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Giant Axons and Escape Swimming in *Euplokamis dunlapae* (Ctenophora: Cydippida)

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Abstract. *Euplokamis dunlapae* responds to anterior stimulation by reversing the beat direction of its comb plate cilia and swimming rapidly backwards. It responds to posterior stimulation by swimming forwards at an accelerated rate. Video playback and laser monitoring were used to analyze changes in the pattern of ciliary beating, while electrical activity was recorded extracellularly. Escape responses occur with latencies of less than 150 ms and involve greatly increased ciliary beat frequencies. Giant axons run longitudinally along each of the eight comb rows, as shown by optical and electron microscopy. They form chains of overlapping neurons, with diameters of about 12 μm in life, and conducting at over 50 cm · s^{-1} as recorded with an extracellular electrode placed directly over the chain. The giant neurons are synaptically linked with smaller neurites of the general ectodermal nerve plexus, with each other, and with the ciliated cells of the comb plates. They appear to constitute a single system mediating rapid conduction of signals in either direction, but a full analysis was not attempted for lack of sufficient material. Electro-physiological examination of two other ctenophores (*Pleurobrachia* and *Beroë*) gives no indication of rapid conduction pathways, and these forms probably lack giant axons.

Introduction

Several cydippid and lobate ctenophores have the ability to reverse the direction of the power stroke of their comb plate cilia. In the best-studied example, *Pleurobrachia* (Tamm and Moss, 1985; Moss, 1986; Moss and Tamm, 1986, 1987), reversals occur unilaterally as part of feeding behavior, and make the animal rotate on its axis. Reversals can also occur simultaneously in all the comb rows, causing the animal to swim backwards. Several cases of reverse swimming have been reported (reviewed by Tamm, 1982), but none have been studied in detail. We here describe the responses of an unusual and interesting cydippid ctenophore, *Euplokamis dunlapae,* that responds rapidly to stimulation by forward and reverse swimming. A novel feature of these responses is that they appear to be mediated in part by giant axons that run under the comb rows.

A taxonomic account of *Euplokamis dunlapae* (Fig. 1). is given by Mills (1987). The species is probably the most abundant midwater ctenophore in the Strait of Georgia and adjacent fjord systems, reaching its greatest density at 250 m; however, specimens are rare above 100 m, and only very infrequently found at the surface (Mackie and Mills, 1983; Mackie, 1985). They are too fragile to be collected and brought to the surface in nets. Thus, opportunities to study them have been few. In 1984 we obtained enough specimens for a study of their prehensile tentilla (Mackie *et al.,* 1988). In 1990 and 1991 we obtained five more specimens, on which this account is based. Having so few specimens necessarily limited the scope of the study, but they were in good physiological condition, and there is no reason to doubt the generality of the findings.

Materials and Methods

Specimens of *Euplokamis dunlapae* Mills 1987 were collected off the dock at the Friday Harbor Laboratories and kept in wide-mouthed glass bottles at 8°C until used. Material for electron microscopy was fixed in 2.5% glutaraldehyde in 0.4 *M* Millonig's phosphate buffer at pH 7.4 for 1 h at room temperature, rinsed, and osmicated

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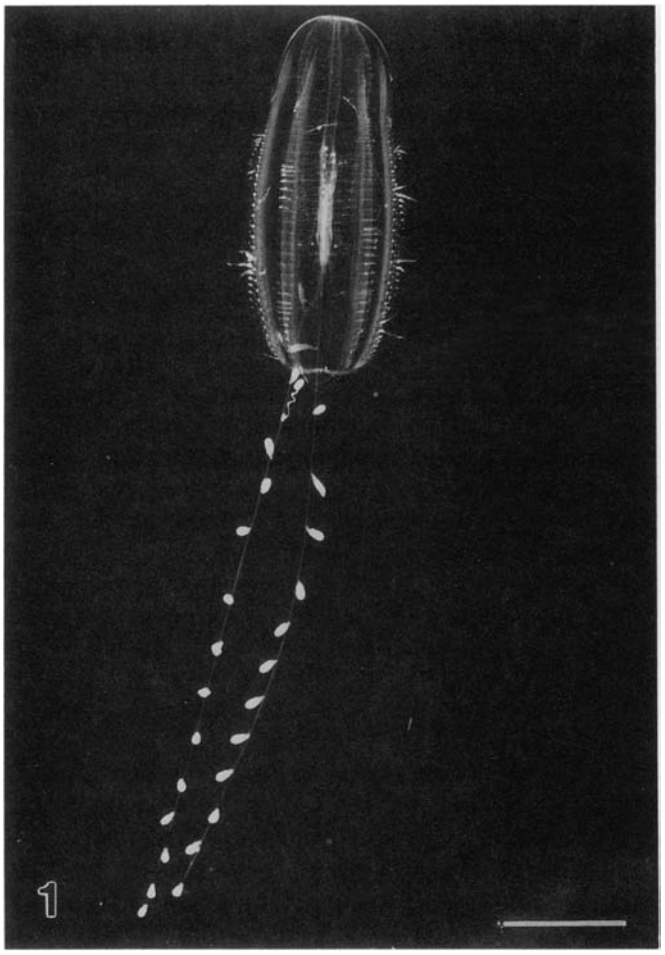


Figure 1. *Euplokamis dunlapae* swimming in an aquarium, oral end up. Scale bar: 5 mm.

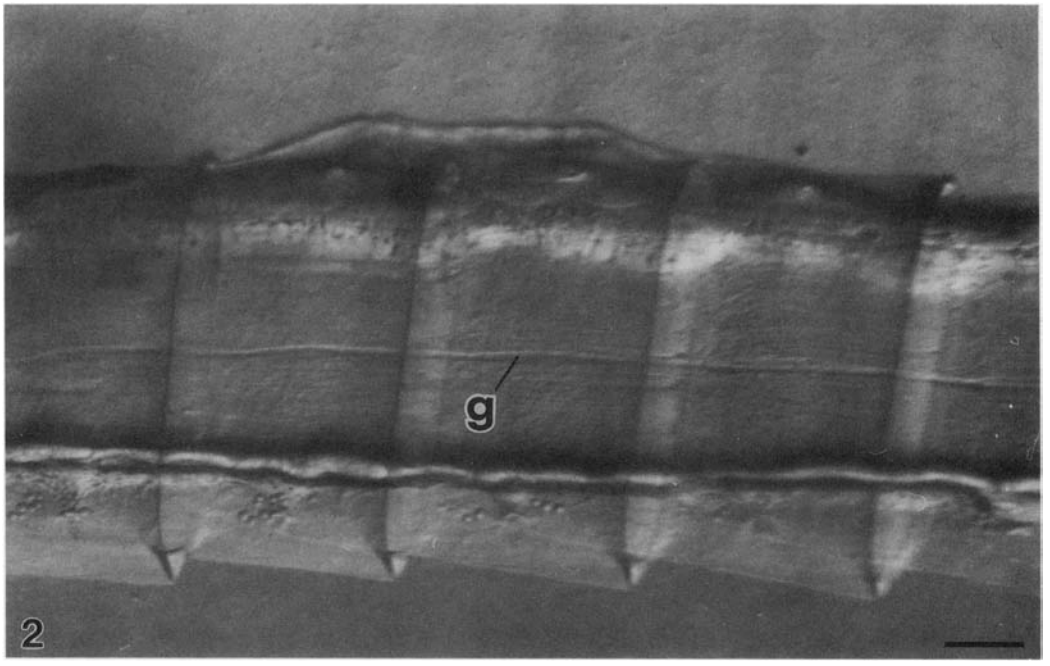
in 1% osmium tetroxide in the same buffer at 4°C for 1 h. Specimens invariably disintegrated during fixation despite every precaution. Fortunately, intact fragments of comb rows, along with some attached underlying tissue, could be retrieved from the debris and processed for electron microscopy. The tissue was dehydrated through graded ethanols and propylene oxide and embedded in Epon 812. Thick sections were stained with Richardson's stain. Thin sections were treated with uranyl acetate and lead citrate. Because the tissues were extremely fragile, we could not prepare whole mounts of living material for examination by Nomarski or phase contrast microscopy. Figure 2, showing giant axons in an intact, living animal, was taken through a dissecting microscope illuminated with a double substage mirror, with the mirror angles arranged to give shadows along the edges of the axons.

Behavior of free-swimming specimens was observed in a 15-l tank, illuminated from the sides, and with a dark background. A Sony CCD video camera (HVM-200), fitted with a Nikon 105 mm macro lens, was used in conjunction with a VCR with frame-by-frame playback for analysis of responses. Recordings of electrical activity were made from specimens pinned down in a Sylgard-lined Pyrex pie dish placed on top of a doughnut-shaped Cambion cooling stage, which allowed light to enter from below. Temperature was maintained at 12°C. Fine polyethylene suction electrodes were attached directly to the body surface using minimal suction to keep them attached. Signals were amplified with capacity-coupled amplifiers and displayed on a digital oscilloscope; conventional extracellular recording procedures were used. Stimuli were delivered through paired metal wires insulated to near the tip. Movement of comb plates was monitored; we used a ruby laser (Spectra Physics Model 155) to project a narrow beam of light across the comb row, and a photomultiplier (International Light 270C) to detect purturbations of the beam caused by the ciliary movement. Laser monitoring of ciliary beating in molluscan veligers is described by Arkett *et al.* (1987). The method, as used here, allowed us to distinguish forward from reverse power strokes by the polarity of the waves recorded.

Histology and Ultrastructure

The comb rows of *Euplokamis* resemble those of other ctenophores as reviewed by Tamm (1982). Each plate consists of thousands of cilia springing from "polster" cells, which are packed with large mitochondria. We have confirmed that gap junctions are present between the polster cells as first reported by Hernandez-Nicaise (1974). The comb plates are richly innervated by fine nerve fibers running among the bases of the polster cells. These fine neurites have diameters in the range of 0.7–2.5 μm ($\bar{X}$ = 1.2, SD = 0.4, n = 13). They appear to represent part of the general ectodermal nerve plexus. This system is well known in *Pleurobrachia* from the investigations of Hernandez-Nicaise (1973a, 1974). Moss (1986) has shown that ciliary reversals in *Pleurobrachia* are mediated by a diffuse conduction system running in the ectoderm, presumably the nerve plexus described by Hernandez-Nicaise in whole or part.

Where *Euplokamis* differs from other known ctenophores is in its possession of bipolar giant axons running along each comb row. These can be seen under the dissecting microscope in the intact, living animal (Fig. 2), and at higher magnifications in thick epon sections (Fig. 3) and electron micrographs (Fig. 4). They are present in all eight comb rows and run roughly down the midline of each comb row from one end to the other. Their cell bodies lie between the comb plates, and there appears to be one cell body between each pair of comb plates. They evidently form a chain of cells arranged end to end, with



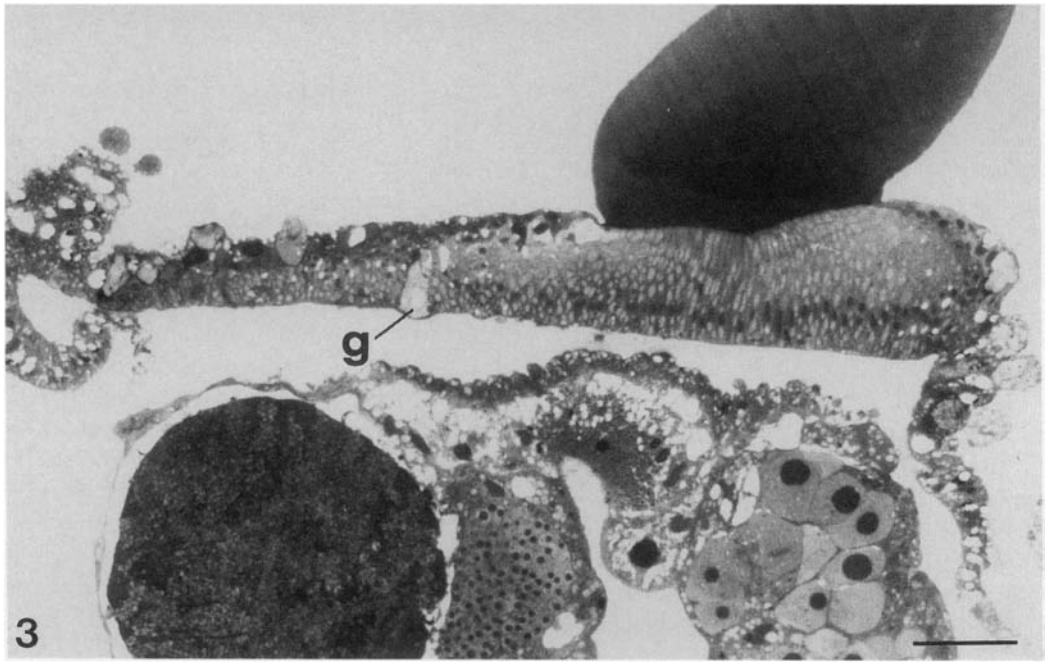


Figure 2. Surface view of a sub-tentacular comb row in living animal, showing four comb plates whose cilia are laid down flat on the surface, and giant axon chain (g). Scale bar: 100 μm.

Figure 3. One (1.0) μm section cut transversely but at a slight angle through a comb plate showing massed cilia (dark mass, upper right) arising from polster cells, and giant axons (g). Gametes are seen in the underlying endodermal canal. Scale bar: 50 μm.

some overlap. In typical sections, one or two axon profiles, but not more, are seen, showing that the neurites must be quite short, probably less than 400 μm. In their thicker regions, they show diameters of approximately 8.5–12.0 μm ($\bar{X}$ = 9.7, SD = 1.02, n = 10), but they taper towards the ends. Allowing for shrinkage during fixation and embedding, the axons are probably at least 12 μm in the living animal, which is also suggested by measurements

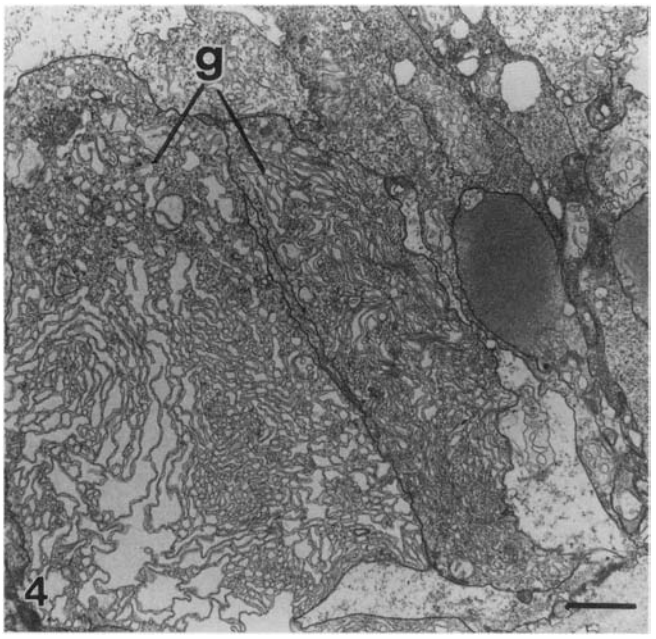


Figure 4. Low power electron micrograph showing cross sections of giant axons (g) surrounded by polster cell bases. Scale bar: 2 μm.

on living preparations. There appears to be only one nucleus per cell; it is long and thin and causes only a slight swelling in the cell body region.

The axoplasm of the giant axons is remarkable for its richly developed smooth endoplasmic reticulum, which appears to form a continuous network of fine canals throughout the entire cell. The axoplasm also contains conspicuous bundles of microtubules. Rows of 100-nm dense-cored vesicles have been seen associated with these bundles (Fig. 5). The area around the nucleus does not differ markedly from other parts of the axoplasm. There is little indication of active protein synthesis. The nucleolus is not prominent, and there is little rough endoplasmic reticulum. Mitochondria and Golgi bodies (one is seen in Fig. 6) are distributed along the whole length of the axon.

We have seen no gap junctions in the nervous system (nor are there any reports of such in the ctenophore literature), but we have observed synapses between neurites of all sizes. There are synapses between fine neurites and giants, and between giants in areas of overlap (Fig. 6). Synapses also occur between these neurons and polster cells (Fig. 7). The synapses resemble those seen in the tentilla of *Euplokamis* (Mackie *et al.*, 1988), as well as those described in other ctenophores. They are characterized by "presynaptic triads" (Hernandez-Nicaise, 1973b, 1974) consisting of a mitochondrion embraced by a flattened ER cisterna with an accompanying row of small (50 nm) synaptic vesicles.

Behavior of Free-Swimming Specimens

When left to its own devices in a large tank, *Euplokamis* shows bouts of forward "cruising" swimming, interspersed with periods of quiescence. During cruising, ciliary metachronal waves run down the comb rows with a frequency of about 5 Hz, driving the animal forward (mouth leading). A specimen 15 mm long cruises at about 2 $cm \cdot s^{-1}$. The tentacles trail behind, partially extended and with their tentilla coiled. When swimming stops, the body swings around passively so that the mouth points up and the tentacles hang down. The animal can hang in this posture with its cilia either arrested or beating irregularly and infrequently. Swimming animals will go into reverse if they hit the walls of the tank, but otherwise they swim forward steadily in the cruising mode.

Stimulation by touch or by an electric shock on any part of the surface alters the pattern of swimming. If the stimulus is applied at the front, for instance on the lips, the animal rapidly changes the direction of the ciliary power stroke in all eight comb rows, goes into reverse, and swims backward for one or two body lengths, pauses, and then resumes forward cruising behavior. During backward swimming, velocities of about 4 $cm \cdot s^{-1}$ were observed, with elevated metachronal beat frequencies. Using frame-by-frame playback on the VCR, the first signs of interruption of the ciliary beat pattern were generally seen within four frames (<130 ms) following the stimulus, with the actual change in the direction of movement occurring one or two frames (<67ms) later.

When stimuli are applied at the rear (statocyst) end, the animal responds by accelerated forward swimming: within four frames (<130 ms) the metachronal rhythm suddenly increases as in the backward escape response, but with no change in the direction of the power stroke, so the animal darts forward rapidly at a velocity of about 5.5 $cm \cdot s^{-1}$. After several seconds, it slows to the normal cruising speed.

During both backward and forward escape swimming, the tentacles contract. The first signs of contraction are seen at about the same time as the first signs of change in the pattern of ciliary beating.

Animals that have been left to swim around the tank without interference respond to stimuli with great alacrity, but after repeated stimulation, responses become less intense and latencies tend to increase.

Electrophysiology

Propagation along the comb rows

If a recording electrode is placed directly over the giant axon chain at any point along its length, and an electrical stimulus is delivered further along the comb row, a pattern

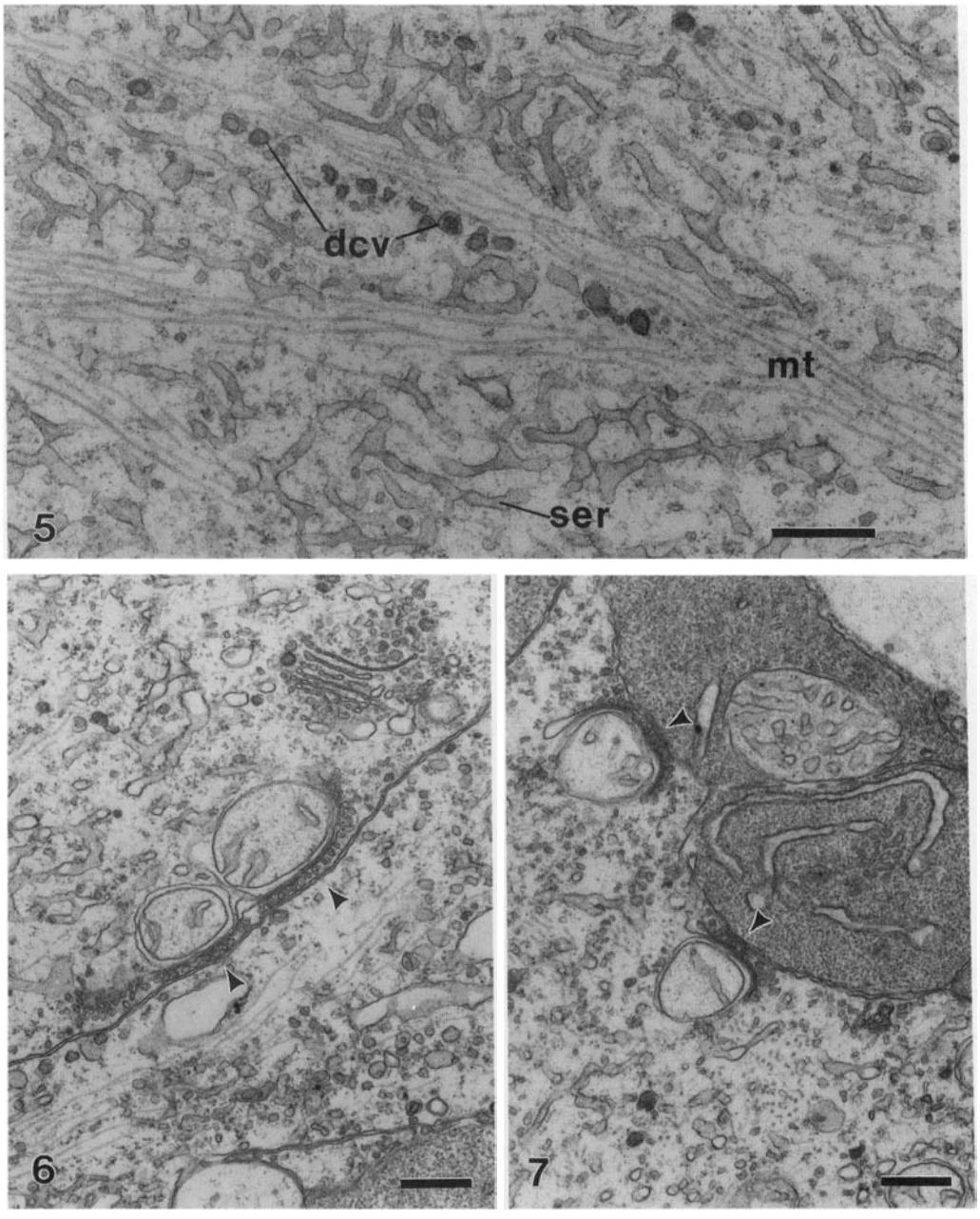


Figure 5. Axoplasm of giant axon cut longitudinally showing dense-cored vesicles (dcv) aligned along microtubule bundle (mt) and richly developed smooth endoplasmic reticulum (ser). Scale bar: 0.5 μm.

Figures 6, 7. Synapses (arrowheads) between giant axons (6) and from giant axon to polster cell (7). Scale bars: 0.5 μm.

of electrical potentials is recorded, which has two readily separable components (Fig. 8). The first component is a small (150 μV), sharp, spikey event conducted at 51–56 $cm \cdot s^{-1}$ ($\bar{X}$ = 53.4, SD = 1.95, n = 10). This event is presumably the extracellular correlate of an action potential propagated in the giant axon chain. The signal is lost when the recording electrode is moved even slightly to one side or the other of the giant axon. The second component is a larger, complex, and variable series of positive and negative-going potentials that presumably represent responses in the ciliated polster cells. This component may be brief or it may take the form of flurries lasting several hundred milliseconds. We were not able to analyze these events in detail, but assume that they include

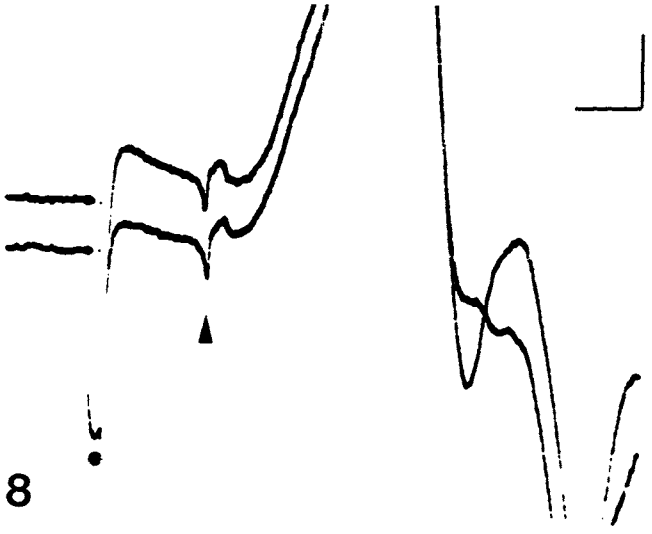


Figure 8. Recording with an electrode placed directly over the giant axon row, two successive sweeps in same position. Following electrical stimulation further along the comb row in the aboral direction (black spot marks shock artifact), a rapidly conducted, sharp, spikey event (arrowhead) is recorded, followed by a much larger, complex, and variable series of potential changes. Scale bar: 10 ms, 200 μV.

summed synaptic events and regenerative events in the polster cells, which, with some of their cilia, were partially engulfed by the electrode. The large biphasic events, which reach amplitudes in the millivolt range, would presumably correspond to the calcium spikes described by Moss and Tamm (1986, 1987) in *Pleurobrachia.*

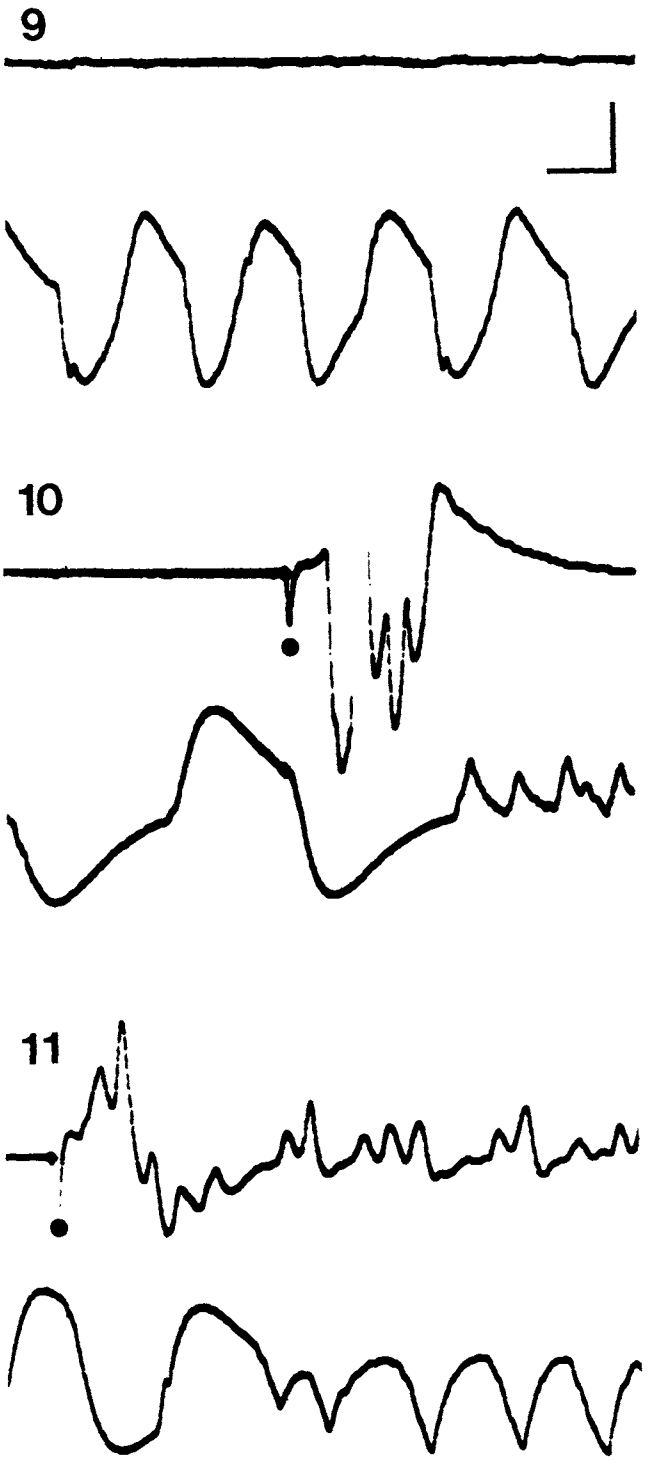
The interval between the arrival of the giant nerve spike at the recording electrode and the start of the polster cell depolarization is quite variable, ranging from 5 to 20 ms ($\bar{X}$ = 12.2 ms, SD = 5.5, n = 7). This may mean that the polster cell depolarization is not initiated at the recording site, but is propagated from an unknown and more or less distant site. Consequently, unless the nerve spike is actually recorded, it is impossible to give an accurate or even consistent estimate of conduction velocity in the nervous system. In spite of this, measurements of the latency between stimulus and polster cell response can give a rough figure (here termed "apparent conduction velocity") serving for comparative purposes in any one location. These estimates assume that conduction always takes place along the most direct route measured between stimulus and recording points, but this too needs verification.

Apparent conduction velocities along the midline of the comb row were reduced to 75% of their original value after a single cut through the giant axon chain. Apparent conduction velocity decreased progressively with further cuts at different levels, reaching 55% of the original value after the axon chain had been destroyed over most of its length. At this point, it must be assumed that excitation was travelling in the general epidermal nerve plexus. The apparent conduction velocity along the comb rows after destruction of giant axons is close to the value obtained for conduction across the ectoderm in regions devoid of giant axons, the mean velocity being 12 $cm \cdot s^{-1}$ (SD = 3.07, n = 17, three specimens). Incisions into the comb row tissue did not reduce apparent conduction velocities unless they intersected the giant axons. The fact that several incisions must be made through the giant axon chain at different levels along the comb row to bring the apparent conduction velocity down to the non-giant velocity is consistent with the idea that impulses can enter the giant axons from a diffuse nerve net at numerous points along the giant axon chain.

Simultaneous recording of polster cell depolarizations and ciliary beat patterns

Animals pinned down in a small dish continue to show normal cruising swimming with a metachronal wave frequency of about 5 Hz (Fig. 9). Using the laser beam method of monitoring ciliary beating, we could, in a few cases, detect the exact moment at which this pattern was interrupted following stimulation and determine the interval between this change and the onset of the electrical response in the polster cells (Figs. 10, 11). The giant axon spike is not seen in these records, as the recording electrode was not placed directly over the midline of the comb row. In Figure 10, following stimulation at the oral end of a comb row, the cilia switched to the reverse pattern of beating 122 ms after the stimulus, and 85 ms after the start of the electrical response recorded from polster cells adjacent to the laser-monitoring site. Beat frequency was 25 Hz immediately after the change of gait, but the frequency declined quickly, and the cilia shortly switched back to beating in the forward cruising mode at 5 Hz. The response latency of 122 ms recorded by this method is consistent with the value of <130 ms obtained from the video-playback analysis.

When the stimulating and recording positions were reversed and a shock was delivered at the aboral end of the comb row, a forward escape swimming response was obtained (Fig. 11). Regular, slow metachronal beating changed suddenly to rapid forward beating starting 143 ms after the shock, and 107 ms after the first component of the electrical response recorded from adjacent polster cells. After two beats at the equivalent of 17 Hz, the frequency declined to 8 Hz and later (off the record) to 5 Hz, as normal cruising was resumed. We could not repeat these experiments enough times to allow a proper statistical analysis and cannot say, therefore, whether the response latency is consistently shorter in the case of reverse escape swimming than in forward, nor how this may vary



Figures 9, 10, and 11. In each figure, the upper trace is a record of electrical activity from a comb plate while the lower is a laser beam record of the ciliary beating at the same spot. Figure 9 is a control (no stimulus). Figure 10 shows the response to a shock delivered orally of the recording electrode. Small upward events on the laser record correspond to reverse ciliary beats. Figure 11 shows the response to shock delivered aborally. Small downward events on the laser record correspond to fast forward beats. Spots mark shock artifacts. Scale bars: 100 ms, 500 μV (upper trace) (9); 50 ms, 500 μV (10); 50 ms, 200 μV (11).

with position along the comb row. In both cases, however, the response latency can clearly be less than 150 ms.

The cilia generally appear to switch directly from one mode of beating to another without a break, but in some cases a short period of inactivity was observed before the new pattern emerged. During these periods, the cilia appeared to be in the "laydown" position described for *Pleurobrachia* (Moss and Tamm, 1986), but this needs to be verified.

Comparison with other species

We know of no previous reports of giant axons in the comb rows of ctenophores. We have looked at living specimens of *Pleurobrachia bachei* and *Beroë sp.* using the optical arrangement that enabled us to see the giant axons in *Euplokamis* (Fig. 2) and could see no comparable structures. We have cut some sections of *Pleurobrachia* and examined them under the EM with the same result, confirming Hernandez-Nicaise (1973a, 1974), who found only small-diameter neurites. Electrophysiological recordings from *Beroë,* made in the same way and at the same temperature (12°C) as *Euplokamis,* show the complex responses associated with excitation of the polster cells but no preceding neural event. Presumably the nerves conducting the excitation are too small and scattered to give a clear extracellular signal. The response latency is also much longer than in *Euplokamis,* with apparent conduction velocities lying in the range of 21–25 $cm \cdot s^{-1}$. This is similar to the mean value of 25 $cm \cdot s^{-1}$ recorded for *Beroë ovata* at 22°C by Hernandez-Nicaise *et al.* (1980). Apparent conduction velocities in *Pleurobrachia* are even slower, in the range of 11–16 $cm \cdot s^{-1}$. It would appear that rapid conduction is peculiar to *Euplokamis* and is presumably made possible by the giant axons found in this species.

Discussion

Giant axons have evolved in many invertebrate groups as mediators of rapid responses serving either for escape or food capture. Such rapid movements are generally muscular, but there is no reason why a streamlined animal swimming by means of powerful cilia should not have undergone selection for fast pathways mediating its locomotory responses. Such appears to be the case with *Euplokamis.* While many details of the ciliary control mechanism remain to be elucidated, there can be little doubt that the giant axons have evolved to bring about rapid changes in ciliary beat frequency and direction in the context of escape. The finding of giant axons in a ctenophore, though interesting, is not likely to require any drastic reconsideration of ctenophore phylogeny and relationships. We agree with Bullock (1984) that giant

fibers have probably evolved independently in many groups.

The giant axons appear to be fairly short, thick, mononucleate structures forming a chain with some overlap. They synapse with each other, suggesting that they constitute a single pathway. They also synapse with elements of the diffuse nerve net, and can be regarded as a specialized pathway within this net, recalling a similar situation in certain hydrozoan coelenterates (Mackie, 1989). Ultrastructurally, the axons are rather unusual. They have an extremely rich smooth endoplasmic reticulum that appears to form a continuous canal network throughout the entire cell. Such a system could serve for intracellular transport (Droz *et al.*, 1975), but we also see EM images of vesicles lined up along microtubule bundles (*e.g.*, Fig. 5), which is suggestive of mechanoenzyme-driven transport (Vallee *et al.*, 1989).

We are not yet in a position to explain exactly how ctenophores control the frequency of ciliary beating or the direction of the power stroke, though much progress has been made in recent years with *Pleurobrachia.* In this genus, the process can be explained without resorting to a dual innervation model (Moss and Tamm, 1986). Moderate depolarization of the polster cells is associated with increased rates of beating in the normal (forward) direction. Larger depolarizations sufficient to cause a regenerative response (calcium spike) are associated with arrest (laydown) and with accelerated beating in the reverse direction. The comb plates can pass fairly abruptly from accelerated reverse to accelerated forward beating, although a period of intermediate beating may intervene. Thus, depending simply on the number and time relationships of input events at a single set of synapses, the polster cells could be maintained at any level of depolarization or spike frequency required for the various locomotory gaits. Moss and Tamm, however, found evidence of two types of excitatory post-synaptic potentials, suggesting that there may actually be two functionally distinct neuronal excitatory pathways, one associated with a short latency response and moderate depolarization of the polster cells, and the other with a more delayed response and facilitating excitatory post-synaptic potentials that generate spikes and cause ciliary arrest or reversal. Recall, in this context, that three subsets of neurites were described in the ectodermal plexus of *Pleurobrachia* from ultrastructural and pharmacological studies (Hernandez-Nicaise, 1974). Pharmacological evidence of two subsets was also presented by Satterlie (1978).

At first sight, our physiological findings on *Euplokamis* favor the dual innervation theory because they show that the direction in which the cilia beat depends on the direction from which the excitation is coming. The easiest way of explaining this observation would be to assume that there are two separate diffuse nerve nets, one receiving sensations from the front and one from the back, and exciting the comb plates in different ways. If so, however, there should be two sets of giant axons, one associated with each net, but this is not the case. The giants appear to constitute a single series in each comb row, adjacent giants being interconnected by synapses. If this is the case, then there is only one cilio-motor innervation, and the giants are part of it, providing the final common pathway for information from all parts of the animal. In this scenario, the way the comb plates respond might depend on impulse frequency differences associated with the excitation of receptors at the two ends, transmitted through the common excitatory pathway. Frequency coding in a single conducting system provides the basis for two sorts of behavior in the sea anemone *Actinia* (McFarlane and Lawn, 1991). However, other explanations are possible, and as the evidence needed to decide between them is not yet available, further speculation on this question is inappropriate. Very probably, however, the control system in *Euplokamis* will prove to be a modified version of the *Pleurobrachia* system, and as *Pleurobrachia* is a rugged and common surface-living species, it is probably better suited for use in exploring these questions than our fragile and elusive midwater species.

Acknowledgments

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