tion that follows the relative refractory period of a single impulse. While significant differences in the magnitude of this variable were seen between cells, differences within cells were slight over time. Thus, for the 23 cells the maximal decrease in antidromic latency varied from 3 to 15 percent of the control antidromic latency, but the total range of variations over time within single cells rarely exceeded 2 percent of the control antidromic latency. However, three of the eight cells that showed progressive increases in antidromic latency also showed a progressive increase in the magnitude of supernormal conduction. Figure 3C shows this measure over days for the three cells represented in Fig. 3, A and B. Note the stability of this measure in cells 1 and 2 and the progressive increase of this measure in cell 3, which also showed a progressive increase in antidromic latency and minimum interspike interval.

These experiments leave questions regarding the underlying mechanism of the slow and progressive increases and decreases in conduction velocity and other conduction properties observed in some axons. Changes in conduction velocity could result from a number of factors (for example, changes in axonal diameter or ion channel density). Such changes may occur naturally, or they may result from subtle pathological changes elicited by the recording (*12*) or stimulating electrode. It seems unlikely, however, that increases in conduction velocity would result from pathological conditions.

I have shown that the physiological properties of individual cerebral axons may be monitored over a period of several months. Three measures of impulse conduction properties (conduction velocity, supernormal conduction velocity, and the minimum interspike interval) were found to be very stable over time or to vary in a progressive and systematic fashion. Systematic changes generally occurred at a rate of less than 1 percent per day and were often not detectable for several days or even weeks. Thus, these three measures provide an unequivocal signature with which to identify a neuron if recordings are obtained at relatively short intervals (2 to 3 days). The stability of the above conduction properties and the slow, systematic nature of the changes that do occur suggest the feasibility of studying the long-term effects of pharmacological, toxicological, or other variables on the conduction properties of individual cerebral axons.

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6. Microelectrodes were usually constructed of platinum-iridium wire (diameter, 25 μm) insulated with Teflon and cut flush. These electrodes were rigidly implanted, with no attempt made to "float" the electrode with respect to the cortical surface. In early experiments, such wires were electrolytically etched and the tips were insulated with a formfitting glass pipette [M. Salkman and M. J. Bak *IEEE Trans. Biomed. Eng.* **20**, 253 (1973)]. However, these electrodes were very difficult to produce and implant and were no more successful in gaining long-term recording stability than the electrodes described above. In each rabbit 5 to 16 microelectrodes were implanted singly or in groups of two to four, each electrode being separated by 0.3 to 0.5 mm. For each electrode (or group of electrodes) a small hole (approximately 0.5 mm in diameter or smaller) was teased into the dura, and the microelectrode was thrust through the pia into the cortex. Stimulation pulses were successively passed through the stimulating electrodes in the corpus callosum as the microelectrode was lowered. When antidromic spikes were elicited, the microelectrode was cemented into place with acrylic cement. All surgery was performed under barbiturate anesthesia.
7. Methods for implanting stimulating electrodes in the splenium of the corpus callosum have been described (*4*). The timing of stimulus pulses was controlled by four-channel digital circuitry (Digitimer).
8. Rabbits were awake and unrestrained during the recording sessions, which were usually held at intervals of 1 to 3 days. Differential signals (between neighboring microelectrodes) were preamplified and led to a digital oscilloscope for display, and the data were stored in digital form.
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12. While most of the brains were processed for the identification of Nissl substance, in one rabbit, after 244 days of recording, a 20 percent solution of horseradish peroxidase (HRP) was infused into the contralateral hemisphere, homotopic to the recording microelectrode. As seen in the stained tissue, the microelectrodes were encapsulated in a glial sheath approximately 25 μm thick. Outside this glial sheath, however, the tissue appeared quite normal. Several HRP-labeled neurons were seen within 50 μm of the microelectrode tips and many were seen within 100 μm of the tips.
13. In addition to the measures reported, other measures of impulse conduction studied included antidromic threshold, a measure of the refractory period (arrowheads in Fig. 2C), and a measure of the period of subnormal impulse conduction which follows a burst of impulses.
14. Previous studies of the callosal system of the rabbit (*2*, *4*) and monkey [H. A. Swadlow, D. L. Rosene, S. G. Waxman, *Exp. Brain. Res.* **33**, 555 (1978)] showed that these decreases in antidromic latency are due to changes in axonal conduction velocity which occur along the length of the callosal axon. Further, these changes in conduction velocity are due to prior impulse activity in the axon under study rather than to ephaptic interactions between axons or other indirect effects of the electrical stimulation. The period of supernormal conduction velocity following a single impulse lasts 20 to 170 msec. In this experiment the interval between the conditioning and test stimuli was that which yielded the maximum supernormal conduction velocity.
15. In one cell it was demonstrated that the progressive increase in antidromic latency results from a decrease in conduction velocity distributed along the axon. The cell showed progressively increasing antidromic latency and could be antidromically activated by stimulating electrodes both at the midline and near the axon terminal. While a progressive increase in latency was seen at both stimulation sites, the magnitude of the increase in latency (in milliseconds) was significantly greater with stimulation of the axon near the terminal.

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Rapid and Precise Down Regulation of Fast Axonal Transport of Transmitter in an Identified Neuron

Abstract. *Within 1 day after the removal of one branch of the bifurcated axon of an identified neuron in* Aplysia, *the cell body reduced its output of transmitter storage vesicles to adjust precisely for the decreased need. This adjustment terminated the initial consequence of the removal, the transport of an inappropriately large number of vesicles to the remaining synapses. The most likely cause of the reduction of transport of transmitter is the loss of information normally provided by the disconnected axon or synapses.*

The axon and synapses of a neuron, which lack ribosomes and most nucleic acids, depend on fast axonal transport for the delivery of newly synthesized organelles from the cell body (*1*). To fully understand expressions of plasticity in axons and synapses, such as those occurring during development and regeneration, it will be important to determine how the cell body is guided to adjust its output of rapidly transported material to meet changing demands from its periphery. We are interested in understanding how the neuron regulates the transport of material destined for use at functioning synapses. Is the normal amount transported in a mature neuron influenced by informational feedback from the neuronal periphery? To address this question we studied the changes in the amount transported when a neuron suddenly had fewer synapses to supply.

For this study we used an identified serotonergic neuron, the giant cerebral neuron (GCN) in the central nervous system of the sea hare *Aplysia californica*. The axon of this monopolar neuron bifurcates within the neuropil of the ganglion, close to the cell body, into branches of similar diameter which course essentially unbranched in separate nerves to innervate separate post-

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Table 1. Effects of transection of CBC on fast transport and protein synthesis in giant cerebral neuron (GCN). A small slit was cut in the ventral body wall of the animal to expose the cerebral ganglion, which contains the symmetrical pair of GCN's. The CBC on one side of the ganglion was transected, the slit was sutured closed, and the animal was returned to a holding tank containing Instant Ocean at 15°C, where it recovered from the $MgCl_2$-induced anesthesia within several hours. The ganglion and nerves were subsequently removed from the animal so that assays could be performed. Transport of [^{3}H]serotonin was measured by microinjecting [^{3}H]serotonin into the cell bodies of the GCN whose CBC axonal branch was transected (E) and the GCN whose axonal tree was untouched (C) (*2, 3*). Total [^{3}H]serotonin transport is expressed as the percentage of the total neuronal [^{3}H]serotonin that has appeared in the PLN and CBC (or 0.5- to 1-mm stump of the transected CBC) in the 3 hours following injection of [^{3}H]serotonin. Transport of PLN [^{3}H]serotonin is the percentage of the total in the PLN alone. In separate ganglia, the transport of [^{3}H]glycoprotein was measured 6 hours after the injection of [^{3}H]fucose into the pair of cell bodies (*5*). Protein synthesis was measured by incubating separate ganglia in 20 μM [^{3}H]leucine for 1 hour at 22° to 23°C (*14*). The GCN cell bodies were removed from the ganglia by microdissection, and acid-precipitable radioactivity was measured. Values are expressed as means ± standard error for N experiments. Those means from experimental cells that are marked with asterisks are significantly different from the corresponding control cell means ($P < .01$) in a paired t-test.

Time after transection (days)	Total [^{3}H]serotonin transport			PLN [^{3}H]serotonin transport			PLN [^{3}H]glycoprotein transport			Protein synthesis (fmole/hour)		
	C	E	N	C	E	N	C	E	N	C	E	N
1	15.9 ± 1.7	13.7 ± 1.4	11	5.4 ± 0.5	10.8 ± 1.1*	11	17.3 ± 1.7	21.7 ± 2.9	10	123 ± 18	106 ± 15	4
2	17.3 ± 1.6	12.7 ± 1.2*	10	6.2 ± 0.5	9.4 ± 0.9*	10				137 ± 35	162 ± 36	8
3	15.9 ± 0.9	9.3 ± 0.9*	10	5.3 ± 0.6	6.3 ± 0.9	10				105 ± 11	119 ± 6	7
7	15.1 ± 1.5	8.1 ± 1.2*	8	4.9 ± 0.6	5.8 ± 0.9	8				84 ± 13	119 ± 22	6

*Significantly different from corresponding control ($P < .01$) in paired t-test.

synaptic targets (Fig. 1A) (*2*). Fast transport of serotonergic storage vesicles can be measured by microinjecting [^{3}H]serotonin into the large (~ 300 μm) cell body (*2*). Within an hour of transecting one of the axonal branches near its point of exit from the ganglion (Fig. 1A), vesicles that would normally have entered that branch for transport are diverted into the intact axonal branch and move by normal fast transport along with the usual complement of vesicles of the intact branch (*2*). We report here that the transport of the abnormally large number of vesicles in the intact axonal branch does not persist, but that the cell body rapidly and precisely reduces its export of vesicles to return transport in that branch to normal. Our results indicate that the production of vesicles is closely related to the extent of the peripheral field and give some indications about the source of regulatory information.

Transport of [^{3}H]serotonin in the symmetrical pair of GCN's was measured 1, 2, 3, and 7 days after transection in vivo, on one side of the ganglion, of the cerebrobuccal connective (CBC), which contains one branch of the bifurcated axon (Fig. 1A). The posterior lip nerve (PLN), which contains the other branch, was left intact. The contralateral CBC was not transected, leaving one GCN as an internal control. One day after transection, transport in the PLN on the side of the ganglion where the CBC had been cut was twice as great as transport in the control contralateral PLN because of the diversion phenomenon (Table 1 and Fig. 1B). Quantitative analysis by electron microscopy confirmed that the number of serotonergic vesicles in the PLN axon (arrows in Fig. 1A) was doubled at this time (Table 2).

Despite the ability of the PLN to move the surplus vesicles by normal fast transport (*2, 3*), total export of [^{3}H]serotonin declined monophasically during the next 2 days to a point at which the amount of [^{3}H]serotonin moving in the PLN was restored essentially to the control level (Table 1, Fig. 1B). Export declined no further during the subsequent 4 days, indicating that it had been adjusted to a new stable level. Total protein synthesis, as measured by the incorporation of [^{3}H]leucine (Table 1), and the amplitudes of the resting and action potentials (data not shown), were essentially unchanged during the week following nerve transection, so the cell did not undergo a general metabolic decline.

If the actual regulatory adjustment by the cell body were a decrease in the synthesis of vesicles, it may have been completed much more rapidly than indicated by the time course of the decrease in [^{3}H]serotonin transport. This is because injected [^{3}H]serotonin labels preexisting as well as newly synthesized vesicles, and there is a large population of vesicles in the cell body which is only slowly depleted following inhibition of protein synthesis (*3*). Extrapolation of the line describing the time course of the decrease in total transport of [^{3}H]serotonin suggests that down regulation be-

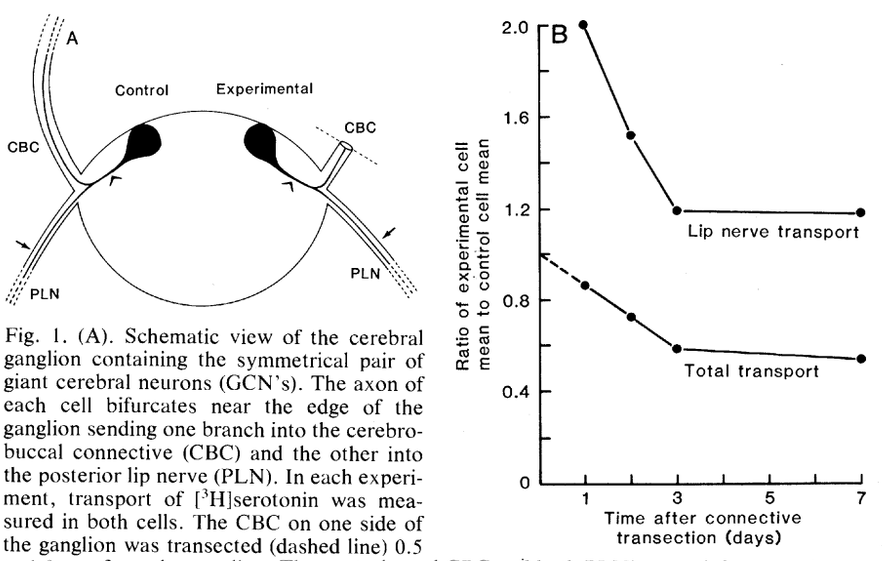

Fig. 1. (A). Schematic view of the cerebral ganglion containing the symmetrical pair of giant cerebral neurons (GCN's). The axon of each cell bifurcates near the edge of the ganglion sending one branch into the cerebrobuccal connective (CBC) and the other into the posterior lip nerve (PLN). In each experiment, transport of [^{3}H]serotonin was measured in both cells. The CBC on one side of the ganglion was transected (dashed line) 0.5 to 1.0 mm from the ganglion. The contralateral CBC and both PLN's were left intact. Arrows and arrowheads identify areas of the axon of GCN prepared for electron microscopic analysis (see Table 2). (B) Ratios of the means for PLN and total [^{3}H]serotonin transport from Table 1. The line joining the points at 1, 2, and 3 days for total transport was fit by least-squares linear regression ($r = -.999$) and extrapolated back to experimental transport/control transport = 1 (dashed line) to estimate when the reduction in total transport began.

gins very soon after transection of the CBC (Fig. 1B), and we have evidence, from injections of [^{3}H]fucose, indicating that the regulatory event in the cell body is indeed largely, if not totally, completed within 1 day. Injected [^{3}H]fucose is incorporated into membrane glycoproteins that in the axon of GCN are initially associated mainly with the storage vesicle (*4*); in contrast to [^{3}H]serotonin, however, [^{3}H]fucose should label only newly synthesized vesicles (*5*). [^{3}H]Glycoprotein exhibits the same diversion phenomenon as [^{3}H]serotonin (*6*) but, 1 day after CBC transection, [^{3}H]glycoprotein transport in the PLN had returned to normal (Table 1).

Several studies of axotomized neurons have demonstrated that, deprived of the peripheral cytoplasm into which they are normally dispersed, some materials exported for anterograde fast transport will accumulate in the cell body (*7*), either by reversing direction at the transection and returning by retrograde transport (*8*) or simply by accumulating centripetally in the axon stump (*9*). It has been suggested that this may be the signal for the changes in export of fast transported material, including neurotransmitter, that have been observed after axotomy (*7, 8*). In the present experiments, however, fast transported material should not have been forced back to the cell body because only part of the axonal tree was removed and material could therefore be diverted into the intact branch (*2*) (Fig. 1B). We confirmed that there was no abnormal return or backup of serotonergic vesicles to the GCN cell body by quantitatively analyzing electron micrographs of the axon of GCN close to the cell body, proximal to its bifurcation (arrowheads in Fig. 1A). Vesicles were counted in micrographs from ganglia fixed either 4 or 24 hours after transection of the CBC, at which times the number of vesicles leaving the cell body is not yet decreased substantially (Fig. 1B), and thus a return or backup of vesicles should be measurable as an increase in the concentration of vesicles in the proximal segment of the axon. There was no difference in concentration at either time point (Table 2). This suggests that return or backup of storage vesicles to the cell body is not the signal for the decrease in fast transport of neurotransmitter that follows nerve transection.

Our results show that when part of the axonal arborization, with associated synapses, of the identified neuron GCN is abruptly removed, the cell body rapidly reduces its output of serotonergic storage vesicles by an amount just sufficient to reestablish the normal supply to the remaining synapses. Without this reduction, those synapses would receive more than twice the normal complement of vesicles, since the vesicles that were originally destined for the disconnected synapses are simply diverted into the intact axonal branch, which apparently has considerable surplus transport capacity (*2, 3*). Axotomy is known to cause reductions in the transmitter content of neurons and in their fast transport of transmitter and transmitter-related enzymes (*7, 10*). Transport of transmitter had not been studied directly, however, in a situation where only part of the axonal tree had been separated from the cell body; that is, where there were still suitable targets—the remaining synapses—to which all the transmitter vesicles would be directed, and where it could be determined whether the reduction, if any, in vesicle transport was congruent with the extent of synaptic loss.

Table 2. Vesicle concentration in the axon of the GCN proximal and distal to the bifurcation after CBC transection. In each of four cerebral ganglia, the CBC axon of one GCN was transected (E) while the other GCN was left intact (C). Ganglia and associated nerves were processed for electron microscopy as described (*15*). The relevant regions of the axon of GCN were identified by cutting serial sections (2 to 8 μm) through the entire ganglion out into the PLN. Cross-sectional electron micrographs were then prepared from the region of the proximal axon about midway between the cell body and the bifurcation (~ 250 μm from the cell body; arrowheads in Fig. 1A) and from the region of the axon in the PLN about 1 mm distal to the bifurcation (arrows in Fig. 1A). For individual micrographs, vesicles were counted and the concentration was expressed as number of vesicles per 100 μm^2 of micrograph area (*15*). All values are means ± standard error from ten random micrographs. The origin of each micrograph was coded and revealed only after counting had been completed.

Time after CBC transection (hours)	Proximal axon		PLN axon	
	C	E	C	E
24	46 ± 6	48 ± 3	50 ± 4	111 ± 6*
24	52 ± 3	43 ± 4	48 ± 4	99 ± 6*
4	44 ± 3	45 ± 3		
4	49 ± 4	45 ± 5		

*Significantly different from corresponding control ($P < .01$) in a *t*-test.

Why does the cell body reduce its output of serotonergic vesicles after transection of the CBC? The reduction could be a response to a novel signal generated by the transection. But we think it is caused by the loss of information normally provided by the disconnected axon or its associated synapses. The signal to which the cell body responds by reducing its output of serotonergic vesicles is rapidly ascertained by the cell body and is sufficiently quantitative to cause a precise adjustment in vesicle output. There seem to be three possible sources for this signal. One is the stump of the axon in the transected CBC. Events in the stump, such as the transient cell depolarization and leakage of axoplasm that may follow axon transection, could occur rapidly enough to elicit the down regulation observed, but it is not easy to see how these events would instruct the cell to reduce output of vesicles just enough to restore transport in the PLN to normal. One event in the stump that could account for the precise reduction would be a turnaround or backup of excess vesicles to the cell body, which would be expected to stop when surplus vesicles were no longer being exported. This event, however, does not appear to occur. A second possible source of the signal for down regulation is the group of synapses at the end of the intact PLN which, upon receiving surplus vesicles diverted from the transected CBC, might send a signal back to the cell body indicating oversupply. Considering the distance from the cell body to the PLN synapses, however, such a signal would be too slow to alone account for the down regulation (*11*).

A third possibility is that the cell body could be signaled to reduce its output of serotonergic vesicles by the loss of information normally provided by the axonal branch in the CBC and its associated synapses. This information could be structural, such as the volume of axoplasm or the extent of the cytoskeleton. Alternatively, an interruption in the ongoing retrograde transport of material from the synapses could be responsible for eliciting at least some of the metabolic changes in the cell body that follow axotomy (*12*). Indeed, many of these changes can be caused by blocking axonal transport pharmacologically rather than by cutting the nerve (*13*). Cessation of the arrival of material from the synapses of the CBC could account for both the speed and precision of the decrease in serotonergic vesicle transport. Were this the signal for down regulation, it would mean that there is a continual

feedback from the periphery influencing the export of transmitter from the cell body and that each branch contributes to this feedback in proportion to the amount of transmitter it ordinarily transports.

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Electric and Magnetic Field Detection in Elasmobranch Fishes

Abstract. *Sharks, skates, and rays receive electrical information about the positions of their prey, the drift of ocean currents, and their magnetic compass headings. At sea, dogfish and blue sharks were observed to execute apparent feeding responses to dipole electric fields designed to mimic prey. In training experiments, stingrays showed the ability to orient relative to uniform electric fields similar to those produced by ocean currents. Voltage gradients of only 5 nanovolts per centimeter would elicit either behavior.*

The shark *Scyliorhinus canicula* and the skate *Raja clavata* have shown a remarkable sensitivity to electric fields in the seawater environment (*1*). The skate exhibited cardiac responses to uniform square-wave fields of 5 Hz even at voltage gradients of 0.01 μV/cm (*2*). The sense organs that detect the electrical stimuli were identified as the ampullae of Lorenzini by the method of selective denervation (*3*) and by recording from the afferent nerve fibers (*4*). Physical theory and behavioral evidence suggest that the elasmobranchs use the electric sense both in predation and for the detection of orientational cues (*5, 6*).

The role of the electric sense in predation was inferred from measurements on the bioelectric fields of fishes and from experiments in which (i) prey fish were shielded with agar to attenuate all but the electrical cues and (ii) the bioelectric fields of prey were simulated by passing electric current between two closely spaced electrodes (*2, 5*). Both *S. canicula* and *R. clavata* executed well-aimed feeding responses to the agar-screened prey and to dipole fields of frequencies from 0 to about 8 Hz (*6*).

In early investigations, the sharks and skates were tested in polyvinyl pools to avoid interference from ambient electric fields. The validity of those results was confirmed by studies at sea, made from a research vessel free of galvanic fields and fitted with a viewing well. Attacks on real and electrically simulated prey were observed in the smooth dogfish *Mustelus canis* and the blue shark *Prionace glauca* in the waters off Cape Cod, Massachusetts (*7*).

Mustelus canis was attracted by liquefied herring released from a tube in the center of the research area. Prey fields were simulated by applying direct current to a pair of electrodes to the right or left of the odor source (Fig. 1A). The tube and electrodes were fitted from beneath through holes in a polyvinyl plate that was set into the bottom and camouflaged by glued-on sand. For young dogfish (30 to 40 cm), an 8-μA current was passed between electrodes 2 cm apart (d_1 electrodes, Fig. 1A) and 15 cm from the odor source. For larger dogfish (90 to 120 cm), the current was switched to electrodes 5 cm apart (d_2 electrodes, Fig. 1A) and 30 cm from the odor source. Tests were conducted at night, when the animals were foraging for food. The setup, located at depths of 2.5 to 3.5 m, was dimly lit by underwater lights.

The attacks usually started with a sudden dive or turn, making them suitable for assessing the distances of response and the corresponding stimulus strengths. With the electric current spreading into half-space, the field along the dipole axis measured $\rho Id/\pi r^3$ μV/cm (for $r >> d$), where ρ is the resistivity of seawater (23 to 26 ohm-cm), I the applied direct current (8 μA), d the electrode spacing (2 or 5 cm), and r the recorded length of the radius vector with the dipole as origin. In the plane normal to the dipole, the field strength was only half that of equidistant points along the dipole axis. The use of a constant-current source and salt bridges (50 cm long) eliminated the adverse effects of polarization at the stainless-steel electrodes. To prevent loss of continuity from air bubbles accumulating, the salt bridges were filled with seawater agar or threaded with seawater-soaked cotton wicks.

The dogfish approached singly or a few at a time and, although motivated by scent, they almost invariably attacked the electrodes between which current was passed and seldom bit the control electrodes or the odor source. In 49 out of 136 responses, small dogfish initiated their well-aimed dives from distances of 15 cm and more, sensing voltage gradients ≤0.033 μV/cm; in 16 of the responses, they struck from 18 cm or more, detecting gradients ≤0.021 μV/cm (Table 1). Larger dogfish initiated 44 out of 112 attacks from 30 cm and farther, where fields measured ≤0.010 μV/cm; in 15 of the responses the distances were in excess of 38 cm, revealing a sensitivity to ~ 0.005 μV/cm or 5 nV/cm (Table 2). Because the field strengths reported were those measured along the dipole axis, 5 nV/cm is a conservative estimate.

The relatively short response distances may suit the shark's feeding strategy (*8*). *Mustelus canis* frequently snapped at small fishes hovering over the sand, but most prey were alarmed in time to escape. The dogfish are not particularly fast-moving and, to catch their prey, they have to strike from close by and with great accuracy. For the detection of prey hiding in loose gravel or sand, it is not so much the distance of response as the penetration power that gives the electric sense an edge over other sensory modalities. It is noteworthy that, when both dipole pairs were activated, small dogfish avidly attacked the closely spaced electrodes, but often

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