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SENSORY ALTERATION OF MOTOR PATTERNS IN THE STOMATOGASTRIC NERVOUS SYSTEM OF THE SPINY LOBSTER *PANULIRUS INTERRUPTUS*

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SUMMARY

1. Stretching the pyloric region of the lobster's stomach in a manner that resembles pyloric dilation triggers a prolonged burst of impulses in two interneurones with axons in the inferior ventricular nerve (IVN). The burst is activated in the oesophageal ganglion by sensory axons that traverse the lateral ventricular nerves, the dorsal ventricular nerve and the stomatogastric nerve. These sensory axons do not appear to make synaptic contacts in the stomatogastric ganglion.

2. Electrical stimulation of sensory branches of the pyloric nerve triggers similar bursts in the IVN interneurones.

3. The burst of impulses in the IVN interneurones lasts from 2 to 30 s and the impulse frequency ranges from 10 to 80 Hz in different parts of the burst. Once triggered, burst structure and burst duration are independent of the intensity or duration of stimuli applied to the sensory nerves.

4. These bursts alter both the gastric and pyloric motor patterns. The IVN interneurones make a complex pattern of synapses with stomatogastric neurones. These are: pyloric dilators (PD) – excitation and slow inhibition; ventricular dilator (VD) – excitation; gastric mill (GM) neurones – inhibition; lateral posterior gastric neurones (LPGN) – inhibition; and Interneurone I (Int 1) – excitation and slow inhibition. The size of the p.s.p.s at each of these synapses depends on the duration and impulse-frequency of the burst in the presynaptic neurones, which in turn alters the firing patterns of the stomatogastric neurones in various ways.

INTRODUCTION

The neurones of the stomatogastric ganglion generate two independent motor patterns: the gastric and pyloric rhythms. These control the chewing movements of the teeth of the gastric mill and the rhythmic contractions of the pyloric filter, respectively (Mulloney & Selverston, 1974*a*, *b*; Selverston & Mulloney, 1974; Maynard & Selverston, 1975; Mulloney, 1977). In a completely deafferented ganglion these motor patterns may continue uninterrupted for hours. The lobster, however,

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Table 1.

Neurone	Neurone number	Location of axon	Muscle innervated	Function
Lateral posterior gastric neurones (LPGN)	2	vLVN	gm3c	Pulls lateral teeth apart
Gastric mill neurones (GM)	4	ALN LVN dLVN	gm1 gm2 gm3a	Pulls medial tooth forward and down
Pyloric dilators (PD)	2	vLVN dLVN	cpv 1 cpv 2b	Dilate pylorus
Anterior burster (AB)	1	SGN	None known	Pattern generation
Ventricular dilator (VD)	1	MVN	cv 1	Pulls open ventral gutter
Interneurone 1 (Int 1)	1	SGN	None	Pattern generation

needs to modulate the two motor patterns so that the movements of the two parts of the stomach are appropriate for proper movement of ingested food through the gut. In the isolated nervous system, stimulation of the Inferior Ventricular Nerve (IVN) and spontaneous bursts in two axons in the IVN each modulate the stomatogastric motor patterns (Dando & Selverston, 1972; Ayers & Selverston, 1977).

This study describes the alteration of the gastric and pyloric rhythms caused by stretch of the pyloric region of the stomach. Sensory input from the pylorus does not act directly on neurones of the stomatogastric ganglion; instead, sensory stimulation initiates a burst in two rostrally originating interneurones with axons in the IVN that then synapse on stomatogastric neurones to change gastric and pyloric rhythms in a characteristic way. This paper also describes the sensory stimulus that triggers this burst of impulses in the IVN interneurones, and extends our knowledge of the connexions of these premotor interneurones in the stomatogastric ganglion. The functional significance of this sensory modulation is discussed.

ANATOMY

The external anatomy of the lobster stomach is shown in Fig. 1. The stomach is divided into three anatomical and functionally separate regions: the cardiac sac (where food is stored), the gastric mill (where food is chewed by a set of teeth), and the pyloric region (where food particles are filtered and sent into the midgut or the hepatopancreatic ducts).

The anatomy of the neuromuscular system has been described by Maynard & Dando (1974). The muscles and their innervation relevant to this study are shown in Fig. 1 and their characteristics are summarized in Table 1. The inferior ventricular nerve (IVN) is a single median nerve that runs from the supra-oesophageal ganglion (not shown) to the oesophageal ganglion (Figs. 1, 2). The IVN contains two axons called the IVN through-fibres by Dando & Selverston (1972), that send axons down the stomatogastric nerve (SGN). Only these two axons project from the IVN to the StG. These two interneurones also send axons into the superior and inferior oesophageal nerves (Kushner, 1979) and have integrative regions in the oesophageal

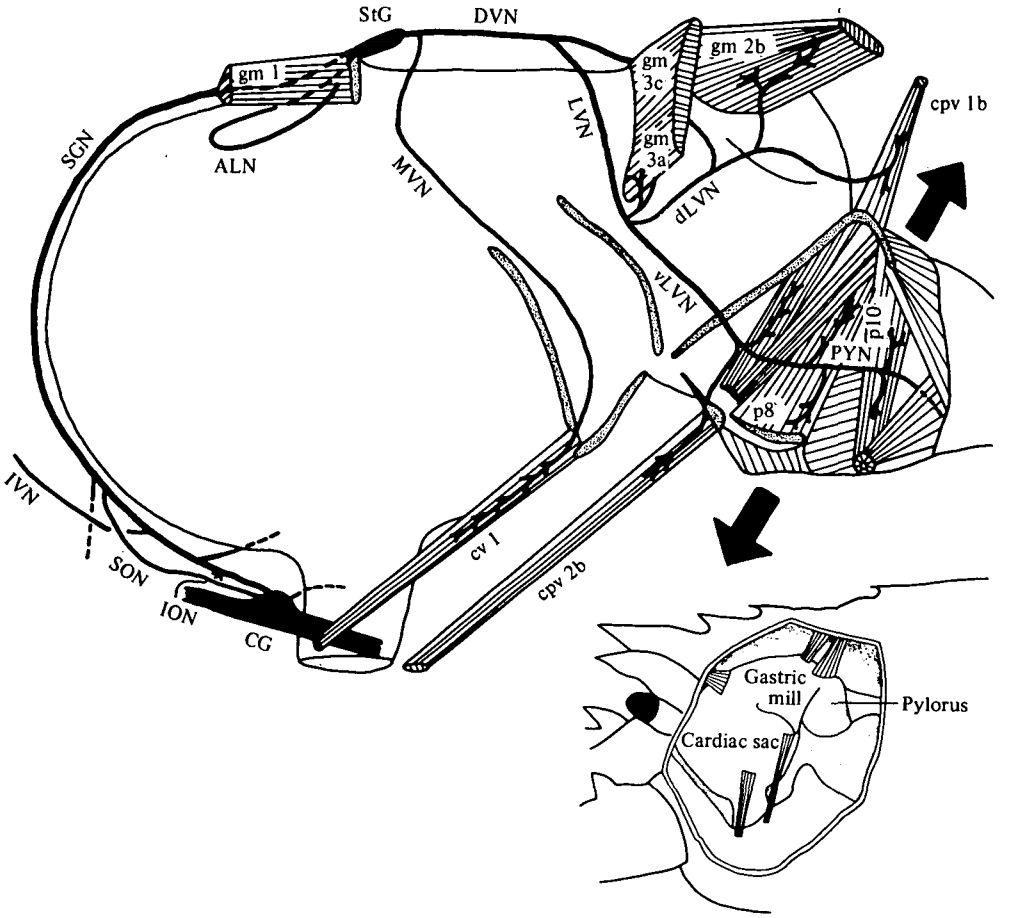

Fig. 1. Diagram of a lateral view of the stomach of *P. interruptus* illustrating the muscles and portions of the stomatogastric nervous system relevant to this study (adapted from Maynard & Dando, 1974). The inset is a lateral view of the animal with part of the exoskeleton cut away to show the position of the stomach in the thorax. The arrows indicate the direction of stretch of the stomach that mimics the effect of contraction of the muscles innervated by the pyloric dilator (PD) neurones – muscles cpv 1b and 2b. Parts of the stomatogastric nervous system listed from most central to most peripheral include: IVN, inferior ventricular nerve; CG, commissural ganglion; SON-superior oesophageal nerve; ION, inferior oesophageal nerve; SGN, stomatogastric nerve; StG, stomatogastric ganglion; ALN, anterior lateral nerve; DVN, dorsal ventricular nerve; MVN, median ventricular nerve; LVN, lateral ventricular nerve; dLVN-dorsal lateral ventricular nerve; vLVN, ventral lateral ventricular nerve; PYN-pyloric nerve. The four muscle groups represented are: gm, gastric mill muscles; cv, ventral cardiac muscles; cpv, cardio-pyloric valve muscles; p, pyloric muscles.

anglion (Selverston *et al.* 1976). The sensory input from the pyloric region which nitiates an IVN burst travels via the vLVN and dLVN, through the stomatogastric anglion, and reaches the integrative region of the IVN interneurones via the SGN. ensory input from the pylorus is limited, in the preparation used in these experiments see Methods), to those sensory receptors whose axons are in the vLVN. There are ther sensory receptors in this region (Dando & Maynard, 1974) but their axons ravel in the posterior stomach nerve and the posterolateral nerve to the commissural anglion, a pathway which is cut in this preparation.

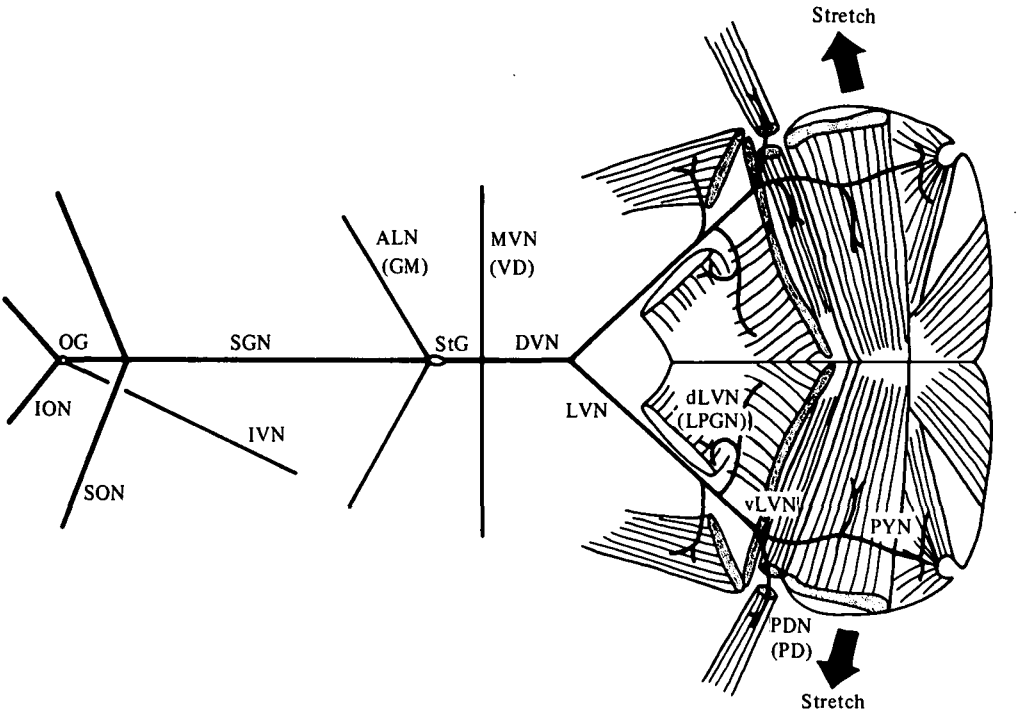

Fig. 2. Diagram of the semi-intact preparation. The posterior region of the stomach including the entire pyloric region is split along the ventral midline and pinned flat. The innervation of this portion of the stomach is left intact. The arrow indicates the direction of mechanical stimulation. The motor neurones whose axons run in a particular nerve are indicated in parentheses. OG, Oesophageal ganglion; GM, gastric mill neurone; VD, ventricular dilator neurone; LPGN, lateral posterior gastric neurone; PDN, pyloric dilator nerve; PD, pyloric dilator neurone. Other abbreviations as in Fig. 1.

METHODS

Spiny lobsters, *Panulirus interruptus*, were purchased from Pacific Biomarine Co., Venice, CA, and kept in aquaria of aerated and circulating seawater at 14–16 °C. Animals weighing 1 kg were normally used although some experiments were done on animals as large as 2·5 kg. The results in this paper were obtained in 25 Expts.

The methods used in these experiments were similar to those described in detail in Mulloney & Selverston (1974*a*). The preparation was semi-intact. The stomach was split along the ventral midline and pinned in a dish (Fig. 2). The anterior portion of the stomatogastric nervous system was dissected free from the surface of the stomach and its connexions to more central portions of the nervous system (the oesophageal ganglion and the commissural ganglia) remained intact. The stomach was usually left intact posterior to the point where the DVN bifurcates, though in some experiments the vLVN was dissected free on one side. This preparation allowed mechanical stimulation of the pyloric region of the stomach.

Mechanical stimulation involved stretching the pyloric region by pulling on the cut edges (ventral midline) of the pylorus as indicated by the arrows in Figs. 1 and 2. This stimulus mimics dilation of the pyloric region similar to that produced

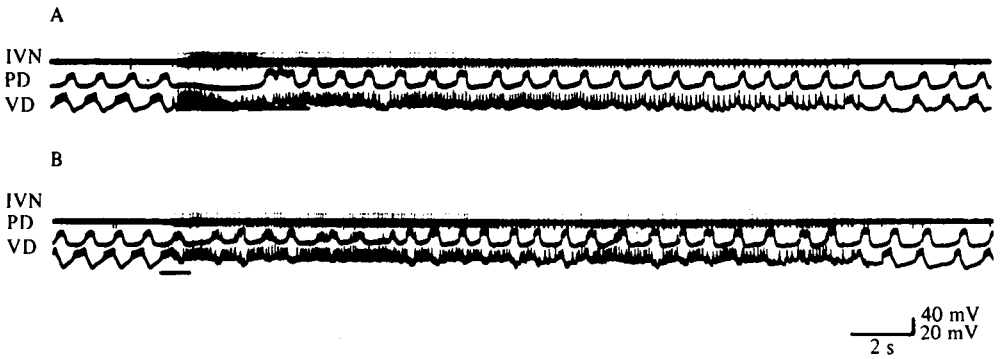

Fig. 3. The motor pattern can be altered by sensory input from the pyloric region and also by electrical stimulation of a sensory branch of the pyloric nerve. (A) Stretch of the pyloric region disrupts the rhythmic output of VD and PD. (B) Stimulation of a sensory nerve innervating the pyloric region produced a similar alteration of the motor pattern. Bars below records indicate period of stimulation. Top trace: extracellular recording from IVN. Middle trace: intracellular recording in PD. Bottom trace: intracellular in VD.

a burst of activity in the pyloric dilator neurones innervating cpv 1 and 2b (cf. Fig. 1).

The preparation was bathed in a saline solution containing 487 mM-Na^+, 12·7 mM-K^+, 13·7 mM-Ca^{2+}, 10 mM-Mg^{2+}, 14 mM-SO_4^- and 519 mM-Cl^- that was buffered to pH 7·4–7·6 with 2 mM-$NaHCO_3$ or 10 mM Tris maleate. Two mM glucose was added at the start of the experiment. The saline was aerated before use and cooled to 16–18 °C during the experiment.

The ganglion was desheathed and transilluminated for intracellular recording. 4 M potassium acetate microelectrodes of 30–50 MΩ were used. Neurones were identified by the peripheral distribution of their axons as described by Mulloney & Selverston (1974*a*). Data were recorded on magnetic tapes or filmed directly.

To block impulse traffic in the SGN reversibly, we built a small well of petroleum jelly around one section of the SGN. When this well was filled with isotonic sucrose solution in distilled water, impulses were blocked, and when normal saline replaced the isotonic sucrose, impulse traffic was restored (Russell, 1979).

RESULTS

Sensory input produces a change in the motor output. Stretch of the pyloric region of the stomach produces a change in the motor output of the stomatogastric ganglion (Fig. 3A). In the example shown, the pyloric dilator (PD) is bursting regularly at 1·0 Hz and the ventricular dilator (VD) is firing alternately with PD. Stretch of the pylorus produces a change in this rhythmic motor pattern. PD is inhibited for approximately 2·5 s, fires a high frequency burst following inhibition and then returns to rhythmic bursting. VD is excited by stretch; stretch produces a long volley of e.p.s.p.s in VD which lasts, in this case, for 23 s and causes VD to spike throughout most of the volley.

The effect of stretch can be mimicked by stimulation of the nerve that innervates the posterior region of the pyloric stomach – a branch of the pyloric nerve (Fig. 1

and 2 PYN). Before stimulating this branch through the suction electrode, we used the same electrode to record the spontaneous activity of the nerve branch. No motor activity of pyloric neurones (PYs) was recorded in this branch, so we conclude that this branch is purely sensory. Stimulation of the sensory nerve produces a response that is similar to stretch (Fig. 3 B). PD bursting is initially disrupted returning to normal after several seconds and VD is excited by a long train of e.p.s.p.s which lasts for 23 s.

Sensory input causes a burst in the IVN interneurones. Both mechanical stimulation of the pylorus and electrical stimulation of the sensory nerve produce a burst in the IVN (Fig. 3). The burst recorded extracellularly is a burst of the IVN interneurones because the spikes in the IVN are always correlated one-for-one with spikes in the SGN as well as the SON. The two IVN interneurones are the only fibres in the IVN that also send an axon down the SGN. Bursts of other IVN axons were never seen in these experiments. Therefore an IVN burst is synonymous with a burst of at least one of the IVN interneurones. The IVN interneurones synapse with a subset of neurones in the stomatogastric ganglion (Dando & Selverston, 1972; Selverston *et al.* 1976; Sigvardt & Mulloney, 1981). Therefore, a burst in the IVN interneurones produces a change in the ongoing activity of the stomatogastric neurones.

Modulation of motor pattern caused by sensory input is a result of a sensory-initiated IVN burst. The conclusion that the effect of mechanical stimulation is not produced directly by sensory synapses on to stomatogastric neurones but instead indirectly by activation of the IVN interneurones is supported by several lines of evidence. First, the onset and duration of the response to stretch of the pylorus or stimulation of the sensory nerve is always correlated with the onset and duration of the IVN burst rather than the onset and duration of the stimulus (Fig. 3).

Second, the effects of spontaneous IVN bursts and the effects of mechanical stimulation are very similar (Fig. 4B, C). The IVN interneurones fire spontaneous bursts in the isolated stomatogastric nervous system if connexions to the oesophageal ganglion are intact (Selverston *et al.* 1976). In the example shown in Fig. 4A, the PD is bursting regularly at 1·4 Hz and the gastric mill neurone is silent. Stretch of the pylorus produces a burst in IVN and a concomitant inhibition of PD and GM (Fig. 4B). Spontaneous IVN bursts occurred occasionally in this preparation and produced changes in PD and GM similar to those produced by stretch (Fig. 4C).

Third, direct stimulation of the IVN at a frequency similar to that in a normal IVN burst produces a response similar to mechanical stimulation. The inhibition of PD is not as complete in Fig. 4D as in Fig. 4B, C because the frequency of IVN stimulation was 20 Hz, which was somewhat less than the frequency within the IVN burst shown in Fig. 4B, C.

Fourth, *every* p.s.p. resulting from stretch is correlated one-for-one with a spike of an IVN interneurone; Fig. 5 shows an expanded portion of Fig. 3A. Stretch produced a burst in IVN and the spikes in IVN are correlated one-for-one with p.s.p.s in PD and VD. This was the case in every experiment; the p.s.p.s in motor neurones produced by sensory stimulation were always correlated one-for-one with spikes of the IVN interneurones. P.s.p.s that were not correlated with IVN interneurone spikes could always be accounted for by known synaptic connexions among stomatogastric neurones.

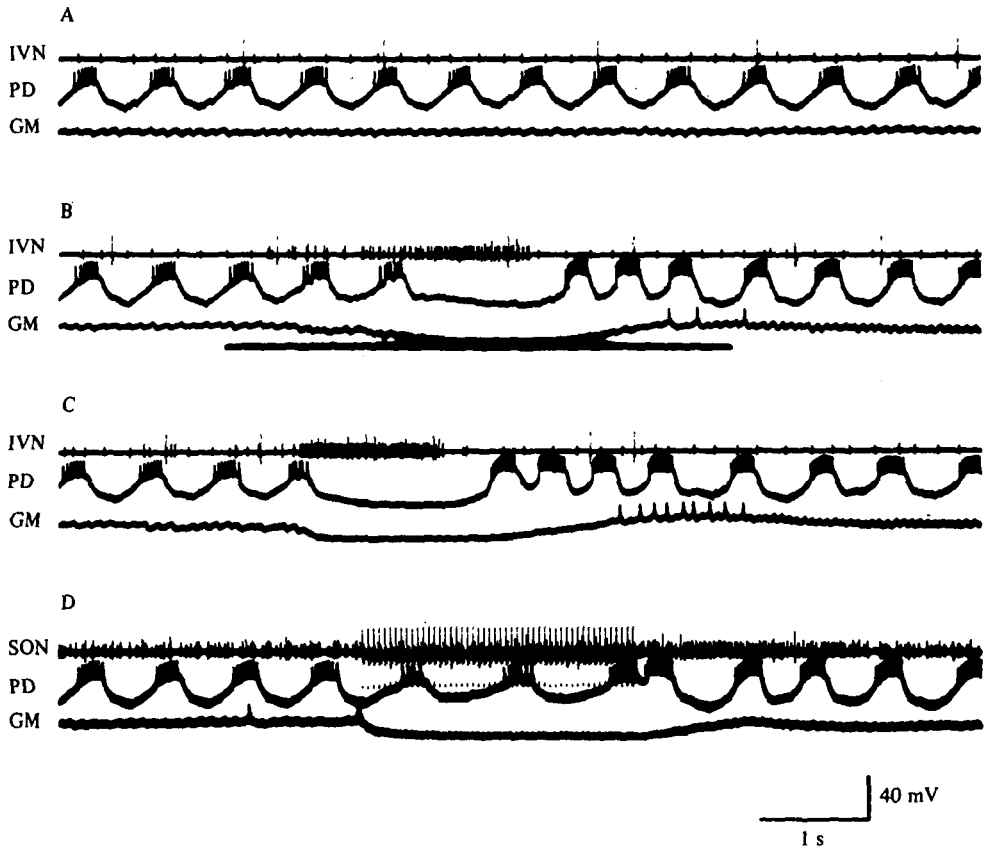

Fig. 4. Alteration of the activity of two stomatogastric motor neurones by stretching the pyloric region of the stomach. (A–C) Top trace: extracellular recording from IVN. Middle trace: intracellular recording from PD. Bottom trace: intracellular recording from GM. (D) Top trace: extracellular recording from SON. Middle and bottom traces: as above. (A) In this semi-intact preparation PD is bursting rhythmically at approximately 1·4 Hz. GM is silent. (B) Stretch of the pylorus (indicated by the bar) produces a burst in IVN that inhibits PD and results in a hyperpolarization of the GM resting potential. GM then fires 3 impulses after the IVN burst stops. (C) A spontaneous IVN burst causes the same response in PD and GM as does the mechanical stimulation in B. (D) Direct stimulation the inferior ventricular nerve at 20 Hz (stimulus artifacts) causes a similar change.

Finally, when the stomatogastric nerve is cut or blocked with sucrose, stretch of the pyloric region has no effect on the motor pattern (Fig. 6). If the effect were direct, then blocking the SGN should still allow modulation of the pattern since the dorsal ventricular nerve, the only direct pathway for sensory input into the ganglion from the pyloric region in this preparation, is intact. Stimulation of the sensory nerve when the SGN was blocked, or under normal conditions, failed to reveal any p.s.p.s in PD or VD that were phase-locked with the stimulus. A sucrose block of the SGN prevents the alteration of the motor patterns by stretch because it blocks the pathway of sensory input from the pyloric mechanoreceptors into the oesophageal integrative region of the IVN interneurones where this sensory input initiates an IVN burst. The sucrose block experiment provides direct evidence only that the

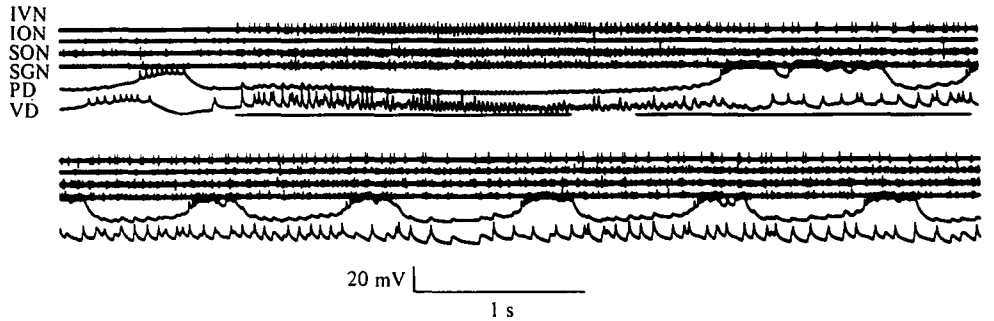

Fig. 5. Stretch of the pylorus (indicated by bar) initiates an IVN burst and a concomitant alteration in the motor patterns of PD and VD. The changes in PD and VD are the result of one-for-one p.s.p.s from the bursting IVN interneuron. Top four traces: extracellular recording from IVN, SON, and SGN, respectively. Fifth trace: intracellular in PD. Bottom trace: intracellular in VD. The two records are sequential, and the same as the early part of Fig. 3A on a faster time base.

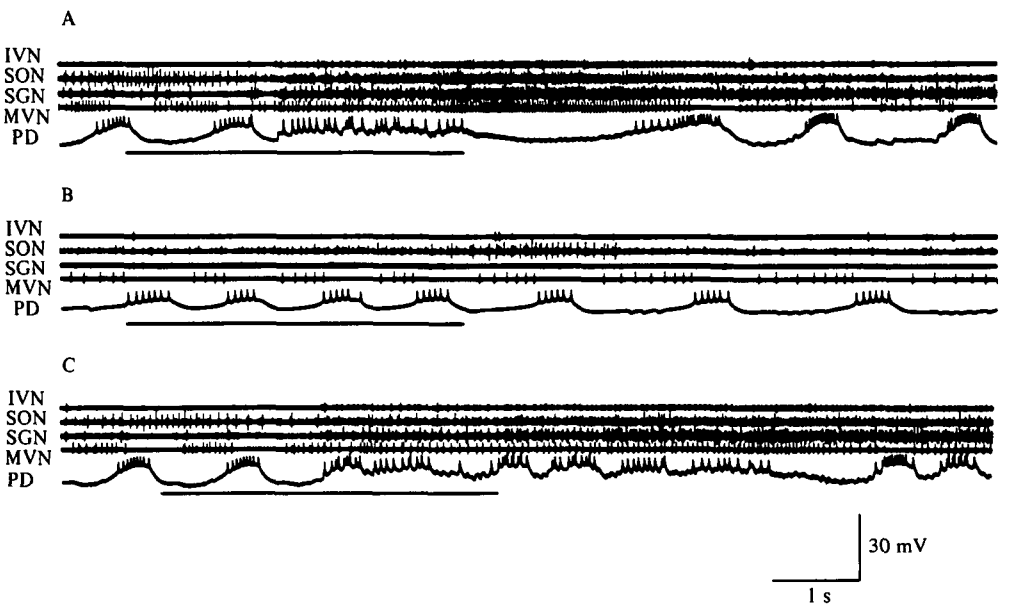

Fig. 6. Sucrose block of the SGN (see Methods) prevents disruption of the motor pattern produced by sensory stimulation (bars). Top four traces: extracellular recordings from IVN, SON, SGN, and MVN, respectively. Bottom trace: intracellular recording from PD. (A) Stretch of the pyloric region of the stomach initiates an IVN burst which disrupts the normal rhythmical bursting of PD and VD (VD is recorded as the large unit on the MVN trace). (B) Block of SGN prevents sensory input from travelling centrally to initiate an IVN burst. There is no direct sensory input on to PD and VD as a result of stretch. (C) Return to normal saline allows conduction of sensory information centrally via the SGN.

effect of mechanical stimulation is mediated centrally via sensory input into the oesophageal ganglion since the block disconnects *all* neurones in the SGN from the circuit. However, since the p.s.p.s produced by stretch are always correlated one-for-one with firing of the IVN interneurones, another unit in SGN producing the change would have to fire one-for-one with the IVN interneuron and make an identical set of synapses.

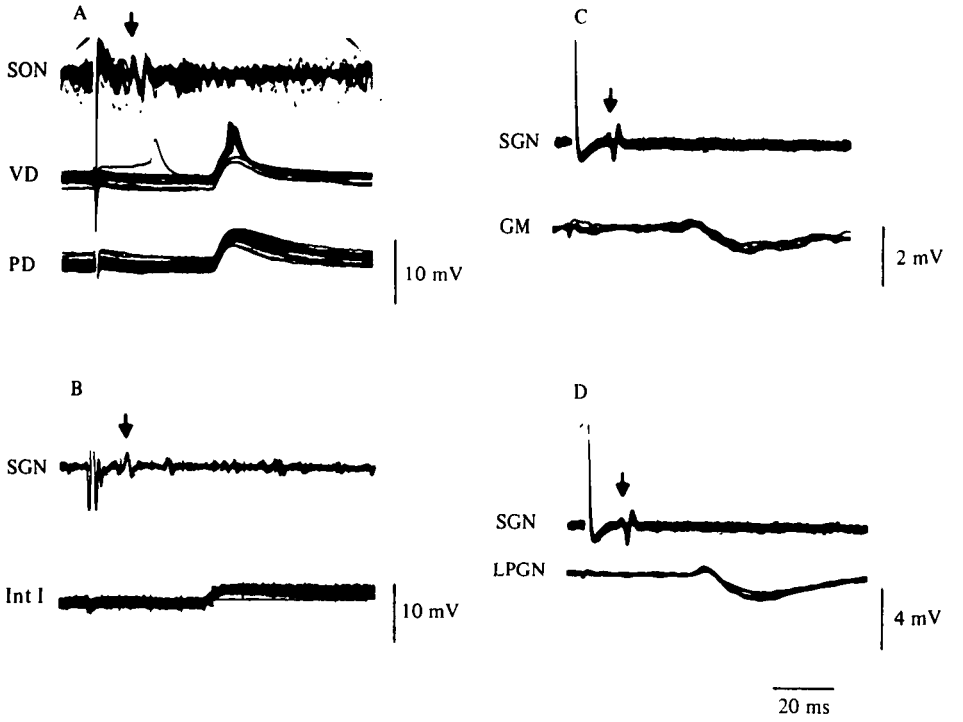

Fig. 7. The IVN interneurones synapse directly on 11 stomatogastric neurones. The IVN spikes (arrows) are recorded extracellularly in SGN or SON. Top traces: extracellular recordings from SON or SGN. Bottom traces: intracellular recordings from VD and PD in A, Int 1 in B, GM in C and LPGN in D. Number of superimposed traces: 7 in A, 3 in B, 4 in C and 4 in D. (A) IVN p.s.p. in VD and PD. There are three PD-AB neurones. All three have similar p.s.p.s. (B) IVN p.s.p. in Int 1. (C) IVN p.s.p. in GM. There are four GM neurones, and all four have similar p.s.p.s. (D) IVN p.s.p. in LPGN. There are two LPGNs, and both have similar p.s.p.s.

Changes in the motor pattern produced by sensory stimulation. The effect of stretching the pyloric region on the motor output of the stomatogastric ganglion depends on the set of synapses made by the IVN interneurones with the neurones of the ganglion. IVN interneurones synapse directly on VD, the two PDs, AB, Int 1, the four GMs and the two LPGNs (Fig. 7 and Dando & Selverston, 1972; Sigvardt & Mulloney, 1981). Each of these neurones has a time-locked, fixed-latency unitary postsynaptic response to IVN stimulation. The IVN interneurones do not synapse with the other neurones of the gastric and pyloric systems (in particular, LGN, AMN, MGN, DGN, LP, and the PYs). The specific effects of an IVN burst on each neurone are:

VD is strongly excited by IVN input; each IVN impulse usually excited VD to fire (Fig. 7A). Therefore, during an IVN burst VD fires at a frequency very similar to the IVN impulse frequency throughout most of the burst (Fig. 5). The electrical coupling between PD and VD is strong (Maynard & Selverston, 1975) so that during the initial part of the IVN burst in Figs. 3A, 5A the strong hyperpolarization of PD increases the VD membrane potential and, therefore, the IVN e.p.s.ps in VD become subthreshold for spike generation. The IVN p.s.p.s in VD are stable over a wide range of impulse frequencies; they do not appear to facilitate or depress.

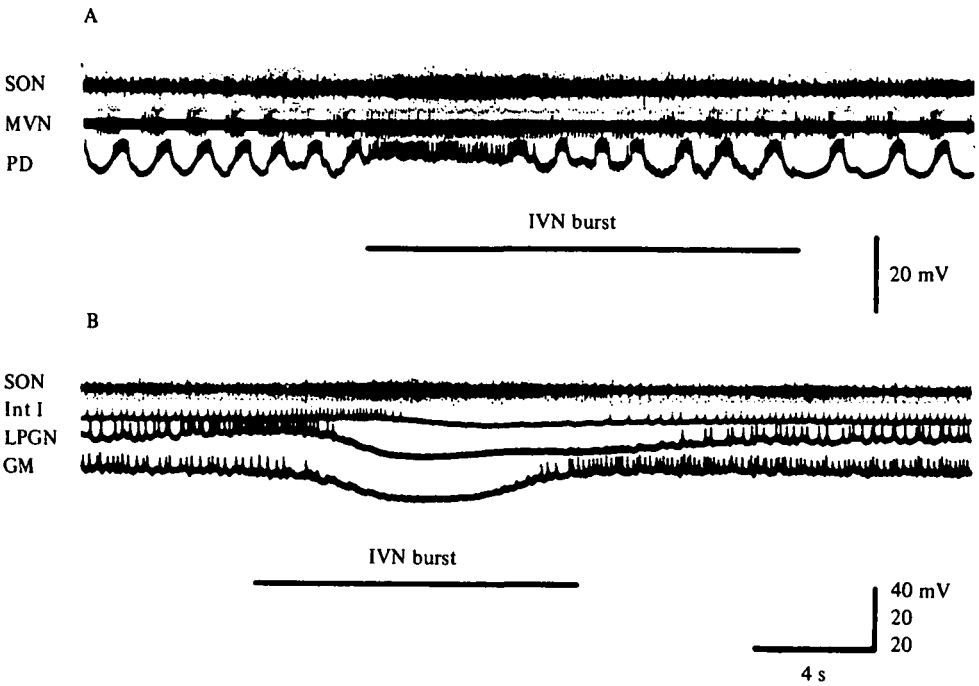

Fig. 8. An IVN burst alters the activity of both the pyloric and gastric systems. (A) The rhythmic bursting of the pyloric neurones, PD and VD is disrupted. VD fires tonically at a frequency similar to that of IVN and PD fires tonically at a slower rate. Top trace: extracellular recording of SON. Middle trace: VD recorded extracellularly in MVN. Bottom trace: PD recorded intracellularly. (B) The tonic firing of several neurones of the gastric system is inhibited by an IVN burst. Top trace: extracellular recording from SON. Second trace: intracellular in Int 1. Third trace: intracellular in LPGN. Bottom trace: intracellular in GM.

The four GMs are strongly inhibited by IVN input (Fig. 7C), so these neurones are silent during most of the IVN burst (Figs. 4, 8B). The IVN p.s.p.s in GM do not vary with frequency and do not depress.

The two LPGNs are inhibited by IVN input (Fig. 7D) although not as strongly as are the GMs (Fig. 8B). The inhibition of the LPGNs does not prevent firing until slightly later in the burst; summation of the IVN p.s.p.s is necessary to hyperpolarize the LPGN membrane potential below threshold. The IVN p.s.p.s in LPGN do not depress.

The response of PD to an IVN burst is more complex than that of the other postsynaptic neurones and depends on the frequency of firing within the IVN burst. At low frequencies the IVN p.s.p. either has little effect on the burst structure (Fig. 3A, later part of burst, where IVN frequency is 5–10 Hz) or the burst structure disappears (Figs. 6A, C, 8A) as PD fires on almost every e.p.s.p. At higher frequencies, PD is inhibited (Figs. 4B, C, 5A). The dependence of the response on frequency can be demonstrated by stimulating IVN directly (Fig. 9). At 20 Hz, PD bursting is unaffected by IVN input. At 40 Hz, PD is inhibited. This frequency-dependent change in the effect of an IVN burst is the result of the biphasic nature of the p.s.p. in PD (Sigvardt & Mulloney, 1981).

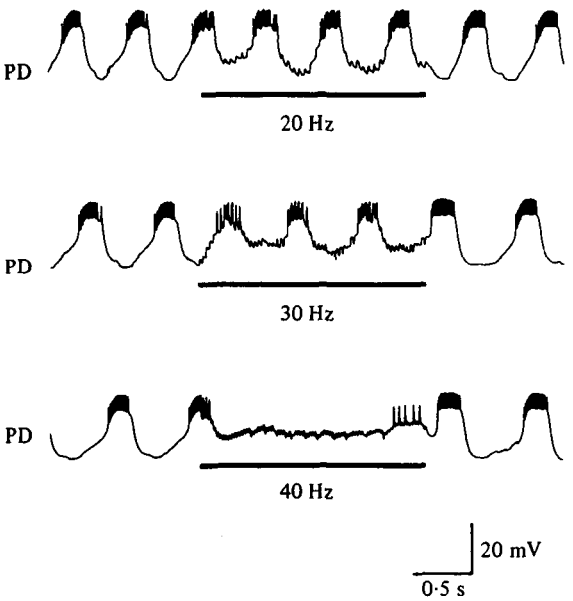

Fig. 9. The response of PD to input from IVN varies with the frequency of firing of IVN. Intracellular recording from PD. Stimulation of IVN (bar) at 20 Hz has very little effect on the PD burst pattern. Stimulation at 30 Hz decreases the number of spikes per burst but does not affect the burst period. Stimulation of IVN at 40 Hz inhibits PD.

The response of Int 1 to IVN input is also complex, again because the IVN interneurone produces a biphasic postsynaptic potential in Int 1 (Sigvardt & Mulloney, 1981). Although the IVN p.s.p. in Int 1 is depolarizing at low frequencies (Fig. 7B), increases in the firing rate result in inhibition of Int 1. The burst shown in Fig. 8B begins slowly, but rapidly reaches high frequency. Because of the frequency-dependent characteristics of the p.s.p. in Int 1, Int 1 first increases its firing rate and then is inhibited.

The change in the motor pattern caused by a sensory-initiated IVN burst is always somewhat variable from burst to burst. This variability is due primarily to the variability of the impulse frequency within the IVN interneurone burst. A typical IVN burst lasts from 20 to 30 s; it begins slowly, reaches a peak frequency of 50–100 Hz after a few s and then slows to a relatively low frequency (5–10 Hz; Figs. 5, 8). A burst can, however, last only a few s (Figs. 4, 6A) but it always contains a high-frequency portion. The change in the motor output of the system is dependent on this burst structure.

A second source of variability in the response to an IVN burst is introduced by the variability in the intensity of the rhythmical cycling of the pyloric and gastric networks in the semi-intact preparation (and probably in the intact animal, as well). For example, the gastric system is often silent, so that the inhibition of gastric motor neurones produced by an IVN burst has little or no effect on their motor output (Fig. 4). The intensity of the cycling is particularly relevant to the effect of a low-frequency IVN burst on PD; when PD is bursting vigorously (1·9 Hz in Fig. 9) stimulation of IVN at 20 Hz has little effect on PD, whereas when PD is bursting

more slowly (1·4 Hz in Fig. 4D), 20 Hz stimulation produces some inhibition (seen as an increase in the interburst interval).

A third source of variability also relevant to the response of PD to low frequency stimulation is a result of the fluctuations in membrane potential in the bursting neurones. If IVN input occurs when PD is in the depolarized phase of its oscillation a p.s.p. may cause a spike (Fig. 8A). If PD is hyperpolarized, however, the p.s.p. will have no effect.

The interplay between the three sources of variability make it difficult to predict exactly what the response of a neurone to a certain rate of firing in the IVN interneurones will be; this is particularly true for PD.

Each IVN interneurone makes an identical set of synapses. Each of the two IVN interneurones appears to make an identical set of synapses on neurones of the stomatogastric ganglion. In experiments where the two axons had slightly different thresholds, the response to stimulation of one fibre alone was consistent with the above pattern, and recruitment of the second axon caused either no change or a slight increase in p.s.p. amplitude. The two axons have similar conduction velocities so that the response to both occurs at the same time in the postsynaptic neurone. We do not yet know if both fibres are always active during an IVN burst.

DISCUSSION

Sensory input

The foregut of the lobster has six major groups of sensory receptors, including chemoreceptors in the lower oesophagus and ventral cardiac sac and mechanoreceptors that monitor movements of the various areas of the foregut (Dando & Maynard, 1974). All of these receptors are probably involved in modulation of the activity of the stomatogastric ganglion and the central circuits that control the movements of other parts of the foregut. This study examines the modulatory role of mechanoreceptors that respond to movement of the pyloric region of the stomach. Stretch of the pyloric region mimics the distension that would occur when food particles enter the pylorus of the intact feeding lobster. The sensory receptor(s) that respond to stretch of the pyloric region are probably muscle receptors associated with muscles p8 and p10 (Fig. 1). The axon(s) of this receptor(s) runs anteriorly toward the stomatogastric ganglion in a branch of the posterior PYN that joins vLVN. Dando & Maynard (1974) described a few bipolar neurons whose dendrites innervate pyloric muscles and whose axons run in the vLVN, but these neurones are more anterior than the receptors described here. We made numerous attempts to locate the receptors on the surface of the muscles using methylene blue staining and cobalt filling of nerves, but did not succeed. Dando & Maynard correctly observed that staining of this region is difficult because of the thick connective tissue that covers this region and the multiple layers of muscle fibres. The existence of mechanosensory neurones in the more distal regions of the foregut has been demonstrated physiologically. Wolfe (1973) found at least 15 and probably 20 or more sensory units in the distal stump of a sectioned LVN; most of these are likely to be mechanoreceptors since 'their activity fluctuated strongly as the foregut and midgut were stimulated with a glass probe'. The axons of these sensory neuron

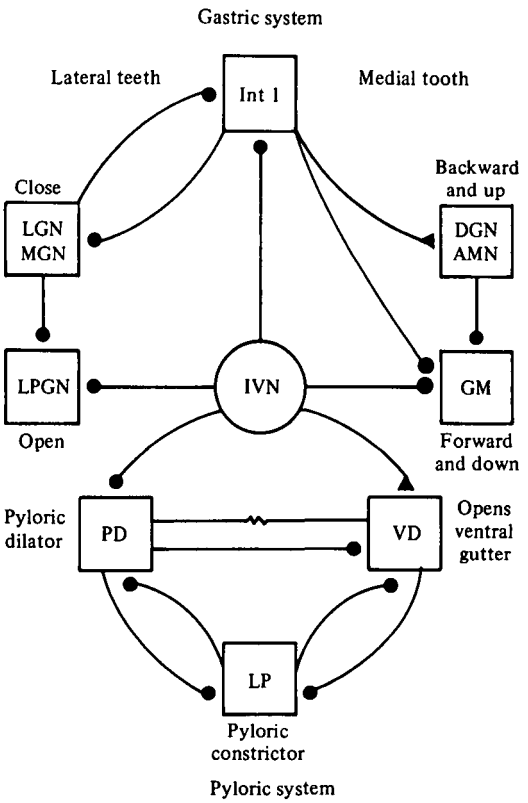

Fig. 10. Diagram of interactions of the IVN interneurones with the neurones of the stomatogastric system. The gastric system coordinates movements of the lateral and medial teeth of the gastric mill. Excitation of LGN and MGN close the lateral teeth and LPGN opens them. The GM neurones cause the medial tooth to move forward and down and DGN and AMN move it backward and up. Coordination of the movements of the lateral teeth and the medial tooth is produced primarily by Int. 1. The pyloric system controls alternate dilations (caused by PD) and constrictions (caused by LP) of the pyloric opening and opening of the ventral gutter produced by VD. —● are inhibitory synapses; —◀ excitatory synapses; and -∿- electrical connexions. The IVN synapse on to PD is inhibitory only at high frequency (see text).

run centrally in the DVN and SGN. Thus, although the receptors have not been identified anatomically, their existence is predictable and their central effects are profound.

Alteration of behaviour

A burst in the IVN interneurones results in a characteristic transformation of the stomatogastric motor patterns with behavioural consequences that can be inferred from the functions of the innervated muscles (Hartline & Maynard, 1975). A summary diagram of the connexions of the IVN interneurones is presented in Fig. 10.

The gastric system. The normal gastric cycle consists of opening and closing of the two lateral teeth and protraction and retraction of the medial tooth, with the movements timed so that the lateral teeth hold pieces of food while the medial tooth moves forward and down to shread the pieces. The lateral teeth then open and the medial tooth retracts. During an IVN burst, the GM motor neurones that innervate

the powerful medial tooth protractor muscles are inhibited, and movement of the tooth stops. The IVN burst also closes the lateral teeth by disinhibiting the closer motor neurones LGN and MGN. (The closer neurones are inhibited by Int 1, which is itself inhibited by IVN.) Lateral tooth closure is ensured by a simultaneous inhibition of the opener motor neurones of the lateral teeth (the LPGNs). Thus, the overall effect of an IVN burst on the behaviour of the gastric mill complex is to hold the lateral teeth closed and prevent the movement of the medial tooth from its retracted position. Thus, IVN not only disrupts a cyclic behaviour, but also induces a stereotyped rest position for the stomach parts. When the gastric mill neurones are not bursting rhythmically, and IVN is silent, Int 1, the LPGNs and DGN fire tonically and the other neurones are usually silent, so the medial tooth is again retracted but the lateral teeth are open. An IVN burst in this circumstance merely closes the lateral teeth.

The pyloric system. The normal pyloric motor pattern results in alternating dilations and constrictions of the pyloric region of the stomach. The PDs innervate muscles that dilate the opening of the pylorus and allow food to enter, the lateral pyloric neurone (LP) constricts the opening, while the eight pyloric neurones (PYs) innervating the muscles of the wall of the pyloric filter cause a peristaltic wave that moves food backward toward the midgut. VD innervates muscles that open the ventral gutters, a pair of channels that pass digestive enzymes anteriorly from the hepatopancreas (Yonge, 1925). VD alternates with the firing of the PDs. The net result of all these movements is to sort the partially digested material that enters the pyloric filter, sending liquid nutrients and minute particles into the hepatopancreas and larger particles into the midgut.

A typical IVN burst lasts from 20 to 30 s; it begins slowly, reaches a peak frequency of 80–100 Hz after several seconds and then slows to a relatively low frequency (10 Hz). During a typical IVN burst, VD is excited to fire continuously at nearly the same frequency as the interneurones, and so the ventral gutter remains open during the entire IVN burst. During low frequency portions of the burst, the PDs and all the pyloric constrictor neurones (LP and PYs) alternate rhythmically as usual because IVN input to the PDs is not strong enough to disrupt its endogenous bursting. When the IVN frequency is high, however, the entrance to the pyloric filter is closed because the PDs are inhibited. Inhibition of the PDs releases the pyloric constrictors from periodic inhibition, and so all the constrictor muscles of the pylorus contract simultaneously, and food particles in the pyloric filter are forced into the midgut. Thus a high-frequency IVN burst initiated by pyloric distension may be a reflex that pushes abnormally large undigestible material out of the pylorus.

Relaxation of extrinsic muscles. The IVN synapses on to neurones of the stomatogastric ganglion are distributed only to those motor neurones that innervate extrinsic muscles of the gastric mill and pylorus – muscles that originate on the body wall and insert on the stomach hold the stomach suspended in the cephalothorax. The motor neurones that innervate intrinsic muscles, those muscles that have their origins and insertions on the stomach, do not receive direct synaptic input from the IVN interneurones. Thus, high-frequency IVN bursts relax all extrinsic muscles of the gastric mill and the pylorus except those that open the ventral gutter.

IVN interneurones as command neurones

These IVN interneurones were first called command fibres by Dando & Selverston (1972), based on their experiments that showed that electrical stimulation of these neurones produced changes in the output of both the gastric and pyloric motor patterns – a method used by many to define command neurones in other systems (e.g. Atwood & Wiersma, 1967). However, the term 'command neurone' implies that the neurone has a critical role in the initiation of a particular normally-occurring behaviour (Kupfermann & Weiss, 1978) and until now this function of the IVN interneurones was unknown (Dando & Selverston, 1972; Selverston *et al.* 1976). It is now clear that an IVN burst results from a specific stimulus and initiates a change in the on-going behaviour, confirming its command status. The criteria for command neurones outlined by Kupfermann & Weiss (1978) have all been met: (1) the IVN interneurones burst in response to a natural stimulus – distension of the pylorus; (2) the response of the stomatogastric ganglion is no longer elicited by the stimulus if the IVN interneurones are disconnected from the circuit; and (3) stimulating these interneurones electrically at frequencies similar to those in a naturally occurring IVN burst produces a response similar to that produced by the normal IVN burst.

There is another neural circuit very similar to the one described here that involves sensory input from the foregut that drives another IVN neurone and results in a change in motor activity of the gut. This is the rectal peristalsis circuit described by Wolfe (1973) in the crayfish *Procambarus clarkii*. The sensory receptors that trigger rectal peristalsis were only partially characterized; their axons enter the oesophageal ganglion in the inferior oesophageal nerve (ION). Stimulation of the ION drives an IVN neurone called the rectal peristalsis interneurone (RPI) and its activation triggers rectal peristalsis. Thus both RPI and the IVN command neurones produce specific motor patterns in response to sensory input from the foregut. It should prove interesting to study mechanisms of coordination between these two visceral command systems.

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