

Phase-Dependent Presynaptic Modulation of Mechanosensory Signals in the Locust Flight System

Ansgar Büschges and Harald Wolf

J Neurophysiol 81:959-962, 1999.

You might find this additional information useful...

This article cites 15 articles, 10 of which you can access free at:

<http://jn.physiology.org/cgi/content/full/81/2/959#BIBL>

This article has been cited by 1 other HighWire hosted article:

Intrinsic noise at synapses between a wing hinge stretch receptor and flight motor neurons in the locust

P. Simmons

J. Exp. Biol., January 1, 2001; 204 (1): 127-138.

[Abstract] [PDF]

Medline items on this article's topics can be found at <http://highwire.stanford.edu/lists/artbytopic.dtl> on the following topics:

Physiology .. Membrane Potential

Physiology .. Motor Neurons

Entomology .. Locusts

Entomology .. Insect Flight

Physiology .. Mechanoreception

Physiology .. Trochacea

Updated information and services including high-resolution figures, can be found at:

<http://jn.physiology.org/cgi/content/full/81/2/959>

Additional material and information about *Journal of Neurophysiology* can be found at:

<http://www.the-aps.org/publications/jn>

This information is current as of March 8, 2007 .

Phase-Dependent Presynaptic Modulation of Mechanosensory Signals in the Locust Flight System

ANSGAR BÜSCHGES¹ AND HARALD WOLF²

¹Fachbereich Biologie, Universität Kaiserslautern, D-67653 Kaiserslautern, Germany; and ²Vergleichende Neurobiologie, Universität Ulm, D-89069 Ulm, Germany

Büschges, Ansgar and Harald Wolf. Phase-dependent presynaptic modulation of mechanosensory signals in the locust flight system. *J. Neurophysiol.* 81: 959–962, 1999. In the locust flight system, afferents of a wing hinge mechanoreceptor, the hindwing tegula, make monosynaptic excitatory connections with motoneurons of the elevator muscles. During flight motor activity, the excitatory postsynaptic potentials (EPSPs) produced by these connections changed in amplitude with the phase of the wingbeat cycle. The largest changes occurred around the phase where elevator motoneurons passed through their minimum membrane potential. This phase-dependent modulation was neither due to flight-related oscillations in motoneuron membrane potential nor to changes in motoneuron input resistance. This indicates that modulation of EPSP amplitude is mediated by presynaptic mechanisms that affect the efficacy of afferent synaptic input. Primary afferent depolarizations (PADs) were recorded in the terminal arborizations of tegula afferents, presynaptic to elevator motoneurons in the same hemiganglion. PADs were attributed to presynaptic inhibitory input because they reduced the input resistance of the afferents and were sensitive to the γ -aminobutyric acid antagonist picrotoxin. PADs occurred either spontaneously or were elicited by spike activity in the tegula afferents. In summary, afferent signaling in the locust flight system appears to be under presynaptic control, a candidate mechanism of which is presynaptic inhibition.

INTRODUCTION

In the control of rhythmic locomotor behavior, central nervous rhythm generators and sensory signals often interact closely to produce a functional motor command (e.g., Bässler and Büschges 1998; Clarac 1991; Grillner 1985; Pearson 1993). Sensory feedback may sculpture a centrally generated pattern, for instance, by resetting the movement cycle. For the locust flight system it has been shown that signals from wing proprioceptors modify centrally generated activity to produce the functional flight motor output. The most detailed understanding of these interactions concerns the role of the hindwing tegula in initiating the elevation phase of the wingbeat cycle (Ramirez and Pearson 1993; Wolf 1993; Wolf and Pearson 1988). Conversely, central commands may subject the transmission and processing of sensory signals to phase-dependent control (Sillar and Skorupski 1986; Skorupski and Sillar 1986). This was demonstrated for the control of walking (El Manira et al. 1991; Gossard et al. 1990; Wolf and Burrows 1995). Currently there is just one example (Reichert and Rowell 1985) of cyclic modulation of afferent signal processing in the locust

flight system. A number of mechanisms exist for such phase-dependent modulation, namely, rhythmic changes in membrane resistance of intercalated neurons, their cyclic de- and hyperpolarization, or presynaptic inhibition of afferent terminals (Burrows and Matheson 1994; see reviews by Clarac and Cattaert 1996; Rudomin et al. 1998). Here we report that presynaptic gating mechanisms function in the locust flight system, modulating monosynaptic input from the hindwing tegula to wing elevator motoneurons in a phase-dependent manner. Presynaptic inhibition is a candidate mechanism because primary afferent depolarizations (PADs), which are sensitive to the γ -aminobutyric acid (GABA) antagonist picrotoxin (PCT), can be recorded in the central terminals of tegula afferents.

METHODS

Fully mature *Locusta migratoria* from a breeding colony in Kaiserslautern were used for all experiments. Animals were dissected from the dorsal side and deafferented according to standard procedures (Robertson and Pearson 1982). Wind stimulation of the head elicited flight motor activity ("fictive" flight). Intracellular recordings were made with an NPI SE101 amplifier (Polder), in either bridge or discontinuous current clamp mode, from the neuropil regions of elevator motoneurons or from tegula axons close to their entrance into the metathoracic ganglion (Fig. 3A outlines the experimental situation). Extracellular hook electrodes for stimulation or en passant recording were used in bipolar configuration. Axons of hindwing tegula afferents were stimulated electrically according to established procedures, at voltages of 1.0–1.2 T (times threshold value) (Pearson and Wolf 1988). PCT was bath applied to the metathoracic ganglion at a final concentration of 1×10^{-5} M.

RESULTS AND DISCUSSION

Efficacy of synaptic transmission from tegula afferents to elevator motoneurons is modulated in the phase of the wingbeat cycle

The hindwing tegulae provide strong and, in the ipsilateral hemiganglion monosynaptic, excitatory input to wing elevator motoneurons (Pearson and Wolf 1988). When recording from metathoracic elevator motoneurons during flight motor activity, we observed phase-dependent modulation of the compound excitatory postsynaptic potentials (EPSPs) elicited by electrical stimulation of tegula afferents (Fig. 1, A and B). EPSPs had the largest amplitudes, reaching up to 15 mV in the recording shown, at phases of ~ 0.4 with regard to depressor muscle activity (Fig. 1, C and D). Smallest EPSP amplitudes were recorded at phase of ~ 0.8 – 1.0 (Fig. 1, C and D). These

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

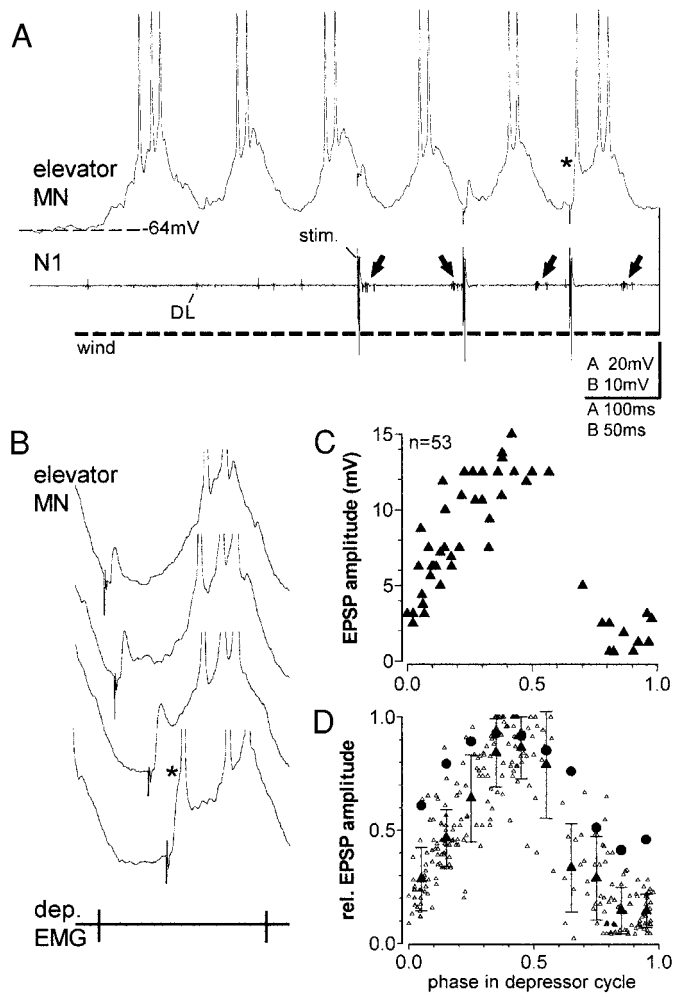


FIG. 1. Synaptic potentials elicited in elevator motoneurons by tegula input vary in amplitude with the phase of the wingbeat cycle. *A*: sample recording; *top trace*, intracellular recording of metathoracic elevator motoneuron; *middle trace*, stimulation of hindwing tegula afferents ("stim." marks afferent volley) monitored in nerve 1 (DL, spikes of dorsal longitudinal motoneurons; arrows mark depressor bursts). Note stimulus-induced compound excitatory postsynaptic potentials (EPSPs) in the motoneuron and spike elicited during 6th wingbeat cycle (*). *B*: four wingbeat periods are superimposed (with vertical offset), taking the depressor cycle as reference (*bottom trace*, digitized EMG potentials mark beginning of depressor discharge); asterisk as in *A*. Note phase-dependent modulation of EPSP amplitude. *C*: EPSP amplitude is plotted vs. phase of the depressor cycle (determined from EMG); each data point represents one EPSP. *D*: relative EPSP amplitude, normalized to the maximum value observed in a flight episode, is plotted vs. depressor phase (open triangles; 272 stimulus presentations, 12 flight episodes). Filled triangles, mean values calculated for phase bins of 0.1 from this data set, standard deviations indicated; same recording as in *A–C*. Filled circles show data from another recording to illustrate the variability of changes in EPSP amplitude and the rather constant phase values of their occurrence.

observations were consistent in 12 recordings, from different metathoracic elevator motoneurons in different animals.

Neither cyclic changes in the membrane potential nor in the input resistance of motoneurons account for modulation of EPSP amplitude

Possible mechanisms of this phase-dependent modulation of EPSP amplitude (see INTRODUCTION) were examined. First, we tested if flight-related oscillations in the motoneurons' mem-

brane potential underlie these changes. At more positive potentials, EPSPs will decrease in amplitude because of decreasing ionic driving forces [a phenomenon observed during depolarizing current injection into motoneurons of quiescent locusts (e.g., Ramirez and Pearson 1991); this holds only if no voltage-dependent ion channels are present in the dendritic region]. Examination of the correlation between EPSP amplitude and membrane potential (Fig. 2*A*) yielded data that were contrary to this expectation. Maximum EPSP amplitudes occurred close to phase 0.4, at a time when the elevator motoneu-

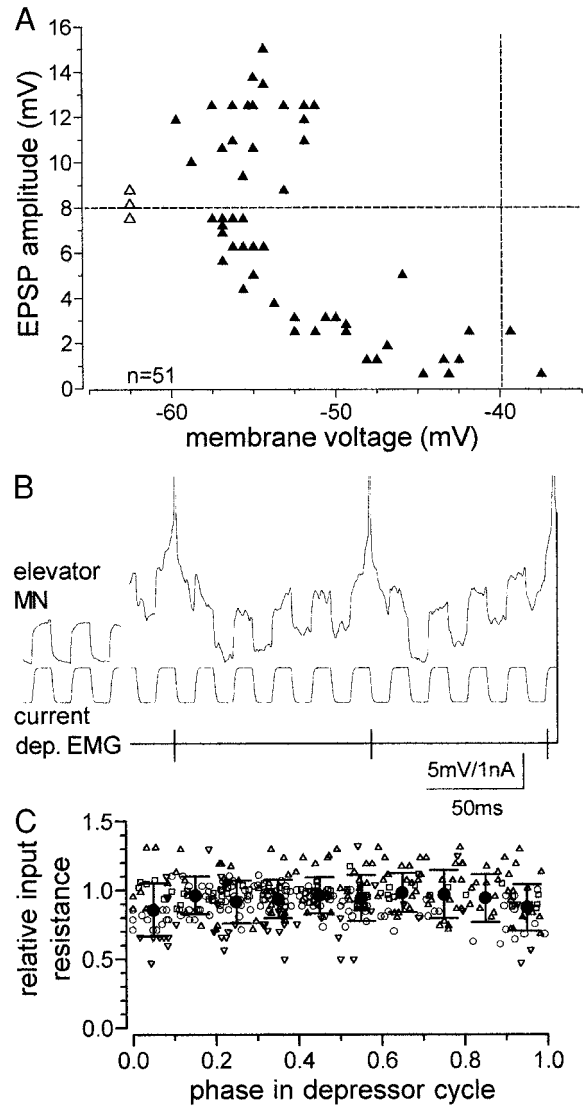


FIG. 2. Modulation in EPSP amplitude is due neither to flight-related oscillations in the motoneurons' membrane potentials (*A*) nor to changes in their input resistances (*B* and *C*). *A*: EPSP amplitude is plotted vs. membrane potential (at the time the PSP was elicited, experimental situation as in Fig. 1, *A* and *B*). Horizontal line indicates average EPSP amplitude before flight (open triangles, sample data points near -62.5 mV in all recordings evaluated); vertical line at -40 mV marks spike threshold. EPSPs may be larger during flight motor activity than at rest. *B*: changes in the input resistance of elevator motoneurons were examined by injection of current pulses (discontinuous current clamp mode, 12.5-ms pulses of -1 nA, 50% duty cycle) during flight motor activity. Motoneurons were held slightly hyperpolarized (-2 to -3 nA) to reduce spike discharge. *C*: relative input resistance, normalized to preflight values, is plotted vs. the phase in the wing depressor discharge period (open triangles, data from 4 animals, 10 flight cycles each; filled circles, mean values calculated for phase bins of 0.1, standard deviations indicated).

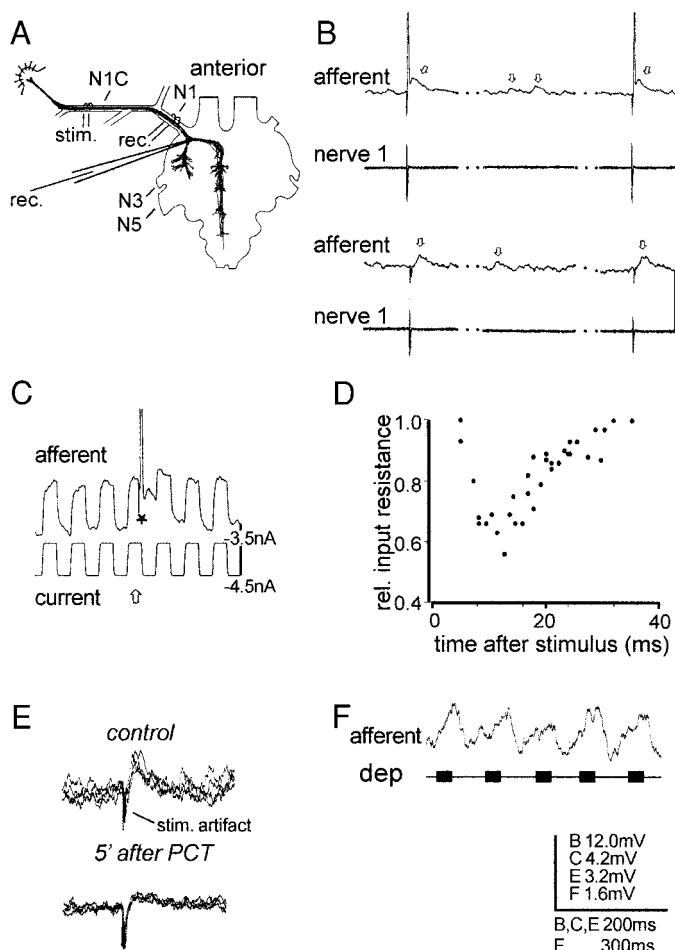


FIG. 3. Primary afferent depolarizations (PADs) occur in the central arborizations of hindwing tegula afferents. *A*: experimental situation. Intracellular recordings from arborizations of hindwing tegula afferents in the metathoracic ganglion (rec.), electric stimulation of tegula axons in nerve 1C1 (stim.), resulting afferent spikes monitored as compound potentials in nerve 1 (rec., asterisk in *C*). *B*: PADs (open arrows, *top traces*) were either related to afferent spike activity (*bottom traces*) or occurred spontaneously. Resting membrane potential of tegula afferents was -61.0 ± 5.9 mV ($n = 6$). Afferents were hyperpolarized to approximately -75 mV to increase the amplitude of PADs (*top traces*) because their reversal potential was close to -60 mV. Stimulus-related PADs also occurred when the recorded afferent itself was not activated by the stimulus (*bottom sample*). *C* and *D*: input resistance of an afferent was determined by injection of 25-ms current pulses (*C*, *bottom trace*; compare Fig. 2*B*); asterisk marks stimulus artifact preceding afferent spike in the intracellular recording (*C*, *top trace*). *D*: relative input resistance, normalized to input resistance at rest, is plotted vs. time after stimulus presentation. A maximum decrease in input resistance, $\sim 40\%$, was reached ~ 13 ms after an afferent spike discharge. *E*: bath application of 0.05 mM picrotoxin (PCT) abolished stimulus-related (and spontaneous, not shown) PADs within 5–10 min. Four sample recordings of stimulus-related activity (note stimulus artifacts) in a tegula afferent are superimposed before (control) and 5 min after PCT application. *F*: recording tegula afferents near their terminal arborizations during flight motor activity showed cyclic fluctuations in membrane potential (dep, digitized depressor EMG; width of bars indicates burst length, compare Fig. 1*A*). No spikes were discharged in the deafferented preparation.

rons already started to depolarize, after the interval of least depolarized membrane potential between phase 0.1 and 0.3 (approximately -56 mV). It was in fact during this interval of relatively constant membrane potential that the most dramatic changes in EPSP amplitude occurred. During flight motor activity, EPSP amplitude was consistently and inversely re-

lated to membrane potential only at more positive potentials (greater than -51 mV). Minimum EPSP amplitudes were observed near phase 0.9, when the elevator motoneurons already started to repolarize. In addition, maximal EPSP size was significantly larger during fictive flight than at rest, despite the more depolarized membrane potentials recorded during flight motor activity (Fig. 2*A*). Similar results were obtained in four other animals.

Second, cyclic changes in membrane resistance, brought about by flight-related synaptic input to the motoneurons, might cause the observed modulation in EPSP amplitude. Possible changes in membrane resistance were examined by injection of constant-current pulses during fictive flight (Fig. 2*B*; $n = 5$). We observed only minor cyclic changes, not remotely sufficient to explain the observed variation in EPSP amplitude (Fig. 2*C*).

Central arborizations of tegula afferents receive presynaptic inhibitory input

Presynaptic inhibition of tegula afferent terminals is the third candidate mechanism, which may be responsible for the cyclic modulation of EPSP amplitude. Indeed, PADs were recorded in the afferent axons of the hindwing tegula, near their terminal arborizations (Fig. 3*A*). In quiescent locusts, PADs occurred spontaneously, but they were also elicited by spike activity in the tegula afferents (Fig. 3*B*). A number of observations suggested that these PADs represent presynaptic inhibitory input (cf. Clarac and Cattaert 1996). 1) The input resistance of afferents, determined by the injection of current pulses (Fig. 3*C*), decreased by 30% during the occurrence of PADs (Fig. 3*D*; $n = 3$). 2) Bath application of PCT, an antagonist of the inhibitory transmitter GABA, abolished PADs (Fig. 3*E*; $n = 3$). 3) The reversal potential of PADs was approximately -60 mV ($n = 4$) (i.e., close to the equilibrium potential of chloride ions, not shown). 4) Finally, recordings from the terminal arborizations of tegula afferents in the metathoracic ganglion revealed cyclic changes in membrane potential during ongoing flight motor activity (Fig. 3*F*). We did not yet determine if these fluctuations in membrane potential indeed reflect cyclic changes in presynaptic input.

In summary, these results provide evidence that the central terminals of hindwing tegula afferents receive presynaptic input modulating their synaptic efficacy in the flight cycle. Presynaptic inhibition is a candidate mechanism for this effect. Further experiments are needed to assess the actual role of presynaptic mechanisms in the phase-dependent modulation of synaptic transmission between tegula afferents and elevator motoneurons and the functional implications for locust flight.

We gratefully acknowledge support of our work by U. Bässler, W. Rathmayer, and R. Wehner. T. Heller and C. Dittrich provided skillful technical assistance. A. Büschges and H. Wolf were Heisenberg fellows of the Deutsche Forschungsgemeinschaft (Bu857 and Wo466) during the experimental work.

Present address of A. Büschges: Zoologisches Institut, Universität Köln, D-50923 Köln, Germany.

Address reprint requests to H. Wolf.

Received 28 May 1998; accepted in final form 1 October 1998.

REFERENCES

- BÄSSLER, U. AND BÜSCHGES, A. Pattern generation for stick insect walking movements—multisensory control of a locomotor program. *Brain Res. Rev.* 27: 65–88, 1998.

- BURROWS, M. AND MATHESON, T. A presynaptic gain control mechanism among sensory neurons of a locust leg proprioceptor. *J. Neurosci.* 14: 272–282, 1994.
- CLARAC, F. How do sensory and motor signals interact during locomotion? In: *Motor Control: Concepts and Issues*, edited by D. R. Humphrey and H. J. Freund. New York: Wiley, 1991, p. 199–221.
- CLARAC, F. AND CATTART, D. Invertebrate presynaptic inhibition and motor control. *Exp. Brain Res.* 112: 163–180, 1996.
- EL MANIRA, A., DICAPRIO, R. A., CATTART, D., AND CLARAC, F. Monosynaptic interjoint reflexes and their central modulation during fictive locomotion in crayfish. *Eur. J. Neurosci.* 3: 1219–1231, 1991.
- GOSSARD, J. P., CABELGUEN, J. M., AND ROSSIGNOL, S. Phase-dependent modulation of primary afferent depolarization in single cutaneous primary afferents evoked by peripheral stimulation during fictive locomotion in the cat. *Brain Res.* 537: 14–23, 1990.
- GRILLNER, S. Neurobiological basis of rhythmic motor acts in vertebrates. *Science* 228: 143–149, 1985.
- PEARSON, K. G. Common principles of motor control in vertebrates and invertebrates. *Annu. Rev. Neurosci.* 16: 265–297, 1993.
- PEARSON, K. G. AND WOLF, H. Connections of hindwing tegulae with flight neurones in the locust, *Locusta migratoria*. *J. Exp. Biol.* 135: 381–409, 1988.
- RAMIREZ, J. M. AND PEARSON, K. G. Octopaminergic modulation of interneurons in the flight system of the locust. *J. Neurophysiol.* 66: 1522–1537, 1991.
- RAMIREZ, J. M. AND PEARSON, K. G. Alteration of bursting properties in interneurons during locust flight. *J. Neurophysiol.* 70: 2148–2160, 1993.
- REICHERT, H. AND ROWELL, C.H.F. Integration of nonphaselocked exteroceptive information in the control of rhythmic flight in the locust. *J. Neurophysiol.* 53: 1201–1218, 1985.
- ROBERTSON, R. M. AND PEARSON, K. G. A preparation for the intracellular analysis of neuronal activity during flight in the locust. *J. Comp. Physiol.* 146: 311–320, 1982.
- RUDOMIN, P., ROMO, R., AND MENDELL, L. *Presynaptic Inhibition and Neural Control*. New York: Oxford Univ. Press 1998.
- SILLAR, K. AND SKORUPSKI, P. Central input to primary afferent neurones in the crayfish, *Pacifastacus leniusculus*, is correlated with rhythmic output of thoracic ganglia. *J. Neurophysiol.* 55: 678–688, 1986.
- SKORUPSKI, P. AND SILLAR, K. Phase-dependent reversal of reflexes mediated by the thoracocoxal muscle receptor organ in the crayfish, *Pacifastacus leniusculus*. *J. Neurophysiol.* 55: 689–695, 1986.
- WOLF, H. The locust tegula: significance for flight rhythm generation, wing movement control and aerodynamic force production. *J. Exp. Biol.* 182: 229–253, 1993.
- WOLF, H. AND BURROWS, M. Proprioceptive sensory neurons of a locust leg receive rhythmic presynaptic inhibition during walking. *J. Neurosci.* 15: 5623–5636, 1995.
- WOLF, H. AND PEARSON, K. G. Proprioceptive input patterns elevator activity in the locust flight system. *J. Neurophysiol.* 59: 1831–1853, 1988.