J. Exp. Biol. (1971), **55**, 569–584
With 9 text-figures
Printed in Great Britain

CENTRAL CONTROL OF AUDITORY INPUT IN THE GOLDFISH

I. EFFECT OF SHOCKS TO THE MIDBRAIN

BY R. W. PIDDINGTON

Neurobiology Unit, Scripps Institution of Oceanography and Department of Neurosciences, University of California, San Diego, La Jolla, California

(*Received 29 March 1971*)

INTRODUCTION

Fish have been shown to be capable of real hearing (van Bergeijk, 1967) and to have a behavioural threshold comparable to that of man (0·001 Å water displacement, Enger, 1966; 10^{-16} W/cm^2 energy flux, Griffin, 1955). However, relative to mammalian or amphibian auditory systems, that of the fish is not a good pitch-analysing system (Kleerekoper & Chagnon, 1954; Enger, 1963) and, in contrast to the lateral-line and visual systems, is non-directional in the sense that it is unable to distinguish right and left symmetrical sources (van Bergeijk, 1964; von Frisch & Dijkgraaf, 1935). Some species communicate with sound (Tavolga, 1964), but the general biological significance of hearing in fish is poorly understood. Piddington (1971) makes a new suggestion on this problem, namely that a fish can tell whether another fish is approaching, receding or moving tangentially to it.

When an object moves through or vibrates in a fluid medium it creates both local hydrodynamic displacements and propagated pressure waves, designated by van Bergeijk (1967) as near-field and far-field sound respectively. The near-field falls off with distance more rapidly than the far-field. The lateral-line system is sensitive only to near-field displacements (Harris & van Bergeijk, 1962; van Bergeijk, 1964, 1967) but the ear (or sacculus) is sensitive to both fields (Enger, 1966, 1968). To stimulate only the ear but not the lateral line requires, therefore, a far-field (with minimum displacement) and this can be achieved by using a loudspeaker in air rather than underwater (Enger, 1966; van Bergeijk, 1967; cf. Harris & van Bergeijk, 1962; Enger, 1963; Groezinger, 1967; Page, 1970). This is the method adopted for the present study. Lateral-line units encountered in this study did not respond to clicks in air but auditory units did.

In the afferent auditory system the single units lock into either or both phases of a sine-wave stimulus (Enger, 1963, 1967; Furukawa, 1966; Furukawa & Ishii, 1967*a*, *b*; Groezinger, 1967; Page, 1970) but may also be influenced by static pressure (Furukawa & Ishii, 1967*b*; see further, Piddington, 1971).

Furukawa (1966) found that excitation of the giant Mauthner cell of the goldfish led to inhibition of single fibres in the auditory nerve, and postulated that efferent control fibres were involved (see Fig. 1). Descending or efferent control has been well documented in the mammalian auditory system (e.g. Galambos, 1956; Fex, 1968) and also recently in a great variety of other sensory systems including the

lateral line (Hasimoto, Katsuki & Yanagisawa, 1970), and olfactory systems in fish (Hara, 1967), the visual system in crustaceans (Aréchiga & Wiersma, 1969), frogs (Branston & Fleming, 1968), birds (Holden, 1968) and insects (Rowell & Horn, 1968), the auditory system in insects (McKay, 1970; Rowell & McKay, 1969), and in the vestibular system of the frog (Llinas & Precht, 1969).

The present experiments show that shocks to higher brain centres reduce or abolish the auditory input following a click. This evidence, with controls to eliminate antidromic or 'back-firing' effects, reveals the action of a descending control system which usually inhibits, but sometimes facilitates transmission along the afferent auditory pathway. Lack of any effect on the receptor potential or 'microphonic' shows that auditory control in fish is mediated neuronally and not mechanically.

This paper deals with the presence and properties of descending control. The following paper (Piddington, 1971) reveals the action of the control system in the electrode-implanted free-swimming animal.

METHODS

A total of 53 goldfish (*Carassius aruatus*) of 10–15 g wt were used in this study. The operation was performed with the animal under light anaesthesia (sodium pentothal 30 mg/kg, i.p., or tricaine methane-sulphonate, MS 222, 120 mg/l). Curare (30 mg/kg, i.p.) was used in conjunction with the pentothal as the latter was effective for only about 1 h. The results were essentially the same with either treatment.

The fish were held in a semi-stereotaxic holder in a freshwater bath that allowed continuous perfusion of the gills via a glass tube in the mouth.

Three modified 'alligator' clips, attached by adjustable arms to the experimental table, were used to clamp the dorsal, caudal and left pectoral fins, and a fourth clamped the mouth to the perfusion tube.

With the above holding method, only the operated area was kept above water level, and the swimbladder and skull were free to vibrate normally. The skull, though not rigidly clamped (the mouth is soft) was nevertheless stable enough to allow unit recordings for up to 3 h.

The operation consisted of scraping the skin from the skull, followed by opening of the skull with scalpel and jeweller's forceps, to reveal the cerebellum and midbrain. The gelatinous intracranial fluid was removed with absorbent tissue and replaced by a pool of saline. A silver wire for grounding was hooked to the anterior border of the skull opening and the electrodes inserted with Narishige micromanipulators.

The x and y axes of the recording manipulator were kept parallel to the antero-posterior and left–right lines respectively; the pitch of the fish was adjusted so that a line joining the top of the pupil to the dorsal tip of the opercular flap was parallel to the water surface, to which the manipulator had been previously aligned normal. For stereotaxic location of active loci the junction of cerebellum and midbrain at the midline was selected as an arbitrary x–y zero point; depth or z values were referred to the cerebellar surface 1 mm posterior to the x–y zero (see Fig. 1). The vernier scales of the micromanipulators were calibrated in 0·1 mm steps.

The laboratory was subject to normal ventilation and equipment noise. Clicks were generated by a 0·2 msec square pulse from a Grass SD 5 stimulator, amplified by a

Heathkit audio amplifier set at constant gain and delivered via a 4 in. loudspeaker located in the air 30 cm from the fish. The click intensity, checked with a hydrophone, was directly proportional to the voltage of the square pulse, and was expressed in arbitrary pressure units, equivalent to the voltage-dial setting.

Shocks to the midbrain were generated by a Grass S 8 stimulator isolated from ground. An additional S 8 stimulator allowed the formulation of any desired combination of shock and click trains. Stimulation and recording electrodes were either metal-filled microelectrodes of 20 μm tip-diameter or of 1 mil (25 μm) insulated stainless-steel wire (California Fine Wire Co.), chopped off square at the tip and held in a glass capillary tube. The simplest electrodes proved to be bipolar assemblies of two such

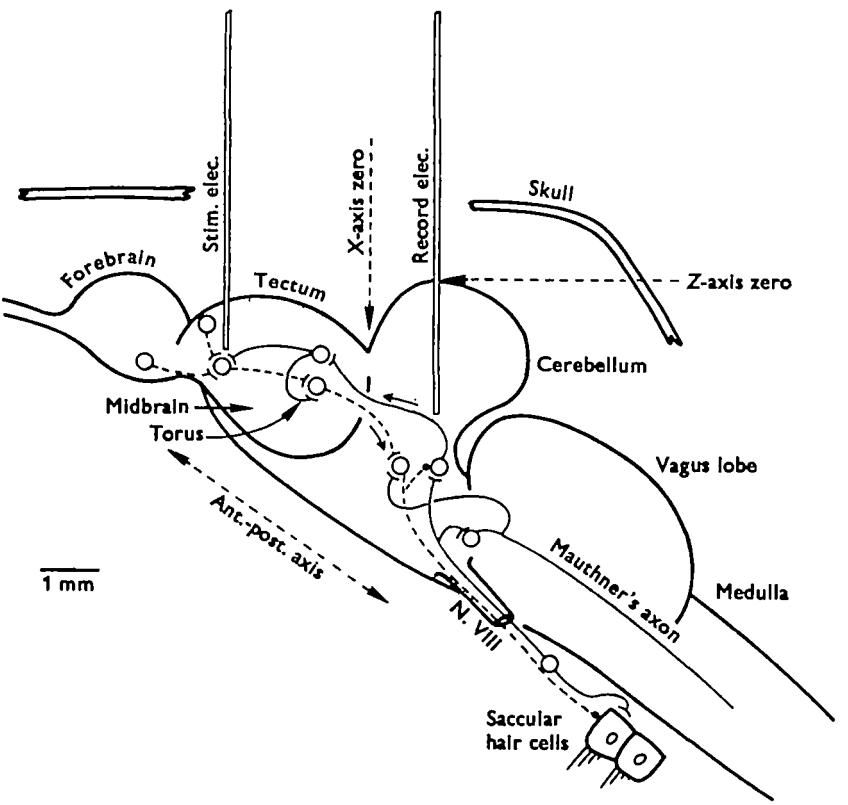

Fig. 1. Anatomical relationships of the ascending and descending auditory pathways. Ascending, solid lines; descending, broken lines. The ascending path to the torus semicircularis, the efferent endings on saccular hair cells, and the inhibitory pathway from Mauthner cell to the sacculus are known. Most of the descending system is inferred from the present experiments. Flat synapses are excitatory; round are inhibitory. The orientation of the brain is the same as it would appear when the animal is correctly positioned for an experiment.

wires twisted and glued together, the tips separated vertically by 2 mm. For best localization of effective midbrain structures (see Fig. 1) the stimulating wires were chopped off level with one another so that the stimulus was highly local. For preliminary localization of areas responding to auditory stimuli, glass micropipettes of 20 μm tip-diameter and filled with 3 M-NaCl were used. The strength and duration values for shocks and clicks were obtained directly from the stimulator dials, the

number of shocks in a train was checked with a monitor oscilloscope, and the delay from a given shock to the following click was measured from the end of the shock artefact to the onset of the click as measured with a Uher microphone at the level of the fish (no air delay).

Auditory potentials were amplified differentially and displayed on a dual-beam oscilloscope. An averaging digital computer was used in conjunction with an x–y plotter; the potential amplitudes were measured from the x–y plots or directly from the tube face of the computer. Direct Polaroid photography of the oscilloscope beam showed that such averaging did not distort the waveform of the response.

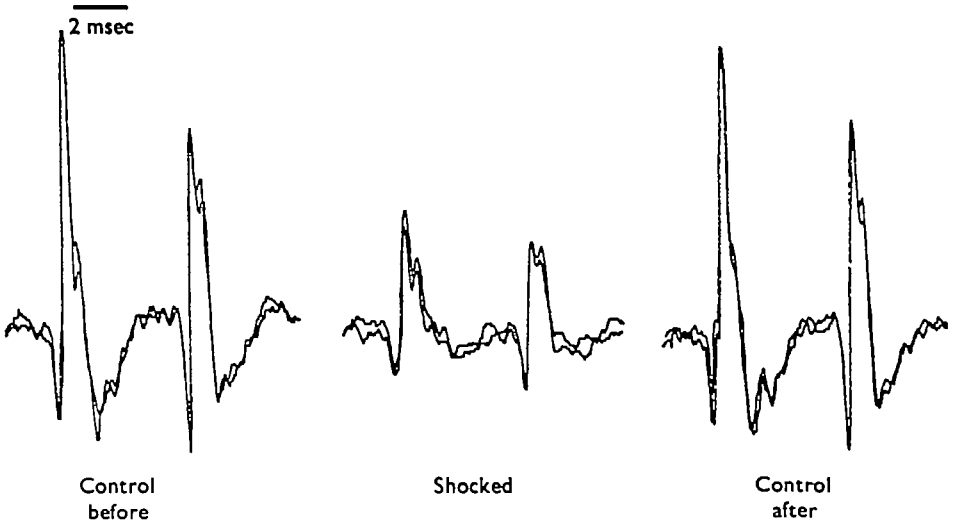

Fig. 2. Suppression of the click-evoked response in the medulla by shocks to the torus semicircularis. The left and right traces show the responses to a 5 msec pair of clicks in the absence of shocks. The centre trace demonstrates the suppressing effect of a previous train of shocks. Ten shocks at 200/sec, 12 V, 0·1 msec duration and 50 msec post-shock delay to first click. Each record shows two superimposed averages of eight trials at 1/sec. Note that the effect is reversible. Click amplitude for this and following figures is at saturation except where specified.

RESULTS

The basic finding of this study is that shocks to certain higher brain centres reduce or abolish the auditory response recorded at an early afferent level in the medulla. Fig. 2 shows that a train of shocks delivered to the torus semicircularis of the midbrain produces a reversible suppression of the medullary response to a pair of closely spaced clicks. The centre trace of Fig. 2 shows that both responses to the click pair following the shocks are significantly reduced relative to their control size in the absence of shocks. Each trace consists of two superimposed averages of eight trials written one over the other, and the reproducibility of the waveforms is readily appreciated.

Fig. 3 (*a*) shows the same result in a slightly different way. Six shocks to the anterior midbrain are now delivered during the interval between two widely spaced clicks and it is apparent that the response to the second click is significantly reduced. Fig. 4 shows that in the absence of shocks the second click of a pair produces a full-sized evoked response if the interval between the clicks is greater than 20 msec. The clicks

in Fig. 3 are separated by 30 msec, and (as Fig. 3*b* shows) when the shock voltage is reduced below a certain critical value, the reduction in amplitude of the second response does not occur. The observed suppression effect is thus caused by the shocks and not by the presence of the first click.

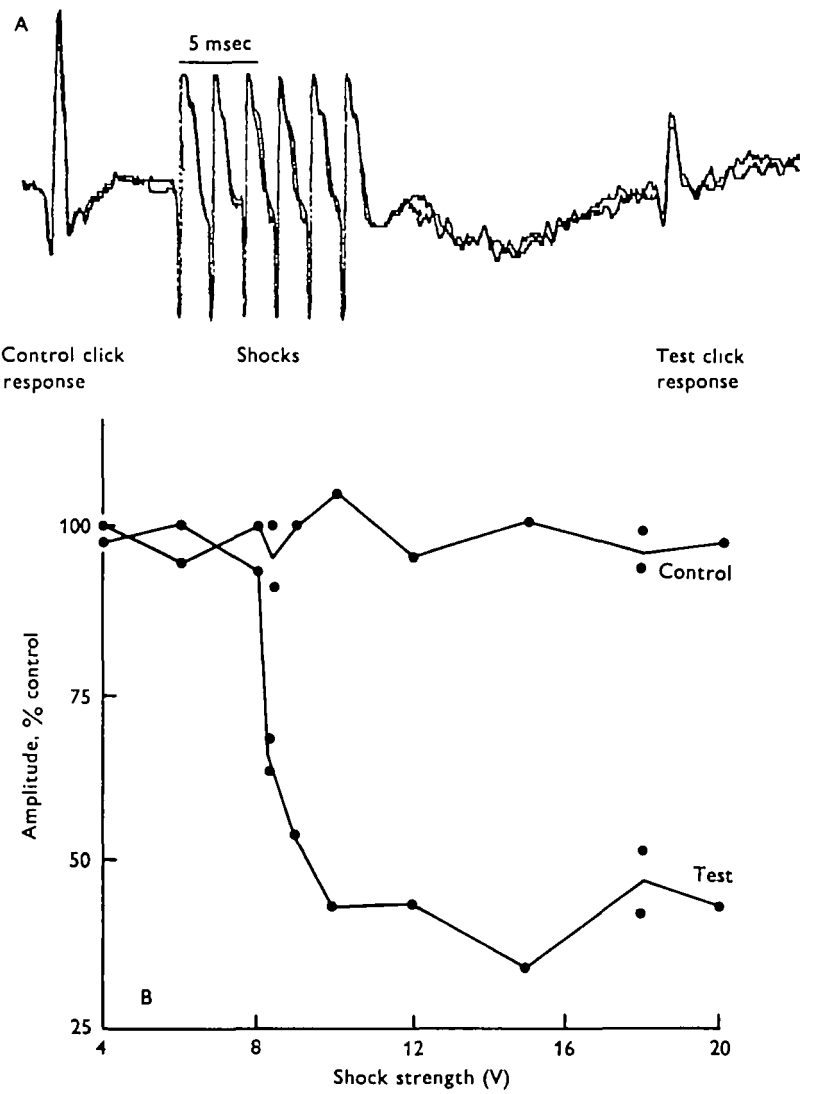

Fig. 3. Suppression of the click response at the medulla by shocks to anterior midbrain.

A. Two clicks separated by 30 msec with six shocks at 500/sec (10 V, 0·1 msec) inserted between them. The second click response is suppressed with respect to the first, and (see B) with respect to the second without shocks. Each record shows two superimposed averages of eight trials at 1/sec.

B. Dependence on shock strength. Control, first click response; test, second response (after shocks). Each point is eight trials at 1/sec averaged, 30 msec between clicks; five shocks at 200/sec, 1·0 msec duration; delay, 15 msec from last shock to test click. The following is true for all figures unless specified: shocks are to anterior midbrain, all data points are averages of eight trials at 1/sec repetition, the same basic relationship of clicks to shocks as Fig. 3(*a*) is used, and amplitude is percentage of control response.

By various experimental manipulations it is possible to show that this result is due to the action of a descending inhibitory pathway, and not due to antidromic activity of the afferent auditory fibres. The constancy of the saccular microphonic during the shock treatment excludes muscular control and a variety of unwanted noise masking effects.

These results will now be considered in detail.

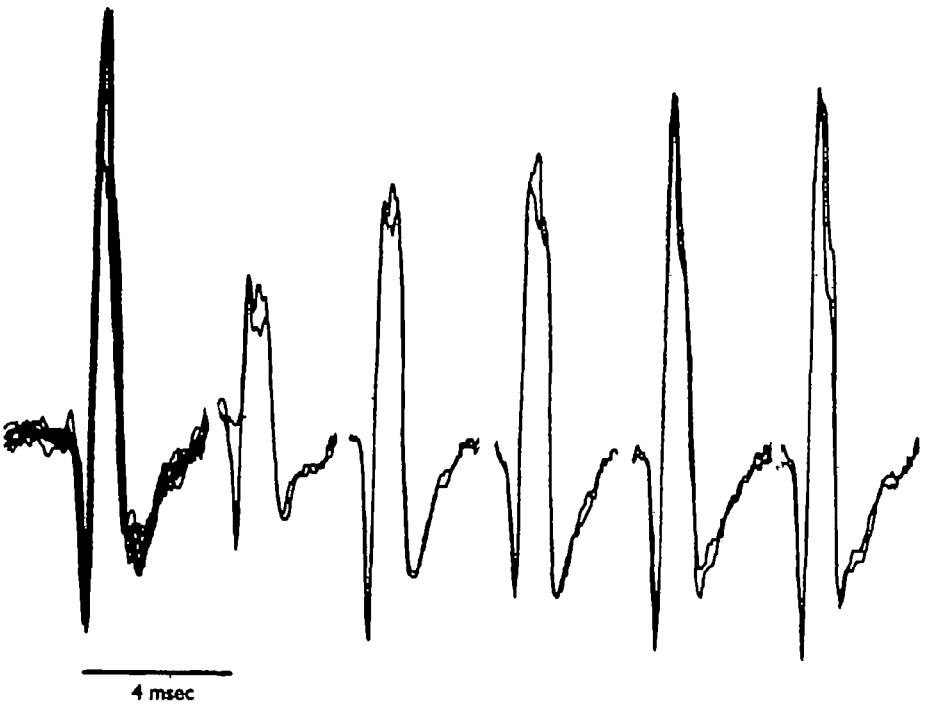

Fig. 4. Effect of interval between two clicks on amplitude of second response, no shocks. Recovery is complete by 20 msec. Eight trials at 1/sec averaged per sweep. All ten averaged responses to first click are superimposed at left, all responses to second click are duplicated (as in Figs. 2, 3*a*).

Recording loci and the type of response

Recordings of auditory evoked activity were obtained from two areas: medulla and torus semicircularis of the midbrain (Fig. 1). Most of the recordings in the medulla were made at the midline so as to simplify localization; that is, by setting y to zero (co-ordinates were x = 1 mm posterior, y = 0, z = 3·5 mm).

The response to a click was typically a sharp compound action potential of amplitude 0·1–0·5 mV, latency 1–3 msec and width of negative spike 0·5–2 msec (Fig. 2). The recordings, except for those with briefest latency, were probably from decussating second-order fibres originating in the auditory nuclei of the medulla and ascending to the torus semicircularis of the midbrain (Cajal, 1908; Pearson, 1936). The region was about 0·5 mm in diameter in the sagittal plane.

It was important to show that the responses were true auditory responses and not lateral-line responses. Units and massed activity, which were probably from the anterior lateral-line nerve, were consistently found about 1 mm dorsal to the auditory area. Such units responded to small water displacements close to the head on one

side only and showed no response to sounds except when the loudspeaker was at maximum intensity and with the base directly contacting the experimental table. By contrast, auditory activity was evoked by human voices and low-pitched whistles, and was not sensitive to small water displacements (0·01 g drops of water from 5–10 cm height were effective but the response showed no directionality). When sounds from the loudspeaker were used, the response was unaffected by whether the loudspeaker base rested on the table or not.

These distinctions between lateral-line and auditory responses are in accord with known properties of both systems (Harris & van Bergeijk, 1962; van Bergeijk, 1964; Enger, 1966).

Evidence for descending inhibitory action

Four regions were found which when shocked produced suppression of the auditory potential at the medulla: (*a*) torus semicircularis of midbrain, (*b*) medulla, close to the recording electrode but not amidst auditory fibres, (*c*) optic tectum, 2 mm anterior to the torus, and (*d*) midbrain, 1·6–1·8 mm below (*c*). Two regions were ineffective: the forebrain and the cerebellum (see Fig. 1).

The first region tried was the torus semicircularis – the assumption being that there were inhibitory fibres from the torus which fed back to the medulla or to the sacculus itself and that these fibres could be directly activated at the torus (see Fig. 1).

The torus was located with a metal electrode by searching for a click-evoked response in the posterior midbrain (see Page, 1970; Groezinger, 1967); shocks were applied via the same electrode and were found to reversibly suppress click-evoked activity at the medulla (Fig. 2). At the end of the experiment a recording was made at the torus, and the evoked response was still present.

The suppressing effect was reversibly abolished if the shocking electrodes were advanced or withdrawn by less than 0·5 mm to loci which showed no click-evoked response. The ineffective regions which were below the torus were actually closer to the medullary recording site. This control eliminates the possibility that direct current spread to the medulla was responsible for response suppression.

The physiological controls necessary to establish true descending inhibition were designed to eliminate the following alternatives: (*a*) shock-induced or voluntary muscular action, causing either masking or direct attenuation of the response, and (*b*) antidromic activation that would either cause critical depolarization of the afferents or activation of collateral inhibitory circuits in the medulla.

(*a*) Muscular action on the mechanical pathway was eliminated by showing that shocks to the midbrain had no effect on the saccular microphonic (see also Piddington, 1971).

(*b*) Antidromic activation was unlikely to have been important because suppression equivalent to that obtained from the torus was also obtained from two areas that were remote from the torus by 2 mm to the anterior. These two areas, anterior optic tectum and midbrain directly below, (1) had no auditory evoked potential, (2) were critically localizable to within 0·2 mm, (3) were separated from the torus and from each other by non-effective areas, (4) gave only a slow-wave response at the medulla but no spike-like response, and (5) showed a latency of 5 msec from shock to suppression in the medulla (Fig. 7*a*). None of these five findings is consistent with antidromic action.

From the above considerations it is evident that the phenomenon described is specific to certain restricted brain regions, rather than being widespread or general, and that it does not depend for its action on the reception of afferent input at the effective shock loci. The results are consistent with the hypothesis that shocks to certain higher brain centres activate a descending pathway that inhibits the click-evoked potential at the medulla by action at the medullary nuclei or at the sacculus itself (see Hama, 1969).

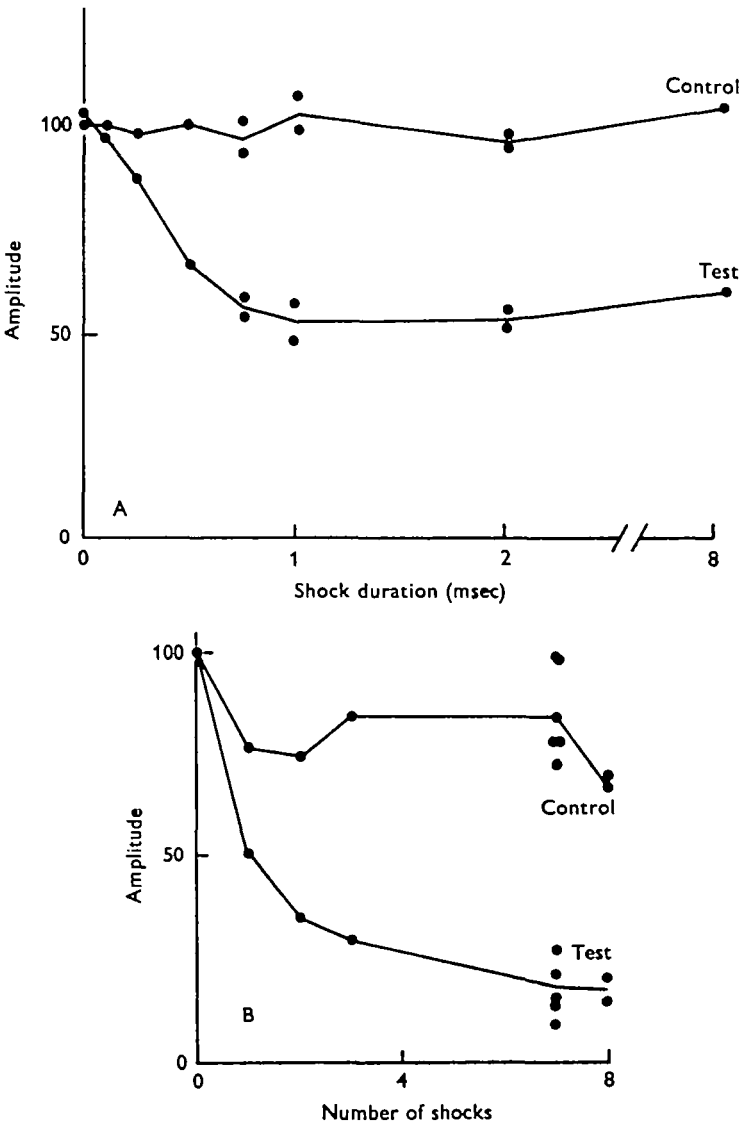

Fig. 5. A. Effect of shock duration on suppression of medullary click response. Optimal duration is 1 msec. Top line, control response, before shocks; bottom line, test response, after shocks. Click separation 60 msec; three shocks at 100/sec and 12 V, 27 msec constant delay from onset of last shock to test click.

B. Effect of number of shocks at fixed shock frequency. About seven shocks are optimal. Shocks at 400/sec, 12 V, 1 msec duration; delay from last shock to test click, 15 msec.

In general, the results were highly repeatable from animal to animal. All correct localizations of the torus (by recording the evoked potential) gave positive shock results and, for the anterior midbrain, the results became completely repeatable after the initial partially successful attempts to find the active locus. The main differences between animals were quantitative and were probably dependent on the precise position of the electrodes (see also Piddington, 1971).

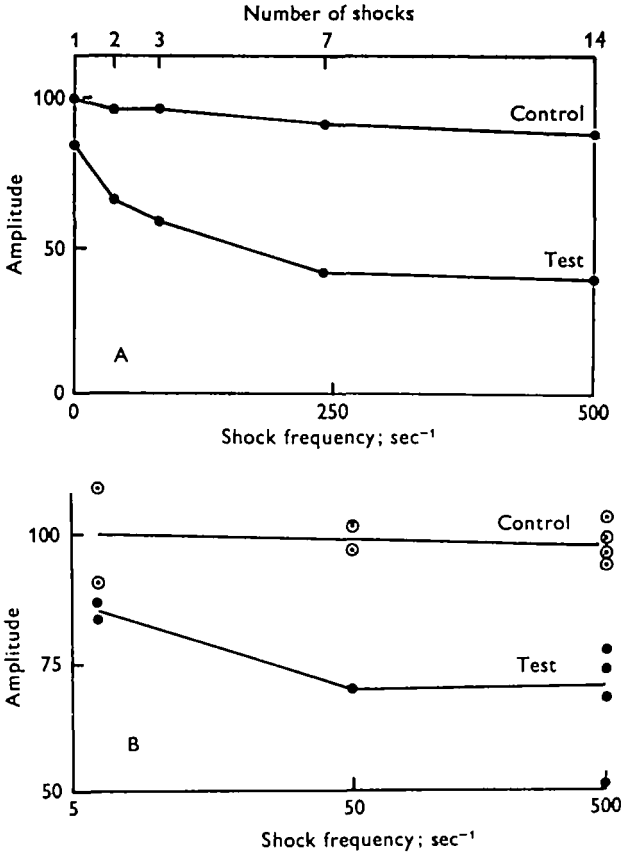

Fig. 6. A. Effect of shock frequency and number at fixed train duration of 30 msec. Seven shocks at 250/sec are optimal. 12 V shocks, 1·0 msec duration, post-shock delay to test click, 15 msec.
B. Effect of frequency at fixed number. 50/sec is the optimal frequency, and at 5/sec the effect is equivalent to a single shock (no summation). Four shocks at 12 V and 0·1 msec duration. Delay from last shock to click, 15 msec.

Dependence of the phenomenon on parameters of shock and click

The anterior midbrain region was used for this study because it gave more pronounced inhibition than the tectum and because it was free of the antidromic objection. For the study of each particular parameter all other parameters were held constant at their respective optimum values.

A. Dependence on shock parameters

The optimal shock settings for maximal suppression were as follows: shock voltage = 10 V (Fig. 3); shock duration = 1 msec (Fig. 5*a*); number of shocks = 7 (Figs. 5*b*, 6*a*); frequency of shocks = 50–200/sec (Figs. 6*a*, *b*); delay from last shock to click = 5 msec (Fig. 7).

The parameters voltage, duration, frequency and number all gave curves exhibiting a certain threshold and saturation level (Figs. 3, 5*a*, *b*, 6*a*). It is notable that a single

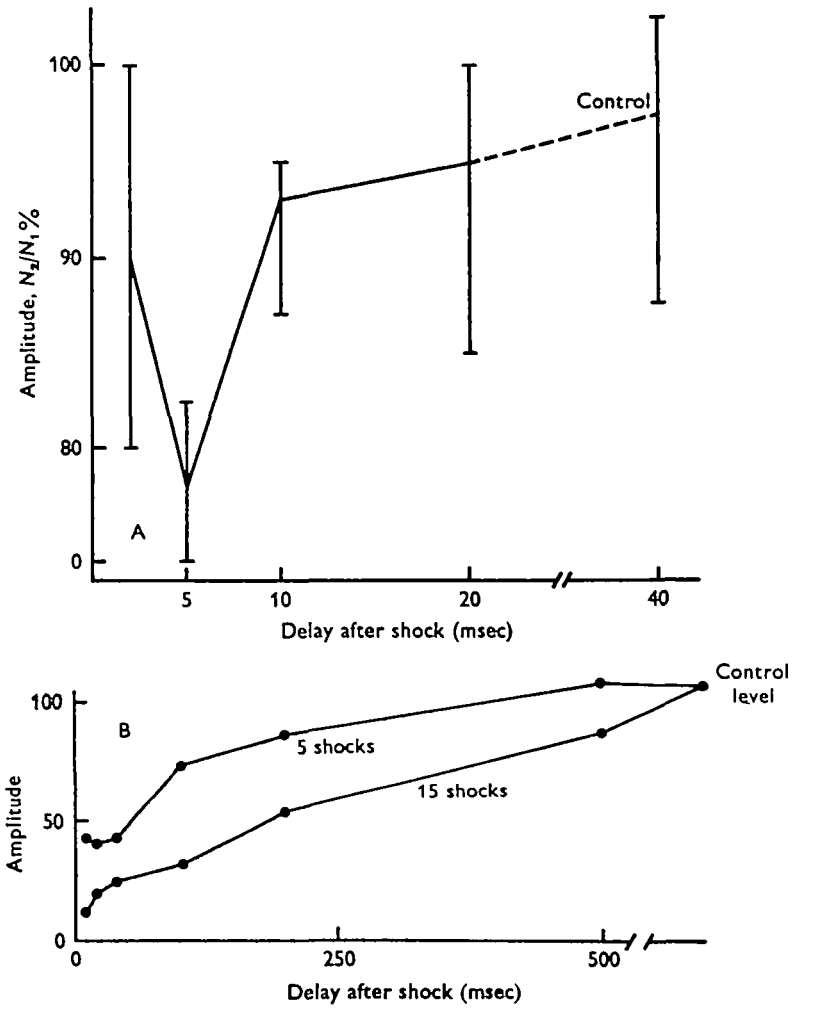

Fig. 7. Time course of inhibitory action.

A. Latency of inhibitory action at medulla following a single shock to anterior midbrain. Inhibition is maximal at 5 msec. The clicks are separated by 40 msec; delay is from last shock to click. Shocks 12 V, 0·1 msec duration. The vertical bars are the ranges for seven separate readings each 32 trials averaged; the curve connects the means. N_1 response to first click, N_2 response to second click; control is no shock.

B. Recovery from shocks; response amplitude as a function of delay from last shock to test click. Fifteen shocks suppress input for more than 500 msec. Top line, recovery following five shocks; bottom, 15 shocks at 200/ sec, 12V, 0·1 msec duration. Control gives amplitude in absence of shocks.

brief shock produced a detectable suppression that was maximal at 5 msec delay but which had decayed by 15–20 msec (Fig. 7*a*). Trains of shocks summated to give more pronounced and more prolonged suppression (Figs. 5*b*, 6*a*, 7*b*); 15 shocks at 200/sec gave suppression that lasted beyond 500 msec (Fig. 7*b*).

B. Dependence on click intensity

In the absence of shocks the amplitude of the evoked potential at the medulla increased continuously with increasing click intensity up to a saturation point (which

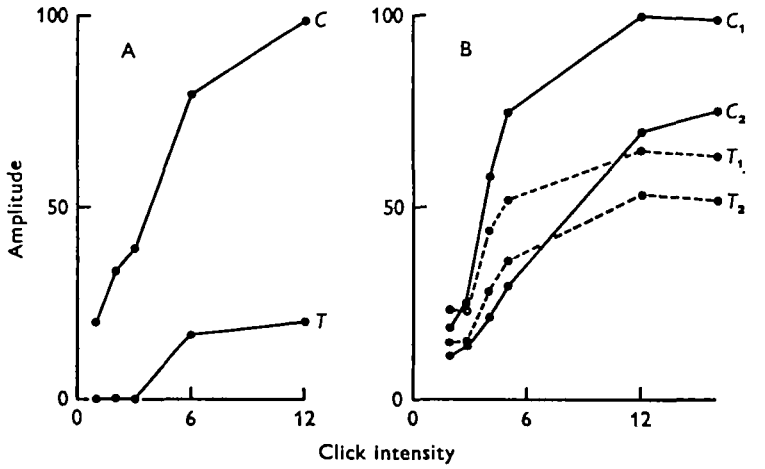

Fig. 8. Dependence of suppression effect on click intensity. In A, the test response is totally abolished at low click intensities, but in B it is enhanced. Click intensity, pressure units (see methods). A, eight shocks at 200/sec, 12 V, 1·0 msec; delay from last shock to test click, 15 msec; *C*, control; *T*, test. B, 14 shocks at 200/sec, 12 V, 0·1 msec duration. T_1 response to first click, T_2 second of a 5 msec click pair that follows the shocks by 10 msec; C_1, C_2 control responses to click pair, no shocks. Two independent averages of eight trials (at 1/sec) averaged per point. Different animal from A.

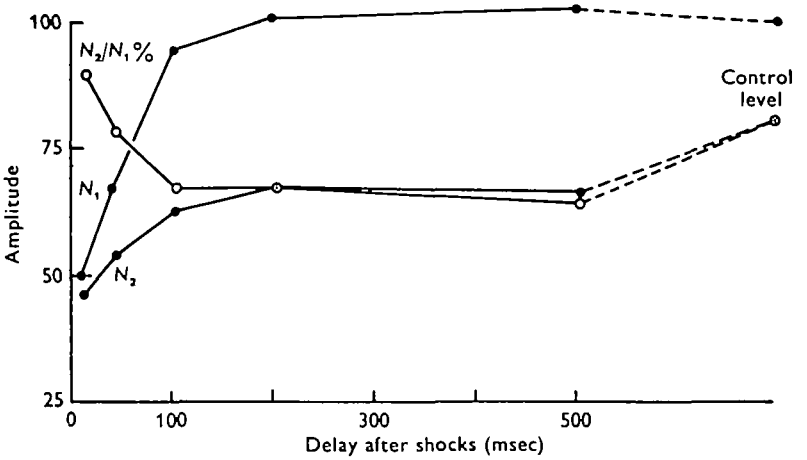

Fig. 9. Amplitude of both responses, N_1, N_2, to a 5 msec click pair as a function of delay from the last shock of a train to the first click of the pair. Fifteen shocks at 200/sec, 12 V, 0·1 msec duration. Each point 16 trials averaged; control values (no shocks), average of three separate readings. At short post-shock delays N_1 is more strongly inhibited than N_2 yet the opposite is true at longer delays.

roughly matched the saturation of the saccular microphonic (Piddington, 1971)). However, with shocks, all preparations exhibited inhibition at high click intensity (that is, at saturation) (Fig. 8). Over the entire intensity range the effect varied from preparation to preparation, often showed complete suppression for weak clicks (Fig. 8*a*), but occasionally a facilitation instead (Fig. 8*b*).

C. Effect on the two-click recovery cycle

In the absence of shocks the second response to a pair of clicks showed complete recovery from the first by 20 msec, but not by 5 msec (Fig. 2; see also Piddington, 1971, fig. 6). With a shock train preceding a 5 msec click pair, the recovery value at this interval was enhanced, provided that the click pair was delivered less than 40 msec after the shocks, but was depressed if delivered after 40 msec (Fig. 9). The depression of the second-click response lasted for more than 500 msec yet the first click response had returned to the control value by 200 msec.

DISCUSSION

The results showed that shocks to the midbrain reversibly reduced or abolished the click-evoked potential at the medulla, that a descending inhibitory pathway was probably responsible and that anti-dromic activation of afferents was not important. It was also shown that clicks in air did not activate the lateral line (see also Enger, 1968).

This paper and the next (Piddington, 1971) are the first demonstrations of central control in the auditory system of a teleost and are in harmony with the recent findings on efferent control in the lateral line (Hashimoto *et al.* 1970), with central control in the vertebrate auditory system (Fex, 1968; Grinnell, 1969), in the insect auditory system (McKay, 1970), and with the general concept of central control of sensory transmission (Livingston, 1959).

The results, which show a peak of inhibition 5 msec after a single shock, suggest that the system is prompt enough to be used in fast reflex suppression, such as following certain brief sounds (Fig. 7*a*), and such feedback action (10 msec latency) can indeed be demonstrated in the free-swimming animal (Piddington, 1971). The long-lasting additive effect of 7–15 shocks (temporal and spatial summation) suggests that the system could work tonically as well as phasically (Figs. 5*b*, 6*a*, 7*b*), and one could predict that, in principle, total inhibition of input could be achieved at the modest rate of only 50 impulses/sec per inhibitory fibre (Results, shock frequency). Habituation to monotonous or irrelevant sounds is a likely function of tonic descending inhibition (Piddington, 1971) and if the animal could direct or channel the inhibition on to or across specific afferent fibres, then in principle the system could work as an attention device (see Hernandez-Peon *et al.* 1961; Buño *et al.* 1966; Piddington, 1971).

All animals studied showed inhibition at high click intensities (Fig. 8) and such inhibition, if directed at the high-threshold fibres, would account for the habituation of such fibres to repeated sounds (see Piddington, 1971). Low-threshold fibres do not habituate and so the present inhibition of these fibres by shocks (Fig. 8*a*) probably relates more to the rapid feedback system (see Piddington, 1971).

How powerful is the inhibitory action, and is inhibition the only kind of descending influence? In none of the preparations was inhibition complete over the entire dynamic range of the sacculus. We cannot conclude, however, that inhibition is therefore weak, because of two factors: (*a*) shocks were applied locally at one side only, whereas the recordings (at the midline) were potentially supplied by both ears; (*b*) examples of 'weak inhibition' could in fact have been the resultant of strong inhibition and (weaker) facilitation. Two animals did show a net facilitation of a component of the evoked potential – one example was seen only at low intensity (Fig. 8*b*) and the other only at high intensity. In the free-swimming animal (Piddington, 1971), inhibitory feedback following a click appears capable of entirely eliminating the afferent response, although it probably operates on the average at less than full strength.

The results do not distinguish between action at the sacculus versus action at the medulla. Efferent endings on saccular hair cells have been described (Hama, 1969), but if these were responsible then the microphonic might have changed in amplitude (see Galambos, 1956; Fex, 1968), yet this did not occur. However, we cannot exclude direct action on the hair cells on this ground alone because (*a*) the geometry and circuitry is not understood, and (*b*) there is separate evidence that efferent inhibition can act on primary fibres (Furukawa, 1966; Piddington, 1971, fig 1*a*). Hama (1969) did not report efferent synapses on the dendrites of the afferent auditory fibres.

Efferent control has been described for several systems which use hair cells as transducers; these include auditory, vestibular and lateral-line systems (e.g. Fex, 1968; Llinas & Precht, 1969; Grinnell, 1969; Hashimoto *et al.* 1970), and to date there are few exceptions. It is likely therefore that the endings on hair cells described by Hama (1969) are indeed efferent and that the cells of origin lie somewhere in the medulla (see Furukawa, 1966) or posterior midbrain (see Fig. 1). If so, this system could be somewhat analogous if not homologous to the cochlear efferent system in mammals (olivocochlear bundle, OCB).

In mammals the cochlear efferents may be activated by a complex chain of neurones that starts in the cortex and projects downwards (Galambos, 1954; Fex, 1962; Dewson, 1968). Shocks to the anterior midbrain of the fish may be activating the efferents via an analogous chain of neurones (Fig. 1), whereas a direct connexion to the sacculus is less likely to be present. (Feedback from the Mauthner cell to the sacculus (Furukawa, 1966) is unlikely to relay through the anterior midbrain.) There are probably additional connexions from the optic tectum to the descending chain (see Results) and also from other important centres such as the forebrain (see Ingle, 1968). (The lack of inhibitory effect from shocking the forebrain could simply mean that anaesthetic or curare blocks the connecting pathway.) The results showing the additive effect of several shocks (Figs. 5*b*, 6*a*) and the effect of changing their frequency (Fig. 6) may pertain to the summating properties of the synaptic links in this chain of neurones or to summation in the final inhibitory synapse. The slow recovery from inhibition following 15 or more shocks (Figs. 7*b*, 9) indicates that accumulation of inhibitory transmitter does occur.

Although inhibitory effects could be explained by an efferent pathway, the facilitatory effects reported here and in the free animal (Piddington, 1971) may depend on more central feedback loops which in the mammal mediate both inhibition and facilitation in the medulla (Whitfield, 1968).

One important remark can be made about auditory control in fish as contrasted to mammals. Structures equivalent to the middle-ear muscles have never been described for fish nor is there any physiological evidence of their existence. We may expect that some functions that are carried out by the middle-ear muscles in mammals are accomplished by neuronal inhibition in fish. Reflex suppression after loud sounds and prevention of stimulation during sound production are good possibilities (Piddington, 1971; see Grinnell, 1969) but protection of the hair cells from damage by intense sound is obviously not. The ostariophysan sacculus may be the only otolith organ with sufficient sensitivity to warrant special protection, but this could be achieved without special muscles by a stop, buffer or elastic limit. Furthermore, all three otoliths (in goldfish and other teleosts) are suspended by fine fibres (von Frisch, 1936; Miyazaki, in Furukawa & Ishii, 1967*a*) in a viscous, cushioning jelly (not a fluid as is commonly implied), and the innervating nerve fan is stuck to the otolith but separated from the capsule wall by a layer of jelly (original). This appears to be a protective arrangement.

The effect of shocks on the interaction between two closely spaced clicks ('recovery-cycle') was complex (Fig. 9). At short delays after the shocks the response to the first click was proportionately more reduced, but the converse was true for longer delays. Thus at short delays the recovery (N_2/N_1) was enhanced; at long delays it was depressed. This was so for shocks to the anterior midbrain (Fig. 9) and for shocks to the torus semicircularis (e.g. Fig. 2).

There are several explanations for this result, but the following is probably the simplest. (*a*) There are two populations of auditory fibres; one normally responds to both clicks and the other only to the first. (*b*) Inhibition acts differently on the two populations.

Shocks initially inhibit both populations but by 100 msec the 'single-responders' have recovered from the inhibition (N_2/N_1 has reached its lowest point). During this early period, and for the next 100 msec, the 'double-responders' recover sufficiently to respond to the first click but not to both clicks (N_1 has recovered; N_2 has not). Not until well after 500 msec does this population give a normal response to both clicks.

In the goldfish auditory nerve there are two groups of auditory fibres, thick (S_1) and thin (S_2) (Furukawa & Ishii, 1967*a*, *b*). The two groups of medullary afferents which I propose could relate to the S_1 and S_2, but which group corresponds to single-responders and which to double-responders is not clear.

The following paper (Piddington, 1971) presents a detailed discussion of the specific uses of descending control in the fish auditory system and of the general biological significance of hearing in fish.

SUMMARY

1. Shocks applied to three midbrain regions reversibly suppressed the click-evoked action potential at the medulla. Clicks in air did not activate lateral-line fibres.

2. Only one midbrain region, the torus semicircularis, showed a distinct afferent auditory response; the other two regions (anterior optic tectum and midbrain below) were used to avoid unwanted antidromic activation of medullary afferents by shocks to the torus.

3. Muscular action on the mechanical auditory pathway was excluded by showing that shocks had no effect on the saccular microphonic or receptor potential.

4. The results are consistent with the action of a descending inhibitory pathway that suppresses auditory input either at the medulla or at the sacculus.

5. One brief shock is effective, but the system is mainly dependent on temporal and spatial summation.

6. The dependency on click intensity may be complex, both high-threshold and low-threshold fibres can be inhibited and occasionally a component of the compound spike shows facilitation instead.

7. Shocks affect the two-click recovery cycle in a complex manner. Recovery is enhanced if the clicks are presented less than 40 msec after the shocks but depressed at longer intervals, even beyond 500 msec.

This study was made in the laboratory of Professor T. H. Bullock, to whom I am extremely grateful. I am also indebted to Dr W. Buño for many initial suggestions, to R. H. Hamstra, Grace Kennedy, Patricia McCorry and Judith Bronowski for technical assistance, and to Professors R. Galambos, A. D. Grinnell, P. F. Scholander and F. N. Spiess for advice and criticism during the course of the study and during the preparation of the manuscript. The work was aided by grants from the National Science Foundation, National Institutes of Health, and Office of Naval Research to Professor T. H. Bullock.

REFERENCES

ARÉCHIGA, H. & WIERSMA, C. A. G. (1969). The effect of motor activity on the reactivity of single visual units in the crayfish. *J. Neurobiol.* **1**, 53–70.

VAN BERGEIJK, W. A. (1964). Directional and non-directional hearing in fish. In *Marine Bioacoustics* (ed. W. N. Tavolga), pp. 281–99. Oxford: Pergamon Press.

VAN BERGEIJK, W. A. (1967). The evolution of vertebrate hearing. In *Contributions to Sensory Physiology* (ed. W. D. Neff), pp. 1–49. New York: Academic Press.

BRANSTON, N. M. & FLEMING, D. C. (1968). Efferent fibres in the frog optic nerve. *Expl Neurol.* **20**, 611–23.

BUÑO, W. JR., VELLUTI, R., HANDLER, P. & GARCIA AUSTT, E. (1966). Neural control of the cochlear input in the wakeful free guinea pig. *Physiol. Behavior* **1**, 23–35.

RAMÓN Y CAJAL, S. (1908). Sur un noyau spécial du nerf vestibulaire des poissons et des oiseaux. *Trab. Lab. de Invest. Biol.* **2**, 129–221.

DEWSON, J. H. (1968). Efferent olivocochlear bundle: some relationships to stimulus discrimination in noise. *J. Neurophysiol.* **31**, 122–30.

ENGER, P. S. (1963). Single unit activity in the peripheral auditory system of a teleost fish. *Acta physiol. scand.* **59** (Suppl.), 210.

ENGER, P. S. (1966). Acoustic threshold in goldfish and its relation to the sound source distance. *Comp. Biochem. Physiol.* **18**, 859–68.

ENGER, P. S. (1967). Hearing in herring. *Comp. Biochem. Physiol.* **22**, 527–38.

ENGER, P. S. (1968). Hearing in fish. In *Hearing Mechanisms in Vertebrates* (ed. A. V. S. de Reuck and J. Knight), pp. 4–10. London: J. A. Churchill.

FEX, J. (1962). Auditory activity in centrifugal and centripetal cochlear fibres in cat. A study of a feedback system. *Acta physiol. scand.* **55**, 5–62 (Suppl. 189).

FEX, J. (1968). Efferent inhibition in the cochlea by the olivocochlear bundle. In *Hearing Mechanisms in Vertebrates* (ed. A. V. S. de Reuck and J. Knight), pp. 169–81. London: J. A. Churchill.

VON FRISCH, K. & DIJKGRAAF, S. (1935). Können Fische die Schallrichtung wahrnemen? *Z. vergl. Physiol.* **22**, 641–55.

VON FRISCH, K. (1936). Über den Gehörsinn der Fische. *Biol. Rev.* **11**, 210–46.

FURUKAWA, T. (1966). Synaptic interaction at the Mauthner cell of goldfish. In *Progress in Britian Research.* A (Correlative Neurosciences). Vol. 2. (Ed. T. Tokizane and J. P. Schade.) New York: Elsevier.

FURUKAWA, T. & ISHII, Y. (1967*a*). Neurophysiological studies on hearing in goldfish. *J. Neurophysiol.* **30**, 1377–403.

FURUKAWA, T. & ISHII, Y. (1967*b*). Effects of static bending of sensory hairs on sound reception in the goldfish. *Jap. J. Physiol.* **17**, 572–88.

GALAMBOS, R. (1954). Neural mechanisms of audition. *Physiol. Rev.* **34**, 497–528.

GALAMBOS, R. (1956). Suppression of auditory nerve activity by stimulation of efferent fibres to cochlea. *J. Neurophysiol.* **19**, 424.

GRIFFIN, D. R. (1955). Hearing and acoustic orientation in marine animals. *Deep-Sea Res.* **3** (Suppl.), 406–17.

GRINNELL, A. D. (1969). Comparative physiology of hearing. *A. Rev. Physiol.* **31**, 545–80.

GROEZINGER, B. (1967). Electrophysiologische Untersuchungen der Hörbahn der Schleie (*Tinca tinca* L.). *Z. vergl. Physiol.* **57**, 44–76.

HAMA, K. (1969). A study on the fine structure of the saccular macula of the goldfish. *Z. Zellforsch. mikrosk. Anat.* **94**, 155–71.

HARA, T. J. (1967). Electrophysiological studies of the olfactory system of the goldfish, *Carassius auratus* L. II. Response patterns of the olfactory bulb neurones to chemical stimulation and their centrifugal control. *Comp. Biochem. Physiol.* **22**, 199–208.

HARRIS, G. G. & VAN BERGEIJK, W. A. (1962). Evidence that the lateral-line organ responds to near-field displacements of sound sources in water. *J. Acoust. Soc. Am.* **34**, 1831–41.

HASHIMOTO, T., KATSUKI, Y. & YANAGISAWA, K. (1970). Efferent system of lateral-line organ of fish. *Comp. Biochem. Physiol.* **33**, 405–22.

HERNANDEZ-PEON, R., BRUST CARMONA, H. PENALOZA ROJAS, J. & BACH-Y-RITA, G. (1961). The efferent control of afferent signals entering the central nervous system. *Ann. N.Y. Acad. Sci.* **89**, 866–82.

HOLDEN, A. L. (1968). The centrifugal system running to the pigeon retina. *J. Physiol., Lond.* **197**, 199.

INGLE, D. (1968). *The Central Nervous System and Fish Behavior*. Chicago University Press.

KLEEREKOPER, H. & CHAGNON, E. L. (1954). Hearing in fish with special reference to *Semotilus a. atromaculatus* (Mitchell). *J. Fish. Res. Bd. Can.* **11** (2), 130–52.

LIVINGSTON, R. B. (1959). Central control of receptors and sensory transmission systems. In *Handbook of Physiology*. Section I: Neurophysiology. Vol. 1 (ed. J. Field), pp. 741–60.

LLINAS, R. & PRECHT, W. (1969). The inhibitory vestibular efferent system and its relation to the cerebellum of the frog. *Exp. Brain Res.* **9**, 16–29.

MCKAY, J. M. (1970). Central control of an insect sensory interneurone. *J. exp. Biol.* **53**, 137–46.

PAGE, C. H. (1970). Electrophysiological study of auditory responses in the goldfish brain. *J. Neurophysiol.* **33**, 116–28.

PEARSON, A A. (1936). The acoustico-lateral nervous system in fishes. *J. comp. Neurol.* **64**, 235–73.

PIDDINGTON, R. W. (1971). Central control of auditory input in the goldfish. II. Evidence of action in the free-swimming animal. *J. exp. Biol.* **55**, 585–610.

ROWELL, C. H. F. & HORN, G. (1968). Dishabituation and arousal in the response of single nerve cells in an insect brain. *J. exp. Biol.* **49**, 171–83.

ROWELL, C. H. F. & MCKAY, J. M. (1969). An acridid auditory inter-neurone. II. Habituation, variation in response level, and central control. *J. exp. Biol.* **51**, 247–60.

TAVOLGA, W. N. (1964). Sonic characteristics and mechanisms in marine fishes. In *Marine Bioacoustics* (ed. W. N. Tavolga), pp. 195–211. Oxford: Pergamon Press.

WHITFIELD, I. C. (1968). Centrifugal control mechanisms of the auditory pathway. In *Hearing Mechanisms in vertebrates* (ed. A. V. S. de Reuck and J. Knight), pp. 246–53. London: J. A. Churchill.