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MODULATED VIBRATION-SENSITIVITY OF LAMPREY MAUTHNER NEURONES

BY S. N. CURRIE* AND R. C. CARLSEN

Department of Human Physiology, University of California, Davis, CA 95616, USA

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SUMMARY

The vibration-sensitivity of larval lamprey Mauthner (Mth) neurones is dependent on behavioural state. Animals are maximally vibration-sensitive when at rest and less so when active or aroused. To demonstrate this effect in freely behaving larvae, we provided repeated vibratory or electrical stimuli to the vestibular labyrinths while animals made transitions between rest and activity. Stimuli which were adequate to elicit Mth spikes 100% of the time in a resting animal (recorded extracellularly from the spinal cord) were consistently subthreshold while the animal was swimming. The same effect was seen in semi-intact preparations, both moving and curarized, while recording intracellularly from Mth cell bodies. Mth vibration-sensitivity decreased abruptly with the onset of 'arousal', defined here by the presence of tonic, descending spinal cord discharge. During arousal, the Mth soma exhibited a slight depolarization (2–8 mV), an increased membrane conductance, and a strong depression of vibration-evoked excitatory postsynaptic potential (EPSP) amplitude. This Mth PSP depression (MPD) appears to underlie altered vibration-sensitivity.

INTRODUCTION

A pair of giant reticulospinal interneurones, called Mauthner cells, is known to initiate the startle response or 'tailflip' reaction of teleost fish and amphibian larvae (Eaton, Bombardieri & Meyer, 1977; Eaton & Bombardieri, 1978; Eaton & Hackett, 1984; Hackett & Faber, 1983; Rock, 1980; Rock, Hackett & Brown, 1981; Zottoli, 1977). Mauthner somata are present in the brain stem of most anamniote vertebrates, including amphibians, bony and cartilagenous fishes, and lampreys (Rovainen, 1967, 1979; Zottoli, 1978). We recently reported that larval sea lampreys (*Petromyzon marinus*) exhibit a rapid startle response following vibration of the otic capsules (Currie, 1984*a*,*b*; Currie & Carlsen, 1985). Lamprey startle behaviour involves a bilateral body contraction (Currie & Carlsen, 1985; S. Currie & R. C. Carlsen, in preparation), unlike the one-sided tailflip of fish and tadpoles.

Lampreys possess two pairs of Mauthner neurones (Rovainen, 1967, 1979), one pair with large, anteriorly situated cell bodies (Mth) and a smaller, posterior pair

* Present address: Department of Biology, Washington University, St Louis, MO 63130, USA.

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(Mth'). In lampreys, vibration of the otic capsules excites the Mth neurones bilaterally (S. Currie & R. C. Carlsen, in preparation; Rovainen, 1979), because of the absence of crossed inhibition (Rovainen, 1979). Simultaneous action potentials in both of the anterior Mth cells are sufficient to initiate the startle response electromyogram (EMG) in semi-intact lamprey preparations (Currie, 1984*b*; S. Currie & R. C. Carlsen, in preparation).

In the present study, we show that the vibration-sensitivity of lamprey Mth cells depends on behavioural state; sensitivity is maximal when animals are at rest, but decreases during periods of muscular activity or arousal. The basis for this effect is an arousal-associated depression of vestibular synaptic input to the Mauthner cell.

MATERIALS AND METHODS

Larval sea lampreys (*Petromyzon marinus*), measuring 9–15 cm in length, were used. Animals were held in a large, sand-bottomed tank which was maintained at 15 °C.

Freely behaving animals

To record spinal cord activity in intact animals, larvae were anaesthetized in ice-cold tricaine methanosulphonate (MS 222, $0{\cdot}3\,g\,l^{-1}$) and pinned out in lamprey saline (Wickelgren, 1977*a*). A 4–5 mm dorsolongitudinal incision was made near the last gill pore to expose the spinal cord, and a monopolar wire electrode (25 μm diameter stainless steel), insulated to within 0·5 mm of its tip, was inserted posteriorly down the spinal canal for about 2 cm. The incision was then sutured closed around the wire. A knot of thread was attached to the wire with glue (Eastman 910) beneath the suture and prevented the electrode from pulling out. Animals were then placed in a shallow aquarium containing lamprey saline and allowed to recover from the anaesthetic. Recordings were made relative to an indifferent bath electrode.

Either vibratory or electrical stimuli were used to evoke Mauthner spikes. Vibrations were produced by lightly tapping the top of the aquarium wall with a rubber-coated ballpeen hammer. For vibratory stimulation, the depth of saline in the aquarium was lowered to about 5 mm, so that the animal, which was 7 mm wide in the gill region, was only partially submerged. This ensured that the animal remained in contact with the aquarium floor, which transmitted the vibration, whether swimming or at rest. Aquarium vibrations were monitored with a calibrated phonograph stylus (Shure 3X), which contacted the outer aquarium wall. Electrical stimuli were produced by implanting 25 μm diameter wire stimulating electrodes in an otic capsule. A small incision was made to expose the dorsal aspect of a capsule and small holes were made with an insect pin in the anterior and posterior ends of the cartilagenous casing of the capsule. The electrode tips were inserted into these holes, and glued in place, and the skin incision was sutured closed around the wires. The labyrinth was stimulated with brief electrical pulses (3–5 V, 0·5 ms, cathode anterior).

Identification of extracellular Mauthner spikes

Mauthner axons are the largest diameter fibres in the lamprey spinal cord (Rovainen, 1967). We identified extracellular Mth axon spikes (Fig. 1) based on their extremely large amplitude and short latency following vibratory stimuli. These first and largest spinal units follow vibratory pulses to an otic capsule one-for-one at frequencies up to 50 Hz (Currie & Carlsen, 1985) an ability unique to the Mth cell (S. Currie & R. C. Carlsen, in preparation) and due to its monosynaptic, predominantly electrical connection with vestibular afferents (Rovainen, 1979). This monosynaptic input also contributes to the short latency of vibration-evoked Mth spikes. Vibration-sensitive Müller neurones (e.g. B_1 and B_2), which are excited *via* a longer latency, polysynaptic pathway (Rovainen, 1979), cannot follow vibratory pulses above 5 Hz (S. Currie & R. C. Carlsen, in preparation).

Semi-intact preparations

We used fully (Figs 3–5) or partially (Fig. 2) immobilized preparations when recording intracellularly in the brain. The former were injected intramuscularly with 50–100 μg of D-tubocurarine. When paralysis was complete, they were transferred to tricaine for 10 min. The animal was then eviscerated and the ventral aspect of its head cut away so that the brain could be transilluminated through the ventral cranium. The larva was pinned out, dorsal side up, and its spinal cord, brain and otic capsules were exposed by cutting away the overlying tissues. A Vaseline-insulated hook electrode was used to record activity from the whole spinal cord, just behind the gill chamber.

The above preparation was sometimes modified so that only the head was paralysed, thus permitting intracellular recording in the brain during active movement of the posterior body. The basic dissection was the same as above except that several body segments were removed behind the gills, leaving a slack loop of spinal cord to connect the head and posterior body. A hook electrode recorded descending activity from the isolated spinal segments. The head alone was then curarized with small, intramuscular injections. Electromyograms were recorded with bipolar, silver wire electrodes (76 μm in diameter) insulated to within 0·5 mm of their tips. Bath temperature was kept between 14 and 16 °C by circulating the saline between the dish and an ice bath with a peristaltic pump.

Intracellular recordings

Reticulospinal somata are easily visible beneath the floors of the third and fourth ventricles after removing the overlying choroid plexus and transilluminating the brain. Intracellular recordings were made with single- and double-barrelled microelectrodes, back-filled with either 3 mol l^{-1} KCl or potassium acetate. Single-barrelled electrode resistances were 30–50 MΩ. Double-barrelled electrodes were pulled from fused (1 mm o.d.) glass and had resistances of 20–30 MΩ per barrel. One barrel delivered current, using the current-passing circuit of a Getting model 5

amplifier, and the other recorded membrane potential, using an MEC model AM-II amplifier.

Otic capsule vibration

We vibrated the otic capsules of semi-intact preparations with a thin, electrically driven probe; this was constructed by capping one end of a rigid steel wire (0·5 mm o.d.) with a short length of plastic tubing and then gluing the capped end of the wire to the diaphragm of a small ear-plug speaker. A Pasteur pipette was fitted over the wire and attached to the outer speaker casing so that the wire extended from the pipette by about 4 cm. Vaseline in the end of the pipette kept the probe-movement smooth and unidimensional. The speaker was driven by one cycle of 300 Hz sine wave from a Wavetek function generator, triggered by a WPI stimulator and amplified by a Heathkit AA-18 audioamplifier. Probe vibrations were monitored with a calibrated gramophone stylus.

RESULTS

Modulated vibration-sensitivity in intact animals

The responsiveness of Mauthner neurones to labyrinth vibration is high while an animal is at rest but decreases while the animal is swimming, as could be shown by using a fine monopolar electrode in the spinal canal (see Materials and Methods) to monitor spinal cord activity in the intact animal (Fig. 1). In the resting animal, Mth axon spikes were observed in response to light taps on the aquarium wall (Fig. 1A). When the animal began to swim the stimulus-locked Mth spike disappeared (Fig. 1B), despite an increased stimulus intensity. Note the spinal cord activity which accompanies swimming. The water level in the test chamber was lowered to a depth of 5 mm for this recording, so the animal remained in contact with the aquarium floor and the otic capsules experienced a roughly constant stimulus whether the animal was swimming or at rest. The floor was covered with a smooth Plexiglas sheet to make swimming easier and the area of the chamber was reduced with a removable barrier.

As an alternative way of achieving constant stimulation, we implanted electrodes in an otic capsule of one larva for direct electrical stimulation of the labyrinth. Again, the short-latency, stimulus-locked spike of the resting animal (Fig. 1C) disappeared while the animal was swimming (Fig. 1D). This unit was identical in amplitude and waveform to the vibration-evoked Mth spike (inset).

Effect of arousal on vibration-evoked synaptic potentials

Arousal reduced Mth vibration-sensitivity in semi-intact preparations, as shown by intracellular recordings from a Mth soma (Fig. 2). The criterion we used to define 'arousal' was the onset of intense spinal cord activity, as seen in the figure. Recordings made simultaneously from two distant sites on the spinal cord showed

that the bulk of this activity originated from long, descending axons (not shown). Note in Fig. 2A that periodic EMG activity from trunk musculature accompanied the spinal discharge. Constant-amplitude vibration pulses (0·75 Hz) were applied to an otic capsule while recording from the ipsilateral Mth cell body. The vibration-evoked startle response, which usually follows the first few such stimuli, had habituated by the time these recordings were made. Vibration-evoked action potentials ceased immediately during the transition from rest to arousal (Fig. 2A), but resumed with the return to rest (Fig. 2B). During arousal, there was a small, maintained depolarization of the Mth soma and a marked decrease in the amplitude of vibration-evoked EPSPs. EPSPs failed to reach threshold for an action potential during arousal, except where a transient slowing of descending spinal cord activity

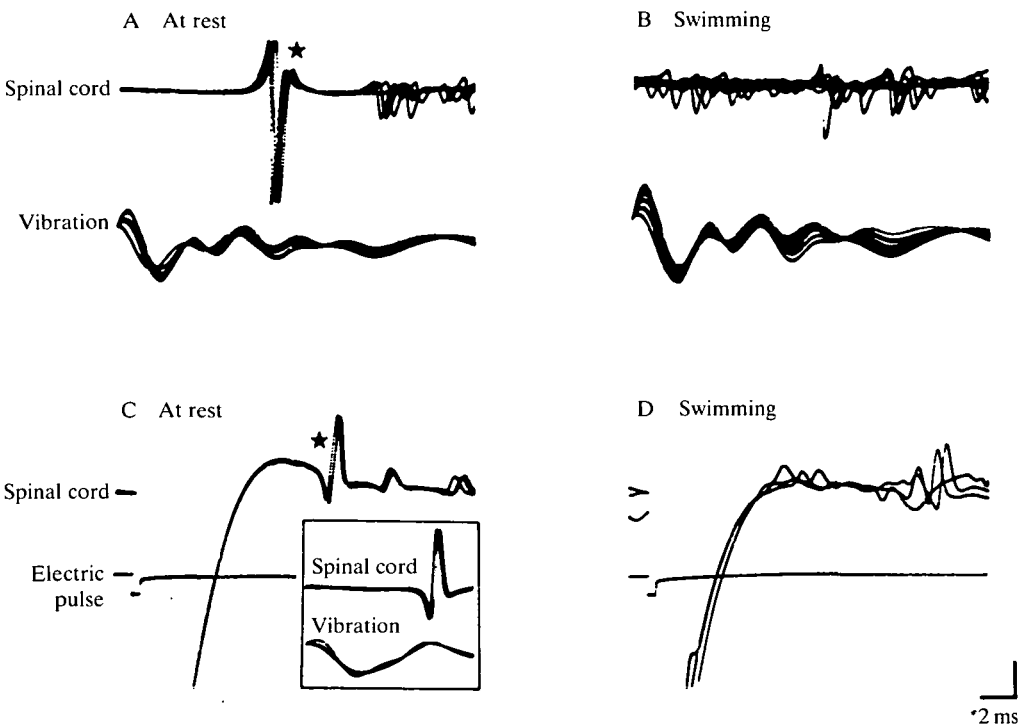

Fig. 1. Modulated Mauthner sensitivity to vibration (A,B) and electrical stimulation (C,D) of an otic capsule in freely behaving animals. Spinal cord activity (upper traces) was recorded by a fine wire electrode in the spinal canal. Mauthner axon spikes are indicated by stars. The animal was observed in a shallow aquarium containing 5 mm of lamprey saline. (A) Mauthner spike resulting from light taps to the aquarium wall while animal was at rest. (B) Vibration-evoked Mauthner spike is lost during swimming. Lower traces, vibration monitor. In C and D, fine wire electrodes were implanted in an otic capsule, glued in place, and used to stimulate the labyrinth electrically. (C) Spinal cord responses to electrical stimuli while animal was at rest. Inset, an identical unit to that in C could be elicited by vibration. (D) Responses obtained while animal was swimming. Lower traces, voltage applied to stimulating electrodes. Five superimposed responses are shown in A,B and three in C,D, each elicited at 2- to 3-s intervals. Vertical calibrations: upper traces (A–D, inset) 0·2 mV, bottom traces (A,B) 7 μm, (C,D) 5 V, inset, 35 μm.

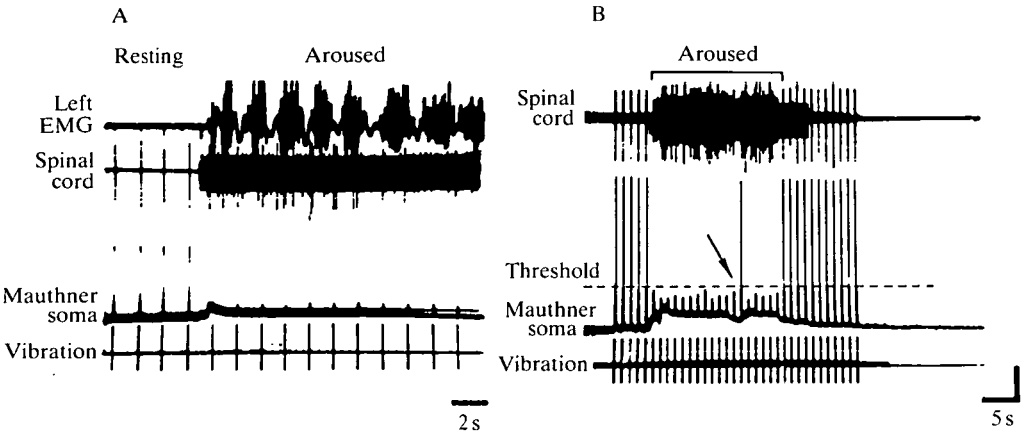

Fig. 2. Loss of vibration-evoked Mauthner spikes during 'arousal' in a semi-intact preparation. Arousal was defined as periods of intense, descending spinal cord activity. A and B show two different sequences from the same animal. Left EMG, electromyogram from left-side of trunk; Mauthner soma, intracellular recording from a Mauthner cell body. Vibration pulses were delivered to the ipsilateral otic capsule. Arousal was induced by tactile stimulation of the oral hood in A and by otic capsule vibration in B. A drop-off in spinal cord activity and partial repolarization of the Mauthner cell was accompanied by an increased PSP amplitude, triggering an action potential (arrow). Vertical calibration: EMG (A), 0·2 mV; spinal cord (A,B), 0·1 mV; Mauthner soma, (A), 50 mV, (B), 20 mV; vibration, (A,B), uncalibrated.

and repolarization of the Mth cell was accompanied by a briefly increased PSP height (arrow). In this and other recordings, the extent of PSP depression was correlated with the intensity of descending spinal cord activity and the amount of Mth depolarization.

The correlation of PSP height and descending spinal cord activity can be seen more clearly in Fig. 3. Arousal was induced by stroking the oral hood with a wooden probe (arrow). Vibratory pulses were delivered at 2·5 Hz, while recording from the ipsilateral Mth cell body. PSP height decreased dramatically on the transition from rest to arousal; however, note the transient PSP increase which occurred during a pause in spinal activity (star). The Mth cell also exhibited some repolarization during this pause.

Müller cells also depolarized during periods of arousal. The most strongly depolarized were the B_1 cells, which fired tonically during arousal and contributed to descending spinal cord activity. Nonetheless, only the Mth cells exhibited an arousal-associated PSP depression. Müller cell PSPs were either unaffected or slightly enhanced during arousal. The Mth resting membrane potential was depolarized by 3–5 mV during arousal and the amplitude of vibration-evoked PSPs was markedly diminished (Fig. 4A,B). (These recordings were obtained with KCl electrodes, but the same depolarization was observed when potassium acetate electrodes were used.) Vibration-evoked EPSPs in the contralateral M_3-Müller cell were actually slightly larger during arousal (Fig. 4C,D).

Increased membrane conductance accompanies Mauthner PSP depression (MPD)

To test for a postsynaptic conductance increase during MPD, we impaled Mth somata with double-barrelled microelectrodes. One barrel passed hyperpolarizing, constant-current pulses (−4·8 nA) while the other recorded Mth membrane potential. Small, vibratory pulses to the ipsilateral otic capsule (approx. 1 μm peak-to-peak) were alternated every 300 ms with current pulses to the cell. The change in membrane potential caused by current pulses was used as a measure of membrane conductance. Conductance values and PSP amplitudes were both normalized relative to their respective resting averages and plotted against time (Fig. 5). PSP depression during arousal was accompanied by a small, but significant, increase in Mth membrane conductance. PSP height changed from a mean of 8·9 mV at rest ($N = 7$, S.D. = 0·50) to a mean of 3·2 mV during arousal ($N = 25$, S.D. = 0·79), a 65% decrease. Membrane conductance changed from a mean of 610 nS (nanosiemens) at rest ($N = 8$, S.D. = 15·3) to a mean of 696 nS during arousal ($N = 25$, S.D. = 31·3), an increase of 14·1%. The changes in PSP height and membrane conductance were both statistically significant at $P < 0·001$, using Student's t-test.

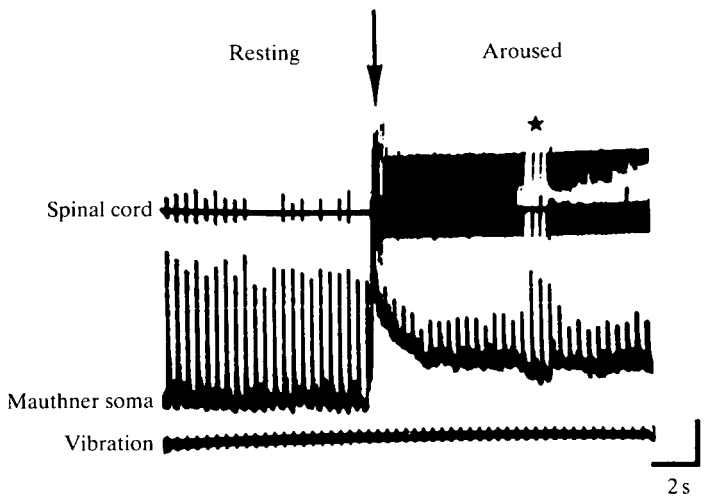

Fig. 3. The degree of Mauthner PSP depression during arousal is correlated with the intensity of firing in descending spinal axons. Upper trace, spinal cord activity; middle trace, intracellular recording from a Mauthner soma; bottom trace, vibration monitor. Vibration-evoked Mauthner PSPs were subthreshold for spike activation; the small spinal cord units which sometimes accompanied these PSPs reflect the activity of other vibration-sensitive neurones. Arousal was induced by tactile stimulation of the oral hood (arrow), which elicited a burst of several Mauthner spikes. The Mauthner soma exhibited a maintained depolarization of 2–5 mV during the ensuing period of arousal. A brief pause in spinal cord activity (star) was accompanied by a small repolarization of Mauthner membrane potential and an increase in PSP amplitude. Vertical calibrations: upper trace, 0·5 mV; middle trace, 5 mV; bottom trace, 7 μm.

DISCUSSION

We found that the vibration-sensitivity of larval lamprey Mauthner cells is modulated by behavioural state; intact animals are most sensitive to vibration while quiescent, and less so while they are swimming (Fig. 1). We noted a similar decrease in vibration-sensitivity in semi-intact preparations during periods of arousal, while recording intracellularly from Mth somata (Fig. 2). We defined 'arousal' in this study as periods of intense, descending spinal cord activity.

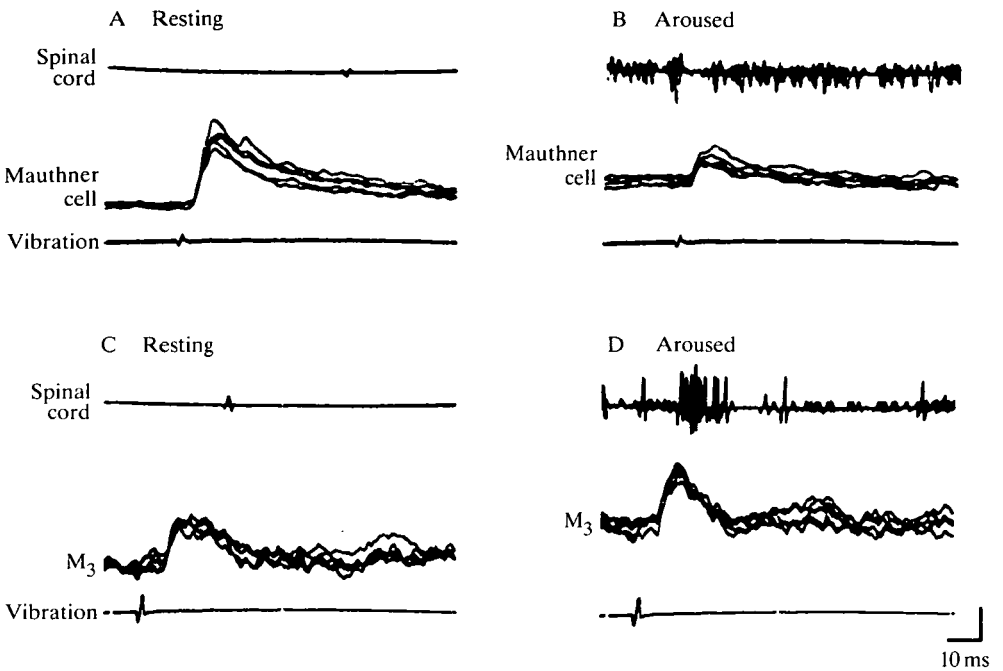

Fig. 4. The vibration-evoked EPSP of Mauthner cells decreases in amplitude during arousal (A,B), but that of the contralateral M_3 cell (and other Müller neurones) does not (C,D). Recordings obtained from a curarized preparation. A small depolarization accompanies arousal in both cells. Mauthner cells were the only reticulospinal cells which exhibited depressed PSPs during arousal. Top traces, spinal cord activity; middle traces, intracellular PSP recordings; bottom traces, vibration monitor. Vertical calibrations: top traces, 0·1 mV; middle traces (A,B), 5 mV, (C,D), 2 mV; bottom traces, 7 μm.

Fig. 5. Mauthner (Mth) cells exhibit an increased membrane conductance during arousal which accompanies PSP depression. (A) Experimental design. (B) Data from an experiment in which vibration-evoked EPSPs were rapidly alternated with hyperpolarizing current pulses in a Mth neurone. Current pulses were delivered through one side of a double-barrelled microelectrode while recording from the other barrel. The amount of hyperpolarization caused by each current pulse was used as a measure of membrane conductance. Vibratory pulses to the ipsilateral otic capsule were alternated with current pulses every 300 ms during a transition from rest to arousal in a curarized preparation. Conductance values and PSP amplitudes were normalized relative to their respective resting averages. During arousal, PSP amplitudes decreased by an average of 65·0 % and membrane conductance increased by 14·1 %.

Vibration-evoked PSPs were strongly depressed in Mth cells during arousal, but were unaffected or even enhanced in vibration-sensitive Müller neurones (Fig. 4). Thus, Mauthner PSP depression (MPD) appears to underlie the decreased

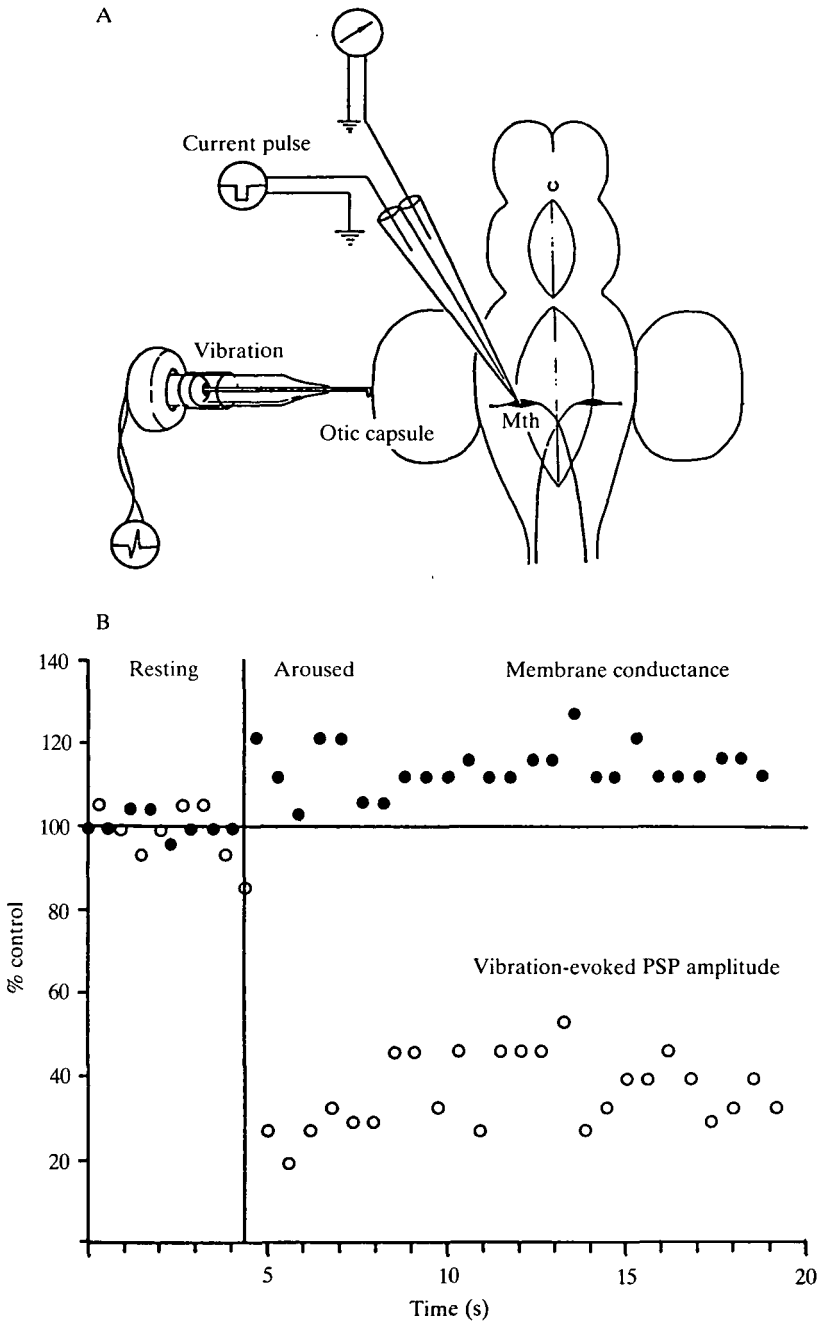

Fig. 5

vibration-sensitivity that accompanies arousal. Completely curarized larvae also exhibit MPD (Figs 3–5), indicating that the effect is not caused by movement-related feedback, but is central in origin.

The amount of MPD during arousal is closely correlated with the level of excitation in other descending brain stem neurones, as judged by the intensity of descending spinal cord activity (Fig. 3); hence both effects may be attributable to a common arousal-inducing network. All of the giant reticulospinal neurones from which we recorded depolarized during arousal. B_1 was the most strongly depolarized (10–15 mV) and fired tonically at up to 30 Hz, thus contributing to the spinal cord activity which defined arousal. Mauthner cells usually depolarized no more than 2–8 mV. What we have called 'arousal' in this study corresponds closely to what Wickelgren (1977*b*) described as 'synaptic afterdischarge', a long-lasting synaptic depolarization of Müller cells which could occur spontaneously or follow electrical stimulation of a cranial nerve. In the present study, we were able to elicit arousal by tactile stimulation of the skin, anywhere on the body, or by repeated vibration of an otic capsule.

MPD is caused, at least in part, by an increase in Mauthner cell membrane conductance. During arousal, we observed a 14·1 % increase in Mth cell membrane conductance (measured in the cell body) which occurred at the same time as a 65 % decrease in vibration-evoked PSP amplitude (Fig. 5). This relatively small conductance increase could reflect a larger, but localized conductance change in the Mth lateral dendrite. EPSPs generated at more distal dendritic sites would then be variably shunted prior to invading the Mth cell body. Diamond (1968) described such 'dendritic remote inhibition' in the goldfish Mth cell, although its occurrence there was unrelated to arousal.

Effects similar to the lamprey MPD have been described in other preparations. Command neurones for crayfish backward walking, for instance, have been shown to inhibit lateral giant tailflip circuitry (Edwards & Simon, 1985). Taylor (1970) described modulated sensitivity of another crayfish interneurone to water-borne vibrations, showing that vibration-sensitivity was strongly decreased during movement of the appendages, struggling and tailflips. In addition, a recent report described the 'switching-off' of interneuronal auditory responses during stridulation in a grasshopper (Wolf & von Helverson, 1986).

Few similar studies have been made to elucidate the effect of behavioural context on the excitability of Mauthner neurones. One case was provided by Mueller (1978, 1981), who described pre-stimulus 'set' conditions in goldfish, which affected the subsequent 'release' of startle behaviour. It has also been reported anecdotally that flatfish suppress their startle response while at rest on a substrate (Zottoli, 1981), possibly to maintain camouflage.

Modulated Mth sensitivity may function as a safety mechanism in larval lampreys by preventing self-activation of the startle circuit during swimming, burrowing and other vigorous activity. Lamprey Mth cells can be excited by weak vibrations or by light tactile stimulation of the oral hood (see Fig. 3), yet animals are apparently able to swim and burrow head first into sand without exhibiting self-triggered startle

behaviour. We have yet to determine the precise mechanism by which sensory input to the Mth cell is regulated. This provides a rich area of future study in lampreys, goldfish and other vertebrates which exhibit Mauthner-initiated startle behaviour.

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