27 Olson, G. C., and Krasne, F. B., The crayfish lateral giants are command neurons for escape behavior. Brain Res. *214* (1981) 89–100.
28 Reichert, H., Control of sequences of movements in crayfish escape behavior. Experientia *44* (1988) 395–401.
29 Ritzmann, R. E., Motor responses to paired stimulation of giant interneurons in the cockroach *Periplaneta americana*. II. The ventral giant interneurons. J. comp. Physiol. *143* (1981) 71–80.
30 Ritzmann, R., The cockroach escape response, in: Neural Mechanisms of Startle Behavior, pp. 93–131. Ed. R. C. Eaton, Plenum Press, New York 1984.
31 Ritzmann, R. E., and Pollack, A. J., Identification of thoracic interneurons that mediate giant interneuron-to-motor pathways in the cockroach. J. comp. Physiol. *159* (1986) 639–654.
32 Spiram M. E., Parnas, I., and Bergmann, F., Histological and electrophysiological studies on the giant axons of the cockroach *Periplaneta americana*. J. exp. Biol. *50* (1969) 629–634.
33 Tobias, M. L., and Ritzmann, R. E., Responses of mesothoracic motor neurons to giant interneuron stimulation in the cockroach. J. comp. Physiol. *154* (1984) 633–640.
34 Vardi, N., and Camhi, J. M., Functional recovery from lesions in the escape system of the cockroach. I. Behavioral recovery. J. comp. Physiol. *146* (1982) 291–298.
35 Vardi, N., and Camhi, J. M., Functional recovery from lesions in the escape system of the cockroach. II. Physiological recovery of the giant interneurons. J. comp. Physiol. *146* (1982) 299–309.
36 Westin, J., Responses to wind recorded from the cercal nerve of the cockroach *Periplaneta americana*. I. Response properties of single sensory neurons. J. comp. Physiol. *133* (1979) 97–102.
37 Westin, J., Langberg, J. J., and Camhi, J. M., Responses of giant interneurons of the cockroach *Periplaneta americana* to wind puffs of different directions and velocities. J. comp. Physiol. *121* (1977) 307–324.
38 Wiersma, C. A. G., Giant nerve fiber system of the crayfish. A contribution to comparative physiology of synapse. J. Neurophysiol. *10* (1947) 23–38.
39 Wine, J. J., and Krasne, F. B., The organization of the escape behavior in the crayfish. J. exp. Biol. *56* (1972) 1–18.
40 Wine, J. J., and Krasne, F. B., The cellular organization of crayfish escape behavior, in: The Biology of Crustacea, vol. 4, pp. 241–292. Eds D. E. Bliss, H. Atwood and D. Sandeman. Academic Press, New York 1982.
41 Yost, W. A., and Nielsen, D. M., Fundamentals of Hearing. Holt, Rinehart and Winston, New York 1977.
42 Zill, S. N., Proprioceptive feedback and the control of cockroach walking, in: Feedback and Motor Control in Invertebrates and Vertebrates, pp. 187–208. Croom Helm, London 1985.

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Behavioral and neuronal mechanisms of cricket phonotaxis

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Summary. The auditory communication of crickets provides a model system for the analysis of the neuronal mechanisms underlying complex behavior. The song of male crickets attracts females. The necessary and sufficient parameter of the song for the female phonotaxis has been determined by a quantified behavioral analysis. Neuronal correlates of this pattern recognition exist in the cricket brain and give rise to a hypothesis on the mechanism of song pattern recognition. Causal relationships between the orientation of a cricket during phonotaxis and the activity of single identified neurons were found by monitoring and deactivating single neurons during behavior. The different roles of various identified neurons for sound localization have been tested by this method. The plasticity of the auditory system at both the behavioral and at the neuronal level has been studied after amputation of one ear, and a mechanism for sound localization with only one ear is proposed.
Key words. Insects; crickets; phonotaxis; auditory neurons; sound localization; song pattern recognition.

Male crickets attract their females by the calling song. If a female is sexually responsive and within the acoustic range she will approach the male. As Johannes Regen [18] showed, the phonotactive behavior is elicited only by sound, and other cues are not necessary. If the female arrives at the singing male, the behavior will continue with antennal contacts, the male courtship song, copulation and male guarding. Up to now only the initial steps of the behavioral sequence, the singing of the male and the phonotactic response of the female, have been studied in detail. The female faces two basic problems in analyzing the communication signal: the recognition of the calling song and the localization of the singing male. Both aspects will be described here from the behavioral and from the neurobiological point of view.

Recognition of the conspecific song

The phonotactic behavior of crickets can be studied during tethered flight [14] or during walking by using a walking compensator [28]. In this situation, the animal walks unrestrained on a sphere and in response the sphere is moved in the opposite direction, so that the animal is maintained at a constant position. As a result the angle and the velocity of walking can be measured for a long time. The walking compensator is placed in an anechoic chamber in which two loudspeakers are placed at different angles. With an attractive auditory stimulus the female walks in the direction of the active loudspeaker, meandering around the midline, and immediately follows each change of the sound direction (fig. 1).

Using this system Thorson et al. [27] tested different sound models for their attractiveness in phonotaxis (fig. 2). A typical cricket calling song consists of a series of chirps that are divided into single sound groups called syllables with a carrier frequency at about 5 kHz. Each syllable is about 18 ms long and is repeated in a chirp 4–5 times with an interval of about 35–40 ms. Changing all parameters systematically Thorson et al. [27] found that in *Gryllus campestris* there is no tracking of a burst (an unmodulated 5 kHz tone). Increasing the number of syllables in a chirp up to a continuous trill does not abolish a phonotractic response, and the length of the syllable and the length of the pause between syllables can be varied over a wide range. The only critical parameter was the syllable repetition interval which was phonotactically effective only between about 25 and 55 ms (fig. 5). From these results they concluded that the necessary and sufficient parameter for eliciting phonotaxis is the syllable repetition interval (SRI). Therefore, the recognition of the conspecific signal can be tested by electrophysiological methods without testing all temporal patterns which are physically possible.

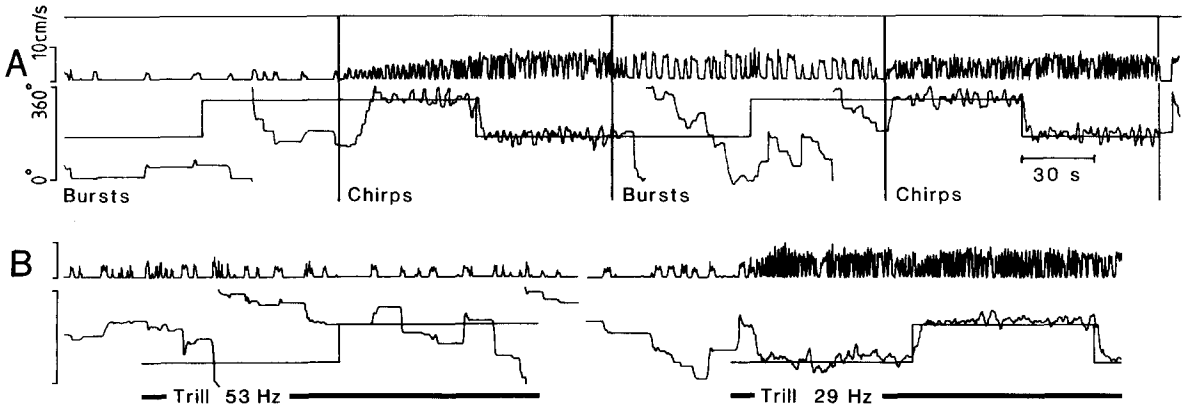

Figure 1. Tracking performance of a cricket on a walking compensator in response to different sound patterns. Each upper trace shows walking speed; each lower trace shows the angle of walking direction, horizontal lines mark the angular position of the active loudspeaker. Each sound condition is presented for 2 min with a switch of loudspeakers after 1 min. *A* Alternate presentation of correct chirps and sound bursts (illustrated in fig. 2); *B* presentation of trills at two different syllable rates (from Huber and Thorson[9], modified).

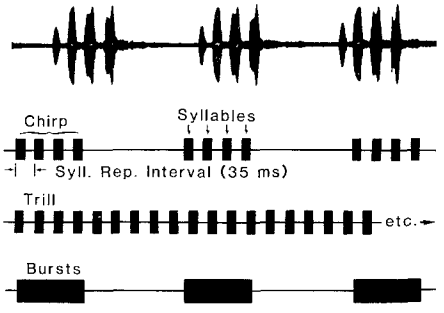

Figure 2. Natural and artificial cricket songs. The male cricket calling song (top) consists of repeated chirps, made up of brief pulses (syllables) with a carrier frequency at about 5 kHz; other traces show various artificial temporal patterns of song used to investigate which features of the song are critical in eliciting female phonotaxis (from Huber and Thorson[9], modified).

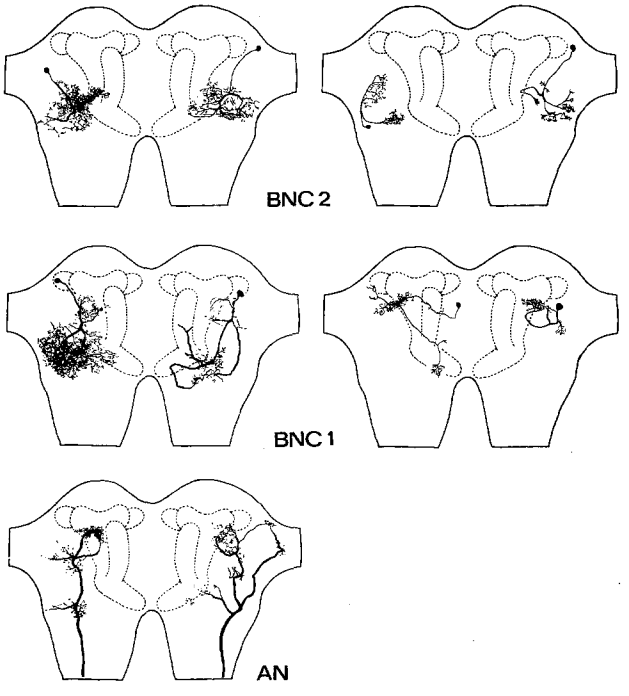

Figure 3. Auditory neurons in the brain. Neurons shown in each left half of the brain are tuned to 5 kHz, neurons in the right half are tuned to higher sound frequencies; AN, ascending neurons; BNC1, brain neurons class 1; BNC2, brain neurons class 2; dashed lines mark the mushroom bodies, specialized neuropil in the insect brain (from Schildberger[21], modified).

Neuronal correlates for recognition

Sensory fibers originate in the tympanal organ in the tibia of the foreleg and project into the prothoracic ganglion where they end ipsilaterally in an area called the auditory neuropil. Auditory receptor cells are tonotopically arranged in the ear, and sensory neurons tuned to the carrier frequency of the calling song are represented in greater numbers than others[16]. In the ganglion, Wohlers and other authors[6, 13, 15, 17, 30 - 32] identified six types of auditory interneurons: two bilateral organized local neurons, the Omega cells (ON1, ON2); two neurons with an ascending axon (AN1, AN2); a through-conducting fiber (TN1) and a descending neuron (DN1). Some of them are sharply tuned to the carrier frequency of the calling song (ON1 and AN1) while the others exhibit more broadly banded tuning. Testing these cells with similar temporal patterns to those used in the phonotaxis experiments revealed that all the cells respond to a wide range of patterns, and no cell responded selectively to or copied only the pattern of the calling song.

The cells AN1 and AN2 ascend to the brain and terminate there in specific areas[4, 21]. Several different types of auditory neurons have been identified in the brain[3, 21, 22]. One may classify these cells on the basis of anatomical criteria such as arborization areas and overlap of projection fields. One can formulate a first hypothesis on the auditory pathway in the brain (fig. 3). The auditory information may pass from the ascending neurons via a class of brain neurons (BNC1) that overlap with them, to a second class (BNC2) that do not overlap with ascending, but do with BNC1 neurons. From here the information may be transferred to descending cells. The hypothesis is supported by the latencies of the respective cell types: ascending neurons having the shortest, followed by BNC1, BNC2 and descending cells with the longest latencies.

Brain neurons were tested for pattern selectivity with model songs that were identical in their temporal pattern, carrier frequency and sound intensity with those of the models tested in the behavioral experiments. Brain cells show a variety

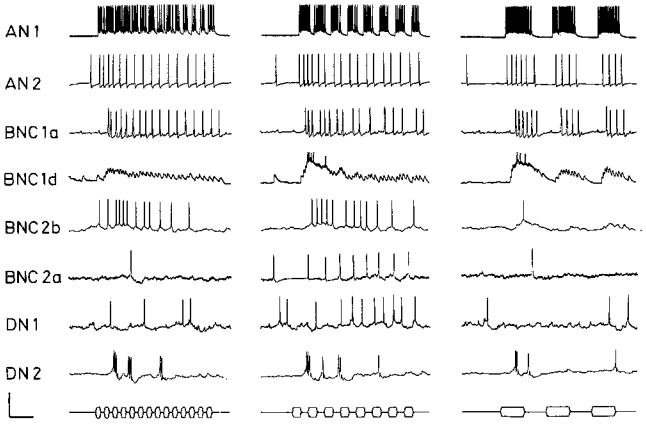

Figure 4. Responses of different brain neurons to chirps with varying syllable repetition interval. Only AN1 copies all patterns; local (BNC) and descending (DN) brain neurons show specific filter properties. SRIs were 18 ms (left), 34 ms (middle) and 98 ms (right) at 5 kHz and 80 dB SPL; calibration: abscissa = 80 ms, ordinate 50 mV (traces 1–3) and 25 mV (traces 4–8); identification labels correspond to neurons of classes shown in figure 3, DN, descending neuron (from Schildberger[22]).

of different response types to the patterns tested (fig. 4). Two response properties were analyzed in detail: the ability of the cells to copy the temporal pattern and the response magnitude, e.g. the number of action potentials per chirp. AN1 cells copy exactly the temporal structure of songs that elicit phonotaxis. When testing different patterns, it was found that the accuracy of copying increases with increasing syllable repetition interval (SRI), the parameter most relevant for recognition, and there is significant copying of temporal patterns that elicit phonotaxis as well as of patterns that are not attractive. BNC1 cells copy less precisely and significant copying is achieved only at long SRIs; BNC2 neurons do not copy the temporal pattern of the chirp. Thus, no evidence was found that conspecific temporal patterns produce higher degrees of synchronization than unattractive ones.

Various relationships between response magnitude and SRI exist in the different neurons. Varying the SRI in chirps with constant energy and constant length has no influence on the response magnitude of ascending and most BNC1 neurons. But in some BNC2 neurons only intermediate SRIs elicit a response while longer and shorter ones fail to do so. This temporal selectivity in the neurons correlates precisely with the behavioral selectivity found in phonotactic experiments and the neurons could be addressed as band-pass cells (fig. 5). In addition, there exist cells in the brain that respond to intermediate and long SRIs but not to short ones (low-pass) and others that respond to intermediate and short but not to long SRIs (high-pass).

On the basis of these findings one may speculate about different models that could be responsible for recognition of the temporal pattern of the conspecific song[9]. The first model, discussed for about 20 years for birds, frogs and crickets, suggests that the female has an inbuilt template to compare with the temporal pattern of incoming male song by some kind of cross-correlation. One possible mechanism would be to synchronize the activity of the presumed internal pattern

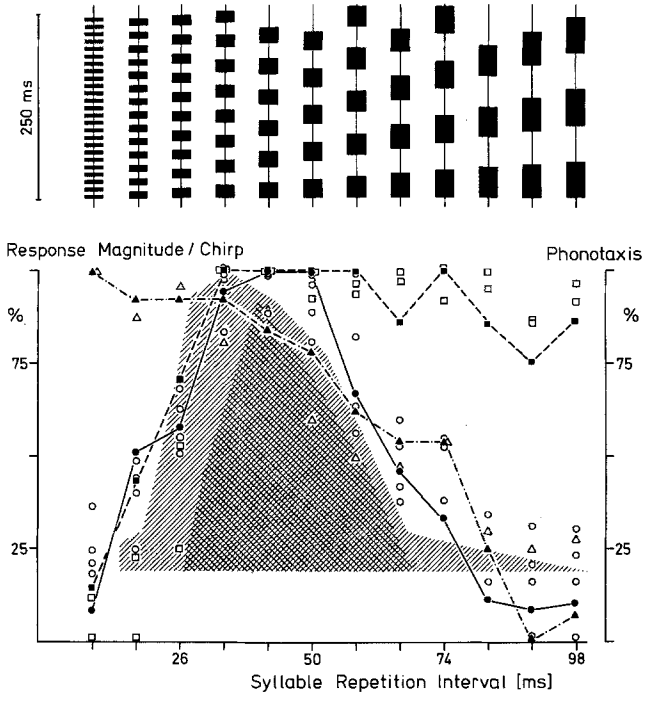

Figure 5. Relative response magnitude of auditory brain neurons with specific filter characteristics to chirps varying in syllable repetition interval. Data points with squares are from the neuron BNC1d, triangles from BNC2b and circles from BNC2a. Closed symbols for three such cells are connected by lines. The open symbols show the responses of other examples of these identified neurons in different animals to indicate variability. The hatched areas show the relative effectiveness (right ordinate) of the syllable repetition intervals in eliciting phonotaxic tracking in *Gryllus campestris* and *Gryllus bimaculatus* (cross-hatched); sound frequency, 5 kHz, intensity, 80 dB SPL, the stimulus configurations are shown on top (from Schildberger[22]).

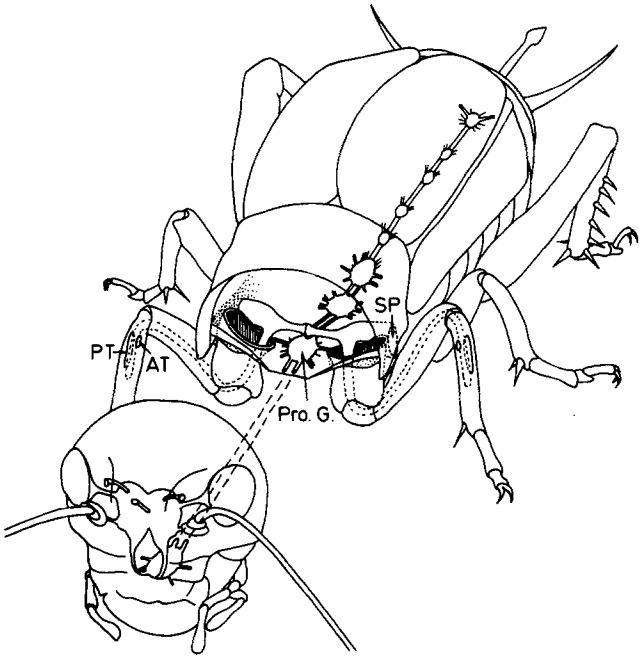

Figure 6. View of a female cricket, showing the H-shaped tracheal tube; funnel-shaped branches lead to openings (spiracles, SP) on the sides of the prothorax; the lower branches descend through the forelegs to the region of the tympana (posterior PT and anterior AT); a chain of 10 ganglia runs longitudinally inside the ventral body wall; fibers of the auditory nerve run through the foreleg and terminate ipsilaterally in the prothoracic ganglion (Pro. G.); (courtesy of T. Weber).

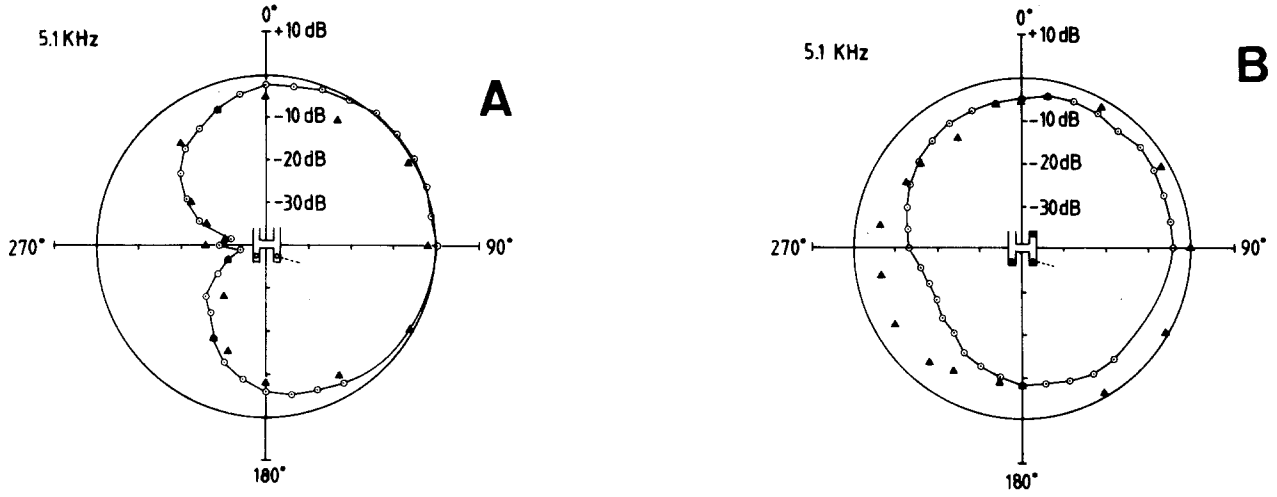

Figure 7. Directionality of the cricket ear obtained with different methods. *A* Directional diagram with all tympana and spiracle openings intact; *B* with ipsilateral acoustic spiracle occluded; open dots are from vibration amplitudes of the posterior tympanal membrane, closed triangles are from receptor cell responses (from Larsen et al.[12]).

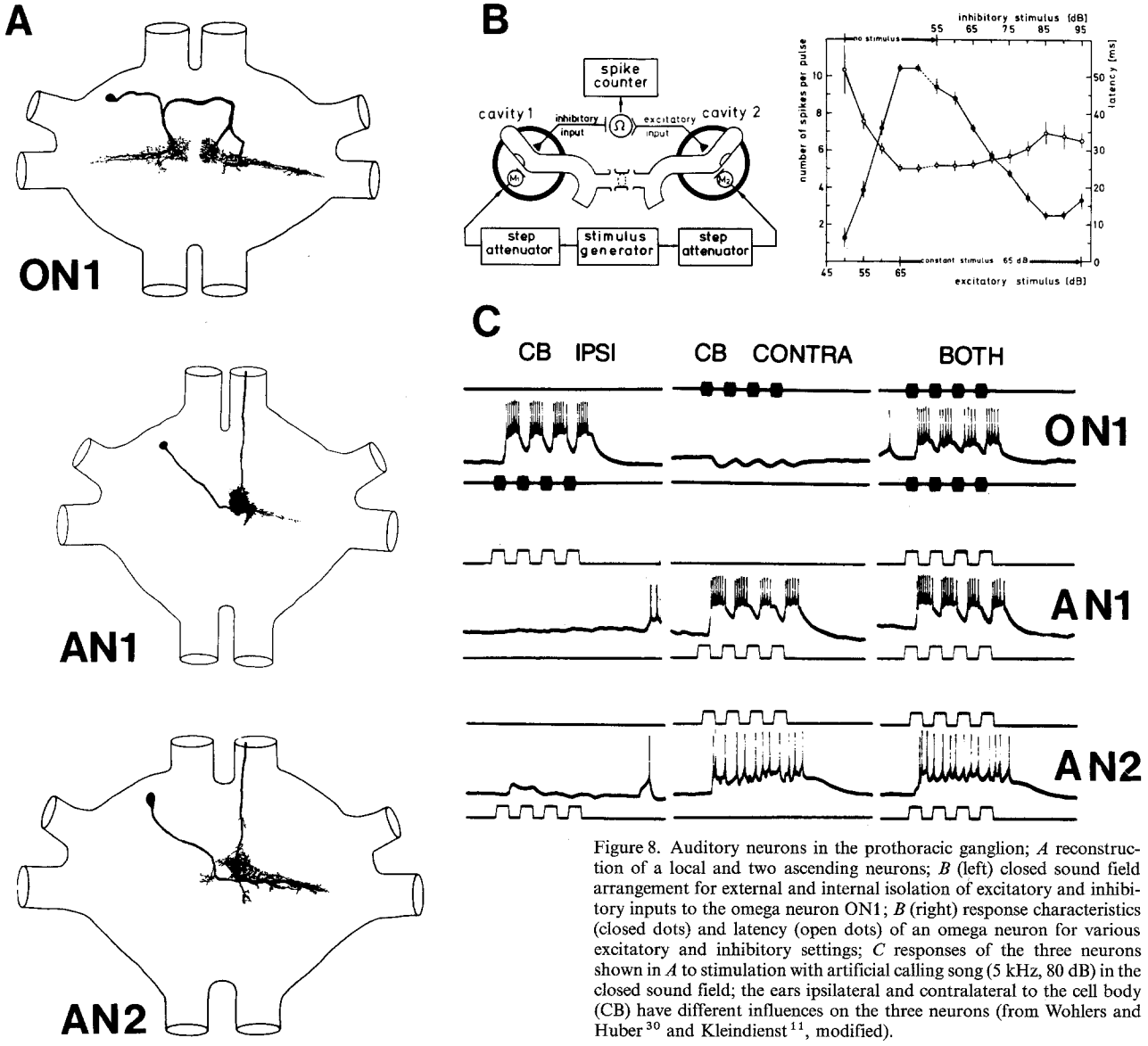

Figure 8. Auditory neurons in the prothoracic ganglion; *A* reconstruction of a local and two ascending neurons; *B* (left) closed sound field arrangement for external and internal isolation of excitatory and inhibitory inputs to the omega neuron ON1; *B* (right) response characteristics (closed dots) and latency (open dots) of an omega neuron for various excitatory and inhibitory settings; *C* responses of the three neurons shown in *A* to stimulation with artificial calling song (5 kHz, 80 dB) in the closed sound field; the ears ipsilateral and contralateral to the cell body (CB) have different influences on the three neurons (from Wohlers and Huber[30] and Kleindienst[11], modified).

generator with the incoming activity. As a result, neuronal responses to attractive temporal patterns should be better synchronized with the stimulus structure than those that are ineffective. Though this cannot be supported by the current data on brain neurons it remains a possible solution.

A second model for recognition of a particular temporal rate was discussed by Reiss [19]. It invokes two pathways by which the signal reaches the recognizer. One pathway involves a specific delay and if the recognizer requires temporal coincidence of events from both pathways then it responds only to certain temporal patterns. The model should recognize higher multiples of the designed rate, because coincident events from the two pathways occur for such multiples as well. But these multiples have been found neither in the behavior nor in the responses of the brain cells.

Another model of recognition, as proposed by Capranica and Rose [5], depends on the discovery in crickets and frogs that band-pass cells are found in close association with high-pass and low-pass cells. Perhaps the nervous system resorts to two steps. First, two sets of intermediate cells decide whether a rate is above a given rate (high-pass) or below (low-pass). In the second step recognizer cells respond only if both the high-pass and the low-pass cells are active. So this model describes a logical AND operation and it is the only one of the three for which we have evidences from behavioral and neurophysiological data, at least in the cricket.

Sound localization

In behavioral experiments, Rheinlaender and Blaetgen [20] showed that crickets always turn to the side of an active loudspeaker when the sound source is at least 20 degrees to the side of the body axis. The percentage of turnings to the 'wrong' side increases at smaller angles. Thus, there is a frontal sector of ambiguity of about 30–40 degrees. This may explain the meandering walking style of a freely moving animal. Because the ears possess a directional sensitivity, the animal turns towards the ear most strongly stimulated thereby crossing the ambiguity sector. At a certain point the other ear becomes more stimulated and the animal turns back to the former side and so on. Measuring the angular velocity in a fixed animal in an open loop situation, Stabel and Wendler [26] found that the turning tendency towards the active loudspeaker increases linearly with increasing stimulus angles up to about 40–50 degrees. The hearing system with its two ears therefore has to provide sufficient information about the direction of sound for these stimulus angles.

The peripheral sound detection system of the cricket, including tympanal membranes, transduces incident sound to excite an array of auditory receptor cells and in turn central nerve cells. The ears are located in the tibiae of the forelegs just below the 'knees' (fig. 6). Two ears are coupled by an air-filled tube and major branches of the tube go to openings on the two sides of the body. Sound pressure acts upon the outside of the tympana directly and the inside via these openings and tubes. The H-shaped tube is an acoustically specialized part of the tracheal system that no longer serves only for respiration. Externally the four ends of the tube system are marked by spiracular openings on either side of the prothorax and by a pair of tympana on the surface of each foreleg tibia. Each auditory organ includes 55–60 sensory cells lined up in a linear array along the wall of an adjacent smaller branch of the main acoustic trachea. The axons of these cells run up the leg, project into the prothoracic ganglion and end in an area called auditory neuropil.

This system of four tympana and associated tubes provide a directional characteristic [1, 12]. Each ear has a maximal directional sensitivity to ipsilateral stimulation and a minimal one to contralateral sound (fig. 7). The directionality critically depends on the different sound entrances. Whereas blocking of the contralateral tympanum has no effect on the directionality of the ipsilateral ear, blocking of the ipsilateral and/or contralateral spiracle openings destroys the directionality. Directional sensitivity in central interneurons [2, 30] is the result of the peripheral directionality and possible inhibitory influences from the contralateral ear mediated by central neurons.

By the use of special 'earphones' [11] one can selectively stimulate one ear and determine the influence of the ipsi- and contralateral ear on an identified interneuron (fig. 8). The omega neuron ON1 receives excitatory input from the ear ipsilateral to its cell body and inhibitory input from the contralateral one. Stimulation of both ears simultaneously causes less excitation than ipsilateral stimulation alone. The ascending neuron AN1 is apparently not influenced by stimulation of the ear ipsilateral to the cell body, but contralateral stimulation – the side of the dendritic field of AN1 – causes excitation. AN2 is also excited by the ear contralateral to the cell body, but the effects of ipsilateral stimulation may vary in individuals from weak suprathreshold excitation to marked inhibition. ON1 may be a candidate for enhancing the binaural contrast by contralateral inhibition. This can be quantitatively analyzed by stimulating the two ears simultaneously with different sound pressures (fig. 8). In this experiment the excitatory stimulus is increased up to 20 dB above threshold and then kept constant. Increasing the inhibitory stimulus causes a decrease in the response to the excitatory stimulus. From these data the enhancement of the binaural contrast is calculated to be about 60% [11]. Selverston and co-workers [25] selectively killed one of the Omega cells. In this case the inhibition in the other remaining Omega cell is no longer detectable. So these two cells are coupled by

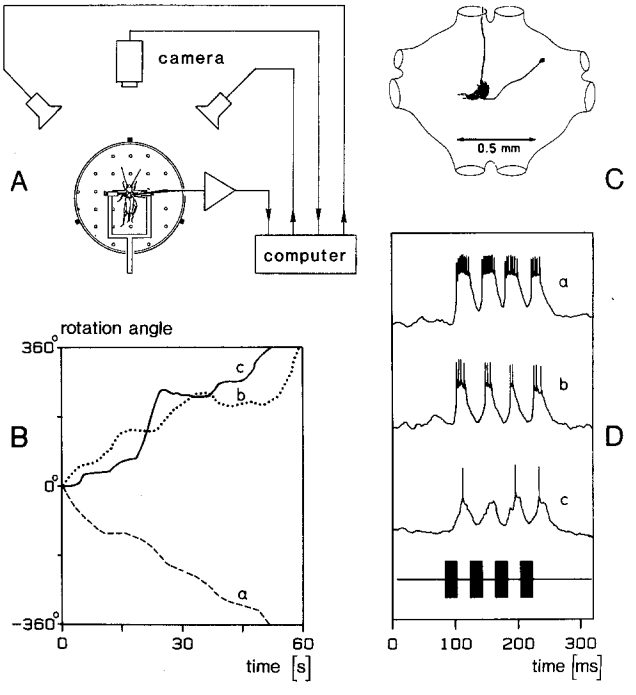

Figure 9. *A* Experimental set up for intracellular recording during walking seen from above. The animal is fixed on a holder but the legs can turn an air-supported styrofoam ball. The camera senses two components of the ball rotation and therefore of the animal's intended rotation and translation. Sound is delivered by 2 loudspeakers, each 50° apart from the longitudinal axis of the animal (not to scale); *B* rotation angle of a cricket during calling song presentation; *a* sound (80 dB) from left, *b* sound from right, *c* sound from left during hyperpolarization of the AN1 shown in *C*; *D* responses of the AN1 to artificial calling song (80 dB), *a*, *b*, and *c* as in *B*.

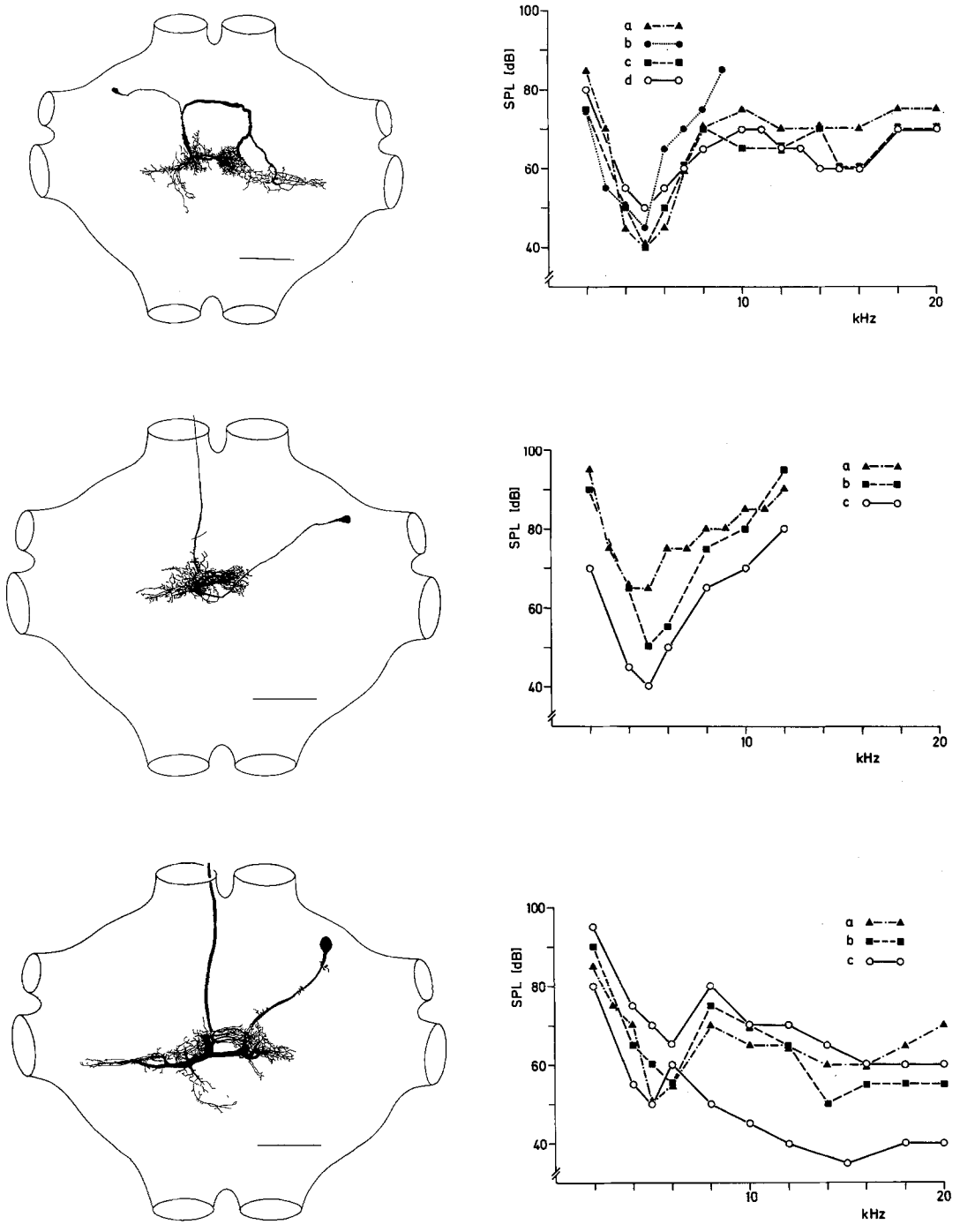

Figure 10. Morphology and physiology of auditory neurons in unilaterally deafferented animals. The left foreleg was amputated in the penultimate larval instar and the neurons recorded in the adult; left column shows the omega neuron ON1 (top), the ascending neuron AN1 (second row) and the ascending neuron AN2 (bottom row) in such animals (compare with the neuron shapes in fig. 8); the right column shows the corresponding threshold curves of different examples of these neurons; threshold curves of AN1 and AN2 with open dots are from intact aniamals, all others are from one-eared crickets (from Schildberger et al.[23], modified).

reciprocal inhibition. The omega neuron also inhibits an ascending neuron AN2 on the contralateral side. Thus, the directional sensitivity of the ear is enhanced in prothoracic neurons and transferred into the brain.

So far we have assumed that the identified neurons described earlier are involved in phonotactic behavior; however, this assumption is based only on the correlation of the behavioral and neuronal characteristics. With a new experimental design we can now study causal relationships between neurons and behavior (fig. 9). Animals are fixed on a holder in such a way that they cannot move their bodies but are free to move their legs. Under these conditions the cricket can turn an air-supported ball and a monitor of the movements of the ball indicates the intended translation and rotation of the animal. Switching on a loudspeaker placed 50 degrees to the left of the body axis causes the animal to turn the ball clockwise and backwards so the intended movement is forward and towards the loudspeaker side. Switching to a second loudspeaker placed symmetrically on the right side causes an intended turning of the animal to the right. The turning tendency increases with increasing sound intensity.

Under the conditions described it is possible to record intracellularly from identified prothoracic auditory interneurons. When the AN1 neuron with the excitatory input on the side of the active speaker is hyperpolarized the responses to sound decrease and the animal turns away from that side. Switching off the negative current with that loudspeaker still active causes the same level of excitation in AN1 as before hyperpolarization and a turning tendency to the side of the active speaker. Turning to the 'wrong' side takes place as long as the response of the AN1 to sound stimulation ipsilateral to its exciting ear during hyperpolarization is weaker than to contralateral stimulation without current injection. One can interpret this experiment in the following way: hyperpolarizing one AN1 changes the balance between the activity of the AN1 cells on each side so that the non-hyperpolarized AN1 is always more strongly excited, independently of the loudspeaker direction, and the animal turns to the side of the more strongly excited AN1. Two conclusions may be drawn; firstly, it is not necessary that both left and right AN1 cells are active to drive the recognizer in the brain because the female shows phonotaxis, and secondly, it is indeed the balance of these two cells that determines the turning direction and therefore the acoustic orientation.

We know almost nothing about directional properties of brain neurons and it remains unclear whether or not the behavioral decisions in phonotaxis may be influenced by further processing of sound direction in the brain.

Phonotaxis with one ear

The hypothesis of sound localization (turn toward the ear most strongly excited) requires two ears and for a long time it was believed that crickets need two ears to orientate. But it was forgotten that the discoverer of orthopteran phonotaxis, J. Regen, reported at the beginning of this century that crickets with only one ear can reach a sound source [18]. Huber and co-workers [8, 10] reexamined his statement by cutting one foreleg in an adult. The animals circled towards the side of the remaining ear. But there is a considerable amount of walking in a loudspeaker-dependent direction. When the amputation is done in a larval instar, the leg will regenerate but without an ear. Some of these animals, tested as adults, tracked without any circling, others circled all the time towards the side of the remaining ear and still others showed periods of tracking interrupted by circling [10, 24]. So the loss of an ear can be compensated in some animals during larval development.

This finding raises the question how an animal with only one ear can detect sound direction. If in such one-eared animals the auditory pathway in the central nervous system is functional only on the side of the remaining ear, orientation would only be possible if the animal measures sound intensity successively in time. But if in one-eared animals both sides of the central auditory pathway are functional, other mechanisms would be possible for localization. In animals operated on in a larval instar we recorded from central auditory neurons that would normally get excitatory input from the now deafferented side and stained the neurons by intracellular dye injection.

Most of the neurons changed their morphology (fig. 10). In contrast to the situation in the intact animal, the cells of the operated side send dendritic branches over the midline and reach the auditory neuropil of the intact side [7, 23]. ON1s that showed this dendritic sprouting over the midline were excited by the remaining ear and the same applies to AN1 and AN2. Threshold and intensity/response functions are very similar to those of intact animals. So these new connections are functional. The consequence is that the responses of auditory interneurons of the operated side are only different from the responses of those of intact animals in the sense that their excitation arises from the 'wrong' ear.

Thus, one-eared animals have functional central pathways on both sides and can localize the sound source. If this localization results from the comparison of excitation of the left and right side of the CNS then a different threshold and different slopes of the intensity/response functions of the left and right side would be sufficient to explain orientation. If the two intensity/response functions do not cross under these conditions the result would be a permanent turning to the side of the more sensitive neuron. If they cross then the excitation is balanced at a specific intensity range and a stable walking course could be maintained at least at these intensities.

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1 Bovd, P., and Lewis, D. B., Peripheral auditory directionality in the cricket. J. comp. Physiol. *153* (1983) 523–532.
2 Bovan, G. S., Directional responses to sound in the central nervous system of the cricket *Teleogryllus commodus*. I. Ascending interneurons. J. comp. Physiol. *130* (1979) 137–150.
3 Bovan, G. S., Auditory neurons in the brain of the cricket *Gryllus bimaculatus*. J. comp. Physiol. *140* (1980) 81–93.
4 Bovan, G. S., and Williams, J. L. D., Auditory neurons in the brain of the cricket *Gryllus bimaculatus* (De Geer): Ascending interneurons. J. Insect Physiol. *28* (1982) 493–501.
5 Capranica, R. R., and Rose, G., Frequency and temporal processing in the auditory system of anurans, in: Neuroethology and Behavioral Physiology, pp. 136–152. Eds F. Huber and H. Markl. Springer, Berlin, Heidelberg and New York 1983.
6 Casaday, G. B., and Hoy, R. R., Auditory interneurons in the cricket *Teleogryllus oceanicus*: physiological and anatomical properties. J. comp. Physiol. *121* (1977) 1–13.
7 Hoy, R. R., Nolan, T. G., and Casaday, G. C., Dendritic sprouting and compensatory synaptogenesis in an identified interneuron following auditory deprivation in a cricket. Proc. natl Acad. Sci. USA *82* (1985) 7772–7776.
8 Huber, F., Plasticity in the auditory system of crickets: Phonotaxis with one ear and neuronal reorganization within the auditory pathway. J. comp. Physiol. *A 161* (1987) 538–604.
9 Huber, F., and Thorson, J., Cricket auditory communication. Sci. Am. *253* (1985) 60–68.
10 Huber, F., Kleindienst, H.-U., Weber, T., and Thorson, J., Auditory behavior of the cricket. III. Tracking of male calling song by surgically and developmentally one-eared females, and the curious role of the anterior tympanum. J. comp. Physiol. *A 155* (1984) 725–738.
11 Kleindienst, H.-U., Koch, U. T., and Wohlers, D. W., Analysis of the cricket auditory system by acoustic stimulation using a closed sound field. J. comp. Physiol. *141* (1981) 283–296.

12 Larsen, O. N., Surlykke, A., and Michelsen, A., Directionality of the cricket ear: a property of the tympanal membrane. Naturwissenschaften *71* (1984) 538.
13 Moiseff, A., and Hoy, R. R., Sensitivity to ultrasound in an identified auditory neuron in the cricket: possible neural link to phonotactic behavior. J. comp. Physiol. *152* (1983) 155–167.
14 Moiseff, A., Pollack, G. S., and Hoy, R. R., Steering responses of flying crickets to sound and ultrasound. Proc. natl Acad. Sci. USA *75* (1978) 4052–4056.
15 Nolen, T. G., and Hoy, R. R., Initiation of behavior by single neurons: the role of behavioral context. Science *226* (1984) 992–994.
16 Oldfield, B. P., Kleindienst, H.-U., and Huber, F., Physiology and tonotopic organization of auditory receptors in the cricket *Gryllus bimaculatus*. J. comp. Physiol. *A 159* (1986) 457–464.
17 Popov, A. V., Markovich, A. M., and Andjan, A. S., Auditory interneurons in the prothoracic ganglion of the cricket, *Gryllus bimaculatus*. I. The large segmental auditory neuron (LSAN). J. comp. Physiol. *126* (1978) 183–192.
18 Regen, J., Über die Anlockung des Weibchens von *Gryllus campestris* L. durch telephonisch übertragene Stridulationslaute des Männchens. Pflügers Arch. *155* (1913) 193–200.
19 Reiss, R. S., A theory and simulation of rhythmic behavior due to reciprocal inhibition in small nerve nets, in: Proc. AFIPS Spring Joint Computer Conf. 21, pp. 171–194. National Press, Palo Alto, CA 1962.
20 Rheinlaender, J., and Blaetgen, G., The precision of auditory lateralization in the cricket *Gryllus bimaculatus*. Physiol. Ent. *7* (1982) 209–218.
21 Schildberger, K., Temporal selectivity of identified auditory neurons in the cricket brain. J. comp. Physiol. *A 155* (1984) 171–185.
22 Schildberger, K., Recognition of temporal patterns by identified auditory neurons in the cricket brain, in: Acoustic and Vibrational Communication in Insects, pp. 41–49. Eds N. Elsner and K. Kalmring. Paul Parey, Berlin 1985.
23 Schildberger, K., Wohlers, D., Schmitz, B., Kleindienst, H., and Huber, F., Morphological and physiological changes in central auditory neurons following unilateral foreleg amputation in larval crickets. J. comp. Physiol. *A 158* (1986) 291–300.
24 Schmitz, B., Compensation of unilateral hearing deficiencies in the acoustic orientation of crickets. Verh. dt. zool. Ges. *79* (1986) 237.
25 Selverston, A. L., Kleindienst, H.-U., and Huber, F., Synaptic connectivity between cricket auditory interneurons as studied by selective photoinactivation. J. Neurosci. *5* (1985) 1283–1292.
26 Stabel, J., and Wendler, G., Akustische Interneurone und das Vorzeichen der Phonotaxis, in: Sensomotorik Identifizierte Neurone, pp. 123. Eds N. Elsner and G. Rathmayer. Thieme, Stuttgart, New York 1986.
27 Thorson, J., Weber, T., and Huber, F., Auditory behavior of the cricket. II. Simplicity of calling song recognition in *Gryllus* and anomalous phonotaxis at abnormal carrier frequencies. J. comp. Physiol. *146* (1982) 361–378.
28 Weber, T., Thorson, J., and Huber, F., Auditory behavior of the cricket. I. Dynamics of compensated walking and discrimination paradigms on the Kramer treadmill. J. comp. Physiol. *141* (1981) 215–232.
29 Wiese, K., and Eilts, K., Evidence for matched frequency dependence of bilateral inhibition in the auditory pathway of *Gryllus bimaculatus*. Zool. Jb. Physiol. *89* (1985) 181–201.
30 Wohlers, D. W., and Huber, F., Intracellular recording and staining of cricket auditory interneurons (*Gryllus campestris* L., *Gryllus bimaculatus* De Geer). J. comp. Physiol. *127* (1978) 11–28.
31 Wohlers, D. W., and Huber, F., Processing of sound signals by six types of neurons in the prothoracic ganglion of the cricket *Gryllus campestris* L. J. comp. Physiol. *146* (1982) 161–173.
32 Wohlers, D. W., and Huber, F., Topographical organization of the auditory pathway within the prothoracic ganglion of the cricket *Gryllus campestris* L. Cell Tissue Res. *239* (1985) 555–565.

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Development of behavior and learning in *Aplysia*

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Summary. A set of fundamental issues in neuroethology concerns the neural mechanisms underlying behavior and behavioral plasticity. We have recently analyzed these issues by combining a simple systems approach in the marine mollusc *Aplysia* with a developmental analysis aimed at examining the emergence and maturation of different forms of behavior and learning. We have focussed on two kinds of questions: 1) How are specific neural circuits developmentally assembled to mediate different types of behaviors? and 2) how is plasticity integrated with these circuits to give rise to different forms of learning? From our analysis of the development of learning and memory in *Aplysia*, several themes have emerged: 1) Different forms of learning emerge according to different developmental timetables. 2) Cellular analogs of learning have the same developmental timetables as their respective forms of behavioral learning. 3) An analysis of non-decremented responses prior to the emergence of sensitization reveals a novel inhibitory process on both behavioral and cellular levels. 4) Sensitization emerges simultaneously in diverse response systems, suggesting an underlying general process. 5) A widespread proliferation of central neurons occurs in the same developmental stage as the emergence of sensitization, raising the possibility that some aspect of the trigger for neuronal proliferation may also contribute to the expression of sensitization.
Key words. Aplysia; development; habituation; dishabituation; sensitization; learning; locomotion; bursting neuron.

Introduction

One of the primary aims in neuroethology is to understand how natural behaviors can be explained in terms of their underlying neural mechanisms. In recent years a number of excellent model systems have been developed that have enabled significant advances in our understanding of the neural substrates of a variety of forms of behavior, especially in invertebrate animals. Two important themes have emerged from this work. First, the characteristic features of diverse behaviors, ranging from simple reflexes to complex fixed-action patterns, can be accounted for by the properties of specific neurons and specific neural circuits (for review see Getting [7] and Kandel [11]). Second, a fundamental property of many behaviors, that they can be modified by experience and learning, can be traced to particular forms of plasticity at specific loci within identified neural circuits (for review see Carew and Sahley [4]).