Sensory Input Induces Long-Lasting Changes in the Output of the Lobster Pyloric Network

S. L. HOOPER, M. MOULINS, AND L. NONNOTTE
Laboratoire de Neurobiologie et Physiologie Comparées, Université de Bordeaux I and CNRS, 33120 Arcachon, France

SUMMARY AND CONCLUSIONS

1. A long-lasting restructuring of the pyloric neural network of the lobster stomatogastric nervous system (STS) by a multisynaptic sensory afferent is described. This restructuring can be obtained either by mechanical stimulation of the pyloric region of the stomach or by brief high-frequency electrical stimulation of a nerve that innervates this region, the lateral posterolateral nerve (lpn). Electron microscopy shows that this nerve contains several thousand very small fibers (~0.3 μm diam), the activation of some subset of which is responsible for the effects of lpn stimulation.

2. These stimulation paradigms result in both short-duration changes in pyloric activity and modulatory effects long outlasting the stimulus end. The long-lasting changes include the cessation of rhythmic ventricular dilator (VD) and lateral pyloric (LP) neuron activity, and thus result in a reduced pyloric pattern in which only the pyloric dilator (PD), inferior cardiac (IC), anterior burster (AB), and pyloric (PY) neurons are active.

3. Tonic low-frequency lpn stimulation, alternatively, results in the VD neuron rhythmically firing long spike bursts with a cycle frequency much slower than that of the pyloric network while an otherwise complete pyloric pattern continues. In this new bursting pattern the VD neuron fires exclusively with an otherwise complete pyloric pattern. In this network, the cardiac sac (CS) network, and thus functionally “switches” from the pyloric to the CS network. This switch of the VD neuron from the pyloric to the CS network occurs when the CS network is spontaneously active.

4. Our results thus demonstrate that sensory input can provoke a long-lasting modification of the functional configuration of a rhythmic neural network. They further extend the concept of flexibility in nervous systems by showing that individual neurons can belong to more than one neural network, “switching” from one to another in response to sensory input or spontaneous central nervous activity.

INTRODUCTION

Rhythmic motor acts, such as walking, scratching, and breathing, constitute a large part of the behavioral repertoire of animals. The basic rhythmicity and pattern of these acts are generated by central neural networks (central pattern generators, CPGs) that can be endogenously active in the absence of patterned sensory input (Delcomyn 1979; Selverston and Moulins 1985). The neuronal characteristics and synaptic connectivities that underlie the endogenous rhythmicity of these networks when isolated have been investigated in many different preparations (Getting 1986; Roberts and Roberts 1983; Selverston and Moulins 1985), and an understanding of the mechanisms involved is gradually emerging. However, in the intact animal these networks are known to receive sensory inputs that can alter their activity in at least two ways. First, sensory afferents can act as a switch, either through triggering or gating mechanisms (Stein 1978), to turn on (and off) CPG output as a whole (Getting 1977; Getting and Dekin 1985; Robertson and Laverack 1979; Robertson and Pearson 1982; Weeks and Kristan 1978). Second, they can act as modifiers of an ongoing rhythm, enhancing the central network’s rhythmicity or inducing cycle by cycle corrective changes in the ongoing pattern (Andersson et al. 1981; Farley and Case 1968; Nagy and Moulins 1981; Pearson et al. 1983; Reichert and Rowell 1985; Wyman 1977).

Recently the CPG concept has further evolved; a CPG can no longer be considered a fixed structure that produces a single pattern. Instead, a single such network can produce several different motor patterns, i.e., its functional configuration can be modified to switch its output from one pattern to another (Altman 1989; Getting and Dekin 1985; Marder 1984; Marder and Hooper 1985; Selverston and Moulins 1987). These restructuring effects have been obtained by the application to the CPG of putative neuromodulators (Dickinson and Marder 1989; Flamm and Harris-Warrick 1986a,b; Heinzel and Selverston 1988; Hooper and Marder 1984, 1987; Marder and Hooper 1985; Nusbaum and Marder 1988; Turrigiano and Selverston 1989, 1990), the activation of identified modulatory interneurons (Cazalets et al. 1990a,b; Dickinson and Nagy 1983; Nagy and Dickinson 1983; Nusbaum and Marder 1989a,b) or mechanosensory neurons (Katz and Harris-Warrick 1990; Sigvardt and Mulloney 1982), or the injection of putative neuromodulators into the animal (Heinzel 1988; Turrigiano and Selverston 1990).

In this and the following article (Hooper and Moulins 1990) we describe the effects of a multisynaptic sensory afferent on the small, well-studied stomatogastric nervous system (STS) of the lobster, Palinurus vulgaris. This afferent induces long-lasting qualitative changes in the neural output of the pyloric network of the STS, activates another of the system’s networks, the cardiac sac (CS) network, and functionally transfers one of the pyloric network’s neurons to the CS network. In this article we describe the preparation and the changes in neuronal activity caused by activation of this afferent; in the following we describe some of the synaptic and cellular mechanisms that underlie these effects. Preliminary reports of some of these data have appeared (Hooper and Moulins 1987, 1989).
METHODS

Preparation

Male and female Palinurus vulgaris (500–1,000 g) were obtained from Prime1 in Plougastell, Bretagne (France) and maintained in aquaria with circulating seawater. Two different preparations were used. In the first (in situ, Fig. 1A) only a portion of the carapace overlying the stomach and the hepatopancreas was removed, allowing access to the stomach in the body cavity. The second (in vitro, Fig. 1B) was the standard dissection for this preparation (Selverston and Moulins 1987) except that care was taken to preserve the dorsal posterior esophageal (dpon), lateral posterolateral (lpn), and hepatopancreatic duct (hpdn) nerves (Maynard and Dando 1974). In all experiments the preparation was continuously superfused (3–10 ml/min) with chilled (12–14°C) aerated saline (in mM: 479 NaCl, 12.8 KCl, 13.7 CaCl₂, 3.9 Na₂SO₄, 10 MgSO₄, 11 Tris, adjusted to pH 7.4–7.6 with NaOH).

Electrophysiology

All electronics and techniques used were standard. Extracellular nerve recordings and stimulations were made with suction electrodes or bipolar stainless steel pin electrodes insulated from the bath with petroleum jelly (Vaseline). Intracellular recordings from neuronal somata were made with the use of glass microelectrodes (resistance 10–20 MΩ) filled with 3 M KCl. Neurons were identified by matching intracellular spikes with action potentials recorded extracellularly from the appropriate nerves. Extracellular and intracellular signals were amplified and displayed on a Gould FS 1000 chart recorder. The results in these two articles are drawn from experiments on 100 animals; unless otherwise noted, all experiments were performed at least three times.

Phase analysis (Fig. 7)

Phase analysis was carried out to determine the average percentages of the cycle period at which any given neuron began and ended its burst. The cycle period was the number of seconds between the first spike of one pyloric dilator (PD) neuron burst and that of the next PD neuron burst. The time of a given event after the beginning of a cycle divided by the cycle period was that event’s phase. Phase averages were calculated over at least 10 cycles. This analysis normalizes with respect to frequency, allowing direct comparison of the phase relations of the pyloric network neurons in preparations with different cycle frequencies.

Morphology

The STS was dissected in saline and fixed overnight with 3% glutaraldehyde in a 0.1-M cacodylate buffer (pH 7.4) with 0.3 M NaCl. The preparation was washed in the same buffer and post-fixed for 1 h in cold 1% osmium tetroxide in cacodylate buffer. After dehydration in graded ethanol solutions, the preparation was embedded in Araldite. Semithin sections (1.5 μm) were stained with toluidine blue and viewed under the light microscope. Ultrathin sections were stained with 5% uranyl acetate in water and with lead citrate (Reynolds 1963) and examined by transmission electron microscopy (Hitachi H600).

Statistics

Statistical tests were performed as described in Bruning and Kintz (1977). In all cases the average is indicated with standard deviation. All t tests were two tailed.

RESULTS

The STS is, anatomically, a relatively separate portion of the lobster nervous system that lies on the surface of the stomach. Figure 1A shows a schematic of the stomach and STS in the in situ preparation, and Fig. 1B the STS after it has been dissected free from the stomach (in vitro preparation). The four ganglia (stomatogastric, esophageal, and paired commissural) of the STS are connected by the stomatogastric (stn), superior esophageal (son), and inferior esophageal (ion) nerves (Fig. 1B). The neuronal cell bodies of the four STS neural networks (pyloric, gastric, esophageal, and CS) are distributed among the system’s four ganglia, and respectively generate the rhythmic motions of the striated muscles of these four stomach regions (the pylorus, gastric mill, esophagus, and cardiac sac). Two other nerves
of interest are the lpln and hpdn, defined sensory nerves (Dando and Maynard 1974) that innervate various regions of the stomach and project centrally through the dpons. The somata of the pyloric network neurons are located in the stomatogastric ganglion, and their activity can be recorded extracellularly in the medial ventricular nerves [mvn; ventricular dilator (VD) and inferior cardiac (IC) neurons], lateral ventricular nerves [lvn; lateral pyloric (LP), PY, and PD neurons], and stomatogastric nerve [anterior burster (AB) neuron], or intracellularly from their somata. As is shown in the top of Fig. 2, the pyloric network produces in vitro a complex approximately triphasic motor pattern with a frequency between 0.5 and 2 Hz. The three phases of the pyloric pattern are shown by the intracellular recordings from the VD, IC, and PD neurons, which fire successively in each cycle. The LP neuron (LP extracellular recording) fires with the IC neuron, and the AB interneuron and PY neurons (not shown here) respectively fire with the PD and VD neurons. This network is almost always endogenously active in vitro and has been intensively studied in the closely related species Panulirus interruptus (Selverston and Moulins 1987; Selverston et al. 1976). Its complete synaptic connectivity (Eisen and Marder 1982; Maynard 1972; Maynard and Selverston 1975) and many of the neuronal characteristics that underlie its rhythmicity (Gola and Selverston 1981; Miller and Selverston 1982a,b; Raper 1979; Russell and Hartline 1982) in Panulirus are well understood; unpublished observations show these data also apply to Palinurus.

The CS network is less well understood, and it is unknown whether all its neurons have been identified. The known CS network neurons are the two cardiac sac dilator neurons CD1 and CD2, and the two inferior ventricular (IV) neurons (Moulins and Vedel 1977). The CD1 and CD2 neuronal somata are respectively located in the esophageal and stomatogastric ganglia; their activity can be recorded intracellularly from their somata or extracellularly from the dpon (Moulins and Vedel 1977; Vedel and Moulins 1977). The IV neuronal somata are located in the brain in Panulirus (Claiborne and Selverston 1984) and are also likely located in the brain in Palinurus (unpublished observations); IV neuron activity is extracellularly recorded from the inferior ventricular nerve (ivn). The CS network is not always spontaneously rhythmically active, but when it is, all the known CS network neurons simultaneously fire long (2- to 10-s duration) bursts of spikes that repeat with a period of 15–70 s, as is shown in the bottom of Fig. 2.

Mechanical stimulation of the pyloric region induces long-lasting changes in the pyloric rhythmic output

In an in situ preparation, i.e., after minimal dissection, pyloric network output can be extracellularly recorded with suction electrodes from the two main pyloric motor nerves, the lvn and mvn; CS network activity can be similarly recorded from the ivn (Fig. 3). In these recordings the rhythmic activity of at least three pyloric network neurons can be seen; in each cycle the PD (large lvn unit), the IC (small mvn unit) and the VD (large mvn unit) neurons fire successively; the CS network was not rhythmically active in this preparation. We mechanically stimulated the pyloric region of the stomach in two ways (arrows, Fig. 1A): either the hepatopancreatic duct was gently and briefly pulled or the stomach wall was stretched by briefly pulling the intestine. Such stimulations always induced dramatic modifications in the activity of the PD, IC, and VD neurons of the pyloric network and of the IV neurons of the CS network (Fig. 3). These modifications can be divided into two classes; short-term effects that only briefly outlast the stimulus and long-term effects that continue for ~30 s after the stimulus end.

All of these neurons are affected in a short-term fashion; the VD and IV neurons are strongly excited, and each fires a long burst of spikes while the PD and IC neurons become silent (the change in IC neuron activity is obscured here by the VD neuron spikes, but can be plainly seen in Fig. 4). The VD and IC neurons also have a long-term response; the VD neuron is totally inactivated after its initial burst of spikes and remains silent for tens of seconds; the IC neuron resumes firing after its initial inhibition but the duration of each burst and the spiking frequency in its bursts are increased. Mechanical stimulation of the pyloric region of the stomach thus induces a long-term restructuring of the pyloric output; the VD neuron becomes silent, and IC neuron firing is increased while the PD neurons continue to cycle normally. These results are still obtained when the lvn and mvns are cut peripherally, but when the dpons are cut the mechanical stimuli become totally ineffective. Thus the neurons activated by the mechanical stimuli project to the STS via the dpons (see Fig. 1).

Electrical stimulation of the lpln induces equivalent modifications of pyloric output

To study the effects induced by mechanical stimulation while recording intracellularly from the pyloric neurons, we developed an in vitro preparation. In a first step, the STS was transferred to a Petri dish with the pyloric region attached to the system via the lpln and the dpon (the lvn and mvn were cut peripherally). In such preparations (2 experiments) mechanical stimulation of the pyloric region again modified the pyloric output as described above. However, the mechanical disturbance introduced by this stimulation prevented intracellular recording from the pyloric network neurons. The mechanical stimulation was therefore replaced with a brief electrical lpln stimulation. Figure 4 shows the effects of 250-ms at 40 Hz lpln stimulation (a stimulation paradigm that we found consistently produced alterations in STS activity comparable to those induced by mechanical stimulation) in the in vitro preparation.

The activity of the same types of neurons (IV, VD, IC, PD) as were recorded in situ in Fig. 3 were monitored in this experiment. Before stimulation all three pyloric network neurons rhythmically depolarized and fired bursts of spikes; the IV neurons were silent. Lpln stimulation (Fig. 4, arrow; bar under PD trace) resulted in changes in their activity essentially identical to those induced by mechanical stimulation in the in situ preparation. The PD neuron was initially strongly depolarized and again fired a long, high-frequency burst of spikes; it then became silent, showed much reduced depolarizing membrane potential.
FIG. 2. In vitro activities of the pyloric and CS networks. Top: intracellular recordings of the VD, IC, and PD neurons show that the pyloric output is a rapid approximately 3-phase rhythmic pattern. The LP neuron extracellular recording (from the ultimate branch of the nerve innervating the corresponding muscle) shows the activity of this neuron; the extracellular Ivn recording shows the activities of the LP, PY, and PD neurons. All 5 recordings were taken simultaneously. Bottom: simultaneous intracellular recordings of the CD1 and CD2 neurons and an extracellular recording of IV neuron activity (ivn trace) show that the CS network output is a slow monophasic rhythmic pattern. The most hyperpolarized points of the neuron’s membrane potentials were as follows: VD, -70 mV; IC, -60 mV; PD, -60 mV; CD1, -70 mV; CD2, -55 mV.
FIG. 3. Mechanical stimulation of the stomach induces long-lasting alterations of the pyloric network's rhythmic output (in situ preparation). Before a gentle pull of the hepatopancreatic duct, the VD neuron (large unit on the mvn) fired bursts of spikes rhythmically with the pyloric pattern. A pull of the duct induced an initial high-frequency burst in the VD neuron and also caused a burst of activity in the ivn. Subsequently, the VD neuron no longer fired with the pyloric pattern, and IC neuron activity increased. Note that rhythmic pyloric activity (mvn, lvn traces) continued during the VD neuron quiescence.  

oscillations, and did not participate in the pyloric neural output for 17 s. Even 26 s after the stimulation (right) the VD neuron still fired less strongly than in the control; full recovery was achieved only 35 s after lpln stimulation. Much longer periods of VD neuron inactivation could be achieved with several 250-ms at 40 Hz lpln stimulations; 10–20 such stimulations always resulted in a VD neuron inactivation that lasted for the remainder of the experiment (5–6 h). The IC neuron was inhibited during the VD neuron burst and, afterwards, showed much reduced hyperpolarizing membrane potential oscillations and an increase in burst duration and spiking frequency during each burst. PD neuron firing again was reduced during the VD neuron burst, but it began to again rhythmically fire spike bursts immediately after the VD neuron burst. In this experiment the amplitude of the PD neuron membrane potential oscillations was augmented for ∼15 s after the lpln stimulation; this effect was of variable duration (although always shorter than the VD neuron inactivation) and was not always seen (see, for instance, the PD traces of Figs. 5B and 7A). Finally, there was again a burst of ivn activity during the long VD neuron burst triggered by the nerve stimulation. We have compared the effects of stimulating the hpdn, the lpln, and the dpon; all three induce these changes in STS activity, including the long-lasting cessation of VD neuron pyloric activity.

**Lpln stimulation results in long-lasting changes in the activity of almost all pyloric network neurons**

These results allowed us to use lpln electrical stimulation to mimic the effects of mechanical stimulation of the pyloric region, and we therefore observed, in the in vitro preparation, the effects of lpln electrical stimulation on each pyloric network neuron type by the use of intracellular recording techniques. This has already been shown for the VD and PD neurons (Fig. 4); the VD neuron shows a characteristic long-term inactivation, whereas the PD neurons do not. The AB neuron is electrically coupled to the PD neurons and fires with them; its response to lpln stimulation was the same as that of the PD neurons (data not shown). The next three figures show the responses of the remaining pyloric network neurons (the LP, IC, and PY neurons); in each case the VD and PD neuron activities were simultaneously recorded to consistently monitor the effects of the stimulation.

Figure 5 shows the effects of lpln stimulation on the LP neuron. Before the stimulation (Fig. 5A) the neuron rhythmically depolarized and fired robust bursts of spikes during
Electrical lpln stimulation (in vitro preparation) induces changes in pyloric activity similar to those resulting from mechanical stimulation (Fig. 3). Simultaneous intracellular recordings of the VD, IC, and PD neurons and an extracellular recording of the IV neurons (ivn trace) are shown; the lpln was stimulated electrically for 250 ms at 40 Hz (arrow; bar under PD trace). This again resulted in 1) simultaneous immediate excitation of the VD and IV neurons and inhibition of PD and IC neuron firing followed by 2) long-lasting VD neuron inactivation, increased IC neuron activity, but no long-lasting change in PD neuron activity. The most hyperpolarized points of the neuron’s membrane potentials (before lpln stimulation) were as follows: VD, -58 mV; PD, -60 mV; IC, -50 mV.

Each pyloric cycle, Lpln electrical stimulation (1 s at 40 Hz, Fig. 5B, bar under PD trace) resulted in an immediate cessation of the rhythmic suprathreshold LP neuron depolarizations, and it therefore stopped firing. This long-lasting inactivation was not preceded by a short-term excitation as it was for the VD neuron and, although of variable duration, was always shorter than the VD neuron inactivation. Even after the LP neuron had resumed firing with the pyloric pattern, it fired weakly, and its depolarizations were of reduced amplitude and duration; in this experiment full recovery took 30 s. Unlike the VD neuron, however, repeated lpln stimulation did not result in increasing LP neuron inactivation; if anything, LP neuron inactivations became shorter with repeated lpln stimulation.

Figure 6 shows the responses of the PY neurons to lpln stimulation. The six to eight PY neurons are not yet individually identifiable, although they have been divided into two subsets on the basis of their responses to stimulation of an afferent in the hpdn that extends to the stomatogastric ganglion via the lvn and dorsal ventricular nerve (Hartline et al. 1987). We have found that these neurons have three different responses to lpln stimulation. One class (Fig. 6A, PY1, 2 of 11 neurons) is immediately inactivated in a long-lasting fashion, remaining inactivated as long as does the VD neuron. This class, like the LP neuron, did not show an increasing period of inactivation with repeated lpln stimulations. A second class (Fig. 6B, PY2, 5 of 11 neurons) immediately depolarizes and remains depolarized, firing high-frequency bursts of spikes with the pyloric rhythm in a long-lasting fashion; we do not know the response of this neuron to repeated lpln stimulations. A third class (Fig. 6C, PY3, 4 of 11 neurons) is almost unaffected by lpln stimulation. We have found PY neurons exhibiting the long term inactivation (type PY1) and long-term depolarization (type PY2) in the same preparation, and those exhibiting no long-lasting response (type PY3) and the long-lasting inactivation (type PY4) in the same preparation, but have not found all three types in the same experiment.

We have primarily described neuronal responses that seem to take a neuron out of functional membership in the pyloric pattern; the VD, LP, and one type of PY neuron all respond to lpln stimulation by becoming silent for varying lengths of time. However, in a multiphase rhythmic pattern like the pyloric, changes in the motor pattern are also
possible; in particular, the relative time at which a given neuron fires in each cycle (its phase) is subject to modification (Eisen and Marder 1984; Hooper and Marder 1987).

As was shown in Figs. 3 and 4, the IC neuron response to lpln stimulation consists of an immediate hyperpolarization during the initial VD neuron burst followed by a long-lasting increase in the duration of, and spiking frequency in, its bursts. Figure 7 shows that this is associated with a change in the phase of IC neuron firing in the pyloric pattern. Figure 7A, left, shows intracellular recordings of the VD, IC, and PD neurons in control conditions; the IC neuron fired bursts of action potentials out of phase with both the PD and VD neurons. After lpln stimulation (1 s at 40 Hz, Fig. 7A right), the amplitude of the membrane potential oscillations of the IC neuron was reduced, and the phase of the IC neuron’s burst of spikes shifted in the pyloric cycle; the IC neuron then began its firing during the PD neuron activity.

This change was quantified by a phase analysis of the data (see METHODS). In the experiment shown in Fig. 7A, the IC neuron fired between phases of ~0.45 and 0.75 before lpln stimulation (left); after lpln stimulation (right) it fired between phases of 0.2 and 0.7. This analysis was performed on seven experiments, and the averaged results are shown in Fig. 7B. Before lpln stimulation (left), the VD neuron fired with the pyloric network, and the IC neuron was rhythmically active out of phase with both the VD and PD neurons. When the VD neuron was silent after lpln stimulation (right), the average of the beginning of the IC neuron activity was shifted forward in the pyloric pattern from 0.4 ± 0.1 (mean ± SD) to 0.1 ± 0.1 (significantly different at P < 0.001, Student’s t test). The IC neuron then began its firing during the PD neuron burst (in some experiments they began their bursts simultaneously) instead of firing strictly out of phase with the PD neuron.

lpln stimulation thus resulted in a qualitative alteration of the phase relationship of the IC and PD neurons. Neither the frequency of the pyloric pattern [1.8 ± 0.5 Hz (before) vs. 1.9 ± 0.5 Hz (after)] nor the phase of the ending of the IC neuron burst [0.8 ± 0.1 (before) vs. 0.7 ± 0.1 (after)] were significantly altered in the seven experiments; lpln stimulation thus also resulted in an increase in IC neuron burst duration [0.2 ± 0.1 s (before) vs. 0.4 ± 0.1 s (after), significantly different at P < 0.01, Student’s t test]. This change in IC neuron activity could, like the VD neuron inactivation, be of extremely long duration; when the VD neuron was inactivated for hours after several bouts of lpln stimulation, the IC neuron always fired early in the pyloric pattern.

In conclusion, lpln stimulation with a brief high-frequency train of pulses has a strong restructuring effect on the pyloric pattern (and no effect on the pyloric frequency). Considering only the long-lasting effects, these are an inactivation (the VD, LP, and some PY neurons), an activation (another class of PY neurons), and a change in the phase of firing within the pyloric rhythm (the IC neuron).

These effects result from the stimulation of small fibers in the lpln

To identify the fiber(s) responsible for these restructuring effects on the STS, we attempted to record units in the dpon that were activated by either mechanical or electrical stimulation and were associated with the described responses of the pyloric network neurons. In either situ or in vitro preparations, dpon recordings showed only two discrete units. These units were generally spontaneously active (Fig. 8A) and responded one for one with constant latency after single electrical shocks to the lpln at very low stimulus voltages (Fig. 8B, 1st and 2nd traces, left). However, lpln stimulation with a train of pulses (1 s at 40 Hz, bar) at these low voltages never induced any response in the pyloric network neurons (Fig. 8B, 1st and 2nd traces, right), and we concluded that these two units were not the fibers of interest. As these two units have a very low threshold they likely correspond to the two large axons (~1 μm diam, arrows) seen in transverse sections of the lpln (Fig. 9, A, B, and C).

When the voltage of the stimulating shocks to the lpln was increased, a compound action potential appeared on the dpon recording (Fig. 8B, 3rd trace, left), and the char-
FIG. 6. PY neuron responses to lpln stimulation (1 s at 40 Hz, bars). A: intracellular recordings of a VD, PY, and PD neuron. B and C: only recordings of the VD and PY neurons (in each case the response of the PD neuron was similar to that seen in A). The PY1 neuron (A) responded to lpln stimulation with a long-lasting inactivation; PY2 (B) with a long-lasting activation, and PY3 (C) showed no long-lasting response. Data in A and B are from the same experiment. The most hyperpolarized points of the neuron's membrane potentials (before lpln stimulation) were as follows: VD, −58 mV (A and B); PD, −60 mV; PY1, −85 mV; PY2, −70 mV; PY3, −80 mV.
FIG. 7. IC neuron response to lpln stimulation (1 s at 40 Hz for all experiments). A: intracellular recordings from the VD, IC, and PD neurons before (left) and after (right) lpln stimulation. The pyloric period is denoted by P; the IC neuron burst duration by D. The most hyperpolarized points of the neuron's membrane potentials (before lpln stimulation) were as follows: VD, −58 mV; IC, −50 mV; PD, −60 mV. B: averaged results of 7 experiments of which the data have been phase analyzed (see METHODS). Lpln stimulation significantly advances the phase of the beginning of IC neuron firing; before lpln stimulation the IC neuron began to fire only after the PD neuron bursts, whereas after lpln stimulation it began to fire during the PD neuron bursts.

characteristic responses of the pyloric network neurons also began to appear. For the VD neuron at medium stimulus voltages, this response is an initial activation with a burst of spikes followed by an inactivation; at this stimulus voltage the VD neuron activation was almost fully achieved, but its inactivation was of only extremely short duration (3rd trace, right). The amplitude of the dpon compound action potential increased when the stimulus voltage was further increased (4th and 5th traces, left), and the VD neuron inactivation also progressively increased to a maximum...
A Spontaneous lPLN activity

B lPLN stimulation

dpon activity after single shock

VD response to train (40Hz, 1s)

Sucrose block on dpon

6ms  8mV
value (4th and 5th traces, right). All the results presented here were obtained by the use of stimulation voltages that gave rise to such maximum responses.

Transverse sections of the lpln (Fig. 9A) showed that this nerve contains only the two large (1 μm) axons mentioned above and bundles of extremely small fibers (<0.3 μm diam, Fig. 9D) that do not have individual glial sheaths (Fig. 9E) as do the two large axons (Fig. 9, B and C). Our electrophysiological results suggest that the fibers responsible for the changes of activity of the pyloric network neurons after lpln stimulation are members of these small fiber bundles. Each fiber is too small to give a discrete action potential at the dpon extracellular recording electrode, and their small diameters argue that they have a higher threshold than the two large axons. The increase in the amplitude of the compound action potential with increasing stimulus voltage suggests that an increasing number of fibers are being recruited with each stimulation; this presumably explains the increase in compound action-potential amplitude and pyloric network neuron response with increasing stimulus voltages. Finally, when conduction was blocked between the lpln stimulating electrode and the dpon recording electrode (Fig. 8B, bottom), the compound action potential (and the response of the pyloric network neurons) disappeared; thus both these responses to lpln stimulation are dependent on action-potential propagation in the lpln and dpon, as opposed to passive spread of current through the bath.

In summary to this point, we have shown that mechanical stimulation of a region of the stomach, or electrical stimulation of the nerves that innervate this region, induces long-lasting changes in the activity of the majority of the neurons of the pyloric network. The effects on the pyloric network are associated with the activation of graded compound action-potential activity in these nerves, and electron microscopy reveals that they contain thousands of extremely small, presumably sensory, fibers. These effects on the pyloric pattern result in an alteration, not the disruption, of pyloric activity; a recognizable but qualitatively different pyloric pattern consisting of the PD/AB, IC, and some PY neurons seems to be specifically selected by lpln stimulation. We therefore believe that we have described a sensory input to the STS, and that the activation of some percentage of the small input fibers of the lpln results in the changes in S1S activity described above.

Low-frequency tonic lpln stimulation induces a new VD neuron bursting pattern

Thus far all lpln stimulations have been done with brief high-frequency trains of pulses (phasic stimulation), as this might be appropriate for a sensory input activated by sudden large mechanical disturbances of the stomach. Such a paradigm was also necessary to show that these effects were long lasting, i.e., continued after the end of the stimulus. However, we have no evidence as to the actual activating stimulus in vivo, and we therefore also tonically stimulated the lplns at low frequencies to mimic long-lasting mechanical disturbances (Fig. 10). On the top are shown simultaneous recordings of IC (taken from a terminal branch of the mvm), PD, and VD neuron activity. Tonic 4-Hz lpln stimulation was begun at the arrow, and the VD neuron was gradually transformed from firing with the pyloric pattern to instead rhythmically firing long bursts of spikes at a low cycle frequency totally different from that of the pyloric network. Associated with these VD neuron bursts were a cessation of IC neuron activity and a decrease in the amplitude of the PD neuron membrane potential oscillations, although both continued to cycle with the normal pyloric frequency between VD neuron bursts.

Tonic lpln stimulation also altered the phase of the IC neuron, as is shown on the bottom. Before tonic lpln stimulation (left), when all three neurons cycled with the pyloric pattern, the IC neuron fired out of phase with the PD neuron. When the VD neuron was silent during tonic lpln stimulation (right), the IC neuron instead fired much earlier in the pyloric pattern, beginning its bursts during the PD neuron bursts. Finally, the bottom middle panel shows that IC neuron firing ceased and PD neuron firing was reduced during the rhythmic long VD neuron bursts induced by tonic lpln stimulation. Thus, with respect to the VD, PD, and IC neurons, the effects obtained with tonic low-frequency lpln stimulation are consistent with those obtained with brief high-frequency lpln stimulations; the VD neuron leaves the pyloric pattern, the PD and IC neurons continue to fire in pyloric time, and the IC neuron activity is phase shifted. The only difference between these two stimulation paradigms is that tonic low-frequency lpln stimulation results in the VD neuron rhythmically firing long bursts of spikes at a low nonpyloric frequency. Subsequent to the tonic lpln stimulation, the VD neuron was generally inactivated for several hours, during which time the IC neuron continued to fire early in the pyloric pattern.

The effects of tonic lpln stimulation on the other pyloric network neurons—the LP and PY neurons—were, however, very different from the effects of phasic lpln stimulation. Figure 11A shows the response of the LP neuron, and Fig. 11B that of a PY neuron that responds to phasic lpln stimulation with a long-term inactivation (type PY1), to tonic lpln stimulation. Although the recordings of the VD

FIG. 8. Analysis of the electrical activity induced in the dpon by electrical lpln stimulation. A: an extracellular recording of the lpln shows only 2 spontaneously active units. B: extracellular recordings of the activity induced in the dpon by a single shock on the lpln (left) and the VD neuron response (right) induced in each case by a 1 s at 40 Hz train of shocks (bar) using the same stimulus voltage as for the single shock. The stimulus voltage was regularly increased from the 1st to the 5th traces. In the 1st trace only 1 of the 2 larger axons (see Fig. 9, A–C) was activated; in the 2nd trace both large axons were activated, and their summed spikes were seen on the dpon. Neither of these 2 units induced any VD neuron response. In the 3rd, 4th, and 5th traces a complex compound action potential appeared, probably because of the recruitment of small fibers in the lpln (see Fig. 9, D and E); the VD neuron response appeared with its characteristic initial activation (which did not increase with the stimulus strength) followed by an inactivation the duration of which increased with stimulus strength. Blocking action-potential propagation in the dpon with a pool of sucrose placed between the stimulating electrode and the recording electrode (bottom) abolished both the dpon and VD neuron responses. The stimulus strength was the same as for the 3rd trace. The most hyperpolarized point of the VD neuron's membrane potential (before lpln stimulation) was −60 mV.
FIG. 9. Morphology of the lpln. A: a semi-thin section observed with the light microscope. Only 2 axons (arrows) are of sufficient diameter to be seen at this magnification. B–E: electron micrographs show that these 2 axons are individually ensheathed by glial cell and connective tissue (B and C) and that the rest of the nerve consists of very small axons (E) ensheathed in fascicles of several hundreds (D).
neurons show that the tonic lpln stimulations were effective in inducing a long-term inactivation of this neuron, neither the LP nor the PY neuron showed any long-lasting inactivation, but instead continuing to cycle with the pyloric pattern between the VD neuron bursts. Although we have not performed a phase analysis comparing the activities of these two neuron types during lpln stimulation and control conditions, there is no obvious change in their firing time. We do not know the effects of tonic lpln stimulation on the PY neurons that respond to phasic lpln stimulation with a long-lasting excitation (PY 1). With this possible exception, however, these results clearly indicate that when the lpln is tonically stimulated, only the VD and IC neuron activities change in a long-lasting manner. The VD neuron leaves the pyloric pattern and fires rhythmic bursts with a low cycle frequency. The IC neuron is silent during these long VD neuron bursts, and between them begins its firing during the PD neuron bursts instead of firing in strict opposition to the PD neurons.

**This new VD neuron bursting activity is associated with rhythmic CS network activity**

The frequency of the new VD neuron bursting activity and the fact that phasic lpln stimulation induces a burst of activity on the ivn (Figs. 3 and 4) suggested that the VD neuron activity might be associated with the induction of rhythmic CS network activity. That this is the case is shown in Fig. 12. Here an ivn extracellular recording and an intracellular CD1 neuron recording monitored CS network activity (see Fig. 2B); the PD neuron monitored pyloric network activity. Originally, the CS network was silent, and the VD neuron cycled as a pyloric network neuron.
FIG. 11. LP and PY neurons continue to cycle in the pyloric pattern during tonic lpin stimulation. Top: the lpin was tonically stimulated at 4 Hz, and the VD neuron again was induced to fire in a non-pyloric pattern. The LP neuron was inhibited and silent during the long VD neuron bursts but, otherwise, continued to cycle with the pyloric pattern. Bottom: the same situation as in the top, but with a PY neuron (compare to Fig. 6). During tonic lpin stimulation this PY neuron type also continued to fire in the pyloric pattern, being only inhibited during the long VD neuron bursts. The most hyperpolarized points of the neuron's membrane potentials (before lpin stimulation) were as follows. Top: VD, −55 mV; LP, −60 mV; PD, −60 mV. Bottom: VD, −55 mV; PY, −65 mV; PD, −55 mV.
FIG. 12. New VD bursting pattern is associated with induction of CS network activity. CS network activity was monitored with an extracellular ivn recording and an intracellular CD1 neuron recording; pyloric activity with an intracellular PD neuron recording. In the control (left) the CS network was silent, and the VD neuron fired rhythmically with the pyloric network. Two Hertz tonic lpln stimulation (started at arrow) induced rhythmic CS network activity. After 30 s of tonic lpln stimulation (right), the VD neuron had left the pyloric pattern and fired long bursts of spikes with the CS network activity. Note that pyloric network activity still continued (PD trace). The most hyperpolarized points of the neuron’s membrane potentials were as follows: CD1, −65 mV; VD, −60 mV; PD, −55 mV.

(left). Two Hertz tonic lpln stimulation was begun at the arrow. The CS network immediately began to cycle rhythmically, and after 30 s of continuous stimulation (right), the VD neuron was transformed to firing exclusively as a CS network neuron. Note that this change was not due to a cessation of pyloric rhythmicity, as the PD neuron continued to cycle with the pyloric pattern.

Spontaneous CS network activity is associated with similar alterations in VD and IC neuron activity

The data shown so far have been taken from experiments in which the CS network was not spontaneously active. However, as shown in Fig. 13, the CS network can spontaneously become active during an experiment, and this is associated with effects on the VD and IC neurons similar to those observed after tonic lpln stimulation in the in vitro preparation. Figure 13A shows this result in an in situ prepa-

razione; the extracellular recordings show the activity of the VD and IC neurons (mvn, large units, VD neuron; small units, IC neuron) and the IV neurons (ivn). Figure 13B shows the same in an in vitro preparation with intracellular recordings of the VD, CD2 (a CS network motorneuron, see Fig. 2), and a PD neuron. In each case the left panels show the activity of the pyloric network when the CS network was silent; the VD neuron cycled with the pyloric pattern. The right panels show the activity of the same neurons (from the same experiments) when the CS network had become spontaneously active (note the rhythmic activity on the ivn in A and the CD2 neuron bursts of spikes in B). The VD neurons were then no longer active with the pyloric rhythm but, instead, fired exclusively with the CS network activity, although pyloric rhythmicity still continued as shown by the IC (A) and PD (B) neuron activities. In each case the IC neuron fired more action potentials per burst and phase shifted forward in the pyloric pattern when the CS network was active [data not shown for the in vitro case, but note the increased length of the IC neuron burst both in terms of time and as a percentage of the pyloric period in A; in this experiment the beginning of the IC neuron burst phase shifted forward from 0.37 (left) to 0.06 (right)]. In the in vitro preparation it was also possible to record the activity of the LP and PY neurons; each continued to cycle with the pyloric pattern during spontaneous CS network activity (data not shown). Spontaneous CS network activity was observed in 2 out of 7 in situ preparations and in some 30% of in vitro preparations.

These results thus show that when the CS network is rhythmically active, either after tonic lpln stimulation or spontaneously, the VD neuron transfers from the pyloric to the CS network. Associated with this change are alterations in IC neuron activity, which is silent during the VD neuron/CS network bursts and fires more vigorously and earlier in the pyloric cycle between them. Finally, the activity of the other pyloric network neurons (AB, PD, LP, and PY) continues largely unchanged between the VD neuron/CS network bursts when the CS network cycles rhythmically.
FIG. 13. The VD neuron switched from the pyloric to the CS networks when the latter was spontaneously active in situ (A) or in vitro (B). A: the activity of the pyloric network (large mvn units, VD neuron; small mvn units, IC neuron) and the CS network (ivn trace) was extracellularly recorded. B: intracellular recordings of a VD, CD2, and PD neuron. A and B: left panels show that the VD neuron cycled with the pyloric pattern when the CS network was silent. Right panels (taken from the same experiments) show that when the CS network was spontaneously active the VD neuron left the pyloric pattern and then fired only during the long CS network bursts. Rhythmic pyloric activity continued when the CS network was active [IC neuron on the mvn trace (A), PD neuron recording (B)]. Note the difference in time scale for the right and left panels in both A and B. The most hyperpolarized points of the neuron's membrane potentials in B (both right and left) were as follows: VD, -70 mV; CD2, -70 mV; PD, -75 mV.

DISCUSSION

Our results show that mechanical stimulation of the stomach wall or electrical stimulation of a nerve (the lpln) that innervates this region of the stomach activate the CS network, transfer the VD neuron from the pyloric network to the CS network in a long-lasting fashion, and induce long-lasting changes in the output of several other pyloric network neurons. We shall discuss three implications of these results. First, the fact that these changes are induced by mechanical stimulation suggests that these changes are due to the activation of a sensory afferent (and thus could occur in vivo in response to mechanical disturbances of the stomach). Second, CS network activation and the changes in pyloric activity always occur together when activated by mechanical or nerve stimulation, and the changes in pyloric activity also always occur during spontaneous CS network activity. This absolute association between CS network activity and changes in pyloric activity suggests that 1) some functional requirement to coordinate the cardiac sac and pyloric motor patterns may exist and 2) at least some of the effects of lpln stimulation on the pyloric network may be the result of CS network input onto the pyloric network (see Hooper and Moulins 1990). Third, the switch of the VD neuron from the pyloric network to the CS network suggests a new flexibility in nervous system design.

Mechanical and electrical stimulations are activating a sensory afferent pathway

Brief mechanical stimulation of the stomach in in situ preparations induced a long-lasting change in pyloric network output consisting of VD neuron inactivation and a phase shift of IC neuron firing without any long-lasting change in PD neuron cycling. The effects of mechanical
stimulation were blocked by cutting the lpln, which innervates this portion of the stomach wall, and electrical stimulation of the lpln in in vitro preparations had similar effects on the VD, IC, and PI neurons. Taken together, these results strongly suggest that the observed changes in pyloric output are induced by a mechanosensory stimulus (which may affect the pyloric network via a polysynaptic pathway involving CS network neurons) and that the responsible sensory fibers run in the lpln. However, three objections can be raised against this conclusion. First, these results do not rule out the possibility that we were actually antidromically stimulating unknown motor axons in the lpln. Second, we were never able to identify by cobalt migration in the cut lpln any discrete sensory structures in the stomach wall. Third, mechanical stimulation of the stomach did not produce in the lpln a spiking response in which individual sensory units could be identified.

We believe that all three of these objections can be countered by our morphological study of the lpln axon profiles. First, there is no example of crustacean motor axons without an individual glial sheath. Only two of the lpln axons are so ensheathed (and thus possible motor axons) and we were able to show that the activation of these axons does not affect pyloric output. Second, the rest of the lpln fibers have very small diameters (<0.3 μm); we have observed in backfilling other STS nerves that small fibers do not take up/transport cobalt well (unpublished observations). Third, the spikes produced by such small fibers would be much too small to record individually with extracellular electrodes. It is therefore our conclusion that the lpln is a pure sensory nerve (with the possible exception of the 2 large axons) and that our lpln electrical stimulations activate a class of mechanosensory afferents.

These results lead us to suggest an extension of the possible effects of sensory inputs onto CPGs. Such inputs can act not only as a switch to turn on and off a CPG but, more generally, can serve to select or promote a particular functional configuration of the CPG out of the several different configurations a single anatomically defined CPG can assume. As noted earlier, CPG restructuring has been often observed in response to the application of neuromodulatory substances to CPGs or the activation of modulatory interneurons of unknown function extending to the CPG. Our results and those of Katz and Harris-Warrick (1989, 1990) showing that CPG restructuring can be induced by sensory inputs suggests strongly that CPG restructuring is a natural attribute of neural networks and may occur in vivo; it follows that the CPG restructurings induced pharmacologically and by interneuron activation may similarly have behavioral relevance.

**Pyloric network restructuring is correlated with activation of another STS network**

Changes in pyloric activity are never observed without CS network activation, and also occur when the CS network is spontaneously active; thus the VD neuron switch and changes in pyloric activity may reflect an underlying functional requirement to alter the pyloric motor pattern when the cardiac sac motor pattern is simultaneously active. It has been proposed (W. Tighe, personal communication in Claiborne and Ayers 1987) that the cardiac sac motor pattern transfers food toward the cardiac sac to allow food to be chewed several times by the gastric mill and to remove large pieces of food from the pyloric filter. We have therefore made visual observations in the semi-intact preparation of the stomach during stretch-induced and spontaneous CS network activity. During CS network/VD neuron bursts, the cardiac sac expands and the pylorus is pulled forward toward the cardiac sac (see Fig. 1). Simultaneously, the IC and, in some cases, the I.P neurons, which constrict the anterior portion of the pylorus, are inhibited. The combination of forward fluid flow to fill the expanded cardiac sac and relaxation of the valve separating the pylorus from the rest of the stomach could well serve to transfer pyloric contents toward the cardiac sac. Our results thus tend to support Tighe's hypothesis, and are consistent with the lpln pathway serving to generally activate the STS and promote vigorous digestive activity in response to stomach distension induced by feeding.

Such an interpretation allows us to assign possible functional significance to the different effects of tonic low-frequency and brief high-frequency (phasic) lpln stimulation on pyloric activity. In this view tonic low-frequency lpln stimulation mimics generalized stomach distension by food, and the associated STS activity (CS network rhythmicity, VD neuron switch, IC neuron phase shift) supports a stomach motor pattern in which food is rhythmically shunted forward from the pyloric filter and gastric mill to the cardiac sac and repeatedly chewed in the gastric mill. Phasic lpln stimulation, alternatively, would mimic an "emergency" situation in which the pylorus was abnormally distended (with, for instance, a large piece of food). The response of the pyloric network to phasic lpln stimulation includes the inactivation of several additional pyloric constrictor neurons (the LP and PY 1 neurons) as well as the changes in VD and IC neuron activity noted above; this increased relaxation of the pylorus (relative to that seen during rhythmic CS network activity) could further facilitate movement of food from the pylorus.

Finally, the tight correlation of CS network activity and pyloric network restructuring suggests that some of the effects of lpln stimulation on the pyloric network may be an indirect result of lpln-induced CS network activation, i.e., result from synaptic input from CS network neurons onto the pyloric network. The role of such multisynaptic pathways is examined in Hooper and Moulins (1990).

**Neurons can switch between different neural networks**

The most dramatic effect of lpln stimulation is on the VD neuron. After a single brief high-frequency lpln stimulation, the VD neuron ceases to participate in the pyloric network for tens of seconds; 10–20 such stimulations can result in the VD neuron leaving the pyloric network for hours. Furthermore, under conditions in which the CS network is active (e.g., tonic lpln stimulation), the VD neuron transfers to the CS network, firing in a pattern radically different in terms of burst length and cycle frequency from that it displays when active with the pyloric network.

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A possible explanation for this switch of the VD neuron from the pyloric to the CS network is suggested by the innervation pattern of the muscles of the stomach. It has been shown both in Homarus (Maynard and Dando 1974) and Palinurus vulgaris (Moulins and Vedel 1977) that the VD neuron innervates a muscle (cv,) that is also innervated by a CS network motoneuron, CD2. This was originally interpreted as an example of a bifunctional muscle that would be active in two different motor patterns. Although this is strictly true, our results indicate that this muscle never simultaneously expresses both CS and pyloric activity patterns, because the VD neuron switches to the CS network whenever the CS network is active. An intriguing possibility is that some functional requirement exists that this muscle not contract with the pyloric rhythm after cardiac sac dilations; this co-use of the same muscle by the two neural networks may be the fundamental reason that the VD neuron transfers to the CS network when the CS network is active.

From a more general point of view, the VD neuron switch adds a new dimension of flexibility to the pyloric network. Considerable evidence indicates that this network receives multiple modulatory inputs that alter the frequency and phase relations of the various pyloric network neurons (Marder and Hooper 1985), restructure the pyloric network so that only a subset of its neurons are cyclically active (Flamm and Harris-Warrick 1986a,b), or abolish pyloric activity entirely (Cazalets et al. 1990a,b). Our results show that this flexibility is not limited to interactions within the pyloric network: not only can single neural networks produce multiple outputs, but individual neurons can be shifted from one neural network to another. Recent work in related species indicates that this sharing of neurons between functionally different networks may not be an isolated occurrence; Meyrand et al. (1988) in Cancer borealis and Dickinson and Marder (1989) in Panulirus interruptus have shown that individual neurons can participate in different neural networks either spontaneously or in response to the application of various neuromodulatory substances to the STS. The work of Dickinson and Marder is particularly relevant, as they showed that the anterior median neuron of the gastric mill network (Maynard and Dando 1974) switches to the CS network when red pigment-concentrating hormone is applied to the STS. Although we did not examine the effects of lpln stimulation on this neuron, as it innervates the intrinsic musculature of the cardiac sac (Maynard and Dando 1974); lpln stimulation does not result in this neuron switching from the gastric mill network, at least in Palinurus (data not shown).

Unfortunately, except for the work here, what triggers these rearrangements is not yet known. Nonetheless, this recent work suggests strongly that the input described here is simply one of many such globally acting inputs onto the STS, each of which ultimately results in a particular rearrangement of its networks. We have shown that a sensory afferent is able to trigger one such rearrangement and described the possible functional consequences of this change in S1S activity; in the following article (Hooper and Moulins 1990) we describe some of the cellular and synaptic bases of this restructuring.

Glossary

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>AB</td>
<td>anterior burster (neuron)</td>
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<tr>
<td>CD1, CD2</td>
<td>cardiac sac dilator 1 and 2 (neurons)</td>
</tr>
<tr>
<td>CPG</td>
<td>central pattern generator</td>
</tr>
<tr>
<td>CS</td>
<td>cardiac sac</td>
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<tr>
<td>dpon</td>
<td>dorsal posterior esophageal nerve</td>
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<tr>
<td>IC</td>
<td>inferior cardiac (neuron)</td>
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<tr>
<td>IC</td>
<td>inferior ventricular (neuron)</td>
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<tr>
<td>ivn</td>
<td>inferior ventricular nerve</td>
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<tr>
<td>LP</td>
<td>lateral pyloric (neuron)</td>
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<tr>
<td>lpln</td>
<td>lateral postero-lateral nerve</td>
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<tr>
<td>lvn</td>
<td>lateral ventricular nerve</td>
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<tr>
<td>mvn</td>
<td>medial ventricular nerve</td>
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<tr>
<td>PD</td>
<td>pyloric dilator (neuron)</td>
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<tr>
<td>PY</td>
<td>pyloric (neuron)</td>
</tr>
<tr>
<td>STS</td>
<td>stomatogastric nervous system</td>
</tr>
<tr>
<td>PD</td>
<td>ventricular dilator (neuron)</td>
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This work was supported by a National Science Foundation-Centre National de la Recherche Scientifique US-French Exchange Program Award #41000 3600 5176 to S. L. Hooper and by a Ministere de la Recherche Technologie Grant #85C1152 to M. Moulins.

Present address and address for reprints: S. L. Hooper, Dept. of Physiology and Biophysics, Box 1218, Mount Sinai Medical Center, 1 Gustave Levy Place, New York, NY 10029.

Received 5 March 1990; accepted in final form 10 July 1990.

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