RAPID COMMUNICATION

Antidromic Modulation of a Proprioceptor Sensory Discharge in Crayfish

MICHELLE BÉVENGUT, FRANÇOIS CLARAC, AND DANIEL CATTAERT
Centre National de la Recherche Scientifique-Unité Propre de Recherche 9011, Neurobiologie et Mouvements, 13402 Marseille Cedex 20, France

Bévengut, Michelle, François Clarac, and Daniel Cattaert. Antidromic modulation of a proprioceptor sensory discharge in crayfish. J. Neurophysiol. 78: 1180–1183, 1997. In the proprioceptive neurons of the coxo-basal chordotonal organ, orthodromic spikes convey the sensory information from the cell somata (located peripherally) to the central output terminals. During fictive locomotion, presynaptic depolarizations of these central terminals elicit bursts of antidromic spikes that travel back to the periphery. To determine whether the antidromic spikes modified the orthodromic activity of the sensory neurons, single identified primary afferents of the proprioceptor were recorded intracellularly and stimulated in vitro preparations of crayfish nervous system. Depolarizing current pulses were delivered in trains whose frequency and duration were controlled to reproduce bursts of antidromic spikes similar to those elicited during fictive locomotion. According to their frequencies, these antidromic bursts reduce or suppress the orthodromic discharges in both position- and movement-sensitive neurons. They induce both a long-lasting silence and a gradual recovery after their occurrences. Neither the collision between the afferent and the efferent messages nor the release of serotonin by the sensory neurons can explain these results. We therefore conclude that antidromic bursts produce a peripheral modulation of the orthodromic activity of the sensory neurons, modifying their sensitivity by mechanisms yet unknown.

INTRODUCTION

In the central nervous system of both vertebrates and invertebrates, presynaptic inhibition of primary afferents is correlated with primary afferent depolarization (PAD) (Écles et al. 1962, 1963; Jiménez et al. 1988; Kennedy et al. 1974; Kirk and Wine 1984; Sillar and Skorupski 1986). Centrally, PADs reduce the amplitudes of the orthodromic sensory spikes and thereby of the transmitter release they induce, thus reducing the excitatory postsynaptic potentials in the postsynaptic neurons (Cattaert et al. 1992, 1994; Hedwig and Burrows 1996; Kirk 1985; Pearson and Goodman 1981).

In vertebrates, PADs are associated with antidromic spikes recorded from leg primary afferents in dorsal root filaments during both fictive (Dubuc et al. 1985, 1988; Gossard et al. 1989, 1991) and normal walking (Belozerova and Rossignol 1994, 1995). In crayfish, during fictive locomotion, 90% of the primary afferents of a leg proprioceptor (the coxo-basal chordotonal organ, CBCO) receive phasic bursts of PADs (4–20 mV in amplitude) locked in phase with the locomotor rhythm (El Manira et al. 1991b). Moreover, PADs of large amplitudes (15–20 mV) seen in 40% of these afferents are able to trigger antidromic spikes that travel toward the periphery in the sensory axons (El Manira et al. 1991b). These spikes have no postsynaptic effect centrally (Cattaert et al. 1994; El Manira et al. 1991b) but their peripheral actions are yet unknown. Therefore, we wanted to test whether the antidromic spikes were able to modify the orthodromic activities of the sensory neurons. In crayfish, the possibility of stimulating and recording intracellularly from identified CBCO afferents enables us to investigate such a mechanism.

METHODS

Experiments were performed in oxygenated physiological saline on 20 in vitro preparations of the nervous system of crayfish, Procambarus clarkii (El Manira et al. 1991a; Sillar and Skorupski 1986). The preparation consisted of the last three thoracic and the first abdominal ganglia of the ventral nerve cord dissected together with all the nerves of the two proximal segments of the left fifth pereiopod (Fig. 1A). The strand of the coxo-basal chordotonal organ (CBCO), containing the sensory cell bodies of this proprioceptor, also was dissected intact, and its distal end was attached to an electromagnetic puller (Ling Dynamic systems, VT101) controlled by a home-made function generator.

Extracellular activity in the CBCO nerve was recorded using “en passant” platinum wire electrodes (200 μm in diameter) insulated with petroleum jelly (Vaseline). The left fifth ganglion was desheathed to allow intracellular recordings from CBCO sensory terminals (CBT) in the neuropile. Glass microelectrodes, filled with 3 M KCl (resistance = 10–12 MΩ), were connected to an Axoclamp 2A amplifier (Axon Instruments). Data were recorded on a digital tape recorder (Biologic-1801) and through appropriate interface and software (Cambridge Electronic Device) directly onto a personal computer.

For each CBT, intracellular stimuli were delivered in trains whose frequency and duration were chosen. For each sequence of n trains at given parameters, a peristimulus histogram was calculated (bins of 20 ms) and normalized by dividing the bin values by n. Thus in the results, normalized histograms express the averaged number of occurrences of the orthodromic spikes per bin against time.

RESULTS

During fictive locomotion, CBTs received spontaneous phasic burst of PADs (Fig. 1B1). These PADs were able to elicit antidromic spikes in the sensory afferents (Fig. 1B2). The instantaneous firing frequency of the antidromic spikes within spontaneous bursts was between 2 and 100 Hz for doublets, with a mean instantaneous firing frequency from 20 to 50 Hz (unpublished data; the mean instantaneous
The activation of which might have altered the specific actions of the antidromic spikes.

Bursts of antidromic spikes in the CBTs (Fig. 1, C and D) were triggered by injecting trains of square pulses of depolarizing current. The amount of injected current and the duration of each pulse were set to trigger only one antidromic spike per current pulse (Fig. 1, C2 and D2). The analysis of the intracellular recording of the CBT traces enabled us to discriminate easily between the orthodromic and the antidromic spikes (respectively Fig. 1C, 1 and 2). Furthermore, the extracellular recordings of the CBCO nerve showed that the triggered antidromic spikes traveled in the nerve up to the sensory neurons of the CBCO even when orthodromic spikes were present (Fig. 1, C and D).

The effects of trains of antidromic spikes on the activity of the different cell types of the CBCO (Mill 1976) were tested for 11 position-sensitive neurons (Fig. 2) and for 10 movement-sensitive ones (Fig. 3). In both figures, under each example of recorded CBTs, a histogram of the averaged number of occurrences of the orthodromic spikes is shown for sequences of trains of stimuli at 10 and 30 Hz. In our experimental conditions, we were able to trigger antidromic spikes in all the recorded CBTs; all of them except two position-sensitive ones showed modifications of their firing activity. For the nine position-sensitive neurons that responded, three results are seen (Fig. 2). First, during stimulation, the number of occurrences of the orthodromic spikes was reduced (Fig. 2A) and then suppressed (Fig. 2B) in the CBTs. Second, the antidromic bursts produced a long-lasting silence of the orthodromic activity (300 ms duration; Fig.

**FIG. 1.** A: In vitro preparation comprises last 3 thoracic (T3–5) and first abdominal (A1) ganglia, motor nerves innervating proximal leg muscles (promotor, PRO; remotor, REM; anterior A and posterior P levator, LEV; depressor, DEP), and coxo-basal chordotonal organ (CBCO) and nerve (CBn). Both extracellular (●) and intracellular (CBT, sensory terminal of a CBCO unit) recording sites are labeled. B: during fictive locomotion, spontaneous bursts of primary afferent depolarizations (PADs) producing antidromic spikes are displayed rhythmically in a CBT (1). Simultaneous recordings from CBn and from a CBT show intracellular and extracellular antidromic spikes (●) during these bursts (2). C: simultaneous recordings from CBn and from a CBT are shown during a triggered burst of antidromic spikes (stim in 1) and demonstrates that antidromic (●) and orthodromic spikes are easily distinguished (2). D: superimposed recordings (n = 5) of CBn and a CBT illustrate time relationships between extracellular and intracellular spikes for orthodromic (1) and antidromic spikes (2).

**FIG. 2.** Spiking activity of a position-sensitive unit of CBCO (CBT) is shown during a triggered burst of antidromic spikes at 10 Hz (A) and at 30 Hz (B). Histograms represent averaged number of occurrences of orthodromic spikes per bin of 20 ms against elapsed time (n gives number of bursts analyzed).
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