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

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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 sculpt 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 SE10l 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 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
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’ membrane 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. 2A) yielded data that were contrary to this expectation. Maximum EPSP amplitudes occurred close to phase 0.4, at a time when the elevator motoneu-
resistance of an afferent was determined by injection of 25-ms current pulses. Bottom sample itself was not activated by the stimulus (Fig. 2C).

2 Afferent spike in the intracellular recording (bottom trace). During flight motor activity, EPSP amplitude was consistently and inversely related 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. 2A). 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. 2B; n = 5). We observed only minor cyclic changes, not remotely sufficient to explain the observed variation in EPSP amplitude (Fig. 2C).

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. 3A). In quiescent locusts, PADs occurred spontaneously, but they were also elicited by spike activity in the tegula afferents (Fig. 3B). 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. 3C), decreased by 30% during the occurrence of PADs (Fig. 3D; n = 3). 2) Bath application of PCT, an antagonist of the inhibitory transmitter GABA, abolished PADs (Fig. 3E; 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. 3F). 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.

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