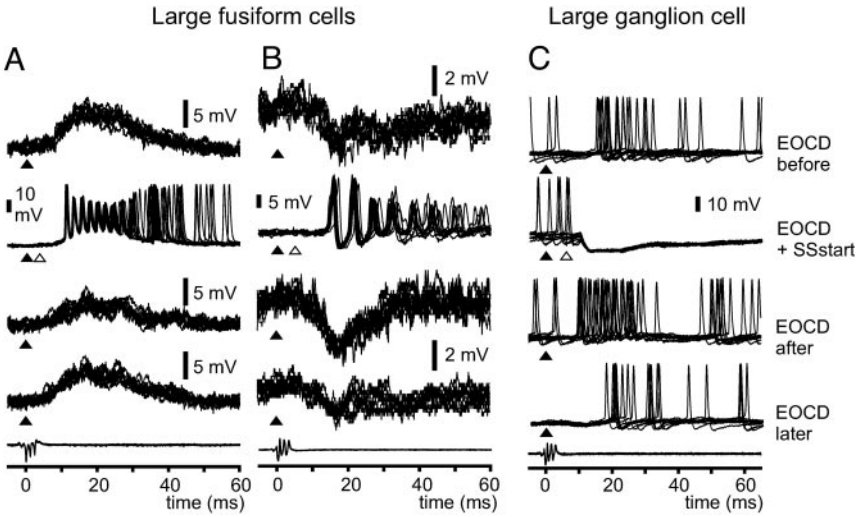

FIG. 12. Plasticity of the EOCD responses of efferent cells. For each of the 3 columns (*A*-*C*), the 1st set of traces show responses of a cell to the EOCD alone before pairing with an electrosensory stimulus (EOCD before), the 2nd set of traces show initial responses to the EOCD plus an electrosensory stimulus at the EOD delay (EOCD + SS start), the 3rd set of traces show responses to the EOCD alone immediately after 2 min of pairing with an electrosensory stimulus (EOCD after), the 4th and final set of traces show the responses to EOCD alone several minutes after the end of pairing (EOCD later). *A*: responses of a large fusiform cell with an EPSP as EOCD response. Note the smaller EOCD-evoked EPSP after pairing with a sensory stimulus that evokes a burst of spikes. *B*: responses of a large fusiform cell with an IPSP as EOCD response. Note the larger EOCD-evoked IPSP after pairing with a sensory stimulus that evokes a burst of spikes. *C*: responses of a large ganglionic cell. Note the more prominent EOCD-evoked burst after pairing with a sensory stimulus that evokes a large IPSP.

known. In this study, we recorded and stained a cell in the medial zone that projected to the dorsolateral zone.

The cell body was in the deep plexiform layer (Fig. 13*A*). A single apical dendrite ascended into the molecular layer, and three basal dendrites descended into the superficial granular layer. The apical dendrite thickened as it ascended and branched repeatedly to form an arbor in the inner half of the molecular layer with some recurrent branches being given off to descend back toward the ganglionic and plexiform layers. All of the dendrites were without spines. The axon of this interzonal cell descended into the deep granular layer where it branched, forming a rather sparse arbor that extended 285 μm in the transverse direction and 200 μm in the rostrocaudal direction. Individual branches ended in distinct clusters of presumed terminals in the deep granular layer. One axonal branch descended further into the deep fiber layer and continued 400 μm rostral and lateral to enter the dorsolateral zone of ELL where it branched to terminate in the superficial and deep granular layers of that zone (Fig. 13*A*, *inset*). The terminal arbor in the dorsolateral zone was more extensive than that in the medial zone. The axonal branch to the dorsolateral zone terminates 400 μm rostral to the location of the cell body in the medial zone. This termination region and the region of the medial zone in which the cell body is located correspond somatotopically; that is, both regions receive input from the same point on the skin surface(the rostral limit of the dorsolateral zone extends beyond the rostral limit of the medial zone).

The EOCD evoked a large EPSP with a latency of 7.7 ms (Fig. 13*B*, *middle*) in this interzonal cell. The EPSP had two peaks with a small spike occurring on top of the first peak. The second peak was followed by a slowly declining depolarization lasting ~70 ms. Electrosensory stimuli given independently of the command evoked a complex response consisting of an initial EPSP with a minimal latency of 2.7 ms, followed by an IPSP with multiple and variable peaks (Fig. 13*B*, *top*). Responses could be evoked from a cluster of five neighboring receptors using low-intensity stimuli. Delivery of the electrosensory stimuli at the EOD delay caused a decrease in the second peak of the EOCD EPSP. Pairing with the electrosensory stimulus for 2 min did not affect the EOCD response.

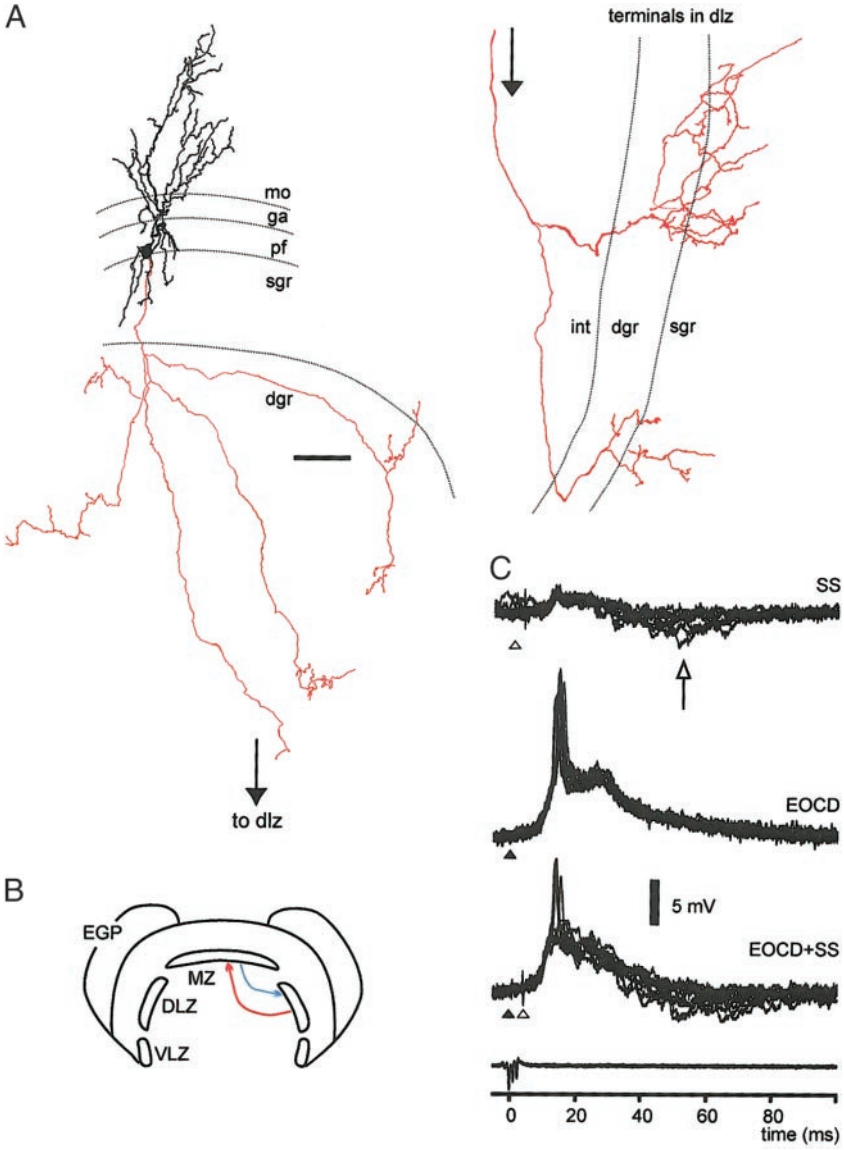

FIG. 13. Morphology and physiology of interzonal cell with a soma in the medial zone of ELL. *A*: morphology of interzonal cell in the medial zone (mz). The axon (in red) sends a branch to the dorsolateral zone (dlz). *Inset*: the termination pattern of this branch in the dorsolateral zone (dlz). Bar: 50 μm. *B*: diagram of ELL to show the interzonal connections between dlz and mz. EGp, eminentia granularis posterior; vlz, ventrolateral zone. *C*: physiology of cell shown in *A*. *Top*: the responses to the electrosensory stimulus alone (SS). The upward arrow points to the variable inhibitory part of the response. *Middle*: the responses to the EOCD alone. *Bottom*: the responses to the EOCD plus an electrosensory stimulus (EOCD+SS). The EPSP and the number of spikes evoked by the EOCD are reduced by the stimulus.

The morphology of the interzonal cell is similar to that of the TSD cell, but there are also important differences. The interzonal cell had several basal dendrites in addition to the one apical dendrite, but the TSD cells did not have any basal dendrites, and none of the TSD cells had axonal branches terminating in the other mormyromast zone. The physiology was also somewhat similar to TSD cells, both cells showing an EPSP to the command that was significantly reduced by an electrosensory stimulus given at the time of the EOD. But the EOCD EPSP of the medium fusiform cell was longer in latency (7.7 ms for the interzonal cell vs. 2.5–4.2 ms for the TSD cells) and electrosensory stimuli elicited a larger and more variable IPSP in the interzonal cell. Given at the time of the EOD, the electrosensory stimulus also affected the first spike of the EOCD response, which was unaffected in TSD cells. These anatomical and physiological differences indicate that the interzonal and TSD cells are distinct cell types.

Large TSD cell

We recorded one example of a new cell type that had not been previously described, either morphologically or physiologically. The cell body (14 × 24 μm) was located in the ganglionic layer (Fig. 14*A*). Three thin apical dendrites extended dorsally from the cell body into the molecular layer where they branched and became considerably thicker. The thick dendrites extended throughout the molecular layer in contrast to the dendrites of TSD cells that were restricted to the inner molecular layer. The dendrites did not have spines. As the thick dendrites approached the outer margin of the molecular layer, they gave off thin branches that penetrated into the preeminential tract, a tract that separates ELL from EGp and conveys fibers from nucleus preeminentialis to ELL and EGp. Some thin dendritic branches were recurrent and extended back into the ganglionic and plexiform layers. The axon of the large TSD cell descended from the cell body down into the deep granular layer where it branched extensively (480 μm in mediolateral and 650 μm in rostrocaudal direction), sending branches into the intermediate, deep granular, superficial granular, and plexiform layers (Fig. 14*A*).

The EOCD evoked an EPSP in this cell with a latency of 8.8 ms. The EPSP in turn triggered a burst of 2–3 spikes (Fig. 14*C*, *top*). The amplitude of the spikes was between 37 and 38 mV. The individual spikes were followed by large afterhyperpolarizations. Electrosensory stimuli given independently of the command evoked an EPSP with a minimum latency of 6.5 ms (Fig. 14*B*, *top*). Increasing stimulus intensity evoked a single spike (Fig. 14*B*, *bottom*). Further increases in intensity reduced the latency of the spike but did not yield more spikes. When given at the time of the EOD, the sensory stimulus decreased the number of spikes from 2–3 to 1–2, but the spikes were evoked at a shorter latency and were more time-locked to the command signal (Fig. 14, *C*, CD+SS, and *D*). Pairing the EOCD with an electrosensory stimulus for 1 min resulted in a response to the EOCD alone after pairing that had a shorter latency and a larger number of spikes than the EOCD response before pairing (Fig. 14, *C* and *D*), suggesting the presence of EOCD plasticity.

The physiology of the large TSD cell is somewhat similar to that of the ordinary TSD cell, but there are also important differences. The EOCD EPSP is longer in latency and smaller in amplitude, and the spikes are larger in the large TSD cell than in the ordinary TSD cell, and electrosensory stimuli alone could evoke spikes in the large TSD cell. These physiological differences together with the morphological differences indicate that the large TSD cell is a distinct cell type.

Primary afferents

Previous studies have shown that synaptic potentials are present in intracellular recordings from mormyromast afferent fibers terminating in ELL (Bell 1990a). The synaptic potentials are evoked by the EOCD and by stimulation of electroreceptors close to the electroreceptor that gives rise to the recorded afferent fiber (as indicated by an incoming spike). The synaptic potentials represent synaptic input to ELL granular cells that is recorded inside the afferent fiber via the electrical synapses between afferents and granular cells (Bell et al. 1989). Our intracellular recordings from primary afferent fibers are described briefly here for pur-

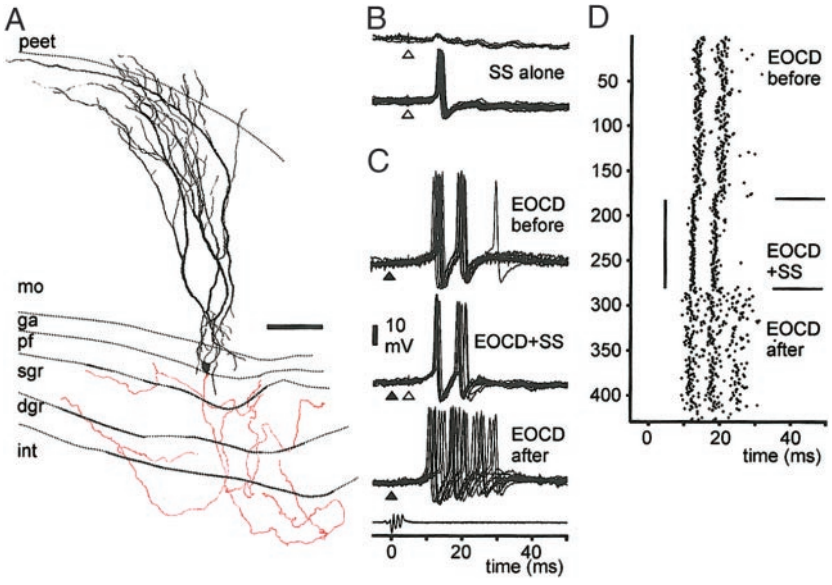

FIG. 14. Morphology and physiology of large thick smooth dendrite cell. *A*: morphology of large thick smooth dendrite cell. Dendrites in black, axon in red. int, intermediate layer Bar: 100 μm. *B*: responses to electrosensory stimuli. A weak electrosensory stimulus evokes a small EPSP (*top*) and a stronger electrosensory stimulus evokes a spike (*bottom*). *C*: plasticity of EOCD responses. *Top*: responses to the EOCD alone before pairing with an electrosensory stimulus (EOCD before). *Middle*: responses to the EOCD plus an electrosensory stimulus (EOCD + SS) during pairing. *Bottom*: responses to the EOCD alone immediately after 40 s of pairing with an electrosensory stimulus (EOCD after). Note the increased number of spikes in the response after pairing. *D*: raster display of EOCD spike responses before, during, and after pairing with an electrosensory stimulus. Derived from the same cell and recordings as shown in *C*.

poses of comparison with EOCD and electrosensory responses in other ELL cells.

We recorded from seven morphologically identified primary mormyromast afferent fibers. An additional 38 recordings were determined to be afferent fibers on the basis of the characteristic spike responses to electrosensory stimuli and the characteristic synaptic responses to both the EOCD and electrosensory stimuli (Bell 1990a). The EOCD evoked brief EPSPs in these cells, which ranged in latency from 4.7 to 6 ms (5.4 ± 0.5). Minimal latencies for spikes from the periphery in response to electrosensory stimuli ranged from 1.1 to 3.4 ms (2.4 ± 0.5 ms, Fig. 15, trace labeled aff).

DISCUSSION

This study extends our previous knowledge of the functional circuitry of the mormyrid ELL (Grant et al. 1998; Han et al. 1999; Meek et al. 1999) by describing the physiological responses of different cell types. Some of these cell types—the two types of MG cells, the TSD cells and the medium fusiform cells—had already been described morphologically, but their responses to sensory and motor signals had not been determined (Bell and Grant 1992; Bell et al. 1997b). Other cell types—the interzonal cell and the large TSD cell—are described here for the first time, both morphologically and physiologically. The following paragraphs first describe the properties of some of these different cell types and then describe some general features of ELL circuitry and the interaction between sensory and motor signals in ELL.

MG cells

The GABAergic MG cells are probably of central importance in the functioning of ELL. Both MG cells and efferent cells have basilar dendrites that receive electrosensory input via ELL granular cells and apical dendrites that extend throughout the molecular layer and receive parallel fiber input. Thus both types of cells integrate these two major inputs to ELL. MG cells, however, are four times as numerous as efferent cells, have twice as many apical dendrites, and have twice as many spines per unit length of apical dendrite (Meek et al. 1996). Parallel fiber synapses on MG cells are therefore ~16 times more numerous than parallel fiber synapses on efferent cells. The somas of efferent cells are densely covered with inhibitory terminals from MG cell axons (Grant et al. 1996), and the output of ELL, as conveyed by efferent cells, is therefore strongly modulated by MG cell activity.

Perhaps the most important finding of the present study with regard to MG cells is that MG1 cells are inhibited by electrosensory stimuli, whereas MG2 cells are excited as previously hypothesized on morphological grounds (Han et al. 1999; Meek et al. 1996). Thus there are E- and I-MG cells, just as there are E- and I-efferent cells (large fusiform and large ganglionic cells, respectively). As originally suggested by Angel Caputi (personal communication) and as further elaborated by Han et al. (1999), the most likely hypothesis is that the I-MG cells (MG1) inhibit the E-efferent cells (large fusiform) and that the E-MG cells (MG2) inhibit the I-efferent cells (large ganglionic). Such connectivity makes functional sense in that inhibition of the efferent cell by the opposite type of MG cell would reinforce or enhance the sensory response of the efferent cell. In contrast, inhibition of an E-or I-efferent cell by an MG cell of the same type would minimize or cancel the sensory response of the efferent cell. Anatomical findings also suggest that MG1 cells end preferentially on large fusiform cells and that MG2 cells end preferentially on large ganglionic cells (Han et al. 1999).

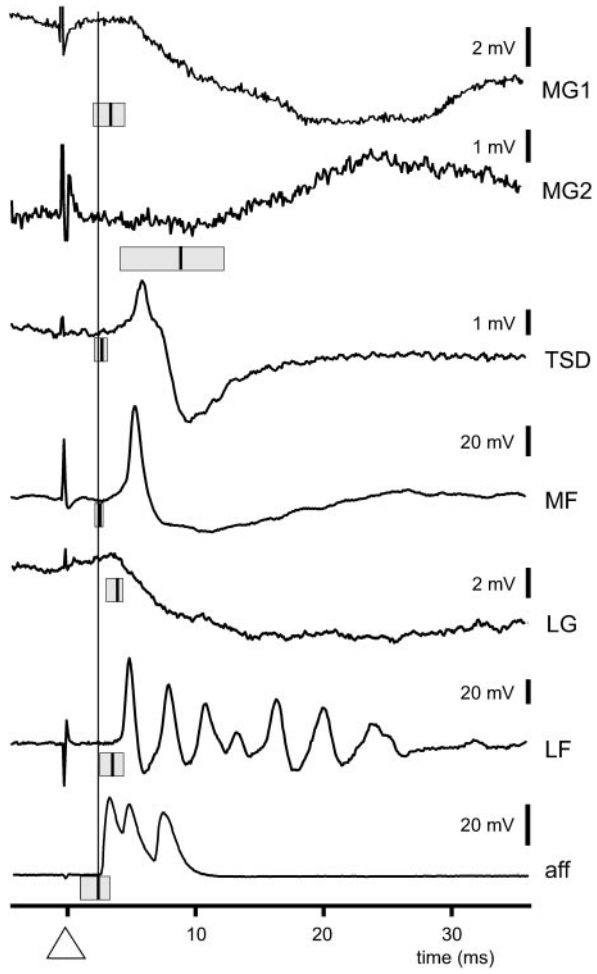

FIG. 15. Relative timing of electrosensory responses in major cell types. Representative examples of responses of different ELL cells to electrosensory stimuli. Stimuli were given independently of the EOD motor command at intensities that yielded minimal latencies for synaptic responses of the cells or for the onsets of the 1st spike in the mormyromast afferent fiber. The gray bar beneath each trace shows the range of minimal latencies for that cell type, and the black line within each bar indicates the mean minimal latency for that cell type. The thin vertical line indicates the mean minimal latency for mormyromast afferent fibers. MG1, medium ganglionic cell 1; MG2, medium ganglionic cell 2; TSD, thick smooth dendrite cell; MF, medium fusiform cell; LG, large ganglionic cell; LF, large fusiform cell; aff, primary mormyromast afferent fiber.

Our findings demonstrate similarities as well as differences between MG1 and MG2 cells. Both cell types usually respond to the EOD motor command with an EPSP that elicits a burst of small spikes, the first spike being more tightly time-locked than later spikes. In the normal life of the fish, when the motor command is followed by the fish's EOD, the EOCD driven

bursts of spikes would provide a background level of excitation that allows for both increases and decreases in activity in response to changes in afferent input from electroreceptors. The importance of such background excitation is clearest for MG1 cells. MG1 cells are inhibited by electrosensory afferents and there must be a background level of activity for the inhibition to act on and be revealed.

Both cell types also show the apparently paradoxical phenomenon of an EOCD-evoked inhibition of broad spikes occurring at about the same time as the EOCD-evoked EPSP and burst of small spikes. This inhibition or elevation of broad spike threshold may be necessary for maintaining the EOCD-evoked EPSP and burst of axon spikes. Associative synaptic depression that depends on the timing of the broad spike has been demonstrated at the parallel fiber to MG cell synapse (Bell et al. 1997c). The associative depression opposes and minimizes *all* depolarizations that evoke broad spikes and are consistently time locked to the EOD motor command. Elevation of the broad spike threshold during the EOCD-evoked EPSP blocks the broad spike and the associative depression that depends on it, allowing the EPSP to be maintained. Modeling studies of such a role for an elevation of broad spike threshold have been presented elsewhere (Roberts and Bell 2003). These modeling studies showed that an elevation of the broad spike threshold is a necessary condition for the maintenance of an EOCD-evoked EPSP in the presence of the type of synaptic plasticity described by Bell et al. (1997c). Synaptic plasticity that depends on the relative timing of presynaptic input and dendritic spikes is present in other systems also (Knudsen and Feldman 1998; Markram et al. 1997), and precisely timed control over the threshold of dendritic spikes provides a general mechanism for modulating such plasticity.

Plasticity of EOCD responses

EOCD plasticity was most marked and most consistent in ELL efferent cells. Pairing with an inhibitory sensory stimulus induced an excitatory EOCD response in large ganglionic cells, and pairing with an excitatory sensory stimulus resulted in a decrease in EOCD excitation or an increase in EOCD inhibition in large fusiform cells. Thus the effect of the pairing was to bring about an EOCD response that was opposite to the effect of the paired stimulus. As in the ampullary region of ELL (Bell 1982) and in the cerebellum-like structures of other fish (Bastian 1995; Bodznick and Montgomery 1993), the newly developed EOCD responses of efferent cells in the mormyromast regions of ELL may be described as negative images of the sensory responses that are predicted to follow the EOD motor command. As in the other systems, addition of the negative image minimizes predictable sensory responses, allowing small unpredictable features to stand out more clearly.

EOCD plasticity in ELL is probably due to synaptic plasticity at the synapses between EOCD conveying parallel fibers and molecular layer dendrites of MG cells and efferent cells (Bell et al. 1997a; Han et al. 2000). Such plasticity has been demonstrated in vitro for both MG cells (Bell et al. 1997c; Han et al. 2000) and efferent cells (V. Z. Han and C. C. Bell, unpublished observations), although the plasticity at synapses on MG cells appeared to be more prominent and consistent. This difference between the two cell types, the fact that parallel fiber synapses are 16 times more frequent on MG cells than on efferent cells, and the strong synaptic connections between MG cells and efferent cells (Grant et al. 1996; Meek et al. 1996) have led us to hypothesize that most of the EOCD plasticity observed in efferent cells is a consequence of synaptic plasticity and changes in the responsiveness of MG cells (Bell et al. 1997b). The EOCD plasticity we observed in MG1 cells is consistent with this hypothesis. The EOCD-evoked EPSP in MG1 cells and the number of spikes elicited by the EPSP increased after pairing with inhibitory sensory stimuli, a change that could be described as the formation of an EOCD-driven negative image, as in the efferent cells. The increase in EOCD excitation of MG1 cells could also explain the pairing-induced increase in EOCD inhibition and decrease in EOCD excitation of large fusiform cells under the hypothesis that MG1 cells selectively inhibit large fusiform cells (see Fig. 16).

The plasticity of MG2 cells that we observed was less marked than that in MG1 cells and not as easily interpreted. The slight decrease in the EOCD-evoked EPSP of MG2 cells after pairing with an excitatory stimulus is consistent with the overall hypothesis of negative image formation. The number of spikes evoked by the EPSP increased slightly after pairing in our recordings, however, and such an increase is not consistent with negative image formation. An increase in the number of spikes evoked by the EOCD in MG2 cells is also not consistent with the increased EOCD excitation of large ganglionic cells after pairing with an inhibitory sensory stimulus under the hypothesis that MG2 cells selectively inhibit large ganglionic cells. It is possible that the MG2 cells, which we recorded, were damaged in some way even though the membrane potential remained constant. The increased number of spikes in the presence of a reduced EPSP would then be a recording artifact, occurring in recorded cells but not in all the unrecorded MG2 cells of the same local circuit. Alternatively, our hypothesis that removing MG2 cell inhibition is an important source for the increased EOCD-driven activation of large ganglionic cells following pairing with a sensory stimulus may need to be revised.

Circuitry of ELL

TSD AND MEDIUM FUSIFORM CELLS. The TSD cells and medium fusiform cells are morphologically similar in that most of the dendritic surface area for both cell types is in the deep molecular layer and both cell types have sparse axonal arbors of similar extent that terminate in the deep and superficial granular layers. Granular cells are by far the most numerous cell type in the granular layers and are presumed to be the major postsynaptic target of both TSD and medium fusiform cells. In vitro studies show that both TSD and medium fusiform cells are excited by parallel fiber input (Grant et al. 1998), and TSD cells are excited by input to the preeminential nucleus to the deep molecular layer (Mohr et al. 2003). Thus both cell types appear to allow for the modulation of granular cell activity by descending input to the deep molecular layer of ELL.

The medium fusiform cells are GABAergic and presumed to be inhibitory, whereas the TSD cells are nonGABAergic and presumed to be excitatory (Meek et al. 1996). The two cells types may nevertheless act in concert. In medium fusiform cells, the EOCD and electrosensory stimuli both cause EPSPs

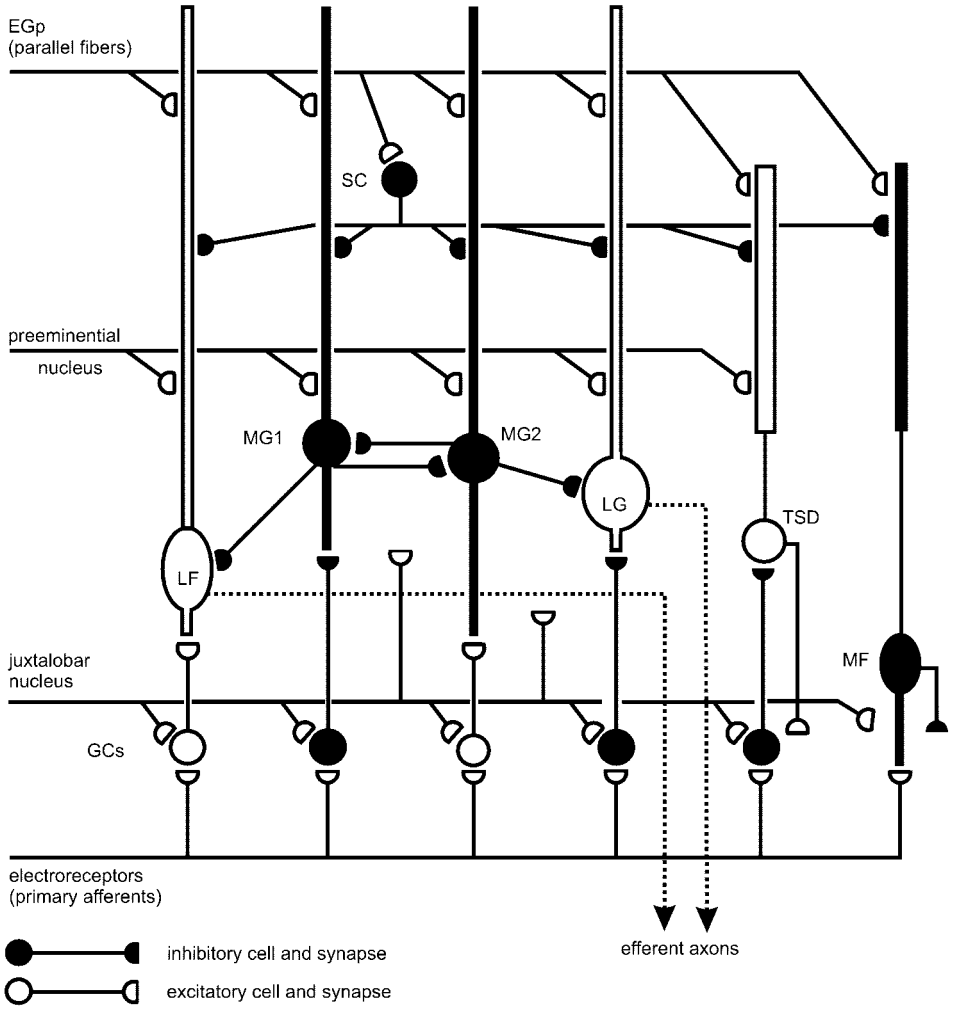

FIG. 16. EOCD circuitry. EGp, eminentia granularis posterior; GC, granular cell; SC, stellate cell. Revised from Han et al. 1999.

and the two EPSPs sum when the stimuli are given at the EOD delay. If the medium fusiform cells were inhibitory, then a local increase in the transcutaneous current evoked by the EOD (due to the presence of a conductive object) would increase the inhibition caused by medium fusiform cells. The EOCD-evoked EPSP in TSD cells elicits a brief burst of spikes that is reduced in number by sensory stimuli given at the EOD delay. If the TSD cells were excitatory, then a local increase in the EOD would reduce excitation by TSD cells. Medium fusiform and TSD cells would thus act in concert on their cellular targets in the granular layer, increasing inhibition and decreasing excitation respectively, following increases in local EOD amplitude (Fig. 16). Effects of the two cell types would also be in concert following decreases in local EOD amplitude. However, only the TSD cell showed plasticity of the EOCD response after pairing with a sensory stimulus.

NONLINEAR INTERACTIONS AND ELL CIRCUITRY. Interactions between the EOCD and electrosensory stimuli were markedly nonlinear in MG cells, TSD cells, large fusiform cells, and large ganglionic cells. In MG2 cells and large fusiform cells, electrosensory stimuli given at long delays after the EOD motor command could have small excitatory or even inhibitory synaptic effects (Bell et al. 1997b), whereas the same stimuli given at the EOD delay could result in a strong additional excitation. In MG1 cells, TSD cells, and large ganglionic cells electrosensory stimuli given at long delays could cause a small inhibition or have no effect, whereas the same stimuli given at the EOD delay could evoke a large inhibition.

The nonlinear interactions suggest that most of the electrosensory input to these cells is transmitted from the periphery through ELL interneurons, interneurons that are themselves excited by both the EOCD and by electrosensory input. Some nonlinear interactions could perhaps be mediated by intrinsic voltage responses of the cells or by *N*-methyl-D-aspartate receptors (Berman et al. 2001), but such mechanisms cannot explain the conversion of an inhibitory response into an excitatory response (Fig. 4*D*) or the enhancement of an inhibitory response (Fig. 11*B*) by giving a stimulus at a short delay after the EOD command. In addition, inhibitory electrosensory responses of ELL cells require an interneuron because primary afferent fibers are excitatory (Bell et al. 1989). ELL granular cells probably mediate the electrosensory responses of MG cells, TSD cells and efferent cells and are probably responsible for the nonlinear interactions between EOCD and electrosensory inputs in the higher-order cells. Granular cells are excited by both EOCD and primary afferent input (Bell 1990a). If granular cells mediate the electrosensory responses of higher-order ELL cells, then some of their effects must be excitatory and others inhibitory because both effects are observed (Fig.

16), but it is not yet known if these distinct effects are mediated by separate classes of granular cells. Medium fusiform cells, in contrast to the other cell types, probably receive primary afferent input directly because they are located in the granular layers and the synaptic responses to the EOCD and electrosensory stimuli sum linearly.

LATENCIES OF ELECTROSENSORY RESPONSES AND ELL CIRCUITRY. The minimal latencies of electrosensory responses that we observed in primary afferents and in ELL cells are consistent with the presence of ELL interneurons between primary afferent input and most higher-order ELL cells. Minimal latencies of primary afferent responses to electrosensory stimuli ranged from 1.1 to 3.4 ms (mean: 2.4 ms) in our recordings from ELL (Fig. 15). The ranges and means of minimal latencies that we recorded for MG1 cells (2.8–5.1 ms, mean: 3.9 ms), MG2 cells (4.9–12.9 ms, mean: 9.6 ms), large ganglionic cells (2.4–3.6 ms, mean: 3.2 ms), and large fusiform cells (3.3–4 ms, mean: 3.7 ms) were consistently longer than the range and minimal latencies of primary afferents.

In contrast, the minimal latencies for EPSPs in medium fusiform cells (2–2.8 ms, mean: 2.4) and TSD cells (1.9–2.9 ms, mean: 2.5) were similar to those of primary afferents, suggesting a direct effect of the afferents on these cells. For medium fusiform cells, this suggestion of a direct effect is consistent with the linear interaction between EOCD and electrosensory responses of these responses and with the location of their somas and basilar dendrites in the granular layers where primary afferent fibers end. In contrast, the somas and dendrites of TSD cells are largely external to the granular layers. Some of the descending dendrites of TSD cells may reach the external surface of the superficial granular layer (Fig. 7*C*), however, where they could be contacted by primary afferent fibers, accounting for the small, short-latency electrosensory EPSP observed in TSD cells. The IPSP evoked by electrosensory stimuli in TSD cells showed strong nonlinear interaction with the EOCD, but the EPSP did not, a result that is consistent with the IPSP, but not the EPSP, being mediated by an interneuron.

Inferences about ELL circuitry can be derived by comparing not only the latencies of electrosensory responses in different ELL cells but also by comparing the latencies of EOCD responses. The implications of the latencies of EOCD responses for ELL circuitry are discussed in the second paper of this series (Mohr et al. 2003) following the description of our results on the responses of ELL cells to central inputs that convey EOCD signals to ELL.

DISCLOSURES

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