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# The Afferent Auditory Pathway in the Ventral Cord of *Locusta migratoria* (Acrididae)*

## I. Synaptic Connectivity and Information Processing among the Auditory Neurons of the Ventral Cord

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*Summary.* The afferent auditory pathway in the region of the ventral cord of *Locusta migratoria* has been studied electrophysiologically. Various recording, stimulating and surgical techniques have been used to infer the circuitry underlying the response patterns of the ventral-cord neurons ascending to the supraesophageal ganglion.

The stimuli used were artificial sounds (white noise and pure tones varying in frequency, intensity, duration, source direction and repetition rate). The responses of the auditory ventral-cord neurons over the range of frequencies and intensities are described. Presentation of two sound stimuli occurring simultaneously or displaced in time permits the demonstration of inhibitory and subthreshold facilitatory influences which, with extracellular recording, cannot be observed directly *via* changes in membrane potential (Figs. 3 and 4).

On the basis of comparisons of responses before and after inactivation of a tympanal organ additional inferences are made concerning the relative contributions of the two tympanal organs to the formation of the response-patterns (Figs. 6, 11, 16, 21, 25).

The geometry, location and course of certain auditory ventral-cord neurons have been revealed by a combined recording and staining technique (Fig. 7); transection of connectives has made it possible to determine the origin of certain response-patterns and whether these change at various stations of the ventral cord as a result of corresponding synaptic input.

The results of such experiments, together with the knowledge of the course and location of endings of the tympanal receptor fibers in the ventral cord, have been used to construct sufficient connectivity diagrams for the ascending ventral-cord neurons (Figs. 8, 12, 17, 22, 26). The following general properties have been revealed: 1. Between receptor cells and ascending auditory ventral-cord neurons, as a rule, interneurons are interposed; this accounts for the observation that the postsynaptic structures of the ventral-cord neurons are for the most part situated outside the auditory neuropile. 2. Six basic connectivity patterns underlie the responses of the 14 ventral-cord neurons described; that is, the same response patterns are produced in sets of two or three ventral-cord neurons, owing to synaptic connectivities involving the same interneurons. The distinctions between the response patterns of the 14 neurons are interpretable as stemming from the addition of another interneuron to, or by elimination of a facilitatory or inhibitory connection from, the basic connectivity patterns.

### A. Introduction

Auditory communication among the Orthoptera serves primarily to assist in finding a sexual partner, in a biotope presenting severe obstacles to visual contact. Even communication by sound signals, however, must be done in the presence of a variety of songs and noises produced by animals of other species. Under such

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conditions, the task of recognizing species-specific signals and localizing a sound source makes great demands upon the auditory systems of these animals.

The number of neurons comprising the central auditory pathway of grasshoppers and crickets is small. In the ventral cord of *Locusta migratoria* only 15 to 20 auditory neurons have been found which ascend to the supraesophageal ganglion. This parsimony is particularly striking if compared with the numbers at the lower levels of the auditory pathway in mammals and birds; for example, the human cochlear nucleus has about 88,000 cells.

Even bearing in mind the relatively modest auditory performance of the grasshopper, especially with respect to frequency discrimination, it is of considerable interest to ask how so few neurons can achieve the recognition of species-specific songs and their localization in a noisy biotope.

As Hughes (1965) has suggested, the explanation may lie in the very complex processing known to occur in arthropod interneurons. He concludes that the function of single insect central neurons is comparable to that of entire chains of neurons in vertebrates.

Recent investigations have been focussed on the structure and function of the tympanal organs and the afferent auditory pathway in the ventral cord of *Locusta migratoria*. The 60–80 receptor cells of the tympanal organ, according to Gray (1960), comprise four anatomical groups: a-, b-, c- and d-cells. Popov (1965) did extirpation experiments on the basis of which he distinguished low-frequency and high-frequency receptors. Michelsen (1971 a, b, c) clarified to a great extent the operation of the receptor organ, by his studies of the frequency-sensitivity of single cells and the acoustic properties of the tympanal organ. It is very likely that signals proceeding from the receptor cells to the ventral cord undergo some alteration as a result of interactions between the axons in a "sub-receptor plexus" described by Popov and Svetlogorskaja (1971). Electrophysiological studies by Römer (personal communication) of the receptor cells in the metathoracic ganglion show that the four anatomical groups of Gray (1960) all fall into the two categories described as low-frequency and high-frequency receptors. However, there are differences in the threshold curves within both groups of neurons; that is, some neurons have low thresholds, others are intermediate, and in a few the thresholds are high. All the receptor cells exhibit a tonic response to auditory stimuli over a range of intensities. Their characteristic curves of response *vs.* intensity rise steeply for 20–30 dB above the threshold level and then flatten out asymptotically.

Histological evidence of the course of the tympanal nerve fibers, with their end-branches in the neuropile of the thoracic ventral cord, was presented by Kalmring, Rheinlaender and Rehbein (1972) and Rehbein (1972). Electrophysiological studies of the auditory neurons in the ventral cord have been made by Suga (1963), Popov (1967, 1969, 1971), Kalmring, Rheinlaender and Rehbein (1972) and Kalmring, Rheinlaender and Römer (1972). Although Suga failed to demonstrate interactions between the auditory neurons on the two sides of the ventral cord, such interactions have been demonstrated by the other authors for almost all the neurons investigated. Popov described two neurons ascending to the supraesophageal ganglion, and Kalmring (1971) described four in the subesophageal ganglion. Even in the first synapses of the auditory pathway there is

a complex pre-processing of the parameters intensity, duration, repetition rate, frequency and direction (Kalmring, Rheinlaender and Rehbein, 1972; Kalmring, Rheinlaender and Römer, 1972). A fundamental property is that each of the nine ventral-cord neurons described in detail encodes several of these parameters simultaneously. Inhibition among ipsilateral and/or contralateral neurons leads to contrast-enhancement. As a result, some parameters are emphasized in the response, while others are attenuated during encoding.

Using a new combined recording and staining technique (the CoS method), Rehbein, Kalmring and Römer (1974), Kalmring, Römer and Rehbein (1974) and Rehbein, Kalmring and Schwartzkopff (1974) succeeded not only in recording from auditory ventral-cord neurons (as had been done before), but in simultaneously revealing their shapes and the courses travelled by the fibers. The results of these studies indicate that there is a significant amount of processing in the first synapses of the auditory pathway. That is, all the neurons for which the shape and fiber positions have been demonstrated thus far, and which form a link between the receptor cells and the auditory association centers in the supraesophageal ganglion, in addition make direct connections by way of collaterals with non-auditory neuronal centers in the various ganglia of the ventral cord. In some cases, these connections are with motoneurons.

Clarification of the synaptic connectivity in the ventral cord is of course a prerequisite for an understanding of the neural processing of auditory information that takes place there. Connections responsible for the response-patterns of the ventral-cord neurons can be detected if the neurons' shapes and anatomical courses are known, by the combination of electrophysiological experiments with various methods for stimulating and deactivating the neurons.

The response-patterns of the ventral-cord neurons are complicated in comparison with those of the receptor cells. Whereas the latter respond to sound stimuli with tonic or phasic-tonic response-patterns, the response-patterns of the ventral-cord auditory neurons are very labile, changing as a result of variation of only one stimulus parameter. Tonic response-patterns give way to phasic, and vice versa. The phasic responses may consist of on- or off-responses, or of a combination of both. As a rule, inhibitory influences participate in the formation of complicated response-patterns. Another interesting aspect is the way in which the boundaries of the response regions in the frequency/intensity field of single neurons are established. Do they arise from the fact that with stimuli outside these limits there is no input from neurons earlier in the circuit? Alternatively, if subthreshold inputs are still present, are these inhibitory or facilitatory? Since intracellular recordings are not practicable because of the relatively small diameter of the fibers, potentials underlying the spike response must be inferred from suitable experiments—e.g., by stimulating with two sound signals, either simultaneously or in succession (Suga, 1964).

A further point in need of clarification is the contribution of each of the two tympanal organs to the overall response of a neuron. By excluding one and then the other tympanal organ, in different experiments, their roles can be determined. But in most cases it remains unclear whether the effects are exerted directly *via* the receptor fibers or involve intermediate neurons. This can frequently be determined on the basis of the topography (shape, course of fibers, branching) of the ventral-

cord neurons if the position of the receptor endings in the thoracic ventral cord is known. When transections of the connectives are performed in addition, inferences can be drawn about the site of origin and possible changes in the response-pattern at the way-stations *en route* to the supraesophageal ganglion.

In many cases application of the above research techniques permits the eventual construction of circuit diagrams for the auditory ventral-cord neurons. Comparisons of the different connectivities provide insights into the fundamental principles of processing in the first synapses of the auditory pathway.

## B. Materials and Methods

Both male and female imagines of the migratory locust (*Locusta migratoria*) were used. Before the experiments, the animals' legs and wings were removed. The locusts were attached to a metal holder, dorsal surface down, with a mixture of wax and colophonium. Depending upon the region from which recordings were to be made, the ventral cord was exposed by removal of the ventral body wall near the metathoracic, mesothoracic, prothoracic or subesophageal ganglion. The microelectrode was normally introduced into the rostral and caudal connective roots of these ganglia, on both the side on which the sound source was located and on the contralateral side. Prior to recording, the abdominal cord was routinely isolated from the thoracic cord by transection anterior to the first abdominal ganglion.

In many cases further transections were made during the experiment, to eliminate one of the two tympanal organs or to interrupt, unilaterally or bilaterally, connectives between thoracic ganglia or in the head region. Such surgical interventions serve two purposes; on the one hand, comparison of the response-patterns before and after such operations can reveal the separate influences of the two tympanal organs upon the response-patterns of neurons at various levels, and on the other they permit determination of the site of formation of certain response-patterns.

Moreover, it is possible to determine whether the response-patterns change at different levels in the ventral cord as a result of additional synaptic connections.

The recording electrodes were glass microcapillaries filled with 3 M KCl or 3 M $CoCl_2$ (with resistances of 4—10 MΩ). A silver wire inserted in the abdomen of the animal served as an indifferent electrode. $CoCl_2$-filled electrodes were used in order that the auditory neurons studied physiologically in an experiment could subsequently be revealed histologically by the precipitation of CoS; the method has been described in detail by Rehbein, Kalmring and Römer (1974).

The recording electrodes were connected *via* cathode follower (WP Instruments VF 1) and preamplifier (Tektronix, Type 122) to an oscilloscope (Tektronix 510 3N), where the action potentials were displayed together with the auditory stimulus; the two traces were photographed with a Tönnies Recordine camera.

Sound stimuli were provided by two acoustic stimulators (Burchard II) and a noise generator (General Radio Company 1382, frequency range from 20 Hz to 50 kHz). Stimulus frequencies between 1 and 40 kHz, at intensities of 30–100 dB, were used; the stimulus duration was varied from 1 to 100 ms, 20 ms being most often selected. The repetition rate ranged from 1 to 20 per sec, with 2/s most common. Rise and fall times of the stimulus envelope normally were 1 ms. Sound intensities are given as dB *re* 0.0002 dyn/cm$^2$.

The experiments were done in an anechoic chamber (from the firm of Grünzweig & Hartmann), designed for frequencies above 100 Hz.

Ordinarily the stimuli were presented *via* a high-frequency loudspeaker (Audax, TW 8 spz.) with a range of 1–40 kHz. The speaker was mounted on a calibrated framework such that it could be moved around the animal in the horizontal plane, in steps of 30°. The distance between speaker and animal was 66 cm (conduction time 2 ms; sound field linear). In order to avoid distortions in the sound field, the locust was mounted on a flat micromanipulator completely imbedded in a layer of glass-wool.

On account of the small diameter of most of the auditory neurons of the ventral cord, only extracellular single-cell recordings can be made. These do however permit the demonstration

of subthreshold facilitatory and inhibitory influences upon the ventral-cord neurons, by the use of simultaneous or temporally shifted stimulation with two sound signals (Suga, 1964). Such experiments involved two loudspeakers at the same distance and approximately in the same direction with respect to the animal; these were driven by one acoustic-stimulus generator and two amplifiers (for stimuli of the same frequency and different intensities) or by two stimulus generators (for tones of different frequencies and the same or different intensities).

Nearly 400 recordings of single neurons were done. The half of all neurons could be tested by the complete stimulus program with more than 200 stimulus-parameter-combinations. Each of the large auditory neurons (groups B and G) were recorded 50 times; the other groups are represented by 20–30 recorded cells.

## C. Results

The classification procedure adopted here is to designate groups of neurons similar in response by capital letters. Certain specific neurons (that is, those of which there is one only on either side of the ventral cord) are further designated as to "type" (actually a pair) if they can be distinguished from the other neurons in their group by their physiological properties and (as far as has been determined) by their shape. Such types are designated by the group letter plus an index number.

### *1. The B-Neurons*

The B-neurons, because of their large-diameter fibers and their topography, lend themselves particularly well to this sort of study. The group consists of two neurons on either side of the ventral cord—types $B_1$ and $B_2$.

The $B_1$ neurons are characterized by tonic responses to sounds in the near-threshold intensity range. Above 70 dB, with stimuli 20 ms in duration, they become phasic—i.e., they exhibit on-responses (Fig. 1). With stimuli 100 ms in duration, the on-responses are followed, after a pause of 40–60 ms, by additional discharges.

In the frequency/intensity graph of the responses of this neuron type (Fig. 2; black columns represent responses to ipsilateral stimulation and white, those to contralateral stimulation), two things are particularly noteworthy. With high-intensity sounds at low frequencies, no responses are observable. Second, contralateral stimulation in the near-threshold region of intensity (in contrast to ipsilateral stimulation) elicits only slight responses. The smaller responses to contralateral stimulation cannot be entirely explained by the effect of diffraction or sound attenuation by the animal's body (Michelsen, 1971c), for the tympanal receptor neurons themselves display a "threshold shift" (when the responses to ipsilateral and contralateral stimulation are compared) of only 10–20 dB (Autrum, Schwartzkopff and Swoboda, 1961; Römer, pers. comm.).

It is to be assumed that tonic responses result from an above-threshold excitatory postsynaptic potential (EPSP), as is diagrammed in Fig. 3(I). The on-responses, on the other hand, could be brought about by three conceivable postsynaptic events: 1. a brief (1–2 ms) above-threshold EPSP, 2. a brief above-threshold EPSP followed by a depolarizing but subthreshold potential, and 3. a brief above-threshold EPSP followed by an inhibitory postsynaptic potential (IPSP) (Fig. 3(III)a, b, c).

Simultaneous stimulation with tones producing tonic responses and those producing on-responses permits one to decide which of these three sequences

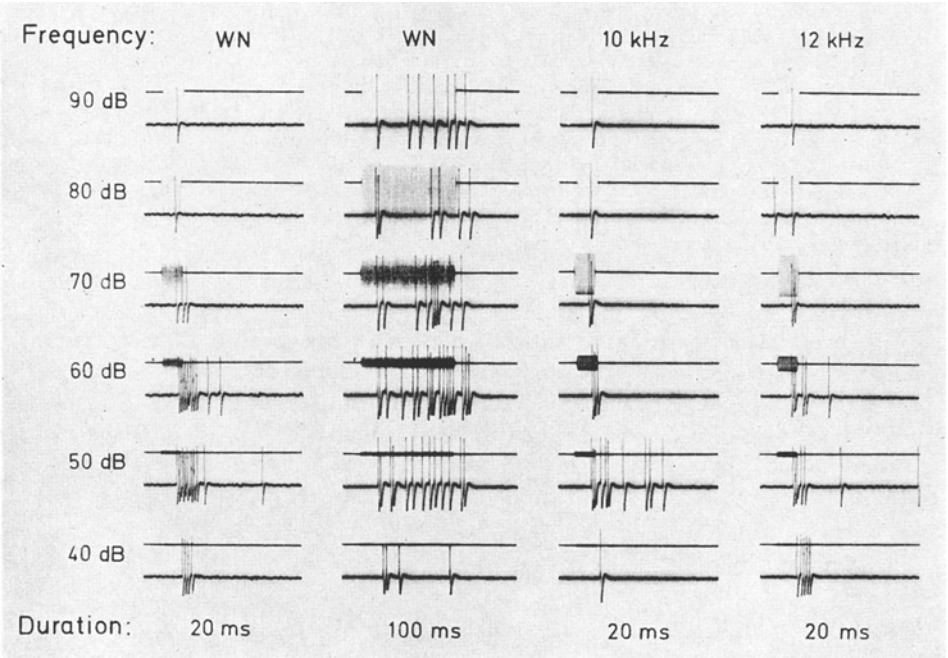


Fig. 1. The response pattern of a $B_1$-neuron, recorded from the frontal region of the mesothoracic ganglion with the auditory system intact, to ipsilateral sound stimulation. Stimulus repetition rate, 2/s; *WN* white noise

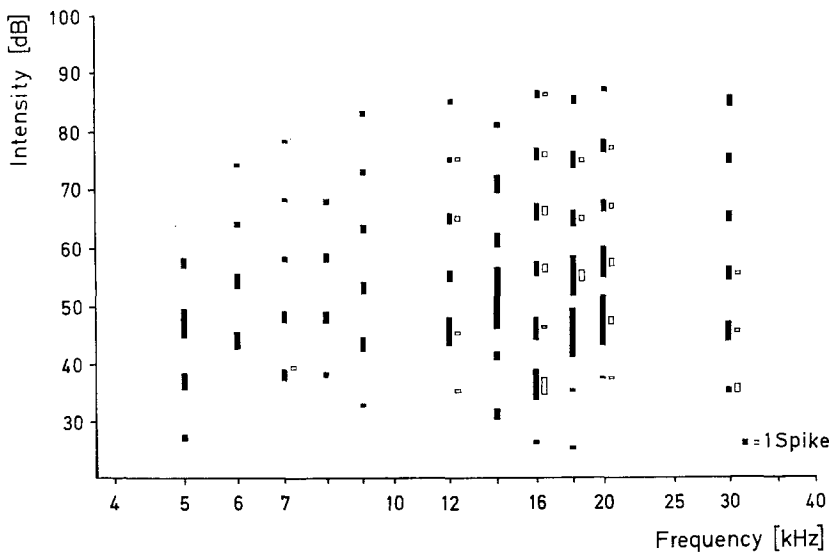


Fig. 2. Responses of a $B_1$-neuron over the range of frequencies and intensities studied. The recording is from the frontal region of the subesophageal ganglion, with auditory system intact. Stimulus duration, 20 ms; repetition rate, 2/s. Black bars: ipsilateral sound; white bars: contralateral sound. At the lower right corner of the picture is shown the bar height corresponding to one impulse per stimulus (the number of impulses are averaged over 4 recordings in each case). This format is used in all diagrams of this kind that follow

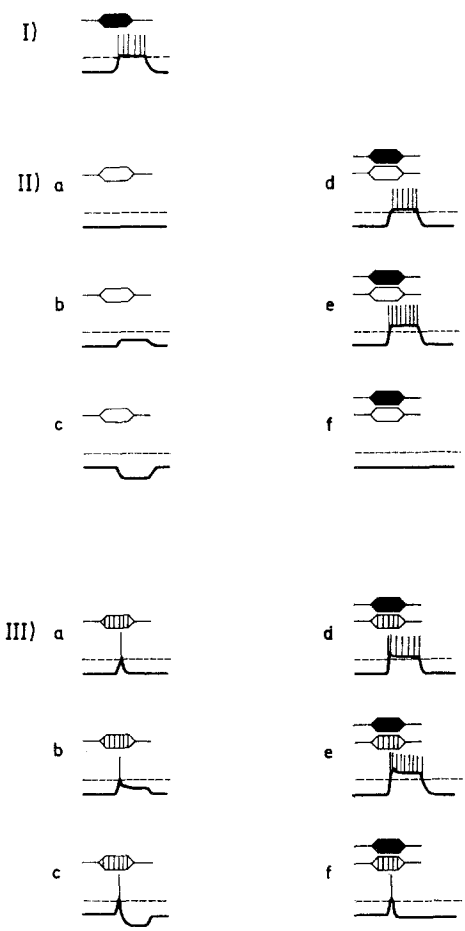


Fig. 3. Schematic representation of postsynaptic potentials which might possibly underlie the various responses. The upper line in each drawing indicates the stimulus; where there are two upper lines (parts d, e and f) two tones are presented simultaneously. The thin dashed line in the middle represents the threshold of the neuron, and the lower, heavy line the postsynaptic potential elicited. (I): Potential underlying the spike pattern of a B-neuron in the near-threshold, tonic-response part of its range. (II) a, b and c: Possible potential changes produced by tones in the low-frequency, intermediate- to high-intensity range producing no spike response. (III) a, b and c: Possible potential changes associated with on-responses of the B-neuron. Parts d, e and f of (II) and (III): The postsynaptic potentials and spike patterns to be expected as a result of simultaneous stimulation with tones producing tonic responses and tones producing no responses or on-responses, respectively

underlies the on-response. The responses, to such stimulus combinations, that would be expected in each case are shown in Fig. 3(III)d, e, f.

The fact that $B_1$-neurons do not respond to low-frequency tones at high intensities (3–6 kHz above 60–70 dB) can also be explained by three possible underlying events: 1. an unchanged resting membrane potential (i.e., no synaptic potential is produced), 2. a subthreshold EPSP, and 3. an IPSP (Fig. 3(II)a,b,c).

If the stimulus combinations shown in Fig. 3(II)d–f are presented, it should be possible to determine from the responses which sort of potential in fact occurs.

Simultaneous stimulation with two sound signals, one in the low-frequency high-intensity range (4 kHz, 75 dB) which produces no impulse response and one (white noise, 55 dB) which produces a tonic response, is followed by no response of the $B_1$-neurons (Fig. 4A: a, b, c). On the basis of the expected responses shown in Fig. 3(II)f and c, one may infer that the neurons of Type $B_1$ exposed to stimuli in this range are influenced by presynaptic inhibitory neurons which produce a long-lasting IPSP. This is made more clearly evident when a stimulus causing a tonic response (WN, 55 dB) is presented 10, 20 and 50 ms after the onset of a stimulus with the postulated inhibitory effect (4 kHz, 75 dB) (Fig. 4A: d, e, f). Under these conditions, the responses of the $B_1$-neurons were suppressed even 50 ms after the end of the inhibitory stimulus.

The method of simultaneous stimulation with two sources to infer postsynaptic potentials encounters difficulties in the case of the on-responses of the $B_1$-neurons. These appear only in response to stimulation at intermediate or high intensities, whereas the tonic responses appear only in the threshold region. Two stimuli at a given frequency with intensities, for example, of 75 dB (on-response) and 55 dB (tonic response) have the same effect as the former alone, apart from a slight increase in intensity. Hence the response is an on-response (Fig. 4B: a, b, c). When the stimuli have two different frequencies or comprise a tone and white noise, presented simultaneously, the effect of the high-intensity stimulus predominates over that of the lower-intensity stimulus, and an on-response results.

Nevertheless, the underlying events can be inferred if two such sound stimuli are displaced in time. For example, if following the end of a stimulus at 14 kHz and 55 dB (tonic response), a second stimulus at 14 kHz and 75 dB (on-response) is presented, the responses to the two stimuli add (Fig. 4B: d). On the other hand, if the two stimuli are exchanged so that the on-response is first elicited, and 20 ms later a stimulus producing a tonic response is given, the two responses are separately visible (Fig. 4B: e). The tonic response is slightly diminished and appears with a longer latency. If a stimulus producing an on-response comes 5 ms after one producing a tonic response, the result—as would be expected from the responses to the previous sets of stimuli—is that the tonic response is partially inhibited (Fig. 4B: f). In view of the predictions of Fig. 3(III)f and c, these findings indicate that underlying the on-response of the $B_1$-neurons is an above-threshold EPSP lasting about 1–2 ms and followed by an IPSP. It may be assumed that the delay in onset of inhibition is caused by an extra (as compared with the facilitatory pathway) synapse between the presynaptic neurons ("forward self-inhibition"). An argument against the possibility that the delayed onset of inhibition is caused entirely by a slower rise of the IPSP is that on-responses appear even at the highest intensities. In such cases the latencies between stimulus and excitation are shortened by a few ms. Thus the temporal relationship between facilitation and inhibition remains unchanged.

On the basis of these results, it is possible to suggest a model regarding the receptor neurons involved in producing the response. Fig. 5 shows the way in which the various types of receptor cells can be assumed to form the response-pattern of the $B_1$-neurons in the frequency/intensity field, when the auditory

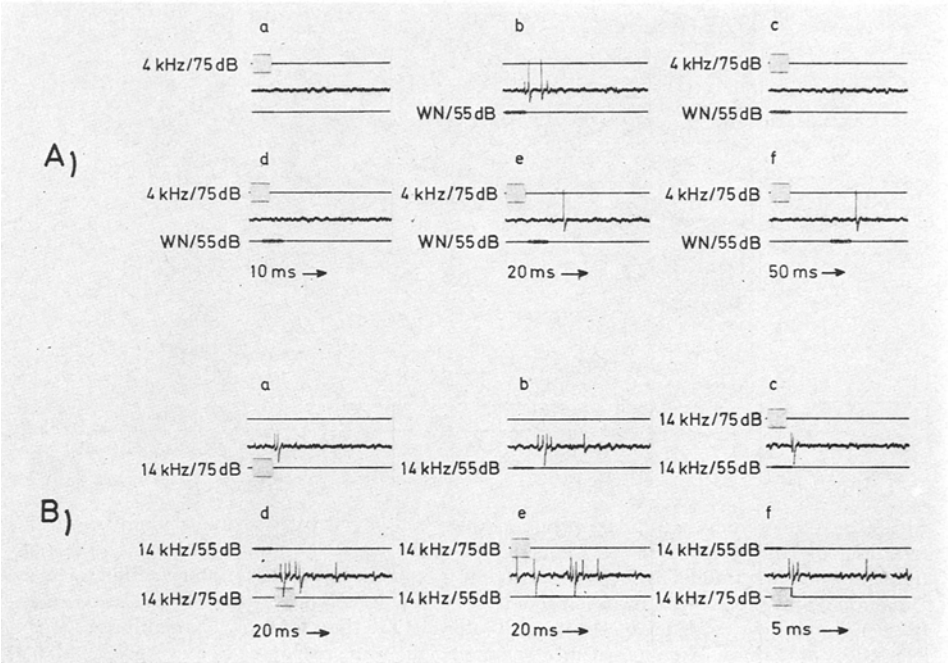


Fig. 4. Simultaneous responses of a $B_1$- and a $G_1$-neuron to stimulation with one or two sounds ($B_1$-neuron: small spike amplitude; $G_1$-neuron: large spikes). In recordings a and b, only one sound signal is presented. In c two stimuli are given simultaneously, and in d, e and f the stimulus shown on the lower trace is presented 5 ms, 10 ms, 20 ms or 50 ms later than that on the upper trace. Stimulus duration, 20 ms; repetition rate, 2/s

system is intact. High-frequency and low-frequency receptors with low thresholds produce the near-threshold tonic-response region (horizontally shaded area). Receptor neurons of intermediate threshold (50–70 dB) at low and high frequencies exert an inhibitory effect on the $B_1$-neuron *via* an additional synapse (1–2 ms delay), and bring about the on-responses. Low-frequency receptors with very high thresholds, which have an inhibitory effect upon the $B_1$-neuron, suppress any response in this region. This model holds for the intact auditory system.

But it is so far not clear which of the two tympanal organs exerts the greatest influence in forming the typical response-pattern. This can be determined if one of the organs is eliminated during an experiment. The results of such studies are shown in Fig. 6, in which the intensity characteristics for the $B_1$-neurons with ipsilateral and contralateral stimulation are compared before and after the input from the contralateral tympanal organ is interrupted. With the auditory system intact, the characteristic curve for ipsilateral stimulation (i/o, thick line) represents the typical response-pattern of a $B_1$-neuron (cf. Figs. 1 and 2). With contralateral stimulation, only a small residue of the response remains, in the threshold region (c/o, thick dashed line). If the tympanal organ contralateral to the neuron is destroyed during the experiment and sound is presented to the ipsilateral organ (i/c, thin line), two things are evident: 1. The "basic pattern" of the response,

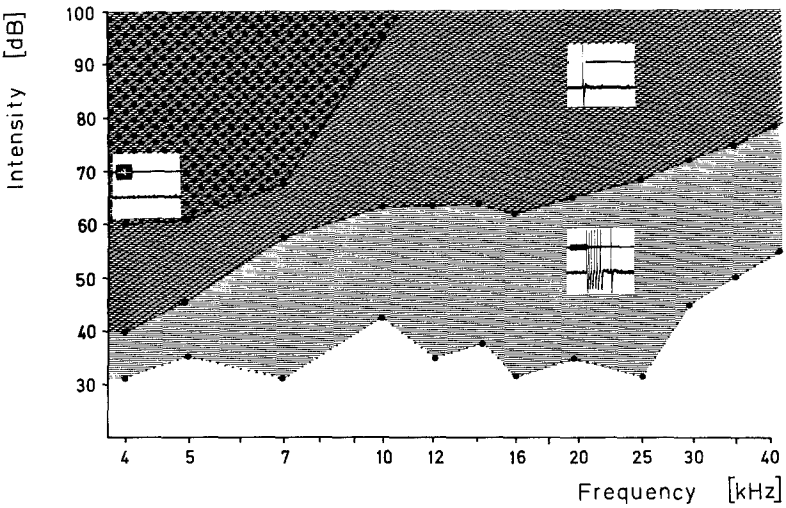


Fig. 5. Semidiagrammatic representation of the receptor-cell activity underlying the various responses of $B_1$-neurons over the frequency/intensity range (auditory system intact). High-frequency and low-frequency receptors with low thresholds have an excitatory action upon the B-neurons, and produce its near-threshold tonic responses (horizontally shaded area); high-frequency and low-frequency receptors with intermediate thresholds have an inhibitory effect, exerted by way of an extra interneuron, changing the tonic responses to on-responses (diagonally shaded area); inhibitory low-frequency receptors with high thresholds suppress the responses to 4–7 kHz tones at intensities above 60–80 dB (dotted area)

with a tonic response near threshold and on-responses at intermediate and high intensities, arises entirely as a result of influences from the ipsilateral tympanal organ. 2. With the intact auditory system, there is a strong facilitation in the near-threshold tonic-response region, which originates in the contralateral tympanal organ. This becomes apparent in the reduction of activity after the corresponding tympanal organ is destroyed (vertically shaded area in Fig. 6).

When the sound stimulus is applied contralaterally after destruction of the contralateral tympanal organ (Fig. 6; c/c, thin dashed line), it becomes evident that the extensive reduction in response of a $B_1$-neuron when sound is presented to the contralateral tympanal organ is brought about by inhibition originating in that organ. The amount of this inhibition is indicated by the diagonal shading in Fig. 6.

By comparing the two characteristic curves obtained after elimination of the contralateral tympanal organ one can obtain a measure of the attenuation of sound by the animal's body; this corresponds to the difference in intensity at the onset of the responses to ipsilateral and contralateral stimulation (thin solid curve i/c and thin dashed curve c/c, respectively), and is read off the abscissa in dB. At 12 kHz the attenuation amounts to 15–20 dB. It must be kept in mind that the physiological responses shown here, which are used as an indication of attenuation, reflect not only events at the receptors but additional synaptic processing as well.

Fig. 7 shows the shape, the course of the fibers and the location of branched endings for the two large ascending ventral-cord neurons—the $B_1$-neuron on the

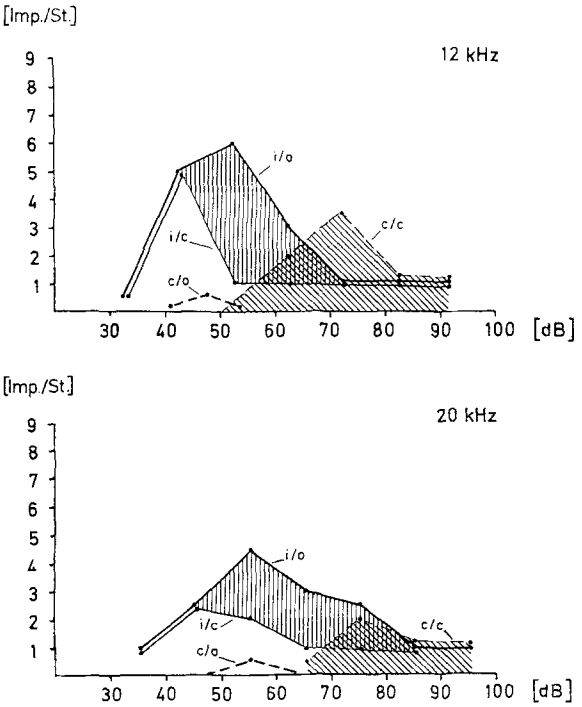


Fig. 6. Intensity characteristics of a $B_1$-neuron for ipsilateral and contralateral sound presentation before (the solid curve i/o and dashed curve c/o, respectively) and after (thin solid and dashed curves i/c and c/c) destruction of the contralateral tympanal organ. Vertically shaded areas: the amount of facilitation exerted by the contralateral tympanal organ when the sound source is ipsilateral to the $B_1$-neuron (derived from the difference in activity before and after inactivation of the organ); diagonally shaded areas: amount of inhibition exerted by the contralateral tympanal organ when the sound source is contralateral to the $B_1$-neuron (again equivalent to the difference in activity before and after inactivation of the organ). That is, for a given sound direction the $B_1$-neuron on the same side as the sound receives facilitatory inputs from the opposite organ, and the $B_1$-neuron on the side away from the sound receives inhibitory inputs from the organ opposite to it. Stimulus duration, 20 ms; repetition rate, 2/s

left, and the $G_1$-neuron on the right. For the moment consider only the $B_1$-neuron. The soma lies in the metathoracic ganglion, on the side opposite that along which the axon runs. The main influences producing the response-pattern of this neuron come from the side ipsilateral to the axon. In the vicinity of the soma, on its side of the cord, there is much branching in the frontal region of the ganglion. From there the axon passes through a commissure to the opposite side and ascends (ipsilaterally, from a functional standpoint) to the supraesophageal ganglion. Several medially directed branchings can be demonstrated in each of the mesothoracic, prothoracic and subesophageal ganglia; in no case do these branches cross the midline. The various branches of the B-neuron differ in structure; on those running medially one can find small swellings which are absent on the branches near the soma.

The complete response-pattern of the $B_1$-neuron can be recorded even in the metathoracic ganglion. Moreover, it does not change when the connection to the

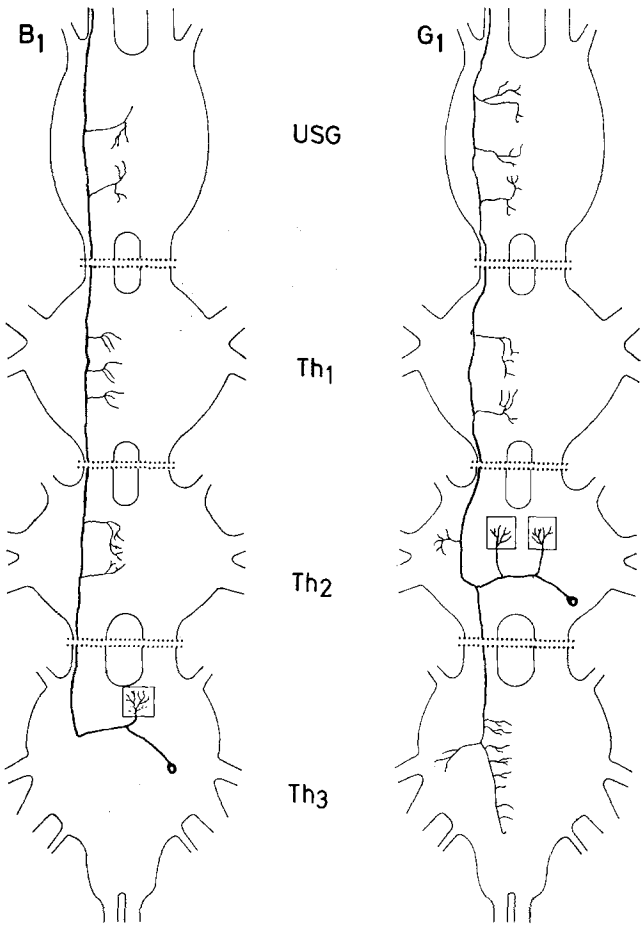


Fig. 7. Arrangement of two auditory ventral-cord neurons. Left: $B_1$-neuron; right: $G_1$-neuron. USG, $Th_1$, $Th_2$, $Th_3$ = subesophageal, prothoracic, mesothoracic, and metathoracic ganglia. The postsynaptic branching regions near the soma are denoted by a square. The topography of the neurons was worked out in collaboration with H. G. Rehbein (cf. Rehbein, Kalmring and Römer, 1974)

more anterior ganglia is destroyed (by transection of both meso-metathoracic connectives). Thus the branches near the soma alone can be considered as adequate post synaptic (dendritic) structures of the B-neurons. Synaptic connections with the axon as it passes through the commissure or in the fiber bundle of the ganglion and the connective are unlikely, since the fibers there are closely packed and no branching could be demonstrated. Evidence against the presence of electrical synapses is provided by the latencies measured. Nor could any indication of such synapses be found in the structural analysis. Though the branches near the soma are in the vicinity of the auditory neuropile formed by the end-branches of the receptor fibers, they lie for the most part outside it.

At the various stations in the ventral cord between the metathoracic and supraesophageal ganglia, the response-pattern of the $B_1$-neuron undergoes no

change. It must be noted, of course, that under these experimental conditions nothing can be said about the possible influence of organs other than the tympanum upon the $B_1$-neuron, since legs and wings had been removed before the experiment and the neural connection to the abdomen had been interrupted. Yanagisawa, Hashimoto and Katsuki (1967) showed that inhibition in the low-frequency range (below 4 kHz) was eliminated after transection of the segmental nerves in the mesothorax and prothorax and after separation of the thoracic from the abdominal ventral cord. Such influences may well be present, especially in the B-neurons described here. Own experiments also demonstrated facilitatory influences of hair sensilla (after elimination of both tympanal organs) upon the B- and C-neurons in the thoracic cord; their effect was to extend the response range of these neurons toward lower frequencies (i.e., the hair sensilla were acting as auditory receptors). Connections to the cercal giant fibers and the hair sensilla of the abdominal wall could be located in the branches near the soma. Inputs from the leg and wing nerves, on the other hand, probably arrive by way of the medial branches. It is thought, however, that their main function is to transmit rather than receive information.

The results presented so far indicate that the $B_1$-neurons are not second-order auditory neurons; that is, they cannot be in direct synaptic connection with receptor cells—at least as far as formation of the "basic pattern" is concerned. This is evident from the fact that their postsynaptic (information-receiving) branches are on the contralateral side, whereas the chief influence in the formation of the response-pattern comes from the ipsilateral tympanal organ (as shown by the extirpation experiments illustrated in Fig. 6). Since it is known from earlier work (Kalmring, Rheinlaender and Rehbein, 1972) that the tympanal receptor fibers all end in the auditory neuropile on the ipsilateral side, and send no collaterals to the opposite side, it must be concluded that the facilitatory influences contributing to the near-threshold tonic-response region also reach the synaptic region of the B-neurons *via* at least one interneuron. Thus the B-neurons are auditory neurons of third or even fourth order (cf. also Rehbein, Kalmring and Römer, 1974). Even the facilitatory influence from the contralateral tympanal organ, which according to the preceding discussion could be exerted by way of direct connections between the receptor fibers and the $B_1$-neuron, is probably transmitted through an interneuron. This view is supported by the latency measurements following elimination of the ipsilateral tympanal organ.

Fig. 8 is a diagram of the postulated circuitry involving the $B_1$-neurons, based on these considerations. Low-frequency and high-frequency receptors having low thresholds, from the tympanal organ contralateral to the synaptic region, synapse with facilitatory interneurons which run to the postsynaptic branch system of the B-neuron and there produce the near-threshold tonic responses (arrows with black triangles: sensitive high-frequency receptors; arrows with light triangles: sensitive low-frequency receptors; for simplicity, the full connectivity is shown for only one of the pair of B-neurons, in this case the left one). From these receptor neurons the same excitation passes through collaterals to inhibitory interneurons, which affect the other (right) $B_1$-neuron (the postsynaptic region of which is located on the left). With the sound source ipsilateral to these inhibitory interneurons, they suppress to a great extent the facilitatory influence of the contra-

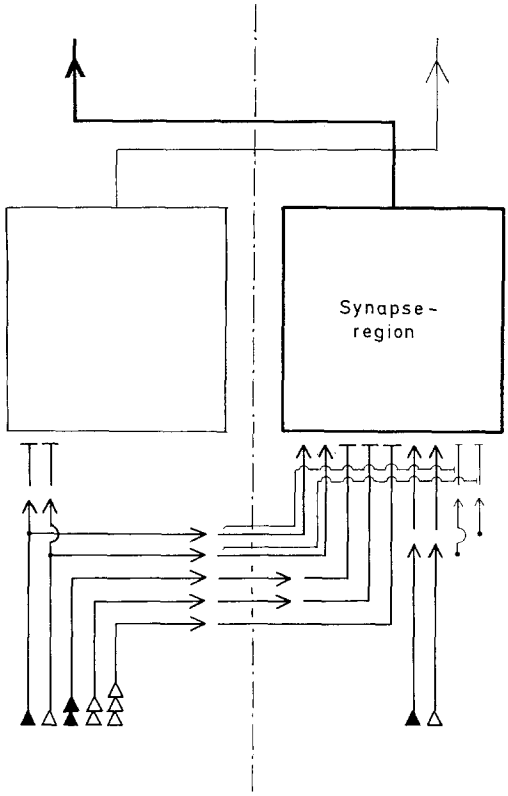


Fig. 8. Connectivity diagram for $B_1$-neurons. One neuron, together with all its inputs, is represented by the heavy lines; its (contralaterally located) postsynaptic region and the course of its axon in the metathoracic ganglion are shown. The thin lines indicate the corresponding structures of the other neuron of the pair. ▶→ high-frequency receptors; ▷→ low-frequency receptors. One, two and three triangles indicate receptors with low, intermediate and high thresholds, respectively. → facilitatory interneurons; —| inhibitory interneurons. The same symbols are used in subsequent illustrations of this kind. For clarity, the complete connectivity diagram is given for only one of the two neurons

lateral tympanal organ; the latter begins to appear only when the stimulus intensity is about 10–15 dB greater, due to the attenuation of sound by the body. The net result is the suppression, to a considerable extent, of the response in the contralateral $B_1$-neuron axon.

The on-responses in a given $B_1$-neuron are considered to be brought about *via* an additional synapse (by the interpolation of a second interneuron) activated by ipsilateral high-frequency and low-frequency receptors with intermediate threshold (arrows with the two black or light triangles). The inhibition in the low-frequency response region at high intensities, shown here by high-threshold low-frequency receptor cells (arrow with three light triangles), also comes from the ipsilateral side and requires an inhibitory interneuron. The sensitive low-frequency and high-frequency receptors of the contralateral tympanal organ are responsible, *via* an interneuron in each case, for the facilitation in the threshold region il-

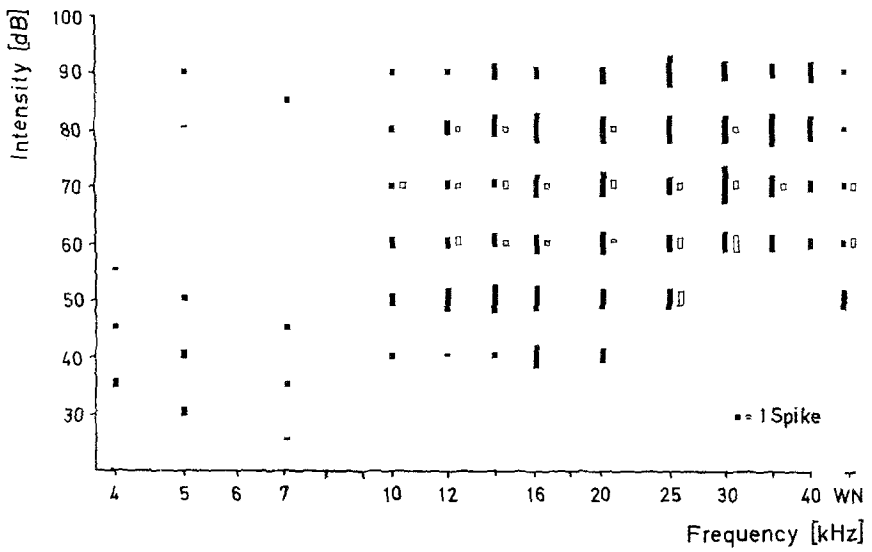


Fig. 9. Responses of a $B_2$-neuron over the frequency/intensity range. The recording is from the frontal region of the mesothoracic ganglion with auditory system intact. Stimulus duration, 20 ms; repetition rate, 2/s. Black bars: ipsilateral sound presentation; white bars: contralateral presentation

lustrated in Fig. 6. In order for this facilitation to be effective, the influence of the inhibitory neurons from the contralateral side must be suppressed; this "simple" connectivity diagram must therefore be extended by the inclusion of appropriate interneurons. Because of this "supplementary circuitry" (interneurons drawn with fine lines), sound originating in front of or behind the animal (i.e., in the long axis of the body) could excite the B-neurons of the two sides to the same degree.

In addition to the $B_1$-neuron described above, there is a second B-neuron on each side of the ventral cord—the $B_2$-neuron. With physiological experiments alone it would be difficult to demonstrate that the two neurons are in fact "related". Only when the physiological results are combined with histologica evidence and transection/extirpation experiments does it become clear that the same "basic connectivity patterns" apply in each case. When several neurons are said to share a "basic connectivity pattern", the implication is that synaptic influences exerted upon or by the same (or at least very similar) receptors and interneurons are responsible for their response-patterns, although these may be modified by additional inputs.

The response of the $B_2$-neuron differs from that of the $B_1$-neuron in the stronger facilitatory influences from the contralateral tympanal organ, the chief effect of which is to obscure the on-response in the intermediate and high intensity range with frequencies above 10 kHz. The difference in behavior is thus explainable in terms of a supplementary facilitatory influence associated with the same basic connectivity pattern.

Fig. 9 presents the responses of the $B_2$-neuron as a function of frequency and intensity (black bars: ipsilateral responses; light bars: contralateral responses).

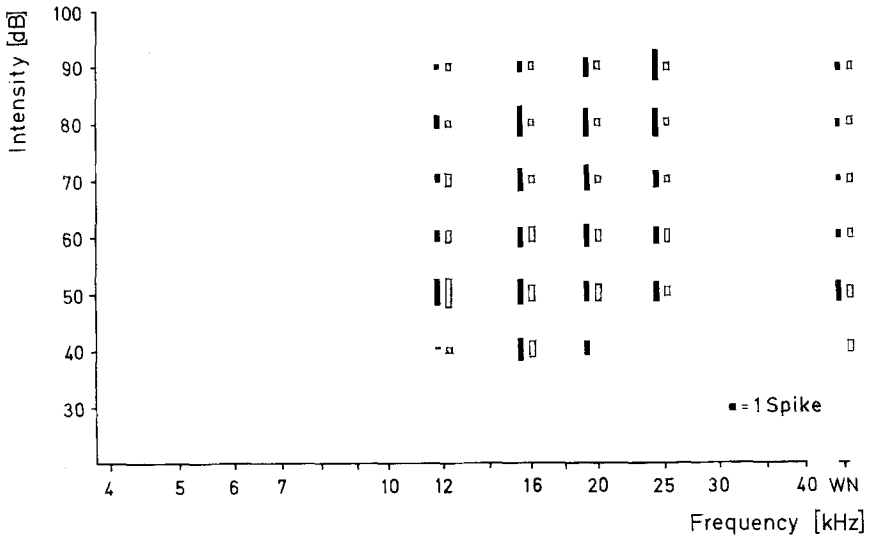


Fig. 10. Responses of a $B_2$-neuron over the frequency/intensity range, recorded from the frontal region of the mesothoracic ganglion ipsilateral to the sound source, before and after elimination of the contralateral tympanal organ. Stimulus duration, 20 ms; repetition rate, 2/s. Black bars: ipsilateral recording before elimination of the contralateral tympanal organ; white bars: after elimination of the organ

Above 10 kHz there are no on-responses. Contralateral stimulation produces smaller responses, as with the $B_1$-neuron. A basic connectivity pattern identical to that of the $B_1$-neuron (as can be discerned from the near-threshold tonic responses, and the on-responses at intermediate and high intensities) becomes apparent when the sound stimulus is presented ipsilaterally after destruction of the contralateral tympanal organ (Fig. 10, white bars). Fig. 10 shows only the responses above 10 kHz. In the intensity characteristics obtained with ipsilateral and contralateral stimulation before and after removal of the contralateral tympanal organ (Fig. 11) it is again possible to see the essential difference between the responses of $B_1$- and $B_2$-neurons; as long as the auditory system is intact, a marked facilitation arising in the contralateral tympanal organ obscures the on-responses (Fig. 11, areas with vertical shading).

Form, position and fiber-course of the $B_2$-neuron differ from those of the $B_1$-neuron in the metathoracic ganglion in two respects: 1. The $B_2$-neuron has two postsynaptic branching regions in the vicinity of the soma; both are in the rostral part of the ganglion, one ipsilateral and the other contralateral. 2. The commissure in which the axon runs from the soma and the postsynaptic branching regions to the opposite side is situated further caudal than in the case of the $B_2$-neuron (Fig. 12, top). In all other details, such as the median branchings and the course of the fibers to the supraesophageal ganglion, the neurons appear similar.

A circuit diagram for the $B_2$-neuron is shown in Fig. 12. Here the "basic connectivity pattern" (cf. also Fig. 8, $B_1$-neuron) is associated with the ipsilateral synapse region. Apart from the fact that the synaptic connectivity is distributed between two branching regions (one ipsilateral and one contralateral), the pattern

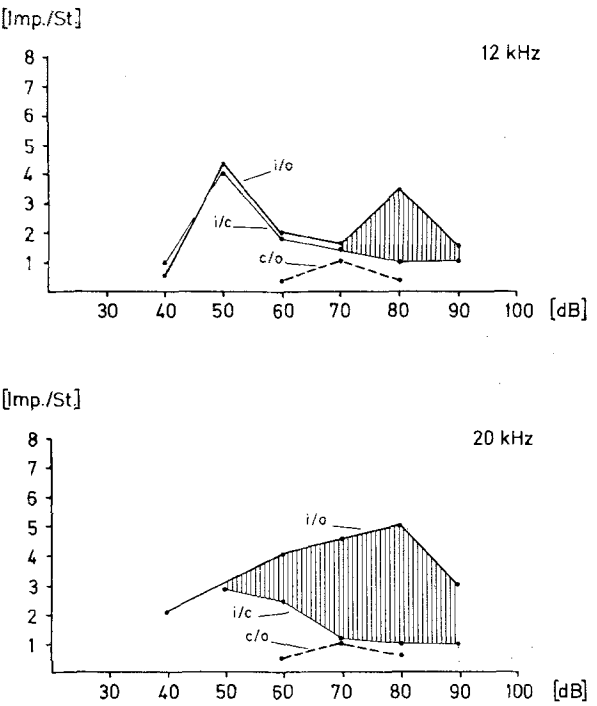


Fig. 11. Intensity characteristics of a $B_2$-neuron with ipsilateral and contralateral sound presentation before elimination of the contralateral tympanal organ (thick solid curve i/o and dashed curve c/o, respectively) and after elimination of the organ (thin solid curve; here only the responses to ipsilateral presentation are shown). The vertically shaded areas indicate the facilitatory influence of the contralateral tympanal organ upon the neuron

is in principle identical to that for the $B_1$-neuron. The two differ in only one detail: the facilitation arising from the contralateral tympanal organ is supplemented in the $B_2$-neuron by another excitatory input in the high-frequency region. It is a likely assumption that the same interneurons supply both B-neurons, so that the same number of interneurons would be involved. In support of this assumption is the fact that during double recordings—that is, when it was possible to record simultaneously from a $B_1$- and a $B_2$-neuron, the action potentials very frequently were coupled, even during spontaneous discharge. Moreover, the two neurons display the same latencies between stimulus and response.

The possibility that the two neurons can influence one another directly is unlikely, since only very small parts of their synaptic regions are superimposed. Moreover, a problem is raised by the fact that the basic connectivity pattern of the $B_1$-neuron is brought about by inputs to the single, contralateral postsynaptic region, whereas that of the $B_2$-neuron arises chiefly in the ipsilateral region. An influence of $B_1$ on $B_2$ might involve the two contralateral postsynaptic regions, but the reverse influence would presumably require transmission from the ipsilateral branches of the $B_2$-neuron to the $B_1$-neuron—a process that one might think would involve serious decremental losses.

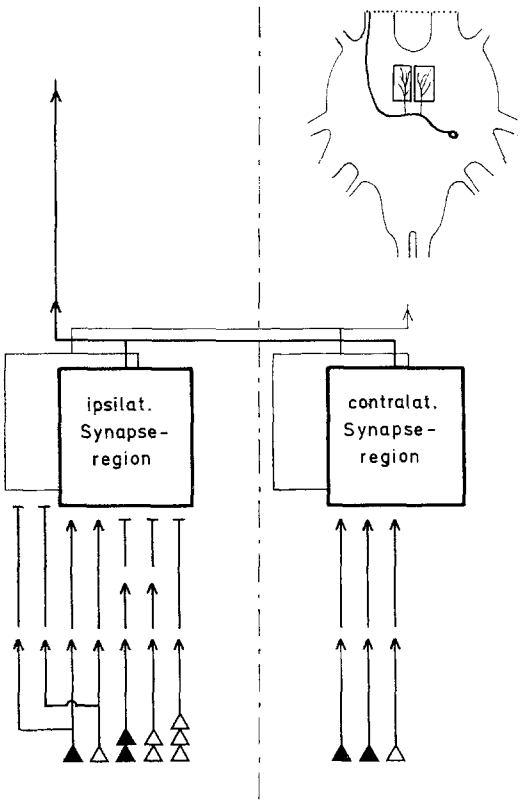


Fig. 12. Connectivity diagram for $B_2$-neurons. The two postsynaptic regions of one neuron are represented by the heavy squares, while those of the contralateral neuron are indicated by light squares. The anatomical diagram (upper right) shows the topography of a $B_2$-neuron in the metathoracic ganglion, with its soma, axon, and postsynaptic branching regions

### *2. The G-Neurons*

The cells termed G-neurons are also found in two types on either side of the ventral cord. The $G_1$-neuron is always situated laterally in the connectives, in the vicinity of the B-neurons, whereas the $G_2$-neuron runs medially in the ventral cord. Both neurons have postsynaptic (dendritic) structures in the mesothoracic ganglion. Whereas the $G_1$-neuron reacts identically to ipsilateral and contralateral stimulation, the responses of the contralateral $G_2$-neuron are entirely suppressed by inhibitory influences from the side ipsilateral to the sound source. These are the factors which distinguish the two G-neurons. Since the topography of the $G_2$-neuron is still unknown, only the $G_1$-neuron will be described in detail here. The G-neurons, together with the B-neurons, constitute the large-diameter auditory neurons in the ventral cord.

Fig. 13 shows typical responses of a $G_1$-neuron to stimulation with white noise and pure tones 20 ms and 100 ms in duration. The responses are phasic in some cases and tonic in others. The entire response range of a $G_1$-neuron in the frequency/intensity field, with both ipsilateral and contralateral presentation of sound, is

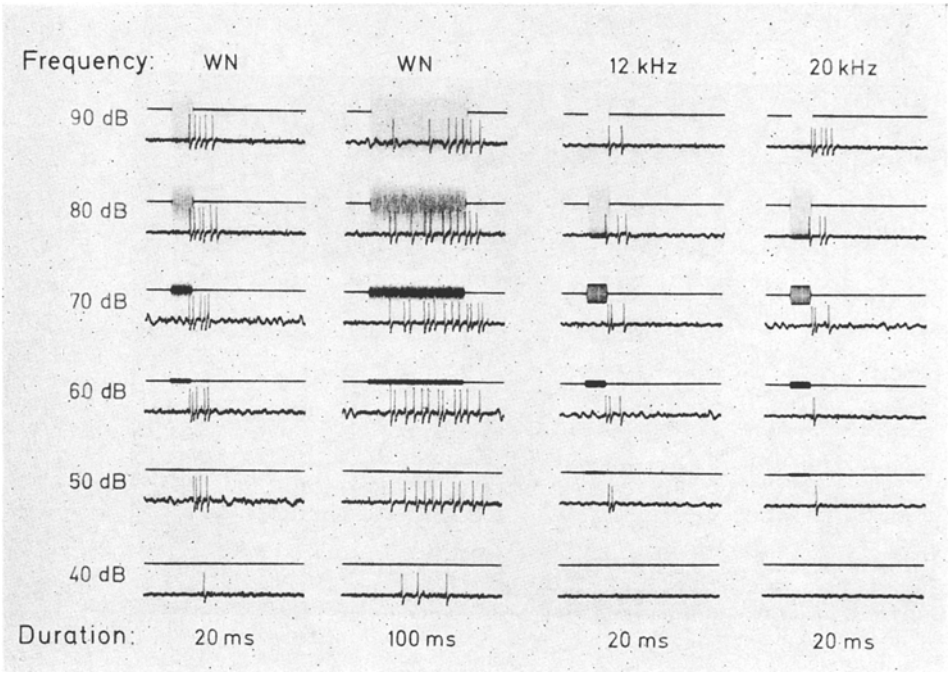


Fig. 13. Response pattern of a $G_1$-neuron recorded from the frontal region of the subesophageal ganglion with ipsilateral sound stimuli (auditory system intact). Stimulus repetition rate, 2/s

shown in the bar diagram of Fig. 14. The most striking aspects of this diagram are as follows. 1. The responses to ipsilateral and contralateral stimulation are about the same. 2. The $G_1$-neurons have relatively high thresholds, as compared with the B-neurons. 3. In the range of low frequencies and high intensities the $G_1$-neurons, like those in group B, do not respond. In the latter case, as with the B-neurons, the response is suppressed by strong inhibitory influences (cf. Fig. 4). This is demonstrated by stimulation with two sound signals.

The receptor influences upon the $G_1$-neuron with the auditory system intact are summarized semidiagrammatically in Fig. 15. Low-frequency neurons with low thresholds, and high-frequency neurons with intermediate thresholds, make facilitatory connections with the $G_1$-neuron. Where the response regions of the receptors overlap (10–12 kHz), the response is especially pronounced. In the low-frequency range the response is suppressed by inhibitory influences from receptor neurons of intermediate threshold. It is still not clear how the phasic to fairly tonic responses of the $G_1$-neurons arise; probably an inhibition acting simultaneously with the facilitation, throughout the response range, is involved.

The characteristic curves in Fig. 16, constructed on the basis of recordings made before and after elimination of the contralateral tympanal organ, make clear that the influence which predominates in forming the response-pattern has its origin in the ipsilateral tympanal organ.

The topography of the $G_1$-neuron resembles in some respects that of the $B_2$-neuron (Fig. 7, right side). As with all the large-diameter auditory neurons

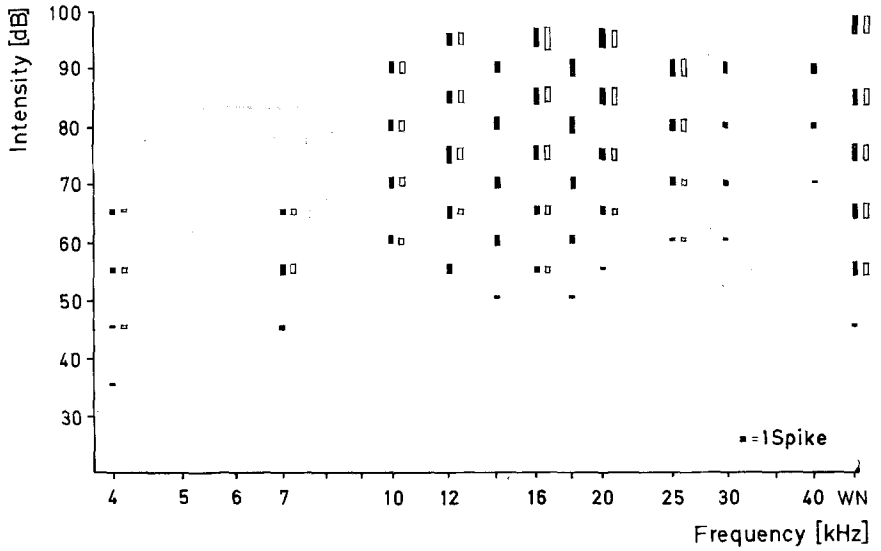


Fig. 14. Responses of a $G_1$-neuron over the frequency/intensity range, recorded from the caudal region of the subesophageal ganglion, with auditory system intact. Stimulus duration, 20 ms; repetition rate, 2/s. Black bars: ipsilateral sound presentation; white bars, contralateral presentation

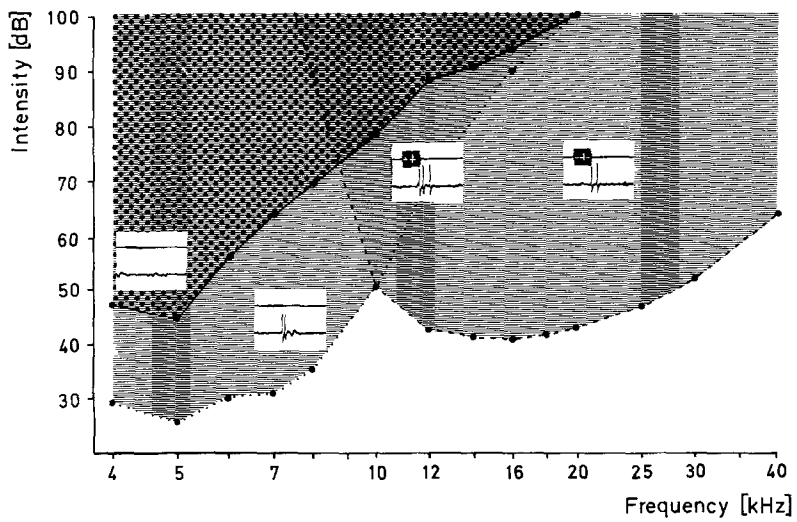


Fig. 15. Semidiagrammatic representation of the receptor-cell activity underlying the various responses of $G_1$-neurons over the frequency/intensity range (auditory system intact). Low-frequency receptors with low thresholds and high-frequency receptors with intermediate thresholds exert a facilitatory influence upon the $G_1$-neurons (horizontally shaded areas); intermediate-threshold low-frequency receptors with an inhibitory effect suppress the responses in the dotted area

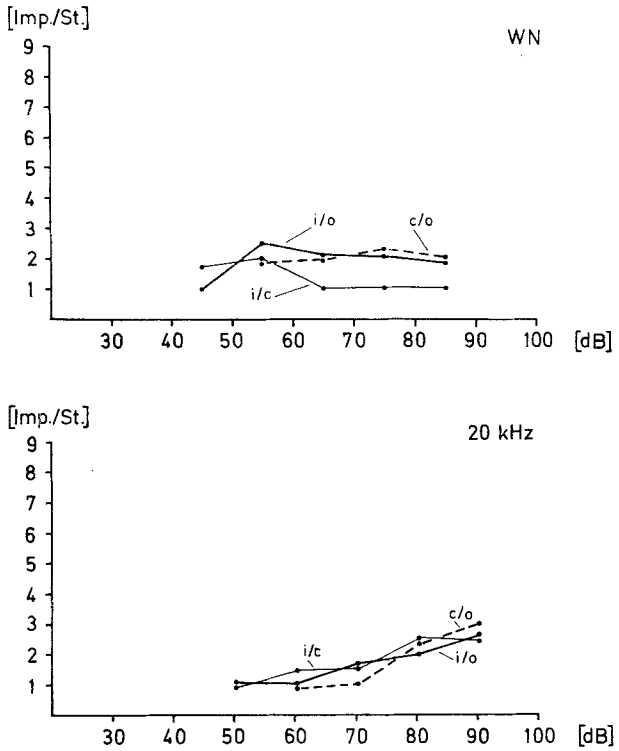


Fig. 16. Intensity characteristics of a $G_1$-neuron with ipsilateral and contralateral sound presentation before (thick solid curve i/o and dashed curve c/o, respectively) and after (thin solid curve i/c; ipsilateral presentation only) elimination of the contralateral tympanal organ

of the ventral cord, the soma is located contralateral to the axon; there are two branching regions near the soma, one on either side of the midline. The median branches and the course of the axon to the supraesophageal ganglion are also very like those of the B-neurons. The G-neurons differ, however, in that their somata lie in the mesothoracic ganglion and the axons divide in the mesothoracic ganglion on the (functionally) ipsilateral side, one trunk ascending to the supraesophageal ganglion and the other descending to the metathoracic ganglion (cf. Rehbein, Kalmring and Schwartzkopff, 1974). In the metathoracic and mesothoracic ganglia the $G_1$-neuron sends out branches laterally; these make direct synaptic contact with motoneurons. In the $G_1$-neuron, as in the B-neurons, the branching regions near the soma are the postsynaptic structures of the neuron. After transection of the ipsilateral meta-mesothoracic connective, which severs the axon trunk running back to the metathoracic ganglion, it is still possible to record a response (though it is diminished) from the $G_1$-neuron in the ipsilateral frontal connective root of the mesothoracic ganglion. Therefore essential inputs to the neuron are in the mesothoracic ganglion. Further, it is implied that some fraction of the influence arising in the ipsilateral tympanal organ travels *via* pathways which cross to the opposite side and pass to the synaptic region of the $G_1$-neuron

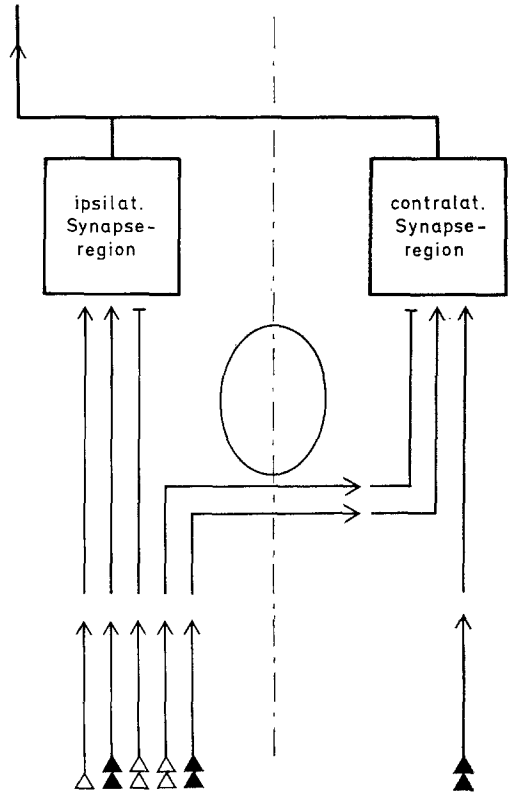


Fig. 17. Connectivity diagram for $G_1$-neurons. The two postsynaptic regions of one of the pair of neurons are shown

by way of the contralateral connective. Experiments in which either the ipsilateral or the contralateral connective between the metathoracic and mesothoracic ganglia are transected and one of the two tympanal organs is destroyed confirm the above results. Latency measurements before and after the surgical interventions indicated that the synaptic connection with the receptor cells in the $G_1$-neuron, as in the B-neurons, involves interneurons.

Fig. 17 shows a corresponding connectivity diagram for the $G_1$-neuron. Since no inhibitory effects arising on a given side are available to reduce the responses in the contralateral neuron, the diagram is limited to the synaptic regions of just one neuron of the pair. The predominant influence, which originates in the ipsilateral tympanal organ, arises in low-frequency receptors with low threshold and high-frequency receptors with intermediate threshold (arrows with one light and two dark triangles, respectively) and is passed by interneurons as facilitation to the ipsilateral synaptic region of the $G_1$-neuron. Strong inhibitory influences from low-frequency receptors with intermediate threshold (arrows with two light triangles) reach both synaptic regions, passing *via* interneurons through both the ipsilateral and the contralateral connective. The contralateral synaptic region is in addition supplied with facilitatory inputs arising in high-frequency neurons of

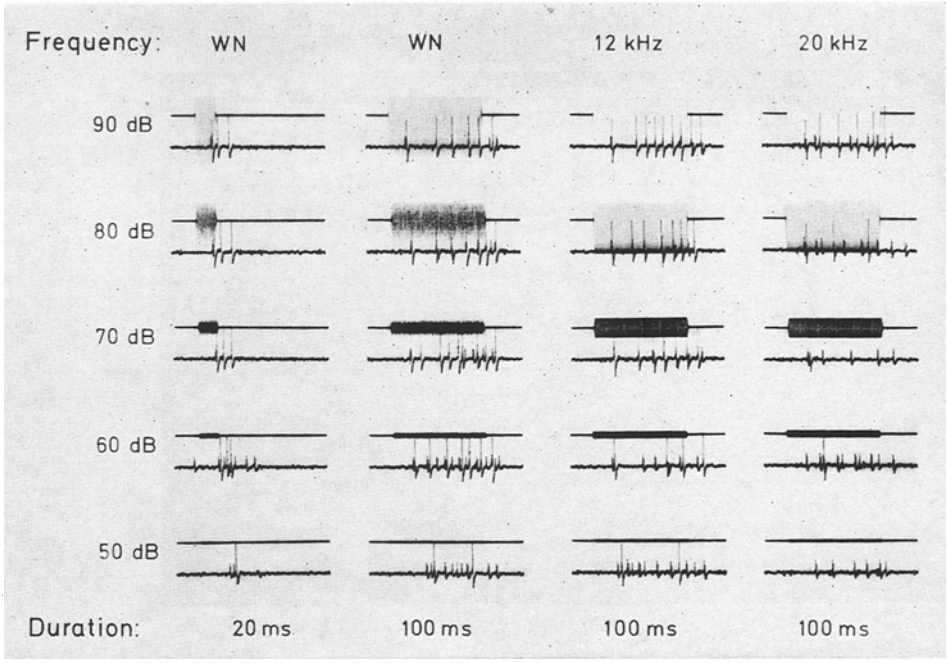


Fig. 18. Response pattern of a $G_1$-neuron (large-amplitude spikes) and a $B_1$-neuron (small spikes) recorded simultaneously from the frontal region of the mesothoracic ganglion; ipsilateral sound stimulation; auditory system intact. Stimulus repetition rate, 2/s

the ipsilateral and contralateral tympanal organs. Wherever connections are made across the midline, an extra interneuron is assumed to be interposed.

A connectivity scheme for the $G_2$-neuron can be derived entirely by the addition of ipsilaterally influenced inhibitory neurons which cause suppression of the responses in the contralateral neuron in each case. The possibility that the $G_1$-neuron is influenced by the B-neurons, and *vice versa*, must be rejected. Fig. 18 shows a double recording from a $G_1$-neuron (large-amplitude spikes) and $B_1$-neuron (smaller spikes). The recording site was the frontal mesothoracic ganglion. As can be seen in these records, the $B_1$-neuron at this point exhibits latencies between stimulus and response which are 1 ms shorter than those of the $G_1$-neuron. This means that the response-pattern of the $B_1$-neuron in the metathoracic ganglion must develop at least 2–3 ms earlier (taking into account the extra conduction time to the mesothoracic ganglion) than that of the $G_1$-neuron. For this reason there can be no "recurrent" influence upon the B-neuron by the $G_1$-neuron (*via* the descending axon trunk); such an effect would require at least another 1 ms conduction time. Nor is it likely that the B-neurons exert a dominant influence upon response-pattern formation in the $G_1$-neuron, since the responses of the $G_1$-neuron are enhanced or at least remain the same when those of the B-neurons change from tonic to on-responses. The differences in detail between the responses of the two categories of neurons make it improbable that influences are exerted in both directions.

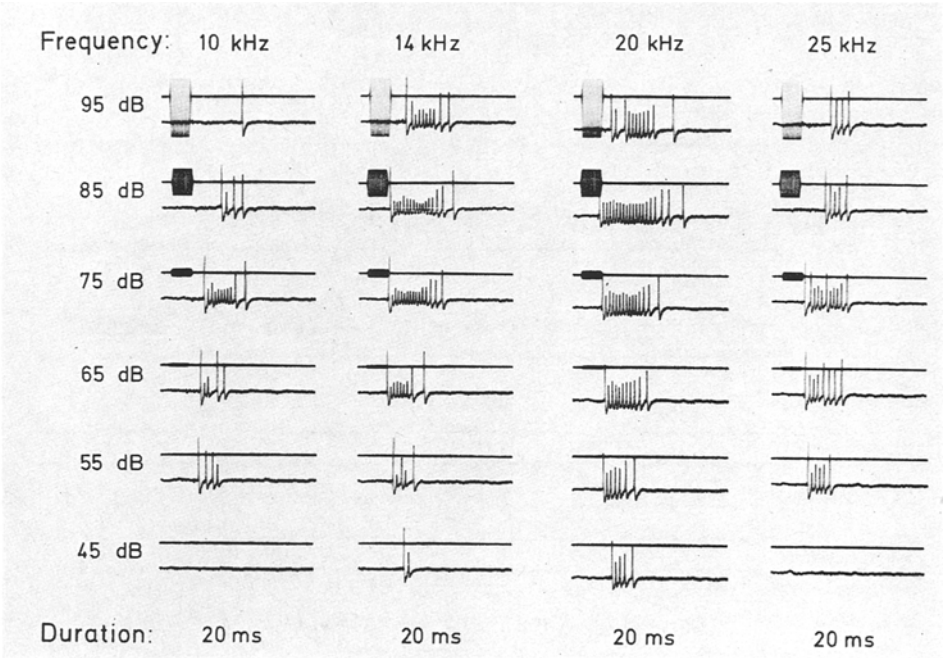


Fig. 19. Response pattern of an $F_2$-neuron, recorded from the frontal region of the subesophageal ganglion, with ipsilateral sound stimulation (auditory system intact). Stimulus repetition rate, 2/s

### *3. The F-Neurons*

There are probably several neurons in this category on each side of the ventral cord. Two of these cells have been demonstrated unambiguously, one with preferential responses below 10 kHz ($F_1$-neuron) and the other responding only above 10 kHz ($F_2$-neuron). As an example of the group, the high-frequency neuron $F_2$ will be described here. The F-neurons display tonic responses, especially at intermediate intensities, to stimuli of different frequencies and 20 ms duration (Fig. 19). However, the response to stimuli of intermediate intensity can outlast the stimulus by three- or fourfold. Above 80–90 dB (and at still lower intensities with frequencies at the edge of the response range) the response is reduced and the latency much increased. The latencies between stimulus and excitation normally amount to 20–22 ms for stimuli at 70 dB, though at an intensity of 95 dB they can be as large as 60 ms. The entire frequency/intensity response field of an $F_2$-neuron is shown in Fig. 20. When the sound stimulus is presented contralaterally, the neurons do not respond.

The intensity-characteristics in Fig. 21, again obtained with ipsilateral and contralateral presentation of sound before and after elimination of the contralateral tympanal organ, reveal strong inhibitory effects between the two sides of the ventral cord. With ipsilateral stimulation after the destruction of the tympanal organ (i/c, thin line), it becomes evident that the response seen with the auditory system intact is much reduced by strong inhibition. The inhibition becomes

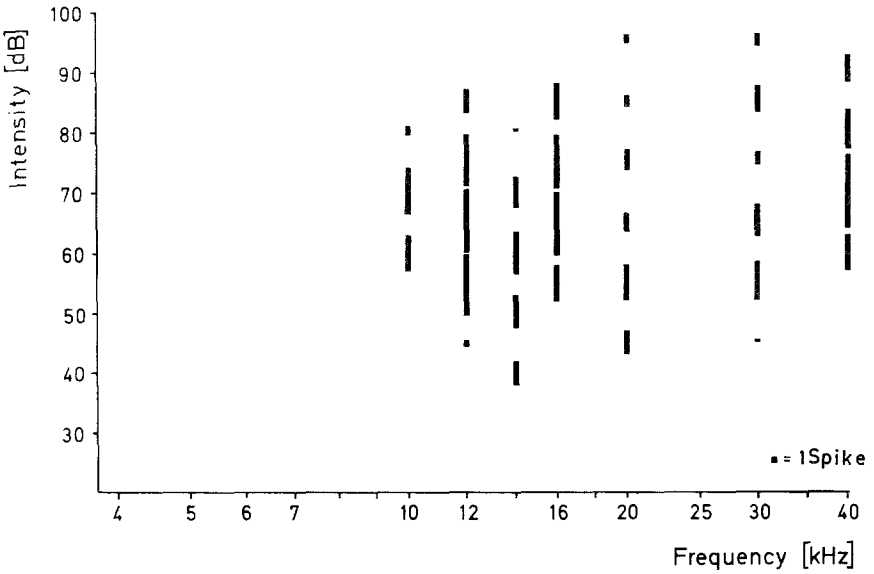


Fig. 20. Responses of an $F_2$-neuron over the frequency/intensity range. Recording from the caudal region of the subesophageal ganglion with auditory system intact. Stimulus duration, 20 ms; repetition rate, 2/s. There is no response to contralateral sound presentation

effective at intensities as low as 60 dB, and increases steadily at higher intensities (Fig. 21, vertically shaded areas). The responses of the contralateral neuron are totally suppressed by inhibitory influences from the ipsilateral side (Fig. 21, diagonally shaded areas).

This reciprocal inhibition brings about a high degree of directional sensitivity in the F-neurons, such that responses appear only to ipsilaterally presented sound. When sound comes from a source on the long axis of the body (either in front of or behind the animal) the responses of the neurons on each side are suppressed completely or at least very much reduced.

Like the $B_1$-neurons, the $F_2$-neurons have a postsynaptic branching region located in the metathoracic ganglion on the contralateral side. The facilitatory input arises only in high-frequency receptors of the ipsilateral tympanal organ. Because of the relatively high thresholds of the F-neurons, the circuit diagram incorporates only receptors of intermediate (Fig. 22; arrows with two black triangles) and higher threshold. The intensity characteristics rise very sharply after removal of the contralateral tympanal organs, but the response of the receptor neurons soon saturates after a brief increase (Römer, pers. comm.); therefore other high-frequency receptors (with high threshold) have a facilitatory effect upon the neuron. These are also shown in the diagram (Fig. 22; arrows with three black triangles). It is necessary that facilitatory interneurons to the contralateral synapse region be interposed, since there are no direct connections of receptor fibers to contralateral structures.

The connectivity diagram is completed by the inclusion of a connection, *via* inhibitor interneurons, between collaterals of the high-frequency receptors and

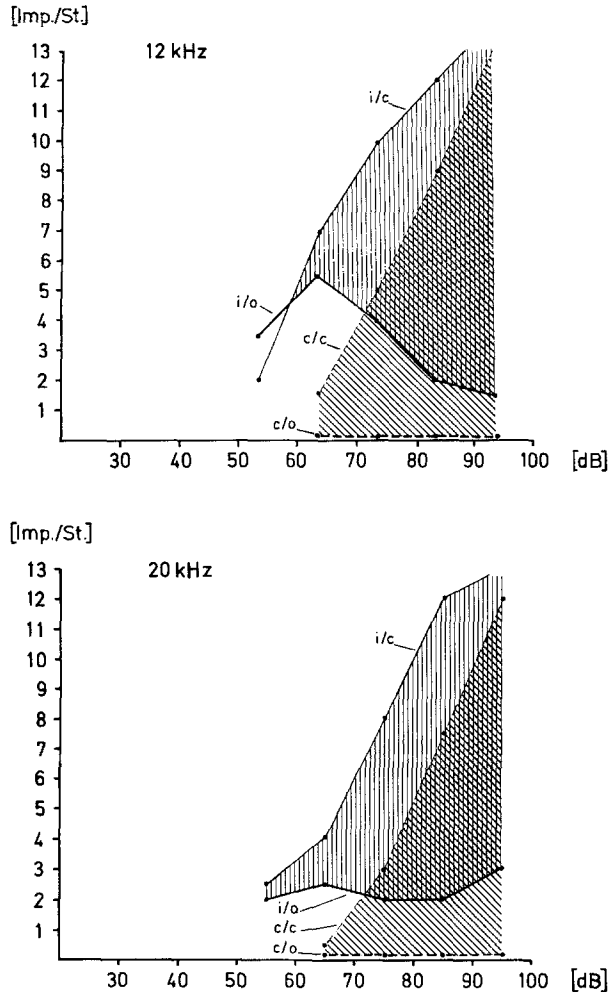


Fig. 21. Intensity characteristics of an $F_2$-neuron with ipsilateral and contralateral sound presentation before (thick solid curve i/o and dashed curve c/o, respectively) and after (thin solid curve i/c and dashed curve c/c) removal of the contralateral tympanal organ. Vertically shaded areas: inhibitory effect of the contralateral tympanal organ during ipsilateral sound presentation; diagonally shaded areas: inhibitory effect of the contralateral tympanal organ during contralateral presentation

the $F_2$-neuron contralateral to them. In Fig. 22 the connectivity diagrams for both sides are shown. No facilitatory influences from the tympanal organ contralateral to an $F_2$-neuron could be demonstrated (Fig. 21). For greater clarity, the facilitatory and inhibitory inputs present in the low-frequency response range have been omitted from the diagram. When these come into play, inhibition prevails and leads to complete suppression of the responses. The connectivity scheme for the $F_1$-neuron can be derived simply by replacing the high-frequency receptors in Fig. 22 by receptors from the low-frequency range.

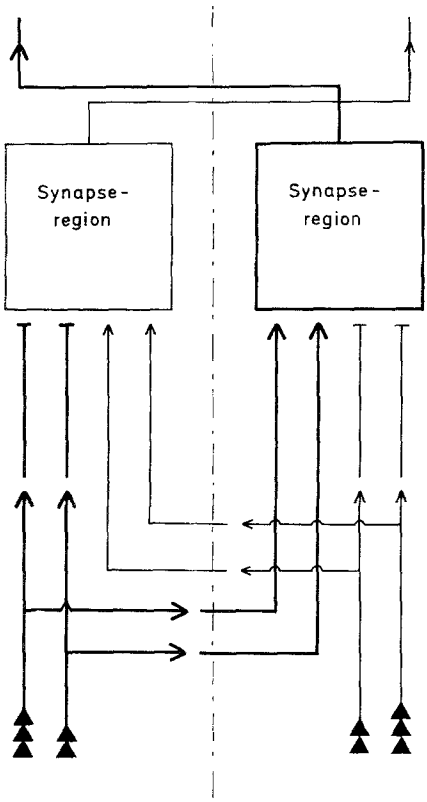


Fig. 22. Connectivity diagrams for the two $F_2$-neurons. Here the heavy lines denote the left neuron and inputs from the left tympanal organ; the thin lines show the corresponding contralateral structures

This connectivity scheme is sufficient to explain the response pattern of the F-neurons. When sound is presented from one side, the ipsilateral neuron is subjected to excitatory, and the contralateral neuron to inhibitory, influences. Since intensities *ca.* 10–15 dB higher are required to excite the neurons of the contralateral side, as a result of attenuation by the animal's body, the inhibition arising on the ipsilateral side is correspondingly stronger and can completely suppress the responses of the contralateral neuron. This remains true even up to the highest intensities, for the inhibition always takes precedence even when it is derived from the higher-threshold receptors. Conversely, excitation of the ipsilateral neuron is correspondingly stronger than the inhibition coming from the contralateral side. As intensity is increased, however, the effect of this inhibition becomes relatively more pronounced, leading to the above-mentioned reductions in response with increase in latency. When the sound source is situated on the long axis of the body, facilitation and inhibition are equally strong in the F-neurons of the two sides, so that they cancel.

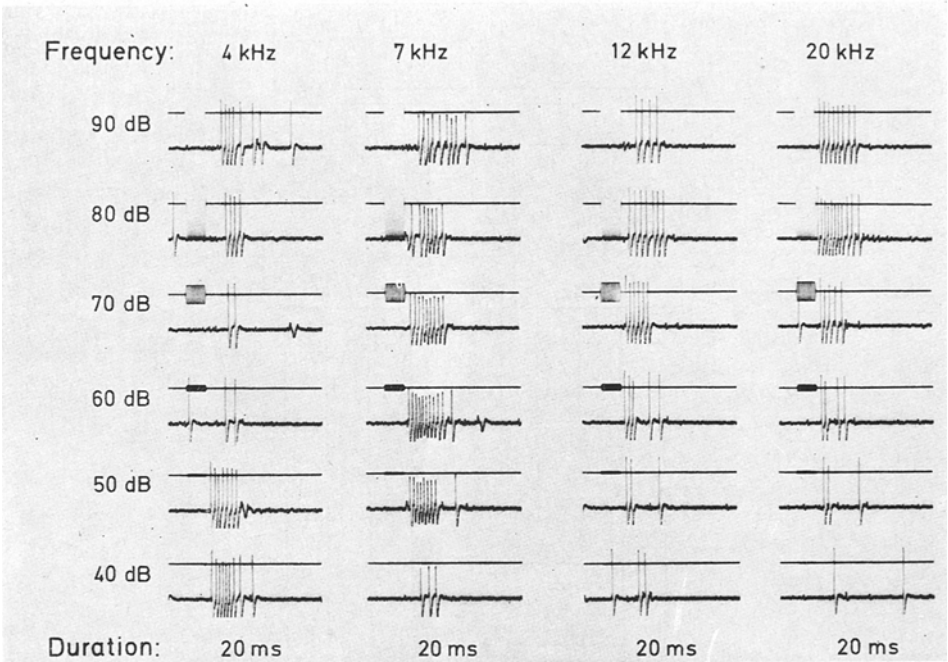


Fig. 23. Response pattern of a $C_1$-neuron, recorded from the caudal region of the subesophageal ganglion with ipsilateral sound presentation (auditory system intact). Stimulus repetition rate, 2/s

### *4. The C-Neurons*

The group designated as C-neurons is distinguished by predominantly tonic responses over the entire response range, under both ipsilateral and contralateral stimulation. Eight such neurons could be identified on each side of the ventral cord. The different response-patterns of these eight neurons can arise from three basic connectivity patterns and are modified by supplementary facilitatory or inhibitory connections.

As an example, the $C_1$-neuron will be described here. Fig. 23 shows the responses of a $C_1$-neuron to stimulation with 20-ms sound stimuli of different frequencies and intensities. It is clear that with different sound frequencies, the maximal response occurs at different intensities. Responses to the "preferred" intensities are characterized by large numbers of impulses, high discharge rates and short latencies. With 4-kHz tones, such responses are elicited in the low-intensity range. At higher frequencies the maxima shift to intermediate and eventually to high intensities. With low frequencies much reduced responses are found just above the response maximum, and with high frequencies the responses below the maximum are greatly diminished. It could be shown that these effects are brought about by strong inhibitory influences, which are manifest not only in the reduction of the responses but also in a considerable increase in latency—especially in the low-frequency range. The latency increase is caused by inhibition,

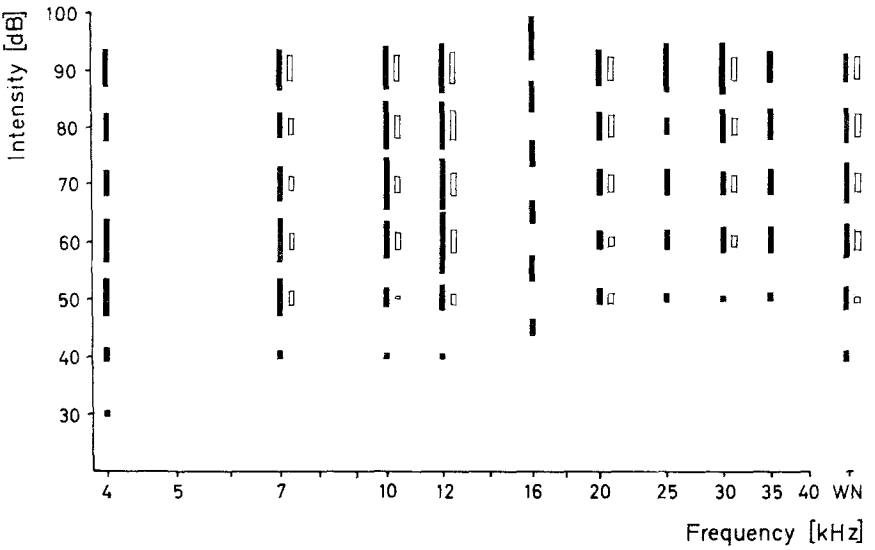


Fig. 24. Responses of a $C_1$-neuron over the frequency/intensity range, recorded from the caudal subesophageal ganglion with auditory system intact. Stimulus duration, 20 ms; repetition rate, 2/s. Black bars: ipsilateral sound presentation; white bars: contralateral presentation

the onset of which is simultaneous with that of the facilitation (cf. the behavior of F-neurons at high intensities).

The responses to ipsilateral and contralateral stimulation, as a function of both frequency and intensity, are shown in Fig. 24. On the contralateral side the responses are reduced by 30–50% as compared with those in the ipsilateral neuron.

On the basis of the intensity characteristics obtained for ipsilateral and contralateral stimulation before and after destruction of the contralateral tympanal organ, it can be shown that the preferred responses are enhanced in the low and intermediate frequency range by facilitatory influences from the contralateral tympanal organ (Fig. 25; thick curve i/o and thin curve i/c for 12 kHz). The inhibitory effects at these frequencies and intermediate or high intensities also arise on the contralateral side. By contrast, the facilitatory influences which bring about a second peak in the characteristic curve at 4 kHz in the high-intensity region come from the ipsilateral tympanal organ and from hair receptors (see Fig. 23). In the high-frequency region (20 kHz) the chief effect of inhibition from the contralateral side is a reduction of the response at low and intermediate intensities. But the responses of the contralateral neuron, with the auditory system intact, are also reduced by inhibitory influences from the tympanal organ.

These results offer a basis for a connectivity diagram of the $C_1$-neuron (Fig. 26). The synaptic regions of this neuron lie in the metathoracic ganglion on the same side as the axon. Inhibitory influences from the contralateral tympanal organ act simultaneously with facilitation from the ipsilateral side. Since, however, this inhibition can reach the synaptic region of the $C_1$-neuron only *via* interneurons,

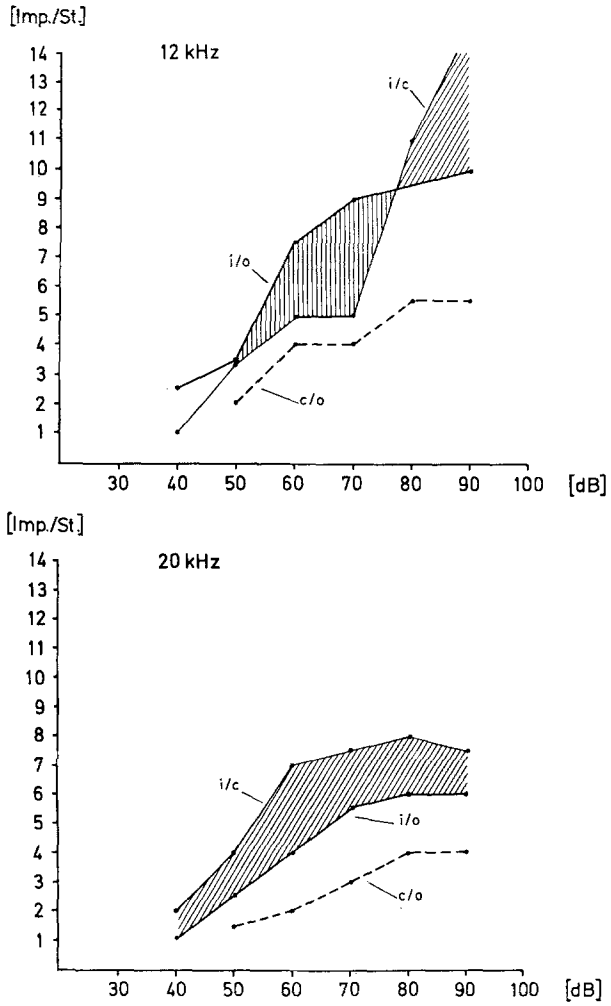


Fig. 25. Intensity characteristics of a $C_1$-neuron with ipsilateral and contralateral sound presentation before (thick solid curve i/o and dashed curve c/o, respectively) and after (thin solid curve i/c; ipsilateral presentation only) removal of the contralateral tympanal organ. Vertically shaded area: facilitatory effect of the contralateral tympanal organ at low frequencies; diagonally shaded areas: inhibitory effect of the contralateral tympanal organ at high frequencies

the facilitatory influences must also be transmitted by interneurons so that the delay in both pathways is the same.

Low-frequency receptors with low and high thresholds, and high-frequency receptors with low and intermediate thresholds, constitute the facilitatory supply from the ipsilateral side to the synaptic region of the $C_1$-neuron. The preferred response regions can be accounted for by supplementary facilitatory influences from contralateral low-frequency receptors with low thresholds. Inhibitory effects originating in low-frequency and high-frequency receptors with intermediate or

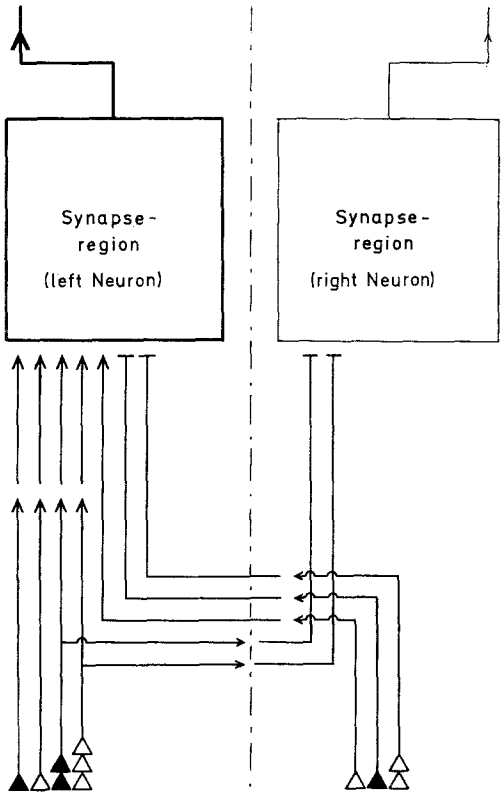


Fig. 26. Connectivity diagram for the $C_1$-neurons. The heavy square represents the post-synaptic region of the left neuron; the light square shows that of the contralateral neuron

low thresholds, respectively, cause the reduction in response. The responses in the contralateral neuron are reduced by inhibitory interneurons supplied *via* collaterals from the ipsilateral high-frequency and low-frequency neurons with intermediate and high (respectively) thresholds.

There is another pair of C-neurons—one cell on each side—that responds with the same kind of discharge as the one just described. The only difference between the two is that the responses of this $C_2$-neuron are always the same on either side of the ventral cord. An identical response by the left and right neurons, independent of the side on which the sound source is located, is possible if the influences responsible for the response-pattern arise in both the ipsilateral and the contralateral tympanal organ.

Fig. 27 shows in diagrammatic form the responses, as functions of frequency and intensity, of the $C_1$- and $C_2$-neurons. The tonic responses are indicated by vertically shaded areas. The intensity characteristics shown below these diagrams are for ipsi- and contralateral responses to tones at 12 kHz, those of the $C_1$-neuron on the left and those of the $C_2$-neuron on the right.

Another nerve cell, the $C_4$-neuron, is characterized by the same basic connectivity pattern and, like neurons $C_1$ and $C_2$, responds over a large part of the

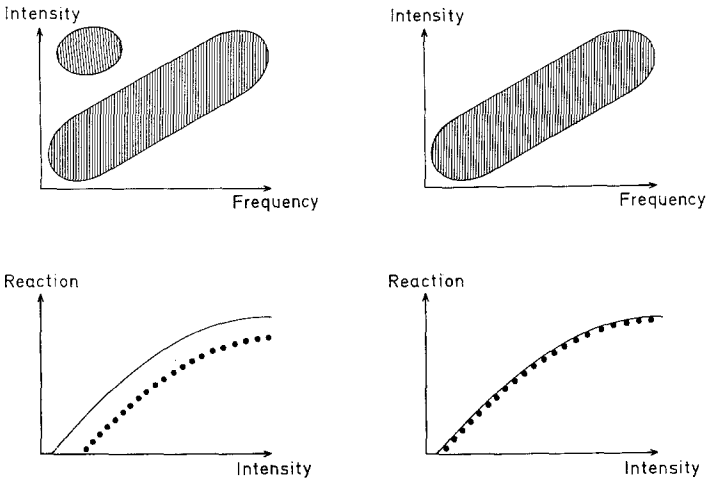


Fig. 27. Upper diagrams: Schematic representation of the preferred response areas in the frequency/intensity field; left, $C_1$- and $C_2$-neurons; right, $C_4$-neurons. Lower diagrams: Intensity characteristics (schematic) for a 12-kHz tone; left, $C_1$-neurons; right, $C_2$-neurons. Solid curves, ipsilateral sound presentation; dotted curves, contralateral presentation

frequency/intensity plot. It differs, however, in that the second peak in the characteristic curve at low frequencies is lacking (Fig. 27, upper right). The sole cause of this difference is the absence of facilitation in this region. The inhibition in the low-frequency range at intermediate and high intensities, which appears very clearly under these conditions, is reminiscent of similar inhibitory influences in the B- and G-neurons.

Another basic response pattern of C-neurons is presented in Fig. 28, in terms of several kinds of diagrams. The responses of neurons of types $C_5$, $C_6$ and $K_1$ to ipsilaterally presented sound increase linearly with the logarithm of the sound pressure over the entire response range of the neurons. With contralateral stimulation, the responses of $C_5$- and $C_6$-neurons are reduced by about 30% (Fig. 28; intensity characteristic on the lower left). In the $C_6$-neurons, responses to tones at frequencies below 10 kHz are weaker than those to tones in the high-frequency region (not shown here). The $K_1$-neurons respond equally to ipsilateral and contralateral stimulation (Fig. 28; lower right). A significant difference between the $K_1$- and C-neurons is that as stimulus duration is increased, the responses of the former saturate, becoming no more prolonged (apart from afterdischarge at high intensities) with stimuli longer than 30 ms. In the C-neurons, on the other hand, the stimulus duration is accurately reflected by that of the response (Fig. 28; upper right).

A basic connectivity scheme adequate for the responses of neurons $C_5$, $C_6$ and $K_1$ is as follows: High-frequency and low-frequency receptors with low, intermediate and high thresholds are connected *via* interneurons to the synaptic regions of the ascending ventral-cord neurons. In the $C_5$- and $C_6$-neurons the reduction of the response to contralateral stimulation is due in part to sound attenuation by the body, and in part to inhibition coming from the side ipsilateral

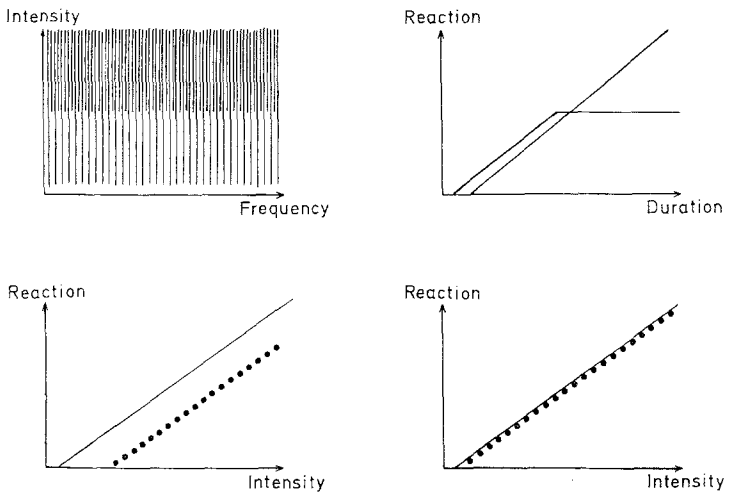


Fig. 28. Upper diagrams: Schematic representation of the responses of $C_5$-, $C_6$- and $K_1$-neurons (left) as a function of frequency and intensity (the denser shaded area indicates stronger reaction). Differing response characteristics of C- and K-neurons as a function of stimulus duration (right). Lower diagrams: Intensity characteristics (schematic) of $C_5$- and $C_6$-neurons (left) and $K_1$-neurons (right) with ipsilateral and contralateral presentation (solid and dotted curves, respectively) of a 12-kHz stimulus

to the sound. The $C_6$-neuron receives an additional inhibitory input from the ipsilateral side in the low-frequency range.

The responses of the ipsilateral and contralateral $K_1$-neurons are the same, regardless of sound-source position, as would follow if the two neurons were connected symmetrically to the receptors on the left and right sides. The saturation of the response at stimulus durations of 20–30 ms is accounted for by inhibitory connections which become effective only after a certain time has elapsed, so that the responses to longer-lasting stimuli are suppressed from that time on.

Fig. 29 gives schematic response diagrams of $C_7$- and $C_8$-neurons. The connectivity pattern of the $C_8$-neuron is very simple; low-frequency and high-frequency receptors with high thresholds make synaptic contact with it *via* interneurons. Since these neurons, too, respond equally to ipsilateral and contralateral stimulation, the connections from either side to the left and right neurons are symmetrical—as in the $K_1$- and $C_2$-neurons (Fig. 29, right).

The connectivity of neuron $C_7$ is the same in the high-frequency region, apart from the absence of bilateral symmetry. In the low-frequency region, it is evident that there are connections to low-threshold low-frequency receptors. Above 50–60 dB supplementary connections with inhibitory interneurons can account for suppression of the responses in this region (Fig. 29, left).

The responses of F- and B-neurons are summarized again in Fig. 30, in diagrams like those for the C-neurons. The F-neurons (upper row) display preferential responses in the intermediate intensity region, when the sound source is ipsilateral. The response to contralateral stimulation is suppressed. The $F_1$-neuron responds in the low and intermediate frequency range, and the $F_2$-neuron responds to inter-

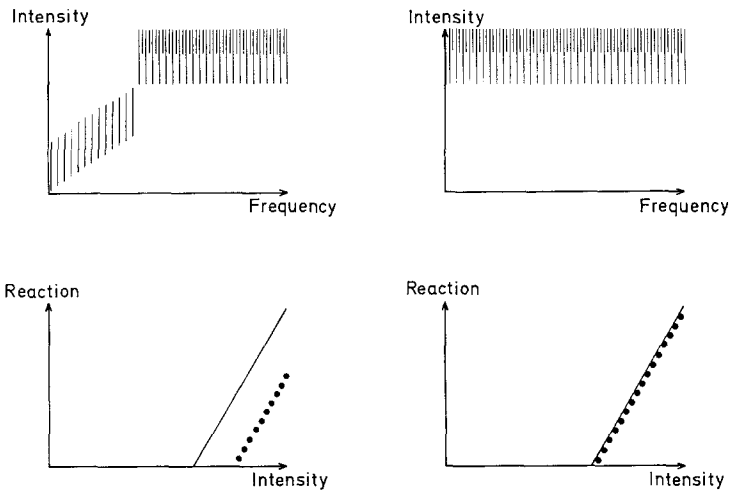


Fig. 29. Upper diagrams: Schematic representation of the responses of $C_7$-neurons (left) and $C_8$-neurons (right) as a function of frequency and intensity (the denser shaded areas indicate stronger reactions). Lower diagrams: intensity characteristics (schematic) of $C_7$-neurons (left) and $C_8$-neurons (right) with ipsilateral and contralateral presentation (solid and dotted curves, respectively) of a 12-kHz stimulus

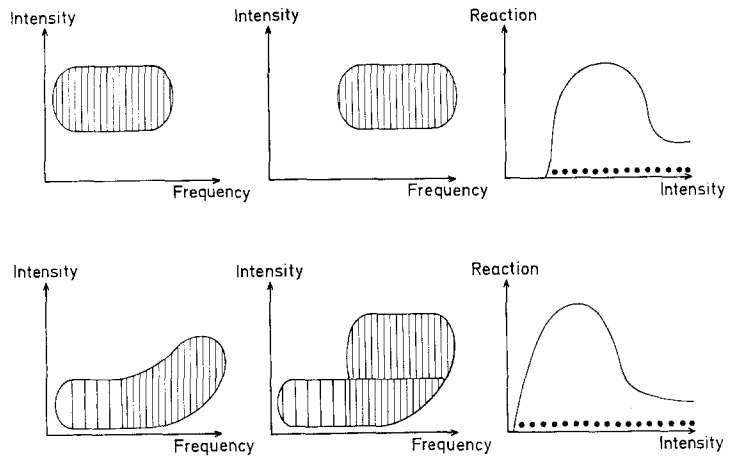


Fig. 30. Schematic representation of preferred response areas in the frequency/intensity field and of the intensity characteristics of F-neurons (upper diagrams) and B-neurons (lower diagrams). $F_1$- and $B_1$-neurons (left); $F_2$- and $B_2$-neurons (center)

mediate and high frequencies. Sound stimuli at 10–12 kHz thus elicit responses from both neurons. The distinctive characteristic of the B-neurons (lower row) is that they respond preferentially in the low-intensity region, stronger responses being produced by frequencies above 10 kHz. The $B_2$-neuron responds in addition to tones of intermediate and high intensity in this frequency range. The response to contralateral stimulation—as with the F-neurons—is suppressed by inhibition.

## D. Discussion

Suga and Katsuki (1961) found a T-shaped auditory neuron in the ventral cord of the bush cricket *Gampsocleis buergeri*. Its axon transmits the same responses to the supraesophageal and to the metathoracic ganglion. The experiments of Rheinlaender and Kalmring (1973) on another tettigonioid, *Decticus verrucivorus*, showed that almost all the auditory information, pre-processed to a great extent in the first synapses of the ventral cord, is also transmitted to the caudal thoracic region. It is supposed that connections between the auditory neurons and motor centers are formed there. Zhantiev and Chukanov (1972) found the situation to be similar in the cricket *Gryllus bimaculatus*.

Using a combined recording and staining method, Rehbein, Kalmring and Römer (1974) showed that in the acridid *Locusta migratoria* the large auditory ventral-cord neurons send multiple branches into non-auditory areas of the CNS. In some cases—*via* the laterally directed branches—there are direct synaptic contacts with motoneurons (Burrows and Rowell, 1973; Hoyle and Burrows 1973; Kalmring, Römer and Rehbein, 1974).

The above results indicate that the pre-processing of auditory information in the synapses of the thorax is highly significant, since this information is not only sent on for further processing in the superordinate association centers of the supraesophageal ganglion, but is also relayed directly to other centers in the ventral cord, including the motor neuropile. Furthermore, these connections in the ventral cord are made by way of neurons ascending to the supraesophageal ganglion, and not simply by receptor fibers or segmental interneurons.

It has also been demonstrated that visual and cercal giant fibers make direct synaptic contact with motoneurons, and interneurons affecting the motor system, in the thoracic ventral cord (Burrows and Rowell, 1973; O'Shea, Rowell and Williams, 1974; Seabrook, 1970, 1971; A. Potente, pers. comm.). The information transmitted by these giant fibers has also undergone considerable pre-processing.

The auditory neurons studied here do not synapse directly with the receptor fibers; the connections are normally made by way of interneurons. This view is compatible with the fact that the postsynaptic branchings of the large ventral-cord neurons, though they are near the auditory neuropile, lie for the most part outside it. Auditory neuropile is formed by the end-branches of the tympanal receptor fibers in the thoracic ventral cord. That the large-neuron postsynaptic branchings are to a great extent outside this region is compatible with the finding that non-auditory receptors, or interneurons associated with them (hair cells of the cerci and the abdominal wall, and receptors on the extremities) make synaptic connections with the auditory ventral-cord neurons (Yanagisawa, Hashimoto and Katsuki, 1967; Kalmring, Rheinlaender and Rehbein, 1972; unpublished results of this author).

In all of the large-diameter ventral-cord neurons so far studied histologically, the cell bodies and part of the—or in some cases the entire—postsynaptic branching are located contralateral to the axon. This is a remarkable feature, considering that the central nervous system of the insect has evolved from a "rope ladder" system. The ganglia on the two sides, joined only by a commissure, develop separately. Since certain other large-diameter fiber systems exhibit peculiarities

like those of the auditory ventral-cord neurons described above, an important neuronal development occurring relatively late in phylogeny can be inferred. The cercal giant fibers and the DCMD-neuron (A. Potente, pers. comm.; O'Shea, Rowell and Williams, 1974) are arranged like the auditory ventral-cord neurons: one finds contralaterally situated cell bodies with postsynaptic branchings in their vicinity, and medially (in some cases also laterally) directed, primarily presynaptic (information-transmitting) branchings in the various ganglia through which the axons run. The latter branchings, in contrast to those near the soma, exhibit swellings.

The receptor neurons of the tympanal organs as a rule make no direct synaptic contact with the ascending auditory ventral-cord neurons. The connection between the two neuron groups is formed by segmental interneurons. One of these (a thoracic low-frequency neuron; Rehbein, Kalmring and Römer, 1974) has been described in detail elsewhere. The physiological data obtained for certain others cannot as yet be accounted for by specific connectivity patterns. It is likely that in some cases, a segmental (i.e., limited to the thorax) interneuron participates in a complex way in the activation of several ascending ventral-cord neurons.

To account for the response patterns of the 14 ascending auditory ventral-cord neurons described here, 6 basic synaptic connectivity patterns have been used. It has been demonstrated that each such pattern is shared by two, or in some cases three, of these neurons. Supplementary circuitry involving an additional interneuron, or the disappearance of a facilitatory or an inhibitory connection, accounts for the variety and distinctness of the responses of the 14 neurons.

Information processing by the ascending ventral-cord neurons thus reveals a principle of economy combined with reliability. The connectivity is economical, since if the same basic pattern is used two or three times, the same segmental interneurons can be employed repeatedly. A few interneurons can even be used for several different basic connectivity patterns. These may include, for example, the inhibitory interneurons with intermediate or high thresholds in the low-frequency range. Therefore only a limited number of interneurons are necessary to form the connectivity patterns inferred in the thoracic ventral cord.

The principle of reliability—through redundancy—is simultaneously evident here, since with a series of stimulus-parameter combinations the same information is transmitted in parallel through the same basic connectivity patterns. For example, after ipsilateral presentation of sound anywhere in the frequency/intensity field, the same responses appear in the $G_1$- and $G_2$-neurons, in the $C_1$- and $C_2$-neurons, and in the $C_5$- and $K_1$-neurons. The same responses to sounds in certain parts of the frequency/intensity field are given by both B-neurons, both F-neurons, the $C_1$-, $C_2$- and $C_4$-neurons, the $C_5$-, $K_1$- and $C_6$-neurons, and the $C_7$- and $C_8$-neurons. There are several neurons ($G_1$, $C_2$, $K_1$, and $C_8$) which respond identically to ipsilateral and contralateral sounds. The $F_1$-, $F_2$- and $G_2$-neurons do not respond to contralateral sounds, and the two B-neurons respond to them only slightly. The duration of the stimulus is "described" by the responses of the eight C-neurons over the whole response range, for sounds on either side; in the B-neurons, however, this information is available only with sounds in the preferred response region (on the ipsilateral side). The K-neurons (there are two in this

category) respond to stimuli for no more than 30 ms. And a number of similar examples could be added to the list.

However, the economy and redundancy characterizing the processing of information here do not mean that the coding need be crude. As the following example will show, appropriate further processing of the auditory information transmitted by the ascending ventral-cord neurons makes possible a relatively precise differentiation of certain combinations of stimulus parameters. If a stimulus of 4 kHz at 40 dB is presented, the neurons of Types $C_1$, $C_2$ and $C_4$ give strong tonic responses with short latencies. This stimulus is also in the preferred response range of the B-neurons, but their responses are phasic-tonic. For neurons of types $C_5$, $C_6$, $C_7$, $K_1$, $F_1$, $G_1$ and $G_2$, the stimulus is in the near-threshold region and evokes only a weak response. The $F_2$- and $C_8$-neurons do not respond at all.

The source of a 4-kHz tone at an intensity of 40 dB can be localized to the left or right of the long axis of the body by the fact that on the side away from the sound source the neurons of Types $C_1$, $C_4$ and $C_7$ give a response reduced by about 30% as compared with that in the same neurons on the other side; the $B_1$-, $B_2$-, $F_1$-, $F_2$, $C_8$- and $G_2$-neurons remain silent. Nor do the contralateral $C_5$- and $C_6$-neurons respond to this stimulus, since it is subthreshold for them. On the other hand, the $C_2$-, $G_1$- and $K_1$-neurons respond, as always, equally strongly on both sides of the body. The responses of the $F_1$-neuron on the same side as the sound source permit a more precise localization. The sound comes from somewhere on the long axis of the body if the $C_1$-, $C_2$-, $C_4$-, $C_5$-, $C_6$-, $C_7$-, $K_1$-, $B_1$-, $B_2$-, $G_1$- and $G_2$-neurons on either side of the body give the same response and if the neurons of Types $F_1$, $F_2$ and $C_8$ do not respond.

The parameter combination 4 kHz, 70 dB can be clearly distinguished from the above stimulus (4 kHz, 40 dB) since the louder stimulus elicits a maximal response in the $F_1$-neuron, while the neurons of types $C_1$, $C_2$ and $C_4$ are strongly inhibited (minimal responses with long latencies). This stimulus is in the "intermediate" response region for the $C_5$-, $C_6$- and $K_1$-neurons. The B- and G-neurons, on the other hand, no longer respond at all at this intensity, nor do the $F_2$-, $C_7$- and $C_8$-neurons. Here the direction of the sound source is represented as described above for the 4-kHz sound at 40 dB.

Just as clearly distinguishable are, for example, 70-dB sounds of other frequencies. With tones at 7 kHz the $C_1$-, $C_2$- and $C_4$-neurons (and of course the $F_1$-neuron as well) give maximal responses with short latencies, as they did with the low-intensity 4-kHz tone, and the B- and G-neurons again respond, but the remainder respond as they did to the louder 4-kHz tone. With tones of 12 kHz, the main difference in the ensemble of responses, as compared with that to the previous stimuli, is that both F-neurons give maximal responses. In this part of the parameter field, the $B_1$-neuron gives on-responses. The G-neurons, too, respond maximally. In this way, all the parameter combinations that can excite the auditory system may be identified by the particular combination of strongly-responding, weakly-responding, and silent neurons with which they are associated. Precision of this sort is only possible, though, if the responses of all or at least most of the 14 ventral-cord neurons are included in the analysis. Resolution of that possibility awaits further study of the superordinate auditory centers. In a comprehensive study Adam (1969) described and analyzed in detail a number of

neurons of the supraesophageal ganglion. Nevertheless, a large fraction of these cells can be placed in the category of ascending ventral-cord neurons, and a great deal remains to be learned about information processing in the superordinate centers.

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