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Functional organization of insect auditory sensilla

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Summary. 1. Electrophysiological recordings from individual auditory sensilla in the tettigoniid (*Caedicia simplex*) ear (Fig. 1) show that spike potentials of two distinct amplitudes occur in the sensory neuron (Fig. 2). Generally, the smaller of the two classes of spike is observed as an inflection on the rising phase of the larger spike. In cases where the larger spike fails or is blocked, the smaller spike becomes distinct. In the attachment cell of the sensillum, negative-going monophasic, or biphasic spikes are recorded (Fig. 3). In biphasic spikes, the positive phase is of smaller amplitude and always follows the negative phase.

2. Simultaneous recordings from the sensory neuron and from its associated attachment cell, using two electrodes, show that the negative-going spike recorded from the attachment cell corresponds with the smaller spike recorded from the neuron (Fig. 4). The occurrence of a large spike in the neuron causes the positive-going potential in the attachment cell, immediately following the negative spike.

3. Injection of negative current into the attachment cell via a recording electrode elicits spikes in the sensory neuron. If large spikes in the neuron fail or are blocked by hyperpolarization of that cell, the injection of negative current into the attachment cell elicits small spikes in the neuron (Fig. 4).

4. Electrical stimulation of the tympanal nerve induces retrograde spikes in the soma of the sensory neuron. Such spikes show no inflection in the rising phase, indicating the absence of the small spike as a precursor to the retrograde spike (Fig. 6). Recordings from the attachment cell, when retrograde spikes are induced, show only a positive-going potential correlated with each retrograde spike. The positive deflections recorded in the attachment cell, resulting from retrograde spikes, generally are of greater amplitude than those specifically associated with large, orthograde spikes occurring in the sensory neuron.

5. These results confirm previous suggestions that, in insect auditory, chordotonal sensilla, spikes of relatively small amplitude occur at the apex of the sensory dendrite and subsequently, trigger spikes of conventional amplitude at a site more proximal in the dendrite. The occurrence of small spikes in the neuron implies novel equilibrium potentials at the apical dendritic membrane, which may result from the scolopale lumen functioning as a receptor lymph cavity.

Introduction

The auditory organs of tettigoniids, located in the proximal part of each foreleg tibia, include the crista acustica, a structure consisting of a single row of chordotonal sensilla positioned in the haemocoel midway between the tympana (Schwabe 1906; Autrum 1940, 1941). Each sensillum is composed of a sensory neuron and its associated attachment, scolopale and enveloping cells (Schumacher 1979).

Electrical potentials derived from sensory transduction in the neuron have been recorded following impalement of the attachment cell of the sensillum (Oldfield 1982). These potentials include negative-going graded potentials, negative monophasic and biphasic spike potentials, in which the initial component is negative and of greater amplitude. Rather similar recordings also have been made from presumed attachment cells in the auditory organ (Müller's organ) of the locust (Hill 1983). The detection of these sensory events by

an electrode in the attachment cell indicates resistive coupling between the attachment cell and the nerve cell membrane.

From electrophysiological studies of sensory transduction in insect epidermal mechanoreceptors, Erler and Thurm (1981) concluded that voltage-dependent channels exist respectively distal and proximal in relation to the 'electrically-tight' epithelium surrounding the sensory neuron. Their technique of transepithelial recording, however, did not permit detailed descriptions of the inferred 'apical' (distal) and 'basal' (proximal) spikes. From considerations of the electrochemical gradients likely to exist at the apical dendritic membrane, they predicted that the apical spike may have novel amplitude and extended time course of repolarization. Furthermore, they predicted that in some insect sensilla, an apical spike may be functionally necessary to convey sensory signals to the base of a long sensory dendrite.

In similar transepithelial recordings from epidermal mechanoreceptors in spiders, negative spikes are interpreted as extracellular recordings of afferent spikes initiated in the apical region of the sensory dendrite (Seyfarth et al. 1982).

Having obtained intracellular recordings from the sensory neurons of the locust auditory organ, Hill (1983) proposed a scheme for the functional organization of the locust auditory sensillum. This hypothesis derived in particular from the model developed by Thurm et al., for the insect epidermal mechanoreceptor (e.g. Erler and Thurm 1981). Hill (1983) observed that in the locust auditory receptor cells, spike potentials of two amplitudes occur, conventional spikes of the order of 65 mV and much smaller spikes up to 30 mV. The resulting model for the locust auditory sensillum invoked the concept that the attachment cell and the adjacent scolopale lumen surrounding the sensory cilium are functionally analogous to the tormogen cell and receptor lymph cavity of the epidermal receptor, respectively (Moulins 1976). Thus, it was proposed (Hill 1983) that the smaller spike recorded in the locust auditory sensillum occurs at the apex of the dendrite, where novel electrochemical gradients exist, the spike thereby being of smaller amplitude and, that the larger spike results from conventional electrochemical gradients towards the base of the dendrite, where the neuron is in contact with conventional interstitial fluid.

The physiological data providing the basis for the Erler and Thurm model of the epidermal receptor are essentially extracellular recordings of potentials across the sensory neuron, between the receptor lymph cavity and the haemocoel ground. In terms of the proposed organization of the auditory chordotonal sensillum (Hill 1983), recordings of sensory potentials obtained from within the attachment cell would be closely analogous to the transepithelial recordings of Erler and Thurm. By combining intracellular recordings from the neuron with recordings of 'transneuronal' potentials, we have further tested some of the propositions in the model of the locust auditory sensillum. This was accomplished by recording from defined parts of the sensillum in the tettigoniid crista acustica, namely the sensory neuron and its associated attachment cell. Simultaneous recording from these two sites, using two electrodes, has enabled unequivocal interpretation of the waveforms recorded in the attachment cell and has further elucidated the organization of the insect auditory sensillum.

Materials and methods

Results were obtained from 27 adult male and female *Caedicia simplex* (Tettigoniidae: Phaneropterinae), caught on vegetation in Canberra. The meso- and metathoracic legs of each insect were removed, the insect was mounted on a perspex stage and the prothoracic legs arranged so that the auditory organs were located in depressions in the stage (0.57 cm^3). One leg was covered with insect Ringer solution (Fielden 1960, plus 3 g/l glucose) and the cuticle covering the crista acustica was cut away (Oldfield 1984). For some experiments, the tympanal nerve was located in the proximal femur and was suspended on a pair of tungsten wire electrodes. Dessication of the tympanal nerve was prevented by covering the nerve and hook electrodes with petroleum jelly. A silver, indifferent electrode was placed in the Ringer solution bath. The preparation was illuminated from below via a glass fibre optic and was viewed using either a stereo dissecting or compound microscope (see also Oldfield 1984).

Electrical potentials associated with either the spontaneous activity, background noise or acoustic stimuli (Oldfield 1984) were recorded using glass microelectrodes (80–100 MΩ), filled with a combination of 5% aqueous Lucifer Yellow CH and 1 mol/l lithium chloride (Stewart 1978). Attachment cell recordings were obtained by impaling the cells under visual control. In addition, positive spike potentials of up to 65 mV were obtained from individual nerve cells in the area occupied by the neural somata, approximately 100 μm to one side and in a different plane to the attachment cells. Using two microelectrodes, recordings were obtained simultaneously from sensory nerve cells and from their associated attachment cells. By visually and acoustically-monitoring recorded spikes from both electrodes, such simultaneous recordings from a single sensillum were recognizable. In some cases, retrograde spikes were induced in the neuron by stimulating the tympanal nerve with voltage-pulse trains (0.1 to 1.0 V, 0.5 ms duration, 5 Hz, applied proximally in the femur). To confirm the sites of recording Lucifer Yellow was expelled from the relevant recording electrode (5 nA negative current pulses at 1 Hz for 5 to 10 min). The prothoracic leg was then removed from the preparation and placed in Ringer solution on a glass microscope slide. The auditory organ was examined by fluorescent microscope.

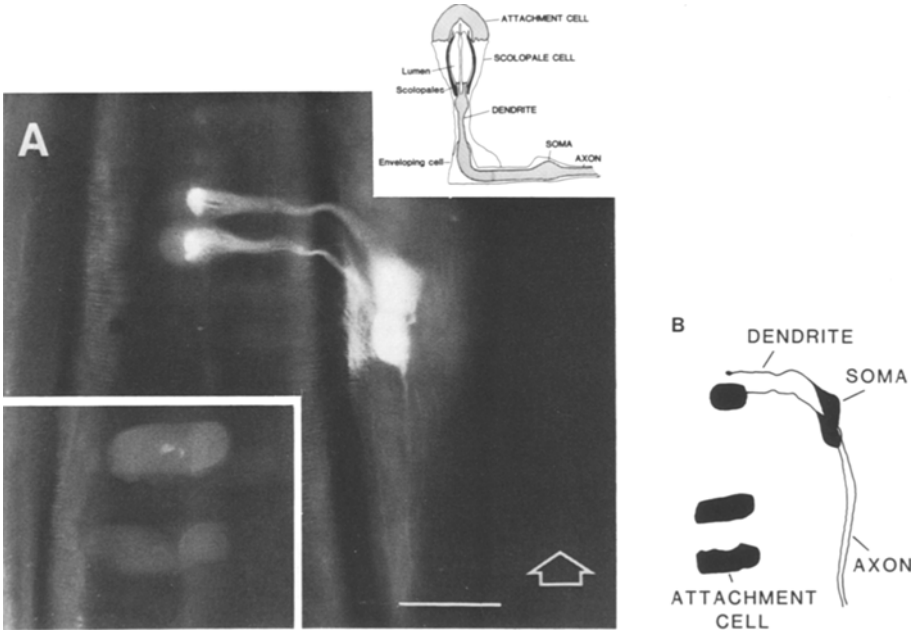

Fig. 1. A Photomicrograph of the crista acustica in *C. simplex* taken at two different planes of focus. Four sensilla are filled with Lucifer Yellow. The most distal sensillum was stained via a microelectrode inserted into the neural soma, the adjacent sensillum via a microelectrodes inserted into the attachment cell and neural soma and the two proximal sensilla via microelectrodes inserted into the attachment cell (arrow indicates distal, scale: 50 μm). Inset: Schematic diagram of a sensillum, in crossection, showing the arrangment of the cells that constitute the receptor (after Schumacher 1979). **B** Line drawing of the sensilla in (**A**) showing the location of the dye revealed in a series of focal planes. Note that dye does not pass directly between sensory neuron and attachment cell

Results

Recording sites in individual sensilla

Figure 1A shows photomicrographs of part of a crista acustica from *C. simplex* in which four of the sensilla have been marked with Lucifer Yellow. In the most distal of the four stained sensilla, the sensory neuron was filled via a microelectrode in the soma. In the adjacent sensillum, both sensory neuron and associated attachment cell were filled by separate microelectrodes. In each of the two proximal, stained sensilla, only the attachment cell has been filled.

The interpretation of the dye marking depends on viewing the crista at several planes of focus. For example, in the most distal, stained sensillum in Fig. 1A, dye appears to have entered the attachment cell in addition to the sensory neuron. This impression is created because the sensory dendrite, having projected across the tracheal surface, bends and projects towards the attachment cell out of the plane of focus. This blurred column of fluorescence directly below the attachment cell has the appearance of dye within it. Actually, there is no indication of spread of dye between the sensory neuron and its associated attachment cell (Fig. 1B) hence, the dye marking reliably confirms the locations of the recording electrodes.

Spike potentials from identified recording sites

Figure 2 shows the form of the spikes recorded from the soma of the sensory neuron in the tettigoniid crista acustica. The soma produced large (to 65 mV) and small (to 20 mV) spikes. A small spike generally appears as an inflection on the rising phase of the large spike (Fig. 2B). Hyperpolarization of the soma by current injection reduces the probability of the large spike and small spikes are then seen as distinct potentials (Fig. 2C). Hyperpolarization of the soma, adequate to block large spikes, does not necessarily reduce the frequency of the small spike potentials (e.g. Fig. 2C), suggesting that small spikes are generated elsewhere in the sensory neuron. Whereas, failure of the large spike and distinct small spikes sometimes occur in apparently normal sensory cells, orthograde large spikes always show the inflection associated with the small spike, indicating that the small spike acts as a precursor to the large spike.

Potentials derived from the sensory neuron, but recorded from its associated attachment cell, are illustrated in Fig. 3. The spike potentials appear

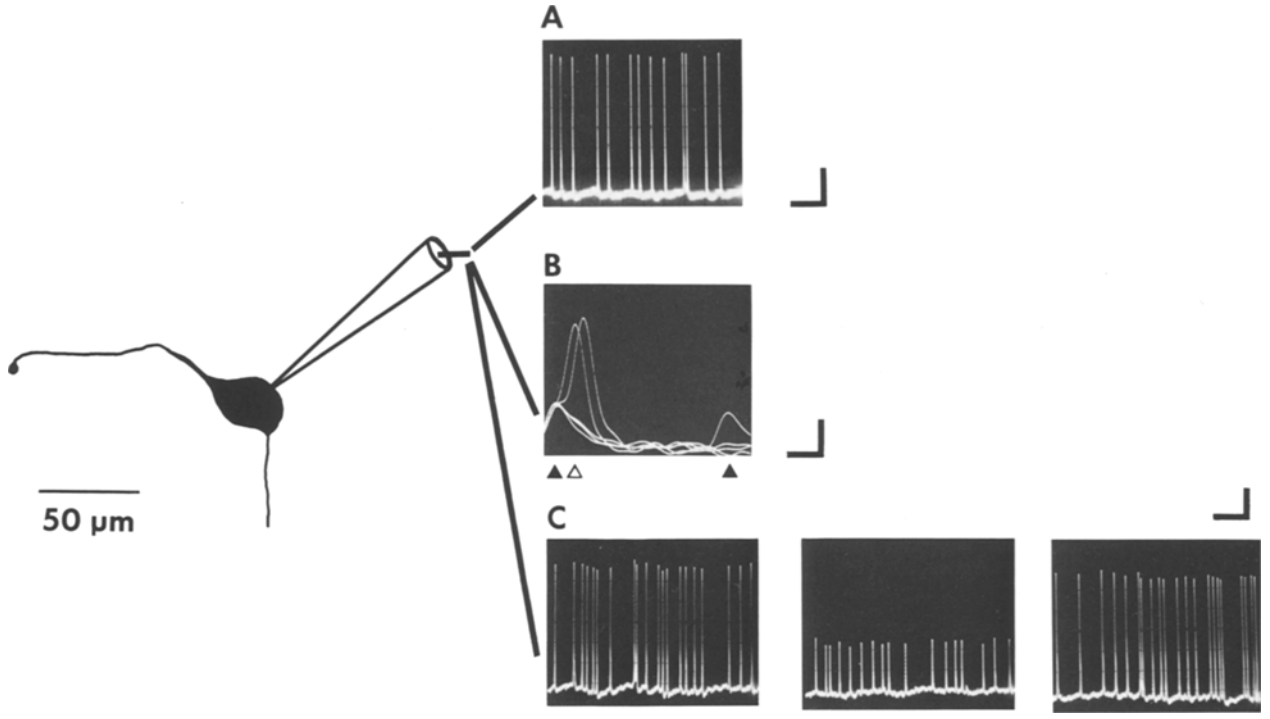

Fig. 2A–C. Spike potentials and site of recording from the neuron in individual sensilla in the crista acustica. **A** Large spike potentials (scale: 50 ms, 10 mV). **B** Superimposed traces of large (△) and small (▲) spike potentials (scale: 1 ms, 10 mV). **C** Spike potentials before (left trace), during (centre trace) and after (right trace) hyperpolarising the neuron by injecting 3 nA of negative current through the recording electrode (scale: 50 ms, 10 mV). Note that hyperpolarization of the neuron blocks the larger spike

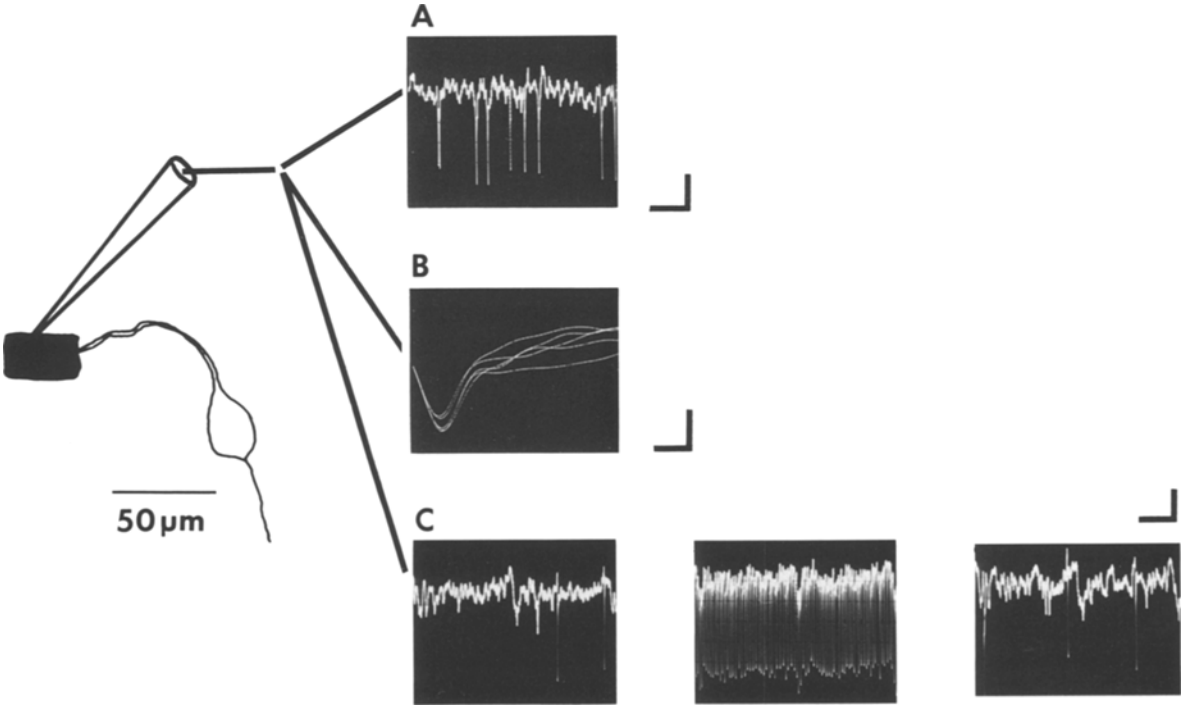

Fig. 3A–C. Spike potentials and site of recording from the attachment cell in individual sensilla in the crista acustica. **A** Spike potentials, negative monophasic and biphasic (scale: 50 ms, 5 mV). **B** Superimposed traces of negative monophasic spikes (scale: 0.5 ms, 5 mV). **C** Records obtained before (left trace), during (centre trace) and after (right trace) the injection of 3 nA of negative current into the attachment cell through the recording electrode (scale: 50 ms, 5 mV). Note the train of evoked spikes during current injection

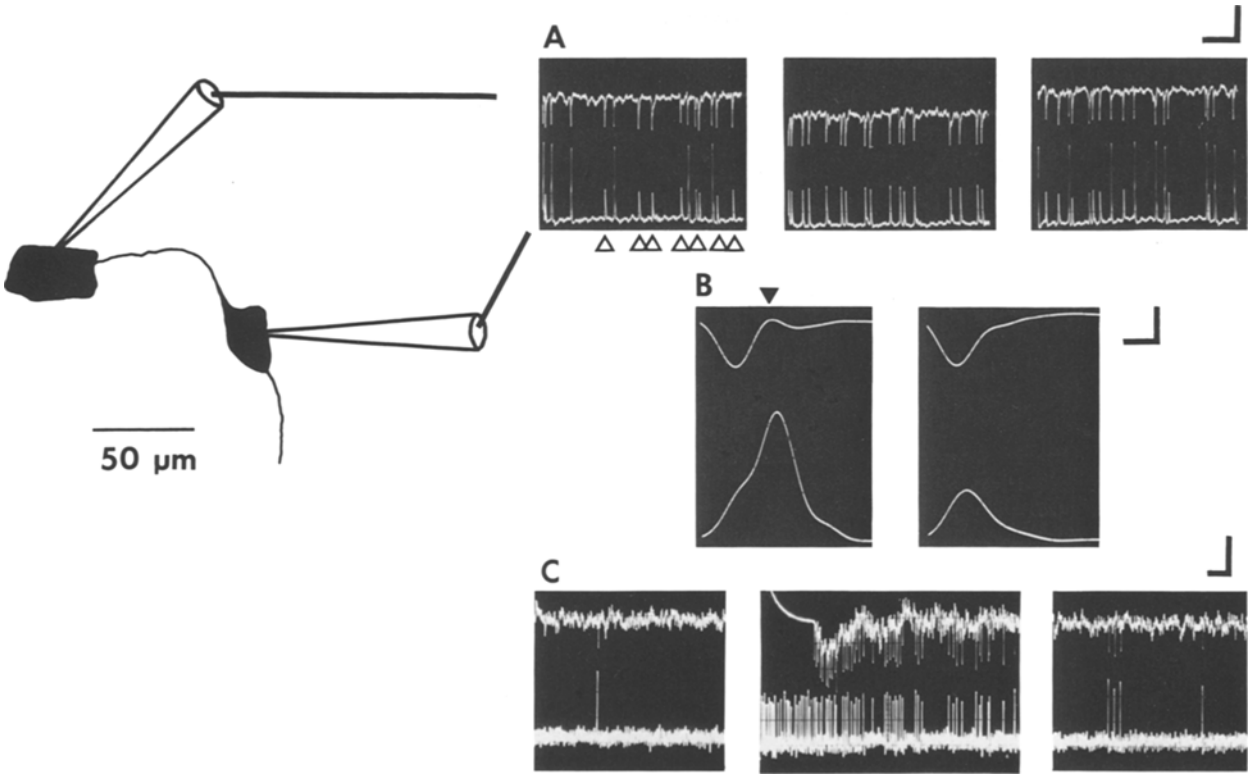

Fig. 4A–C. Spike potentials simultaneously recorded from the attachment cell (upper trace in each series) and neural soma (lower trace in each series), of individual sensilla. **A** Records obtained before (left trace), during (centre trace) and after (right trace) hyperpolarising the neuron by injecting 3 nA of negative current through the appropriate recording electrode (scale: 50 ms, 10 mV). Note that small spikes (▵) recorded in the neuron correspond with negative potentials in the attachment cell. **B** Details of waveforms of neural spikes and corresponding potentials in the attachment cell. Note that a small positive deflection (▾) in the attachment cell corresponds with the large spike recorded in the neuron (scale: 0.5 ms, 10 mV). **C** Records obtained before (left trace), during (center trace) and after (right trace) the injection of 3 nA of negative current into the attachment cell through the appropriate electrode. Note that the current injection evoked a burst of small spikes in the neuron, which was hyperpolarized in order to block large spike production, and corresponding negative spike potentials in the attachment cell. The loss of recording during the initial phase of current injection (middle trace) was due to AC-coupling of the oscilloscope (scale: 200 ms, 10 mV)

either monophasic, negative-going or biphasic, with a much smaller positive component following the negative phase. When a negative-going graded potential is observed (i.e. during acoustic stimulation), the biphasic form is more pronounced (see below).

Simultaneous recordings from sensory neuron and associated attachment cell

Viewed under a compound microscope, particular attachment cells may be impaled by choice. Specific somata of the sensory neurons, however, are not readily seen. When the spikes recorded by two electrodes were monitored with headphones, one channel to each ear, it was immediately obvious, by virtue of coincidence, when the electrodes were recording from the sensory neuron and the associated attachment cell of a single sensillum.

Figure 4A illustrates the one to one correspondence between spikes in the attachment cell and spikes in the neuron. When large spikes in the neuron are blocked by hyperpolarization of the soma (Fig. 4A, centre), negative-going spikes in the attachment cell coincide with the small spikes occurring in the neuron. In Fig. 4B, attachment cell records are compared with records from the neuron, displayed with expanded time scale. The negative spike in the attachment cell corresponds with the onset of the spike in the neuron, to the inflection in the rising phase. The remainder of the large spike in the neuron corresponds with a positive deflection in the attachment cell (Fig. 4B, left). When the large spike fails in the neuron, there is no positive component following the negative-going spike in the attachment cell (Fig. 4B, right).

When negative current is injected into the attachment cell via the impaling electrode, small spikes are evoked in the neuron (Fig. 4C). This becomes clear when large spikes are blocked by hyperpolarization of the neuron (Fig. 4C). In these conditions, in the attachment cell, such evoked

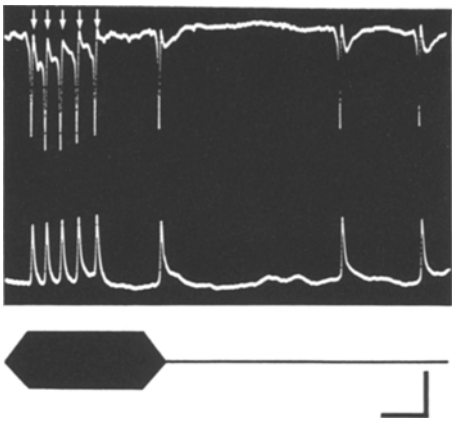

Fig. 5. Spike potentials simultaneously recorded in the attachment cell (upper trace) and neural soma (lower trace) of an individual sensillum, stimulated by sound (35 ms, 14 kHz). Note the change in the relative amplitude of the positive component of biphasic spikes (↓) during adaptation of the receptor potential, seen as a negative slow potential in the attachment cell (scale: 5 mV (upper trace), 40 mV (lower trace), 10 ms)

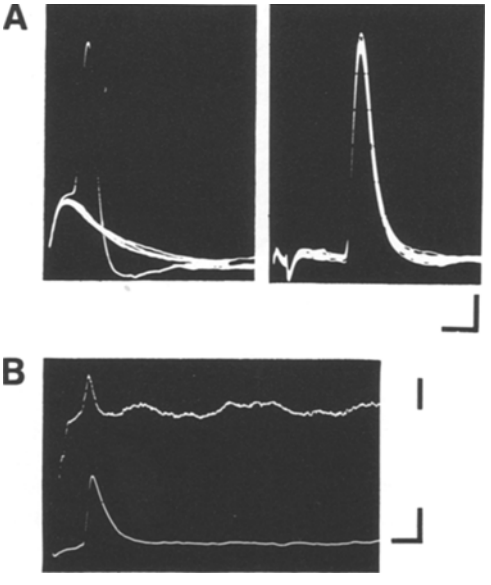

Fig. 6. A Orthograde (left trace) and evoked retrograde spike waveforms (right trace) recorded from the sensory neuron. Note the small apical spikes, one of which initiates the large orthograde spike and that the retrograde spike has no apical spike precursor. The repolarizing time course of the small spikes is longer than that of the large orthograde spike, consistent with ionic conductance mechanisms implied in the model of the sensillum (Hill 1983). The prominent undershoot on the orthograde large spike, when compared with the retrograde spike, suggests that the dendrite is relatively depolarized with respect to the axon (scale: 0.5 ms, 10 mV). **B** Simultaneous records of a retrograde spike in the neuron (lower trace) and the associated monophasic positive deflection recorded in the attachment cell (upper trace) of an individual sensillum (scale: 5 mV (upper trace), 10 mV (lower trace), 1 ms)

spikes are monophasic-negative. Large spikes in the neuron occur in association with almost all evoked small spikes, if the neuron is not hyperpolarized.

In response to an acoustic stimulus, a graded receptor potential generally is not recorded in the neural soma. In the attachment cell, however, the graded potential is apparent, negative-going and is associated with spikes (Fig. 5). Biphasic spikes recorded in the attachment cell, when superimposed on a graded potential, vary with respect to the relative amplitude of the positive component; the greater the amplitude of the graded potential, the greater the relative amplitude of the positive component of the biphasic spike recorded in the attachment cell (Fig. 5).

Spikes evoked by electrical stimulation of the nerve

Stimulating the tympanal nerve in the proximal part of the femur, using trains of voltage pulses, causes retrograde spikes in the sensory neuron. Retrograde spikes were identified as spikes consistently following voltage pulses with fixed delay and at a frequency that could be set many times higher than the background frequency of orthograde spikes. Examples of retrograde spikes recorded in the neural soma are shown in Fig. 6A. In contrast to orthograde spikes in the neuron, retrograde spikes never show the inflection during the rising phase. When recordings were made from the sensory neuron and its associated attachment cell during electrical stimulation of the tympanal nerve, the record from the attachment cell contained monophasic positive deflections, each corresponding to a retrograde spike occurring in the sensory neuron (Fig. 6B).

Discussion

The specialized, chordotonal sensilla of the tettigoniid crista acustica and of Müller's organ in the locust independently have evolved to encode certain properties of incident sound energy in the form of trains of action potentials. Intracellular recordings showed that the sensory neurons of Müller's organ produce spike potentials of two distinct amplitudes, apparently initiated in different parts of the neuron (Hill 1983). We have now found the same phenomenon in the sensory neurons of the tettigoniid crista acustica, thus demonstrating that the functional organization of the auditory, chordotonal sensillum is essentially similar in locust and tettigoniid.

The possibility of spike initiation in the distal part of a long sensory dendrite in an insect sensillum and, that such spikes may have novel waveform, was suggested by Erler and Thurm (1981). In an elongate sensory neuron, depolarization of the more basal part of the dendrite, sufficient to initiate spikes, might not be obtained from the graded receptor potential which appears to be generated at the apex of the dendrite. Regenerative potentials at the apex would constitute amplification of the sensory signal. Such spikes might be expected to have novel amplitude, however, if the fluid in contact with the apex of the dendrite were a specialized receptor lymph, possibly containing elevated potassium and reduced sodium concentrations (Kaissling and Thorson 1980; Thurm and Küppers 1980). It was suggested, therefore, that in the locust auditory sensillum, the smaller spike is initiated at the apex of the dendrite and that it triggers the larger spike at the base of the dendrite (Hill 1983). The model for the functional organization of the locust auditory sensillum was based on that for the epidermal mechanoreceptor (Erler and Thurm 1981) and on the anatomical analogies between epidermal mechanoreceptors and internal chordotonal sensilla (Moulins 1976). The results reported in the present paper confirm the proposals made for the locust auditory sensillum and show the general applicability of the main features of that model to other insect auditory sensilla and perhaps, to chordotonal organs in general.

Apical spikes

The site of initiation of the small spike is now confirmed as the apical part of the sensory dendrite. The attachment cell bounds the scolopale lumen, which surrounds the sensory cilium and apex of the dendrite and which appears to be isolated from extracellular space outside the sensillum (Schumacher 1979). The attachment cell and apical region of the dendrite, therefore, contact this isolated extracellular space. Inward ionic currents at the apex of the dendrite would be signalled by negative voltage deflections in the attachment cell. Simultaneous recordings from attachment cell and sensory neuron of a single sensillum show that negative deflections in the attachment cell correspond with small spikes in the neuron (Fig. 4), hence, the small spikes result from transient, inward cationic currents at the apex of the dendrite. Additional evidence that the generation of the small spike occurs at the apex of the dendrite is provided by current injection into the attachment cell. Some of a negative current injected into the attachment cell would pass to the scolopale lumen and lower its potential. The apical dendritic membrane, therefore, would be depolarized. Such current injection initiates trains of small spikes in the neuron (Fig. 4). We also have noted that injection of positive current into the attachment cell, which would hyperpolarize the apical dendrite, depresses the frequency of spontaneous firing in the neuron. These results correspond to the spike-initiating effect of transepithelial, outward current applied to epidermal mechanoreceptors (Erler and Thurm 1981; Seyfarth et al. 1982). The waveform of orthograde, large spikes recorded in the sensory neuron always is characterized by an inflection on the rising phase, which was shown to correspond with the small spike (Fig. 4). Large spikes, therefore, normally are triggered by the small spikes. This also occurs in the locust auditory sensillum (Hill 1983). Confirmation of this interpretation comes from backfiring the tympanal nerve. The retrograde spikes never show the inflection on the rising phase (Fig. 6).

The positive deflections recorded in the attachment cell correspond with the occurrence of large spikes in the neuron (Fig. 4). Initiation of the large spikes may occur proximal to that part of the dendrite sheathed by the scolopale cell where the dendrite first may contact interstitial fluid. The nature of the positive deflection recorded in the attachment cell depends on the circumstances in which the large spike occurs. For example, the positive phase of the biphasic spike recorded in the attachment cell, when a spontaneous, orthograde spike occurs, is of smaller amplitude than the positive deflection in the attachment cell resulting from a retrograde spike in the neuron. In addition, the amplitude of the positive phase of the biphasic spike recorded in the attachment cell, when a pronounced graded potential is observed, varies according to the amplitude of the graded potential. These observations may be explained in terms of the excitable nature of the apical dendritic membrane and the proposed function of the scolopale lumen as a receptor lymph cavity.

When a large spike occurs in the dendrite, proximal to the scolopale region, the depolarization electrotonically will invade the apical dendrite. This invading depolarization may shift apical membrane potential beyond the local equilibrium potentials for cations (given novel electrochemical gradients). In the case of an orthograde spike in the neuron the invading depolarization would follow an apical spike. Consequently, voltage-depen-

dent channels at the apex would be refractory. Either because of a passive outward current to the lumen, or by reduction of a standing inward current at the apex of the dendrite, the large orthograde spike would be expected to cause a positive deflection in the attachment cell. In the case of a retrograde spike in the neuron, the invading depolarization of the apical dendrite is not preceded by an apical spike and, therefore, would be expected to activate the voltage-dependent channels at the apex. If equilibrium potentials were exceeded, a transient outward current would occur (i.e., a reversed apical spike). Hence, in comparison with the former case, a larger and more transient positive deflection would be expected in the attachment cell.

In the case of a train of spikes superimposed on a sound-evoked graded potential (Fig. 5) the amplitude of the relative, positive shift of potential in the scolopale lumen, due to the invasion of the apical dendrite by the basal spike potential, would increase according to the conductance at the apical dendritic membrane, and decrease in accordance with adaptation in the mechanically-induced graded potential (see also Hill 1983).

As predicted (Erler and Thurm 1981), afferent spikes in primary auditory fibres in locusts and tettigoniids normally depend on spikes occurring at the apex of the sensory dendrite. Only rarely have we recorded potentials in attachment cells not showing the negative component, that signals the occurrence of an apical spike in the sensory neuron (Oldfield 1982; Hill 1983). In abnormal circumstances (e.g. hyperpolarized apical membrane and potentiated mechanically-induced graded potential) basal spikes may be initiated in the absence of apical spikes. The waveform of the confirmed, 'apical' spike implies that in the insect auditory sensillum, the scolopale lumen functions as a receptor lymph cavity, containing ionic concentrations that set a cation equilibrium potential somewhere near -30 mV. Recordings obtained directly from the scolopale lumen will elucidate this question.

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