Résumé. Le dibutyryle et l'AMP cyclique ont apparemment un effet sur la conductibilité membraneuse. Cet effet se marque par une réduction d'amplitude et de vitesse de conductibilité aussi bien que par une augmentation du seuil à atteindre et du retard dans l'élaboration du processus. Ces effets ont été confirmés par la théophylline et la caféine. Les ions de calcium semblent accélérer les effets du AMP cyclique en augmentant la résistance membraneuse (réduction d'amplitude de l'impulsion nerveuse) de 30%.

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Somatotopic Organization of Inhibition in the Crayfish Abdomen of *Procambarus clarkii*

The discovery of identified neurons has made it possible to map the somatotopic organization of small invertebrate ganglia[1]. However no definite principles have been found to govern where a particular identified neuron has its soma located. The somatotopic organization of the neuromuscular system controlling the deep extensor and flexor muscles of the crayfisch abdomen have been studied. It is these muscles which serve to flex and extend the 'tail' during the swimming behavior which propels the animal backwards. All the motoneurons to the deep flexor muscles have been identified, and characterized both electrophysiologically and geometrically[2]. The present study reports both electrophysiological and morphological information regarding the peripheral inhibitor neuron to the deep extensor muscles, in the third abdominal ganglion of the crayfish.

Large *Procambarus clarkii* (5–8 inches) were used. A preparation consisting of a block of deep extensor muscles from 1 hemi-segment, including its intact motor nerve supply (NPM) and a section of the ventral nerve cord (VNC), was isolated in cold crayfish Ringer solution[3].

Antidromic stimuli of 0.1 msec duration were applied to NPM while individual somata within the 3rd abdominal ganglion were sampled one by one via the intracellular electrode. A deep extensor motoneuron soma (E1) located in this fashion was found in close proximity to F1, the flexor motor giant. The cell body is contralateral to the second root via which its axon exists the ganglion. A small (less than 15 mV) soma potential is recorded in response to NPM stimulation. The possibility of a chemical synapse between the NPM stimulating site and the cell body being monitored was ruled out when stimuli were delivered at rates of 100 Hz and above. No known chemical synapses to motor neurons are reported to follow at this high frequency[4]. The soma potential followed NPM stimulation 1:1 at this frequency. Next, depolarizing current pulses were passed through the intracellular soma electrode,

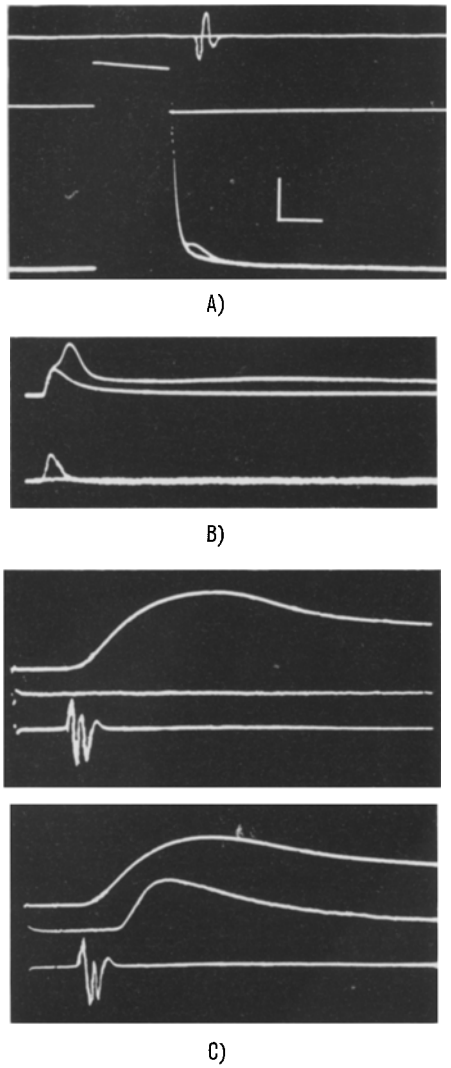

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Fig. 1. A) Depolarization of E1 via intrasomatic electrode. The top and bottom traces show 2 superimposed sweeps. The 1st sweep is subthreshold for E1 activation; the extracellular record of NPM (top trace) is flat while the intracellular potential (bottom trace) measured on a bridge circuit drops smoothly back to baseline. The 2nd sweep shows supra threshold stimulation of E1. An action potential is recorded at NPM, and a soma potential can be seen as a bump in the intrasomatic trace just before the return to baseline. The middle trace is the current monitor, recorded only during the suprathreshold sweep. Demonstration of inhibitory action of E1 on deep extensor muscles. B) 2 superimposed sweeps before and after activation of E1 by NPM stimulation. A 50% reduction of the muscle potential in a fibre of DEAM, the most medial deep extensor subunit (upper trace) occurs concurrently with E1 activity (bottom trace). C) Electrical activity in L2, the most lateral deep extensor subunit (top trace) before and after E1 activation (middle trace) via NPM stimulation. The repolarizations of L2 to 25 mV above resting potential after the peaks of the muscle potentials demonstrate the shortened time course of the excitatory junction potential current with E1 activity. The bottom traces are extracellular recording from proximal R2. Calibrations: A) 5 msec, 5 mV for bottom trace, for middle trace. B) 20 msec, 30 mV upper, 5 mV lower. C) 2 msec, 20 mV upper, 5 mV middle.

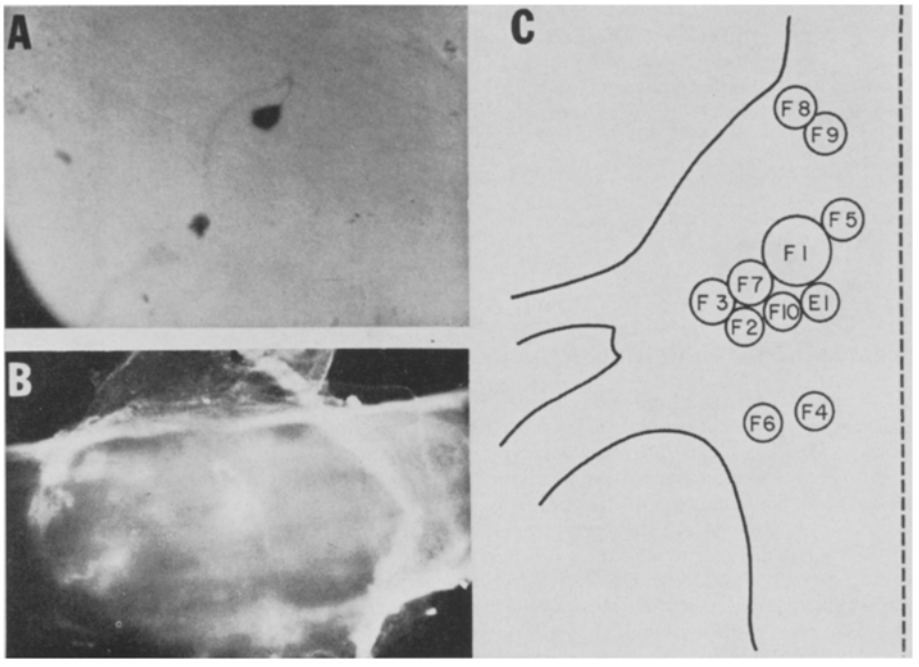

Fig. 2. A) Cobalt-injected E1. This view is looking down upon the ventral surface of the ganglion, in a cleared whole-mount. E1 is at the top of the picture. The filled cell at the bottom of the picture is another cell, unsuccessfully filled. B) Procion Yellow filled E1. This is a cleared whole-mount, viewed with the ganglion tilted toward its edge. C) Topographic locations of E1 the extensor inhibitor, particularly in reference to F-10 the flexor inhibitory and other flexor motoneurons.

and action potentials initiated were recorded at NPM (Figure 1A). Muscle contractions were never observed in the deep extensor muscles in response to activation to E1, leading us to believe that this was the peripheral inhibitor to the deep extensors[5]. E1 and a muscle fibre in one of the deep extensor subunits were impaled simultaneously[6]. NPM was stimulated at a voltage just sufficient to activate 2 excitatory deep extensor motoneurons. A visible twitch occurred, as well as a large (50 mV) excitatory junction potential (EJP). The stimulus current was then raised slightly, so that the E1 was activated along with the excitatory fibres. A marked reduction in muscle twitch occurred as well as a 50% decrease in EJP amplitude (Figure 1B). This is a larger reduction than previously reported[7], but was consistently obtained. The time course of the excitatory response was also shortened (Figure 1C) as previously reported. E1 responses were seen in all 3 subunits of the deep extensor. This indicates that E1 is almost certainly the inhibitory axon[6].

E1 cell bodies were injected iontophretically with Procion Yellow[8] and with the cobalt chloride[9]. The geometry of E1 was quite consistent in many injections. A thin neurite leaves the cell body and first heads away from, and then swings toward the contralateral second root (Figure 2A and B). The cell was markedly free of dendritic branching. This was evident in the whole mounts, and verified in serial cross sections through the ganglion. This lack of branching is in marked contrast to F10, the peripheral inhibitor to the deep flexors, which exhibits a large dendritic field[10].

The most striking feature of E1's topographic position in the ganglion is its close proximity to F10 (Figure 2C). The cell body of E1 was often touching that of F10 when viewed under the dissecting microscope. E1 is generally more closely grouped with F10 than with any of the excitatory motoneurons to the deep extensors which have been identified[11]. A similar grouping of inhibitors was found in lobster abdominal ganglia[12]. This is some further evidence to indicate a grouping of motoneurons according to transmitter biochemistry. In the lobster abdominal flexors and extensors[12], in the crayfish abdominal flexors[13] and extensors, and in leech motor neurons[14], the inhibitory cell bodies are all contralateral. This may be primarily related to the need for electrical coupling but also appears to be a pattern amongst inhibitory motor neurons.

Zusammenfassung. Mittels elektrophysiologischer Methode und Kobaltinjektion wurde der Zellkörper eines als Streckungsinhibitor wirkenden motorischen Neurons im Abdomen des Krebses *Procambarus clarkii* lokalisiert. Es zeigte sich, dass ein schmales, unverzweigtes Neurit zunächst vom Zellkörper weggeht und dann kontralateral zur Ausgangswurzel verläuft.

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