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'Switching-off' of an auditory interneuron during stridulation in the acridid grasshopper *Chorthippus biguttulus* L.

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Summary. In freely moving grasshoppers of the species *Chorthippus biguttulus* compound potentials were recorded from the neck connectives with chronically implanted hook electrodes. The spikes of one large auditory interneuron, known as the G-neuron (Kalmring 1975a, b) were clearly distinguishable in the recordings and the neuron was identified by its physiology and morphology. In quiescent grasshoppers the G-neuron responds to auditory and vibratory stimuli, but responses to both stimuli are suppressed during stridulation in males (Fig. 1, top, Fig. 7). When a male's wings were removed so that the stridulatory movements of its hindlegs produced no sound, the suppression of the G-neuron response still occurred (Fig. 1, bottom). When proprioceptive feedback from the hindlegs was reduced, by forced autotomy of the legs, the switching-off remained incomplete (Fig. 3) (production of stridulatory patterns was inferred on the basis of electromyograms from the relevant thoracic musculature). Imposed movement of the hindlegs, on the other hand, suppressed the G-neuron response in a graded fashion, depending on the frequency of the movement (Figs. 4 and 5). These experiments suggest that the switching-off is brought about by a combination of proprioceptive feedback and central efferences. The switching-off phenomenon could either protect the grasshopper's auditory pathway from undesired effects of overloading by its own intense song (e.g. self-induced habituation as described by Krasne and Wine 1977) and should therefore apply for most auditory neurons. Alternatively it could prevent escape reflexes from being triggered by stridulatory self-stimulation and consequently might apply only for neurons involved in such networks (as the G-neuron might be).

Introduction

Many acridid grasshoppers have evolved an intraspecific acoustic communication system: the males of these species produce songs, which induce receptive conspecific females to answer with songs of their own. The tympanal organs of acridids are located in the first abdominal segment, in the immediate vicinity of the stridulatory apparatus – a toothed ridge on the inside of each hind femur, which rubs against a modified vein on the adjacent elytron. Because the sound-producing apparatus is so close to the auditory organ, its own song provides an intense auditory stimulus for the animal.

The question arises how the grasshopper can cope with this strong self-stimulation; whether, for instance, acoustic events in the environment can still be resolved against the background of its own stridulatory signal. According to the reafference principle invoked by von Holst and Mittelstaedt (Mittelstaedt 1970), this could be achieved, for instance, by subtracting an efference copy of the song pattern – representing the excitation which is expected to be produced in the auditory pathway by the animal's own song – from the total input from the tympanal organs. Such a computation of afferences and efference copy is possible only if external and self-induced stimuli are superimposed without major interference. This applies for most visual stimuli – where, for example, shifting of the retinal image is composed of a linear superposition of both body and environmental move-

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ments. In the case of acoustic stimuli the situation is different; any self-produced sound will interfere with external acoustic events and reduce the signal-to-noise ratio for the perception of these external signals. This problem becomes considerably worsened if the self-produced sound is large compared to some simultaneously received environmental stimulus or if it even exceeds the dynamic range of the sense organ. Furthermore, strong stimuli ordinarily cause adaptation of the sense organ or habituation of higher-order networks in order to adjust the working range of the system to the current stimulus intensity. After the end of such a stimulus, full sensitivity is only gradually restored and the perception of external stimuli remains impaired.

In the experiments described in this paper, the activity of a grasshopper auditory interneuron during stridulation was investigated. The response of this neuron to synthetic stimuli was determined in quiescent and in stridulating animals and it is shown that stridulation suppresses interneuron activity.

Methods

Recordings were taken from the neck connectives of *Chorthippus biguttulus*. The method was partly adopted from Kämper and is described in detail in Kämper and Dambach (1981). Hook electrodes (steel wire with lacquer coating, 30 µm in diameter, insulation removed on the inner surface of the hooks, inside diameter of the hooks about 80 µm) were attached to the neck connectives while the animal was under carbon dioxide anaesthesia. The connectives were easily accessible by cutting a small slit in the ventral membraneous cuticle of the throat. The wire was run up to the pronotum, fixed there by a mixture of wax and rosin (1:2) and suspended from a plug mounted some 20 cm above the floor, allowing the grasshopper to move on a tussock of grass.

To record electromyograms, a hole was pricked into the cuticle above the muscle, and the tip of a steel wire was inserted and fixed with wax-rosin (Elsner 1975). This electrode recorded the discharge of the tergocoxal muscle, M119, and occasionally also lower amplitude potentials from other song muscles. This activity provided a reliable representation of the animal's singing behavior.

The electrodes implanted in the neck recorded compound nerve potentials. The action potentials of single large interneurons could be identified by their conspicuous amplitudes. To enable computer-assisted evaluation, the amplitude and time of occurrence of all spikes were digitized and stored on magnetic tape (Zarnack and Möhl 1977). In an amplitude histogram of all these spikes, an amplitude window could be defined that corresponded to the activity of one particular neuron – a prerequisite for further evaluation.

Usually, the G-neuron was identified by its unique physiological characteristics (see Results). This identification was confirmed by determining the morphology. During recording from the cervical connectives with the hook electrodes, a micropipette was used to probe the thoracic ganglia until an intracellularly impaled cell was found, whose discharge matched that of the neuron of interest in the hook electrode recording. The cell was then injected with Lucifer Yellow (Stewart 1977) and identified by microscopic examination. In this way the auditory neuron which appeared regularly in our recordings has been identified as the G-neuron described by Kalmring (e.g. 1975a, b). This identification was performed only once and is described in more detail elsewhere (Wolf 1986).

In this paper we will retain the old, but generally familiar, term G_1- or simply G-neuron. This neuron has also been called VS2 (Silver et al. 1980), TH2-TC1 (Hedwig 1985), and TN3 (Marquart 1985).

To move the hindlegs of the animal in a manner resembling the stridulatory rhythm, the rotation of a variable-speed electric motor was converted to a linear back-and-forth movement with an approximately sinusoidal waveform by way of a cam and piston arrangement. This movement was transferred to the two hindlegs which were joined together with a small wire attached to the drive rods. Amplitude and range of the leg movement were matched to that observed during natural stridulation. The animal itself was glued to a holder by its anterior and middle legs and by the sternum of the thorax. Animals prepared in this way do not sing spontaneously and electrodes in the song muscles recorded no activity during imposed leg movements. Motor and drive rods were enclosed in a sound-attenuating housing to prevent stimulation of the auditory organs by the motor's noise. Running the motor with the hindlegs uncoupled had no effect on the G-neuron response and imposing movement on the animal's middle legs provided only huge vibratory input to the G-neuron. These control experiments indicated that it was indeed the imposed movement of the hindlegs which mimicked the effects of natural stridulation.

Results

A male grasshopper with chronically implanted hook electrodes is able to move freely (as far as the recording wires permit) and expresses the behavioral repertoire typical of its species. The spikes of the G-neuron are usually clearly identifiable among the potentials recorded from the cervical connectives because of their large amplitudes. In a quiescent animal the G-neuron responds to auditory and vibratory stimuli, in a manner closely resembling the responses of the corresponding neuron in *Locusta migratoria* (e.g. Kalmring 1975a, b; Čokl et al. 1977; for a detailed description in *Chorthippus biguttulus* see Wolf 1986a, b).

Its discharge is mainly phasic, with a latency (at the cervical connective) of 10 to 15 ms and shows marked habituation. The auditory threshold is around 50 dB for noise stimuli (bandwidth ca. 3–45 kHz) but is frequency-dependent for pure tones. The curve of the G-neuron response magnitude vs. intensity is a typical saturating characteristic for noise pulses and high frequency (above 8 kHz) tones (Kalmring and Rheinlaender 1974) but exhibits inhibitory areas for low tones at high intensities. The responses to noise from all directions are about equal in threshold.

1. Response of the G-neuron to the sound of the animal's own stridulation

Although the largest responses are elicited by brief, loud noise pulses, the neuron also responds clearly to the songs of other males and the response stridulation of nearby females. Much greater excitation must be produced in a grasshopper's auditory pathway by its own stridulation than by the songs of other males because the stridulation apparatus is immediately adjacent to the tympanal organ. How does the G-neuron respond to this strong self-excitation?

The very first experiments with implanted electrodes in freely moving males made it clear that no activity correlating with the auditory stimulus could be recorded from the neck connectives while the animal was stridulating. In particular the G-neuron action potentials were no longer to be seen. This observation was confirmed in 11 animals.

2. Response to external sounds during stridulation

Two obvious hypotheses can be proposed to explain the failure of the G-neuron to respond to the animal's own song. One is based on the reafference principle of von Holst and Mittelstaedt; that is, the signals from the tympanal organ could be compared with, and effectively cancelled by, an efference copy of the stridulation. Although it is not easy to imagine how this system would operate when other acoustic signals interfere with the stridulation sounds, the effect would be to permit central interneurons to respond only to sounds of external origin. An alternative hypothesis is that the auditory pathway to the G-neuron is closed to all input during stridulation and the neuron does not respond to any acoustic stimuli while the animal is singing.

To distinguish between these alternatives, the grasshopper was exposed to a steady background stimulus, an uninterrupted train of short, loud noise pulses (7 ms duration, 70 dB SPL, upper traces in Fig. 1). In the quiescent animal each such pulse reliably elicits a G-neuron response of one or occasionally two spikes (second traces in Fig. 1). During stridulation this response of the G-neuron disappears. Evidently the pathway in which the auditory signal is conducted – from tympanal membrane through receptors and interneurons to the G-neuron – has been closed at some point so that the G-neuron is switched off.

The recordings in Fig. 1 show that the stimulus-induced G-neuron discharge stops at about the same time as the leg downstroke occurs and does not reappear until the end of the song, when it is temporarily enhanced (see below). During the song, no spikes from this neuron can be recorded even at stimulus intensities up to 90–100 dB (i.e. 40–50 dB above threshold).

The B_1-neuron (Kalmring 1975b) is switched off during stridulation in the same way but in these compound recordings its spikes are so small as to make quantitative evaluation difficult (see Wolf 1984).

In Fig. 2 the switching off of a G-neuron is represented in the shape of a PST-histogram. Spikes occurring within a certain time interval – covering stimuli presented from ca. 1 s before until ca. 1 s after each song – were accumulated for several songs in a stimulus-related manner. The times of beginning and end of each song, as found from electromyograms from the song muscles (see Methods), could be determined for this stimulus-related histogram with an accuracy of one stimulus interval only (arrow heads below the histogram) because the songs were, of course, completely unsynchronized with the background stimulus and all of different durations.

Before the song the G-neuron responds to each noise pulse (indicated below the histogram) by discharging an average of 1.8 spikes. During the song there is no activity correlated with the stimulus. After the end of stridulation the response magnitude is transiently increased to an average of 3.5 spikes per stimulus (see also Fig. 1). Probably this increase in the G-neuron response following stridulation does not only represent a recovery from habituation induced by the repetitive stimulation prior to the chirp because in the quiescent animal recovery from response decrement is much slower. An increase in response level from 1.8 to 3.5 spikes per stimulus following a repetitive sequence of sound pulses requires usually more than 10 s, whereas the songs of *C. biguttulus* last only for ca. 0.8 to at most 3 s. (For a detailed description of the response decrement properties of the G-neuron see Wolf 1986a).

The background activity in the histograms reflects the discharge of other neurons, the spikes of which occasionally enter the amplitude window set for the G-neuron. The reduction of this activity during stridulation indicates that neurons other than G-neuron are also affected by this behavior.

3. Is the G-neuron switched off by the sound production during stridulation?

Evidently, somewhere in the transmission channel from tympanal membrane to G-neuron, information that stridulation is in progress acts to switch

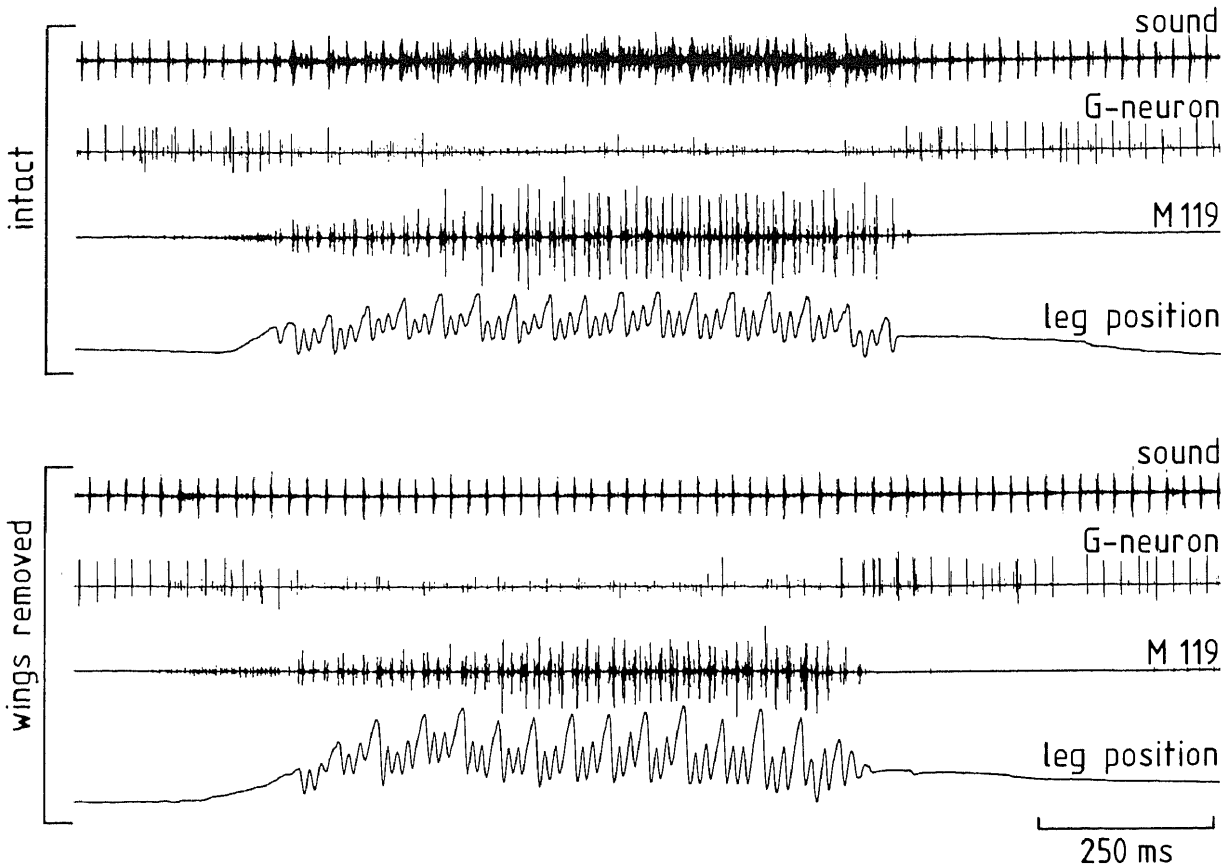

Fig. 1. Switching off of the G-neuron during stridulation. The upper four traces show the effect in an intact animal; in the lower set, the same effect occurs in an animal without wings where stridulatory leg movements do not produce sound. The train of acoustic stimuli (7-ms noise pulses, 70 dB, 18 ms intervals) and the stridulatory sounds were recorded by a microphone (top traces). The second traces are compound recordings from the neck connectives, showing the prominent spikes of the G-neuron. The third traces represent electromyograms from tergocoxal muscles (see Methods); the bottom traces the stridulatory movements of one hindleg, recorded with an optoelectronic camera (von Helversen and Elsner 1977)

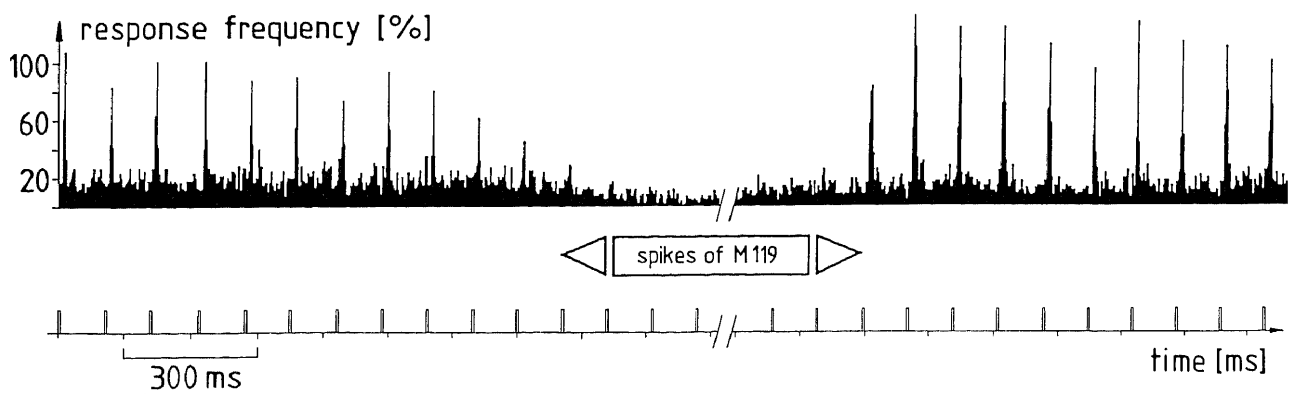

Fig. 2. Switching off of the G-neuron: PST histogram. The suppression of the response, as shown in Fig. 1 for a single chirp, is here (for another animal) presented by a PST histogram in which the activity before, during and after 59 chirps from a long song sequence is accumulated. Beginning and end of each chirp were identified by the first and last action potentials, respectively, in a muscle recording (see Methods) and are indicated below the histogram with an accuracy of one stimulus interval (arrow heads, see text). Underneath, the envelope of the stimulus train (7-ms noise pulses, 70 dB, intervals 100 ms) is indicated. A value of 100% on the ordinate means that for the bin in question (binwidth 3 ms) each of the 59 stimulus presentations was answered by one spike on average

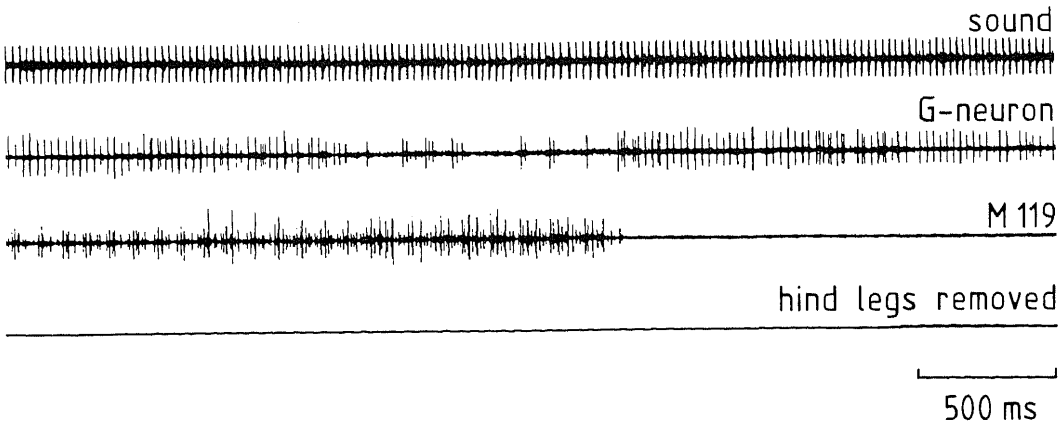

Fig. 3. Incomplete suppression of G-neuron responses after amputation of the hindlegs. Stimulus presentation and recording as in Fig. 1. In a grasshopper that had lost both hindlegs (normally coordinated) stridulatory activity could be inferred by means of electromyograms from song muscles (third trace, see text, and Elsner 1975). Under these conditions only intermittent suppression of the G-neuron response could be observed toward the end of some chirps. This also holds true for an animal whose hindlegs are merely immobilized rather than removed

off the auditory response. This information can have several possible sources: It could be a mechanical suppression of tympanic vibrations, or a feedback signal from proprioceptors (e.g. in the leg joints) in response to leg movement, an output of central nervous networks (e.g. a corollary discharge generated in parallel with the leg motor commands), or activity in sound-sensing structures signalling the intense sound of stridulation (cercal hairs or even the tympanal receptors themselves).

The last of these possibilities can be tested by removing the animal's wings. The hindlegs then carry out normally coordinated singing movements which make no sound (the microphone recording shows only the sound stimulus while stridulation is in progress, Fig. 1). Even during silent stridulation, the G-neuron fails to respond to the noise pulses. Therefore it is not the sound produced by stridulation that causes it to be switched off.

4. Role of central nervous excitation in switching off the G-neuron

The preceding experiment also demonstrates that proprioceptive feedback from the wings (e.g. vibration detected in the wing joint) is not responsible for the effect. A number of receptors in the leg, including the chordotonal organs and stretch receptors in coxa and femur (Bräuning et al. 1981), might provide the necessary information. In fact there is evidence that feedback from these receptors makes an extremely important contribution to the long-term stability of the stridulation pattern (Lindberg and Elsner 1977).

To check the importance of central nervous components, proprioceptive feedback was eliminated which could best be done by removing both hindlegs (the animal was coaxed into autotomizing them). The coxal stumps which remained on the animal were then immobilized with a hard and brittle wax-rosin mixture. Under these conditions, stridulatory activity could be inferred only on the basis of electromyograms from the relevant thoracic musculature, which continued to exhibit normally coordinated stridulatory patterns (Elsner 1975). Even here, however, there remains a possibility that, for instance, changes in tension of the coxal or thoracic cuticle might be detectable by chordotonal organs.

The experiment illustrated in Fig. 3 demonstrates that the G-neuron is not completely switched off during stridulation when both hindlegs are absent. During the first part of the chirp it responds to each of the external sound pulses, but toward the end of the chirp only occasional pulses elicit a response. The degree of suppression varies widely. Often there is no suppression at the beginning of a song sequence but as the sequence progresses and the chirps are longer, suppression of the G-neuron response – always toward the end of a chirp – becomes clearly apparent as in Fig. 3.

Amputation of only one leg has no detectable effect; the G-neuron is switched off during stridulation as in the intact animal.

5. Influence of imposed leg movements

The outcome of the above experiment suggests that central nervous excitation (a 'corollary discharge': Sperry 1950) *and* feedback from the periphery signalling leg movement (e.g. from leg proprioceptors) act together to switch off the G-neuron dur-

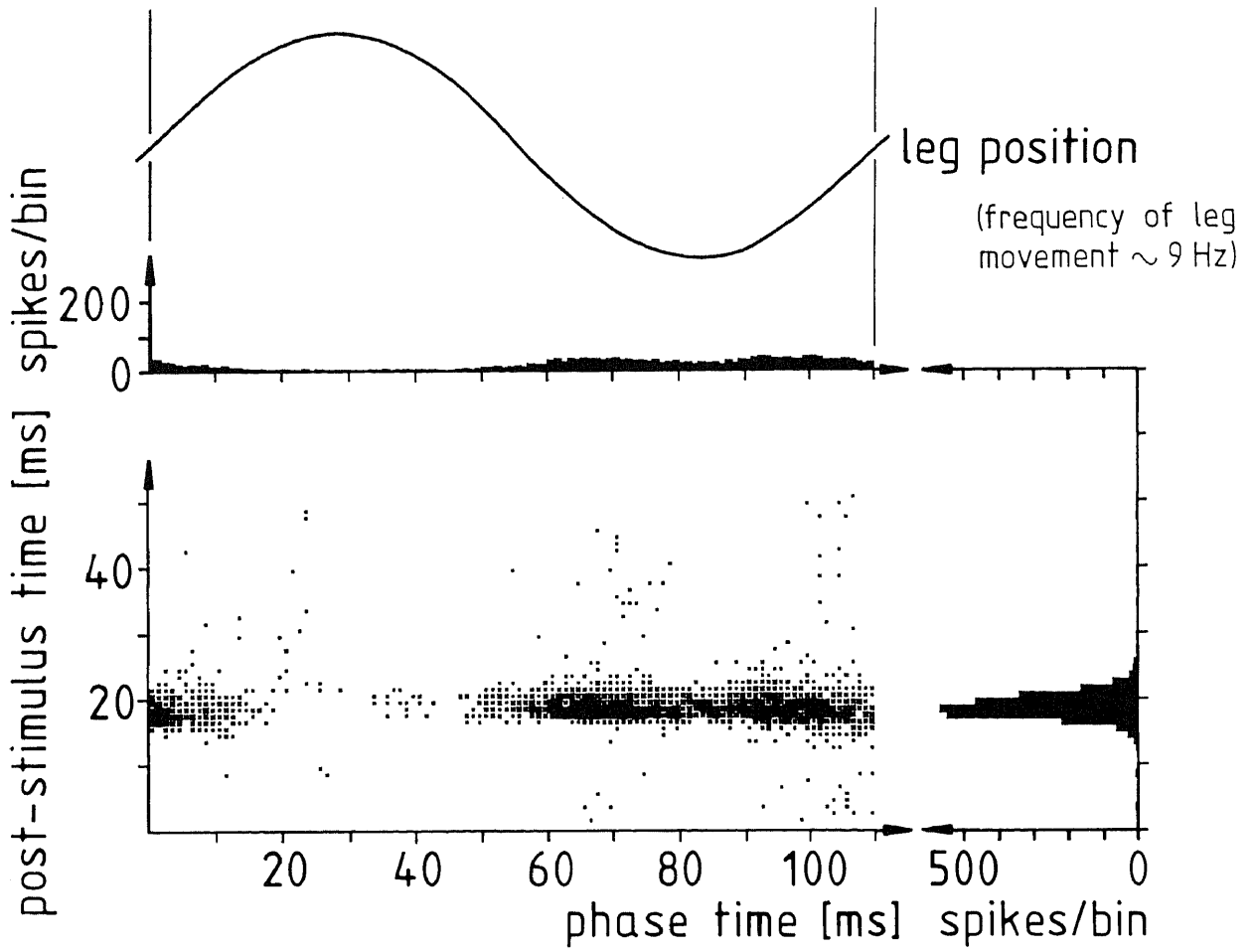

Fig. 4. Phase-coupled suppression of G-neuron responses during imposed hindleg movement. The animal was immobilized except for the hindlegs, which were moved back and forth approximately sinusoidally (see Methods). While the legs moved at ca. 9 Hz, a continuous sequence of noise pulses (7 ms, 70 dB, intervals 50 ms) was presented. Each action potential of the G-neuron is plotted on a coordinate system representing the elapsed time since the immediately preceding acoustic stimulus (ordinate) and the time since the last zero-crossing with positive slope of the leg movement curve (equivalent to phase of leg movement; abscissa). The upper diagram (same scale) shows a spike histogram based on the leg movement cycle and, above it, the leg movement curve. At the lower right data are represented as a PST histogram based on the stimulus interval. Each dot in the coordinate system represents one to 15 spikes (2696 spikes total), depending on its area. These spikes occurred during 2120 cycles of leg movement which had an average period of 110 ms. During this time the stimuli occurred with almost uniform frequency at all phases of the leg movement. Absence of responses during the period around the upper reversal point of the leg movement is not due to a failure of stimuli to occur at this time

ing stridulation. As a check on the possibility that signals from the proprioceptors alone can cause at least partial suppression of the G-neuron response the following experiment was done.

A male grasshopper was held firmly by its forelegs and thorax and its hindlegs moved in a manner resembling the song rhythm by an external force. Presumably in this way the sense organs normally excited by stridulation were activated, although not precisely as they would be during the natural behavior. Sound stimuli were presented while moving the hindlegs.

In the experiment illustrated in Fig. 4, the hindlegs were moved at a frequency of ca. 9 Hz during presentation of a continuous train of noise pulses. In the lower left-hand plot of Fig. 4, for each G-neuron spike recorded under these conditions the time of its occurrence with respect to the leg-movement cycle is plotted on the abscissa, and the time elapsed since the immediately preceding stimulus on the ordinate. The spike distribution with respect to the ordinate (PST histogram on the right) is fairly narrow; G-neuron spikes in response to a stimulus occur at a latency of about 15 ms from stimulus onset. The distribution with respect to the phase of imposed leg movement (top histogram)

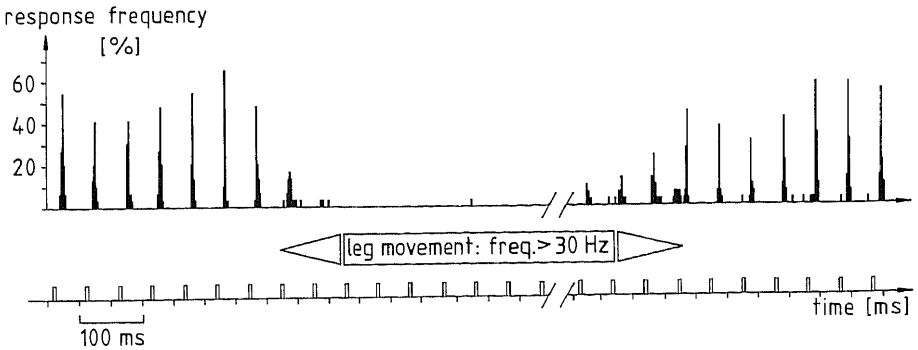

Fig. 5. Complete switching off of the G-neuron during imposed leg movement. Movement was imposed on the hindlegs of the grasshopper as described for Fig. 4 but at a frequency between 30 and 45 Hz. PST-histogram as in Fig. 2. Time during which leg movement occurred was determined from photoelectric recordings of the movement. The driving motor does not reach full velocity or come to a full stop immediately and during these transitions the neuron is not completely switched off (as in Fig. 4). This is reflected in the histogram by the gradual changes in height of the columns near the beginning and end of the leg movement. These points were specified only to within 200 ms, an oszillation with a period of 100 ms was arbitrarily taken as the criterion for beginning and end of leg movement. The histogram represents the spikes occurring during 29 movement episodes, using a 2-ms binwidth

is approximately uniform except for the time from 15 ms before the upper reversal point to 20 ms after it. During this time the response of the G-neuron to the auditory stimulus is almost completely or fully suppressed. Hence proprioceptive feedback with respect to leg movement contributes to the switching off of this neuron.

According to D. von Helversen (1972), at the temperature of these experiments (23 °C) the song of *C. biguttulus* has a characteristic repeat period of ca. 130 ms; each pattern unit consists of a basic movement on which smaller up-and-down movements of triple the basic frequency are superimposed (see Fig. 1, bottom traces). An oscillation at 9 Hz corresponds roughly to the frequency of the basic pattern of the song but not to the actual frequency of the leg movement during stridulation, which is three times higher.

When the imposed leg movement frequency is raised accordingly, to more than 30 Hz, the G-neuron is completely switched off. This effect is illustrated in Fig. 5 by a PST histogram constructed as in Fig. 2. When the driving motor is turned off, the leg movement slows down gradually (due to inertia) and the G-neuron does not 'switch on' as abruptly as after natural singing.

6. Time course of switching off at the onset of stridulation

In the spontaneous calling song and especially in the courtship song, a male normally increases the leg-movement amplitude slowly within a chirp so that often it begins with almost unnoticeable stridulatory movements. Because of this gradual onset it is impossible to specify exactly when a song begins, at least in a computer-assisted analysis based on leg movement or muscle potentials. It is difficult to determine with certainty whether the G-neuron is switched off before the first leg movement or only after it has begun – a decision that bears crucially on the question of whether proprioceptive feedback might be a causative factor.

Rivalry songs, on the other hand, usually begin very abruptly and at full intensity, with a sudden upstroke of the hindlegs. When chirps of such abrupt onset are produced, compound recordings from the neck connectives usually show a sharp burst of activity during the first 100 ms. This phenomenon was analyzed by selecting particularly loud and sudden rivalry chirps from a longer song sequence. The associated neural activity is illustrated in the shape of a PST-histogram in Fig. 6.

This was the same animal that provided the data for Fig. 2. Here too, the action potentials of other neurons occasionally enter the amplitude window set for the G-neuron and produce a 'background level' in the histogram. Therefore the activity peak in the first milliseconds after onset of the song certainly contains other spikes in addition to those of the G-neuron (moreover, during such bursts of intense activity, spikes in different neurons are likely to coincide so that the recorded potentials summate, making amplitude a poor criterion by which to identify a neuron).

Such a burst never appears during silent stridulation by males without wings (see Fig. 1, bottom) whereas acoustic stimuli that happen to occur during the first 100 ms of such a song are answered sometimes. This, and the sharp increase in spike frequency just after the stimulus within the burst (arrow in Fig. 6, top), indicates that external

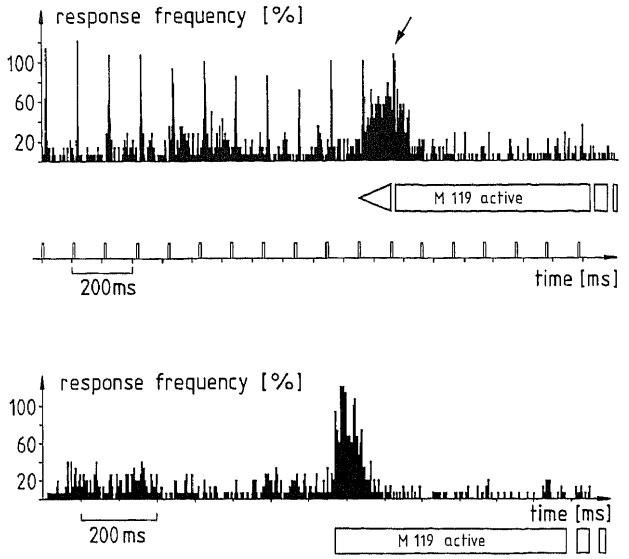

Fig. 6. Time course of switching off of the G-neuron at the onset of singing, demonstrated by selecting rivalry songs that begin abruptly. During the experiment acoustic stimuli (7 ms, 70 dB, intervals 100 ms, outlined as envelopes below the upper histogram) were presented in an uninterrupted train. The upper histogram was constructed like that in Fig. 2 on the basis of the stimulus train. The lower histogram – which accumulates the spikes from the same experiment and in the same amplitude window as for the upper histogram – is based instead on the beginning of stridulation, as determined by the first muscle spike. Hence a stimulus-correlated response is no longer visible but the spike burst at the onset of singing is shown in its correct time course. Both histograms represent the spikes occurring during 14 chirps, with a bin width of 3 ms

acoustic stimuli still elicit a neural response during the first milliseconds of such a rivalry song. The spike burst itself can be interpreted as a response of the G-neuron and other auditory (or, for instance, vibratory) fibres to the first stridulation sounds before they are switched off 80–100 ms after stridulation onset. This latency of the suppression is confirmed by the lower histogram in Fig. 6, in which external stimuli are ignored and the histogram is based on the chirp onset time. The spikes within the same amplitude window used previously are accumulated (but only with respect to the first muscle potential) to give a 'peri-song-time' histogram. Onset of the spike burst coincides exactly with the beginning of muscle activity and the burst lasts 80 to 100 ms.

No spike bursts were ever observed toward the end of a song, indicating that the neurons are switched off at least until the last sound of the song has ceased.

7. Vibration sensitivity of the G-neuron

The G-neuron responds not only to acoustic stimuli but also to vibration of the anterior or middle legs (Čokl et al. 1977). Like the auditory responses, this vibration sensitivity is switched off during stridulation (Fig. 7). Although the stridulatory movements undoubtedly stimulate the vibration receptors, neither these nor external vibratory stimuli are effective during singing.

This finding is interesting inasmuch as the vibratory inputs converge on the G-neuron by way of the leg nerves of the two anterior thoracic ganglia, while the auditory inputs arrive by way of the metathoracic ganglion. An indicator of this difference is the change in response latency, 4–5 ms for vibration and about 10 ms for auditory stimuli. That is, during stridulation the G-neuron does not respond to excitation from two entirely different input structures.

Discussion

1. What can be achieved by switching off an auditory interneuron during stridulation?

In *C. biguttulus,* the forewings – as part of the sound-producing apparatus – lie barely 0.5 mm above the membranes of the tympanal organs. Al-

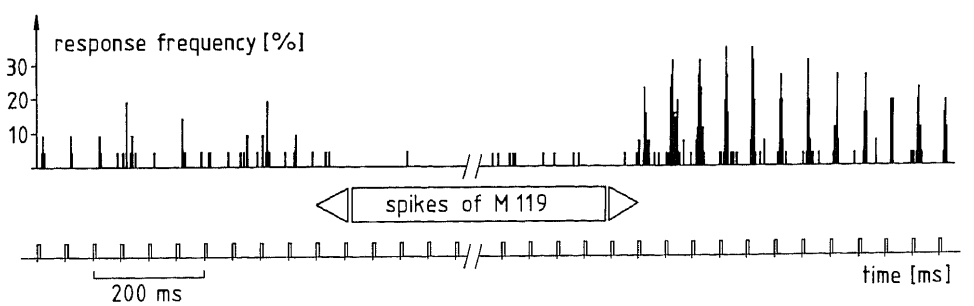

Fig. 7. Switching off of the vibration sensitivity of the G-neuron during stridulation. Experiment and histogram as for Fig. 2, except that stimuli were pulses of vibration (average amplitude ca. 30 μm (RMS), 5-ms noise pulses, intervals 50 ms) rather than sound. G-neuron shows strong habituation to this vibratory stimulus so that the stimuli prior to stridulation rarely elicit responses. The histogram represents the spikes occurring during 24 chirps, using a 2-ms binwidth

though a precise measurement of the sound intensity of the song at the tympanal organ is problematic and has not yet been made, the close proximity of the tympanal organ to the stridulatory apparatus implies that the song is a strong stimulus. Even at a distance of 25 cm the intensity of the *C. biguttulus* song can be as high as 70 dB. The effect of such a stimulus can be gauged by the finding that even the highest-threshold tympanal receptors that have been found in *L. migratoria* are excited to saturation at intensities between 90 and 100 dB (Römer 1976), that is, at sound pressure levels probably far exceeded by the animal's own song.

The switching off of auditory interneurons during stridulation must certainly reduce the effects of self-stimulation, while at the same time sacrificing completeness of auditory information. Two hypotheses can be proposed as to the function of the switching off phenomenon:

(i) The G-neuron might be switched off to prevent overload or habituation of the auditory pathway. The grasshopper's own song could have such high intensity at the tympanal organ that more or less all the tympanal receptors would be excited to saturation. The only information then transmitted is just the fact that a loud song occurs. But for this trivial information a high price has to be paid, that is the habituation at higher levels in the auditory pathway and therefore impairment of auditory reception outlasting the end of stridulation. But it is during that time just after stridulation that a female who is ready for mating is most likely to produce her response song, which is not nearly as loud as the male's song. This is also a dangerous time for the grasshopper to be insensitive to sounds made by approaching predators whose attention may have been attracted by the stridulation.

Thus, when the tympanal organs are overloaded it would be no disadvantage for the auditory neurons to be switched off. At levels within the nervous system central to the site of suppression, adaptation or habituation would be prevented and full auditory sensitivity would be guaranteed as soon as the animal stops singing. If this line of argument actually applies, one would expect most, if not all, auditory neurons to be switched off during stridulation as the G-neuron is.

Protection from overload or habituation by self-stimulation has often been demonstrated; one of the best-known examples is the presynaptic inhibition of habituation-prone afferent synapses in crayfish during tail-flip behavior. This inhibition protects escape reflexes from becoming habituated to stimuli produced by the animal's own tail-flip movements (e.g. Krasne and Wine 1977).

(ii) The G-neuron could be an auditory interneuron involved in triggering escape reflexes, although it is certainly in no way a 'command neuron' for escape, simply because it is active all the time in quiescent animals. However, it might be switched off during stridulation – together with other neurons of similar function – to avoid triggering of these reflexes by the combined acoustic, vibratory and visual stimulation.

Examples of sensory input modulation in this context have been found in other animals. During the animal's own movement, the afferents from hairs sensitive to currents on the tail fan of the crayfish are inhibited presynaptically (e.g. Kennedy and Fricke 1983) and the activity in lateral-line afferents of amphibians and fish is suppressed (Russel 1976; Klinke and Schmidt 1970). In both cases, activation of these receptors by strong external stimuli elicits an escape reflex – a stroke of the crayfish's tail and swimming movements of fish and amphibians. The suppression of (re)afferences from these sense organs probably prevents the triggering of these reflexes by stimuli associated with normal locomotion.

A similar line of reasoning has been suggested with respect to a large, predominantly visual interneuron in grasshoppers, the DCMD. In part because it synapses directly with the fast motoneuron to the extensor muscle of the tibia, a muscle very important in jumping, the possibility that it participates in initiating the escape jump has been discussed (Pearson et al. 1980). The DCMD is switched off during behavior that produces visual stimuli, such as antennal cleaning (Zaretzky and Rowell 1979), optic saccades and nystagmus (Rowell 1971), and probably also movements made in preparation for the escape jump (Heitler 1983).

Like the DCMD, the G-neuron in *L. migratoria* – and most probably also in *C. biguttulus,* in view of the general similiarity of the two species – communicates monosynaptically with the fast motoneuron of the extensor tibiae (Pearson et al. 1980). These connections and the large diameter of the fibre as well as some physiological properties suggest that the G-neuron may be involved in thoracic networks for short-latency initiation of the escape jump (Hedwig 1985).

Hedwig (1985) observed that in *Omocestus viridulus,* a closely related acridid, most auditory interneurons do not respond to external sound during stridulation but often discharge in a manner phase-coupled to the leg movement.

The available data thus do not provide conclusive evidence as yet to rule out either of the above hypotheses. Another question bearing on this point

concerns the level at which the auditory input is blocked during stridulation. Can it be that the tympanal fibres themselves are prevented from responding to acoustic stimuli?

2. At what level in the auditory pathway does the switching off observed in the G-neuron occur?

It is conceivable a priori that the transmission of information could be interrupted at any point in the auditory pathway, for example, by loss of tension in the tympanal membrane, by efferent control of the tympanal receptors, by inhibition of interneurons presynaptic to the G-neuron, or by hyperpolarization of the G-neuron itself.

It has been demonstrated in cicadas that the tympanal membranes are non-functional during sound production (Pringle 1954). A comparable mechanism in bats has been thoroughly investigated; here sound transmission in the middle ear is hampered by the stapedius reflex during the very loud vocalizations the animal produces for echolocation (Suga and Jen 1975). This input attenuation effectively adjusts the dynamic range of the ear. Mechanical effects at the tympanal membrane of the grasshopper might produce analogous damping but there are no anatomical grounds on which to infer such effects. Neither specialized cuticular structures around the membrane nor muscles that could modify the tension in the tympanal membrane are known to exist. The musculature present in the vicinity of the larval tympanal organs has completely regressed in the adult (Pichler 1953, and personal observations). Therefore it is relatively unlikely that peripheral damping of sound transmission occurs in the grasshopper.

Efferent suppression of tympanal-receptor activity, comparable to that in the lateral line organs of amphibians and fish, also seems unlikely for the tympanal nerve is not known to contain any efferent fibres.

Marquart (1985) showed that in *L. migratoria* both excitatory and inhibitory auditory interneurons converge on higher-order ascending fibres, for instance, the B-neuron. Therefore, an anatomical substrate seems to be available for inhibition of auditory information flow at a level presynaptic to the G-neuron.

The suppression of vibratory as well as auditory responsiveness of the G-neuron could occur on a central level after convergence of these inputs or, as well, by suppressing the different inputs separately. From many studies it appears that inhibitory effects on sensory pathways during a motor act are a widespread phenomenon and will probably intersect on several levels of the nervous system (e.g. Wine and Krasne 1982). It is probably a common mechanism, wherever a protection from undesirable effects of self-stimulation is required yet it is not possible to compute afferences and efference copy according to the reafference principle, that sensory pathways are 'nonspecifically' turned off.

Recordings obtained from auditory interneurons in a freely moving grasshopper, motivated to behave in the normal, species-specific manner, document that the auditory pathway can be modulated according to the behavioral context. Given the existence of such controlling elements, it follows that electrophysiological experiments on interneurons (auditory and other) in an immobilized and dissected animal will usually not sample the full range of neuronal properties. In such a preparation not only is feedback from sense organs eliminated but, perhaps more importantly, the behavior-dependent control of neuronal activity cannot be expressed.

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