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PROPRIOCEPTIVE CONTROL OF THE BILATERALLY ORGANIZED RHYTHMIC ACTIVITY OF THE OESOPHAGEAL NEURONAL NETWORK IN THE CAPE LOBSTER *JASUS LALANDII*

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SUMMARY

1. In the lobster *Jasus lalandii* the activity of the oesophageal nervous system (monitored through the firing of its main motor neuron, OD1) is modulated by a pair of proprioceptors, the posterior stomach receptors (PSRs).

2. The *in vitro* preparation used consisted of the oesophageal nervous system, the suboesophageal ganglion and the two PSRs, which provide the only source of sensory input.

3. Stimulation of a PSR activates only the oesophageal oscillator located in the ipsilateral commissural ganglion.

4. When spike conduction is blocked in the ipsilateral connective, the stimulation of a PSR activates the contralateral oesophageal oscillator. Inputs from each PSR project to the different parts of the distributed oesophageal network (in the two commissural ganglia and the oesophageal ganglion), but at a given time only one of the PSRs' projections is effective.

5. The relative efficacy of the PSRs' projections is controlled by the oesophageal motor network itself and requires that the superior oesophageal nerves be intact (*sons*).

6. The PSRs' inputs are integrated in the suboesophageal ganglion before reaching the oesophageal network. However, this premotor step is not involved in the control of the unilaterality of PSRs' effects.

7. The PSRs are stimulated by at least two different rhythmical muscular sequences of the foregut (the gastric mill sequence and the cardiac sac sequence) and provide a source of rhythmical inputs to the CNS.

8. The oesophageal nervous system exhibits a periodically varying sensitivity to the PSRs' inputs, which is illustrated by a phase-response curve.

9. Each oesophageal oscillator can be entrained by the rhythmical PSRs' inputs over a range of period. This range includes the period of the spontaneous gastric rhythm.

10. It is proposed that the PSRs enable the oesophageal and the gastric mill rhythms to be coordinated through a peripheral loop. The participation of PSRs in the coordination of different motor sequences of the foregut is discussed.

INTRODUCTION

Rhythmic motor behaviour is controlled by central pattern generators that can sometimes function without sensory feedback (Wilson, 1968; Grillner, 1975). This is apparent for cases in which the motor output can be recorded in an isolated nervous centre *in vitro* (i.e. after total deafferentation). Such preparations, which allow analysis of motor patterns in terms of the activity of single cells, have been very useful for studying the organization of central pattern generators (see Fentress, 1976).

Nevertheless, in intact animals, the expression of such generators is controlled by sensory activity, notably by proprioceptive feedback (Pearson *et al.*, 1973; Carlson, 1977; Stent *et al.* 1978). Moreover the central nervous system can in turn modulate the sensory pathways, and enhance or decrease the efficacy of sensory inputs on the motor activity during its execution (Stein, 1978). Despite the advantages of isolated preparations for studying the mechanism by which this is achieved, they have seldom been used. This may be due to the technical difficulties involved in isolating the motor and integrative centres, as well as identified and controllable sensory input.

The stomatogastric nervous system of decapod Crustacea, particularly its oesophageal components, is a favourable preparation for such studies. The preceding paper (Moulins & Nagy, 1980) has shown how the oesophageal motor behaviour is organized by a neuronal network distributed in three different centres, and how the activity of this network can be monitored through a single motor neurone functioning with several spike-initiating sites. This preparation has been recently developed by isolating in association with the oesophageal nervous centres, a pair of mechanoreceptors innervating the foregut (the posterior stomach receptors, PSRs) and an integrative centre connecting them with the oesophageal centres (the suboesophageal ganglion) (Nagy, 1977).

In the present work we have analysed the action of the PSRs' inputs on the oesophageal network activity to answer two main questions:

(*a*) How does a single receptor project on a distributed neuronal network, and what are the mechanisms by which the central nervous system controls the efficacy of these projections? The preparation used is favourable because the integrative premotor step is anatomically distinct from the motor step, and the motor network is distributed in three anatomically distinct centres. It is possible to isolate reversibly the different elements. It is shown that a single receptor (each of the two PSRs) projects on to the three parts of the distributed oesophageal network, but that only one of these projections is effective at a given time. This control of efficacy of the PSRs' different projections takes place at the level of the oesophageal motor network itself, although the PSRs' inputs are integrated in the suboesophageal ganglion before reaching the oesophageal network.

(*b*) How does a rhythm pattern generator react to a sensory input rhythmically activated by other motor systems? Here again the preparation is favourable, as the PSRs respond to rhythmical stomacal movements and not to oesophageal ones, although the stomacal motor activity and the oesophageal motor activity are interrelated. It is shown that the oesophageal rhythm can be entrained by rhythmical PSRs' inputs over a range of periods, which can include that of the spontaneo

gastric rhythm. It is thus suggested that the PSRs allow these two rhythms to be coordinated through a peripheral loop. The participation of PSRs in the coordination of different motor sequences of the foregut is also discussed.

MATERIAL AND METHODS

Male and female cape rock lobsters, *Jasus lalandii*, were used. Most of the experiments were performed on isolated nervous systems (*in vitro* preparations), but some were done using minimally dissected animals (semi-intact preparations).

Semi-intact preparation. Animals were restrained in a tank filled with refrigerated and oxygenated artificial sea water. The cephalothorax was opened dorsally to reach the foregut, and nervous activity was recorded with polyethylene suction electrodes. Identification of motor and sensory units was based upon previous anatomical and physiological works (Maynard & Dando, 1974; Selverston *et al.* 1976; Vedel & Moulins, 1977).

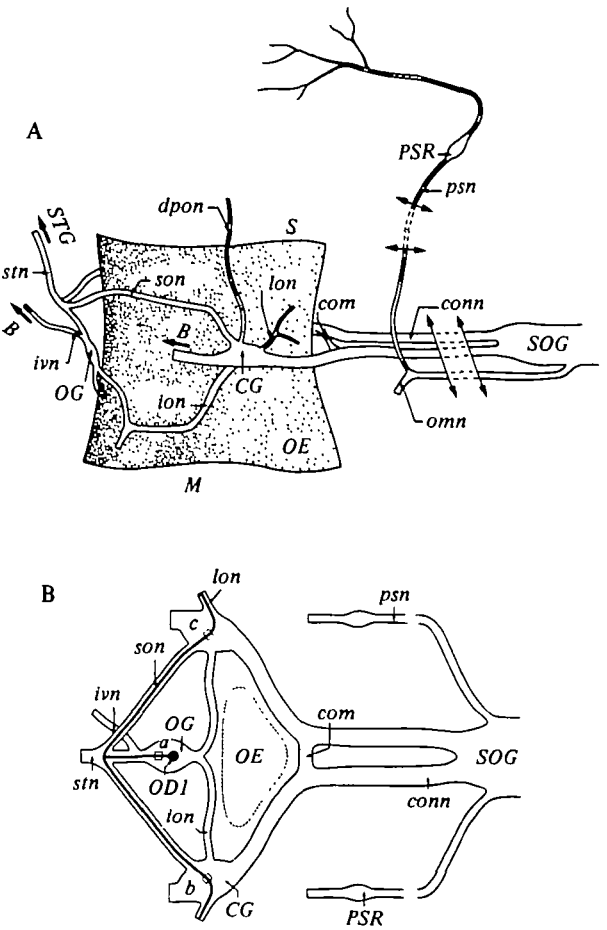
In vitro *preparation.* We enlarged the 'combined preparation' of Selverston *et al.* (1976) (stomagastric ganglion, oesophageal ganglion, commissural ganglia and the nerves connecting them), by the addition of the suboesophageal ganglion and the two posterior stomach nerves (*psns*) (Fig. 1; stomatogastric ganglion not shown). Isolated with each *psn* was a group of sensory cell bodies and 1 cm length of their dendrites, which comprise the posterior stomach receptor (*PSR*). After dorsal opening of the cephalothorax under artificial sea water, the distal part of the PSR dendrites was rapidly severed from the stomach. The stomatogastric ganglion and its nerves were dissected out of the gut and pulled forward, then the stomach was severed from the oesophagus and removed with the hepatopancreas, care being taken to preserve the stomatogastric nerve. The oesophageal and commissural ganglia and their connecting nerves were dissected around the oesophagus. The ventral endophragmal skeleton was removed to dissect out the perioesophageal connectives, suboesophageal ganglion and proximal part of the *psns*. The isolated nervous system was then pinned out in a Sylgard-lined petri dish (Fig. 1 B). Haematopoietic and connective tissues were removed from the nerves. The oesophageal ganglion was desheathed to expose the cell bodies of oesophageal neurones. This dissection takes from 7 to 8 h, but if the saline is kept cold and suitably oxygenated, the oesophageal nervous system can operate for another 6–8 h, with a rhythm similar to that recorded from freshly dissected, semi-intact preparations. For description of recording techniques, see Moulins & Nagy (1980).

Impulse conduction was reversibly blocked along the connectives to determine central pathways of neurones. A small pool of petroleum jelly was made around a previously desheathed segment of the connective. When the pool was perfused with isotonic sucrose solution, conduction was blocked after 3 or 4 min; normal conduction was re-established 3–4 min after saline again flowed through the pool.

RESULTS

The oesophageal rhythm and the main oesophageal dilator motor neurone

The oesophageal rhythm, which consists of regularly alternating bursting activity of constrictor and dilator motor neurones, is organized by a neuronal network distributed in three different centres: two commissural ganglia and the unpaired oesoph-



g. 1. The oesophageal nervous system. (A) Lateral view. (B) Diagram of the isolated nervous item preparation. *a-square*, oesophageal spike initiating zone of OD1, *b- and c-squares* ..nmissural spike initiating zones of OD1; *B*, to brain; *CG*, commissural ganglion; *com*, commissure; *conn*, connective; *dpon*, dorsal posterior oesophageal nerve; *ion*, inferior oesophageal nerve; *ivn*, inferior ventricular nerve; *lon*, lateral oesophageal nerve; *M*, mouth; *OD1*, main oesophageal dilator motor neurone; *OE*, oesophagus; *OG*, oesophageal ganglion; *omn*, outer mandibular nerve; *psn*, posterior stomach nerve; *PSR*, posterior stomach receptor (group of sensory cell bodies); *S*, stomach side of the oesophagus; *SOG*, suboesophageal ganglion; *son*, superior oesophageal nerve; *STG*, to stomatogastric ganglion; *stn*, stomatogastric nerve.

ageal ganglion (Spirito, 1975; Selverston *et al.* 1976; Moulins & Vedel, 1977). The rhythm is generated by an oscillator located in each commissural ganglion. *In vitro*, the activity of each oscillator consists of series of bursts separated by silent periods. The series of busts of one oscillator regularly alternate with those of the contralateral oscillator (Moulins & Nagy, 1980).

The activity of this distributed network can be monitored by that of the main oesophageal dilator motor neurone (OD1), whose anatomy and functioning were described in the preceding paper (Moulins & Nagy, 1980). OD1's cell body lies in the unpaired oesophageal ganglion, and each of the two main branches of its axo

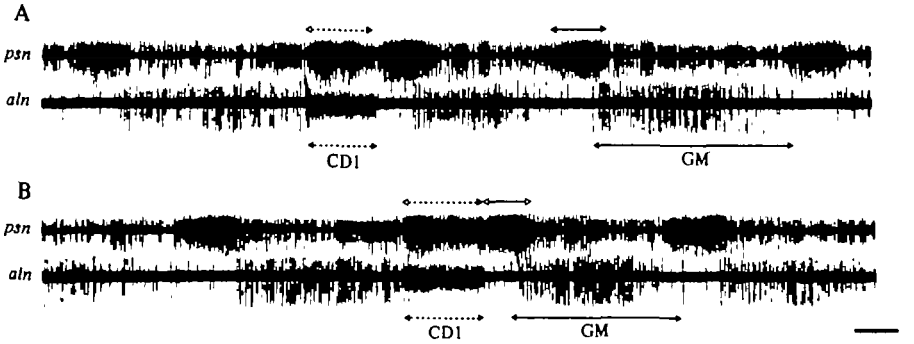


Fig. 2. Posterior stomach receptor (PSR) activity is correlated with the rhythmical motor activity of the gastric mill and of the cardiac sac. The rhythmical activity of the gastric mill is indicated by regular bursts of GM neurones recorded on the anterior lateral nerve (*aln*) of the stomatogastric ganglion (two gastric bursts are marked by solid lines with black arrowheads). The PSR (the activity of which is recorded on the *psn*) driven by the gastric-mill rhythm, fires before each GM burst (solidlines with white arrowheads). The cardiac sac rhythm is monitored by the activity of the CD1 neurone (dilator motor neurone of the cardiac sac), also recorded on the *aln* (dotted lines with black arrowheads). The PSR, driven by cardiac sac rhythm, fires during each CD1 burst (dotted lines with white arrowheads). Panels A and B are not continuous. Calibration: horizontal bar, 2 s.

reaches one commissural ganglion (Fig. 1). OD1 possesses three spike initiating sites, one in each of the three ganglia. Each commissural spike initiating site is driven by the oesophageal oscillator that lies in the corresponding ganglion (Moulins & Nagy, 1980). Thus, by considering the activity of OD1, it is easy to determine which of the two commissural oscillators is active at any time.

The posterior stomach receptors (PSRs)

Each posterior stomach nerve (*psn*) arises as a branch of the outer mandibular nerve, which enters the suboesophageal ganglion (Fig. 1A). The distal part of each *psn* comprises a group of sensory cell bodies and their 2 cm long dendrites, which form the PSR. The dendrites of the PSRs ramify in the soft tissue surrounding the posterior arch of the gastric mill. This arch supports the medial cuticular tooth of the stomach. Because the soft tissue is easily deformed, the PSRs can be stimulated by anything modifying the stomach volume. The records in Fig. 2 show that the PSRs are rhythmically stimulated by the motor activity of the gastric mill (trituration) and by the motor activity of the cardiac sac (temporary food storage). Activity of a *psn* is recorded on a semi-intact preparation in response to spontaneous movements of the foregut, which are driven by stomatogastric motor neurons. Rhythmical discharge of some of these motor neurones is recorded on the anterior lateral nerve (Fig. 2, *aln*). It is possible to recognize: (1) regular bursts of the four electrically coupled motor neurones GM, which drive the power-stroke muscles of the medial tooth of the gastric mill: (2) rhythmical bursts with a longer period. These are comprised of a small unit, CD1, a motor neurone which innervates dilator muscles of the cardiac sac, and fires with the cardiac sac rhythm (Moulins & Vedel, 1977). It appears that the PSR firing is driven by the rhythmical activity of the gastric neuronal network (here in phase opposition to the GM neurons) as well as that of the cardiac sac

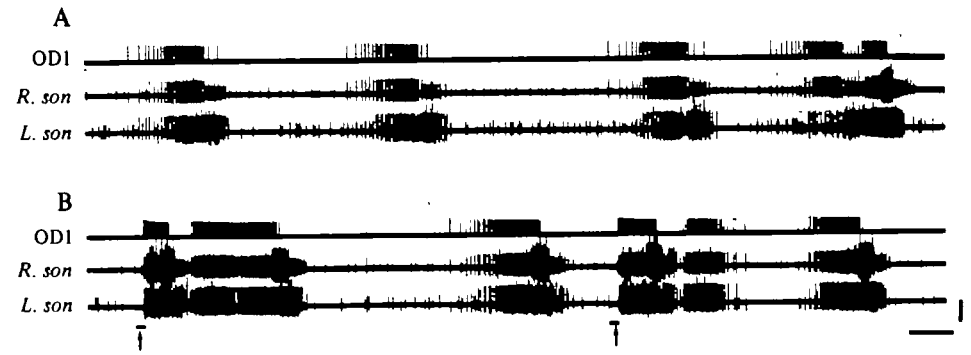


Fig. 3. Electrical stimulation of the *psn* modulates the rhythmic oesophageal motor activity. (A) Spontaneous oesophageal bursting activity recorded intracellularly from the cell body of OD1 and extracellularly on right and left *sons* (*R. son*, *L. son*) in an isolated preparation. (B) A short train of electrical stimulation (arrows) of the PSR dendrites induces a strong burst of oesophageal activity. Calibration: horizontal bar, 1 s; vertical bar: 40 mV.

network (here in phase with CD1). Thus the PSRs have a periodic activity pattern. They provide a rhythmical input to the stomatogastric nervous system, showing several bursts frequencies depending on which neuromuscular system calls them into play.

The central control of the PSRs' inputs to the oesophageal network

In isolated preparations, electrical stimulation of the dendrites of the PSRs modifies the oesophageal activity (Fig. 3). The stimuli we used consisted of short trains (0·15–0·20 s) of 1 ms square pulses at a frequency of 50 Hz. These stimuli were always delivered during the second half of the oesophageal period (see p. 242).

Such a stimulus elicits a burst of OD1 spikes, and all the neurones involved in a normal oesophageal sequence, as recorded extracellularly on the *sons*, are stimulated.

(*a*) *Ipsilateral efficacy of a PSR stimulation*

Stimulation of a PSR activates the oesophageal neuronal network. Because this network is distributed in different centres, we can ask which part of it is stimulated, and which of the two symmetrical oscillators is induced to fire. This question can be answered by looking at the activity of OD1 after PSR stimulation. If the left PSR is stimulated (S1), the elicited OD1 burst originates from spike initiating zone *b* (Fig. 4 A; *b*) in the left commissural ganglion (in Fig. 4 B2, the sequence of arrival of OD1 spikes at the extracellular recording sites is 1, 2, 3, 4). Conversely, if the right PSR is stimulated (S2), the elicited OD1 burst originates from the spike-initiating zone in the right commissural ganglion (Fig. 4A; *c*) and the sequence of extracellular recordings is reversed (Fig. 4C). The two kinds of OD1 burst are similar in duration, number of spikes and latency after stimulation. The terminal part of each record shows that not only OD1, but also most of the oesophageal units recorded in *sons* come from the left ganglion after *S1* stimulation, and from the right one after *S2* stimulation. Therefore, in isolated preparations, the electrical stimulation of a given PSR evokes the response of only one of the two oesophageal oscillators that located in the ipsilateral commissural ganglion.

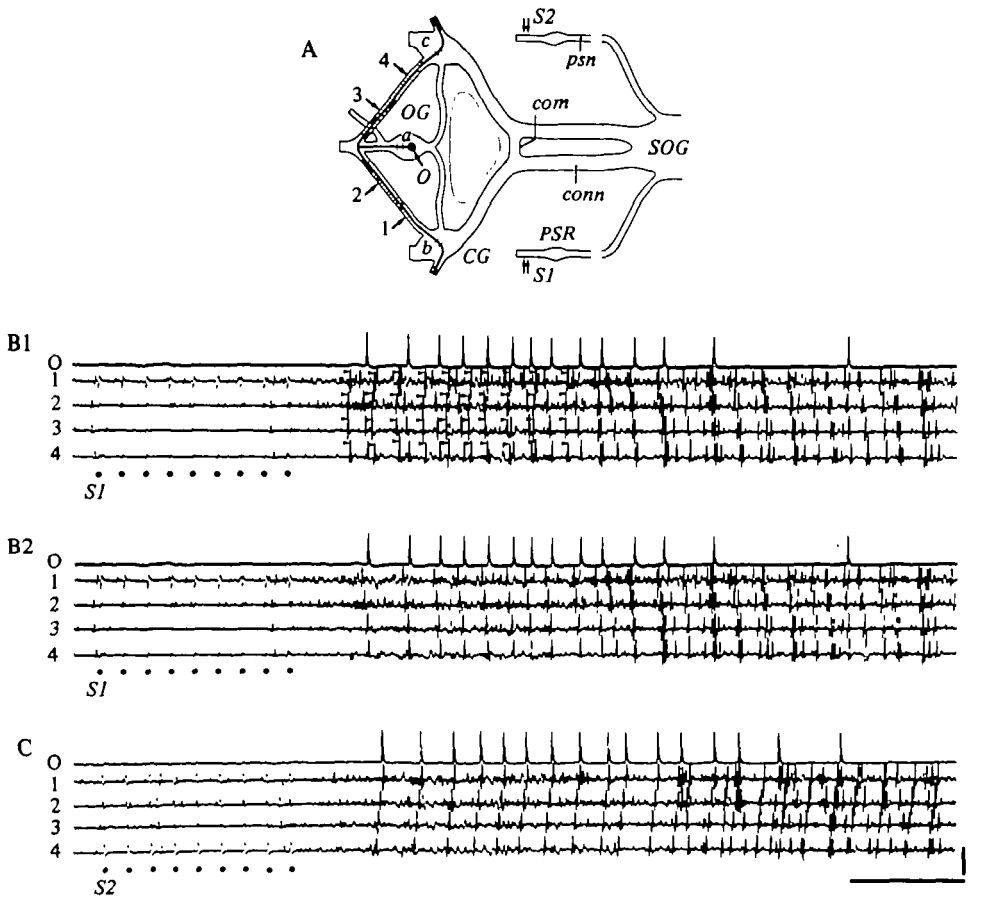


Fig. 4. Stimulation of a PSR elicits an oesophageal burst from the ispilateral commissural ganglion. (A) Diagram of the preparation. For the abbreviations see legend of Fig. 1 (B1–B2). A short train of electrical pulses on dendrites of left PSR (*S1*) elicits a burst of OD1 spikes generated by its spike initiating zone (*b*) in the left commissural ganglion (chronology of extracellular recordings is 1–2–3–4). Other oesophageal units recorded later on *sons* also come from left commissural ganglion (Note that B1 and B2 represent the same record, but that in B1 a large unit (arrowheads) masks the OD1 activity. It is probably not involved in the oesophageal rhythm as it never appears in spontaneous oesophageal bursts. This unit has been erased from B2 and from all high-speed records in subsequent figures to allow easier observation of OD1 activity). (C) Stimulation of right PSR (*S2*) elicits a burst of OD1 spikes generated by its spike initiating zone (*c*) of right commissural ganglion (chronology of extracellular recordings is 4–3–2–1). In both cases delays are similar. Calibration: horizontal bar, 100 ms; vertical bar, 20 mV.

(*b*) *Bilateral projection of a PSR on the oesophageal oscillators*

There are at least two possible explanations for the observation that each PSR activates only the ispilateral oesophageal oscillator: either each PSR projects only on to the ipsilateral part of the oesophageal network; or each PSR projects bilaterally on to the different parts of the oesophageal network, but the contralateral projection is ineffective.

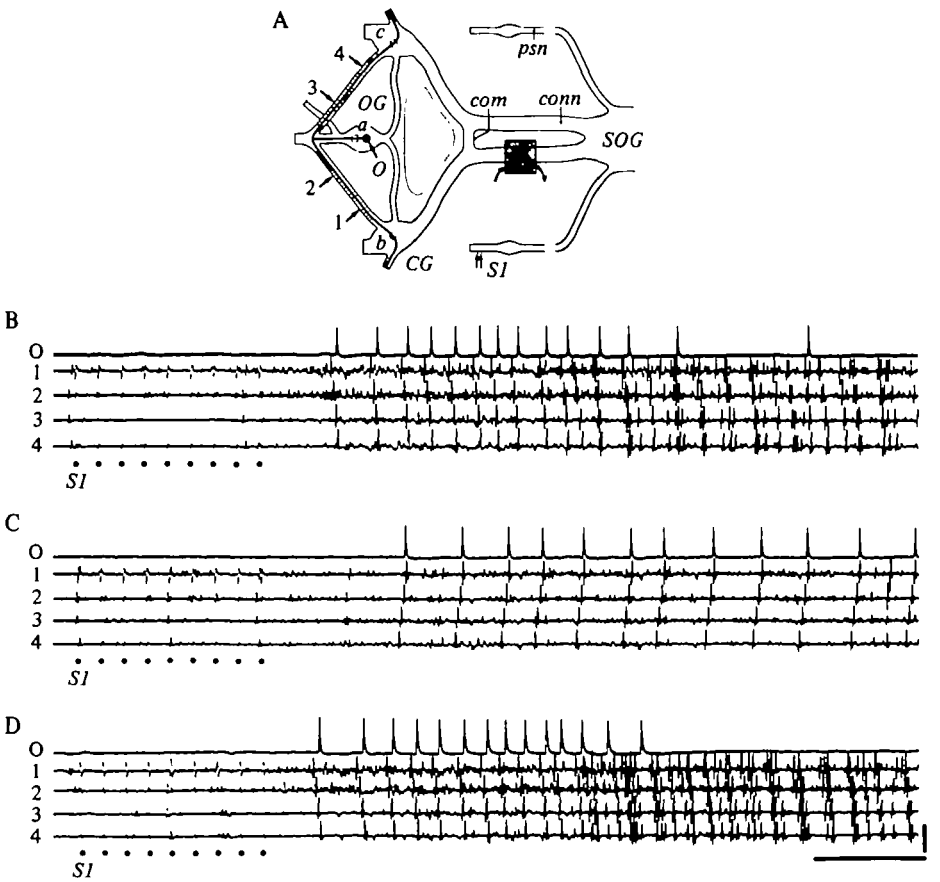


Fig. 5. PSR inputs project bilaterally on the oesophageal network. (A) Diagram of the preparation. Striped square with arrows represents perfused bath of isotonic sucrose solution, whose effects are shown in C. For abbreviations, see legend of Fig. 1. (B) Control experiment: stimulation of left PSR (*S1*) elicits an oesophageal burst from left commissural ganglion (as in Fig. 4). (C) Five minutes after impulse conduction is blocked by a sucrose gap along left connective tract, stimulation of left PSR elicits an oesophageal burst from right (contralateral) commissural ganglion (chronology of extracellular recordings is 4–3–2–1 instead of 1–2–3–4 in control). Note a 25% increase of latency between first electrical pulse and first OD1 spike. (D) After restoring normal impulse conduction along left connective by washing out sucrose solution, *S1* stimulation again elicits an oesophageal burst coming from the left (ipsilateral) ganglion. Calibration: horizontal bar, 100 ms; vertical bar, 20 mV.

It has not been possible to record the activity of the PSR sensory pathways in the connectives between suboesophageal ganglion and the commissural ganglia. It was, however, possible to unmask the contralateral effects of either PSR by reversibly interrupting its ipsilateral pathway. This was done by placing a sucrose gap on one of the connectives between the suboesophageal ganglion and the commissure (Fig. 5A). When conduction in the left connective tract is so blocked, electrical stimulation of the left PSR elicits an OD1 burst which originates at the spike initiating zone in the right (contralateral) commissural ganglion (Fig. 5A, *c*) (In Fig. 5C the spikes of OD1 are recorded extracellularly in the order 4, 3, 2, 1, i.e. the inverse of that in the cont

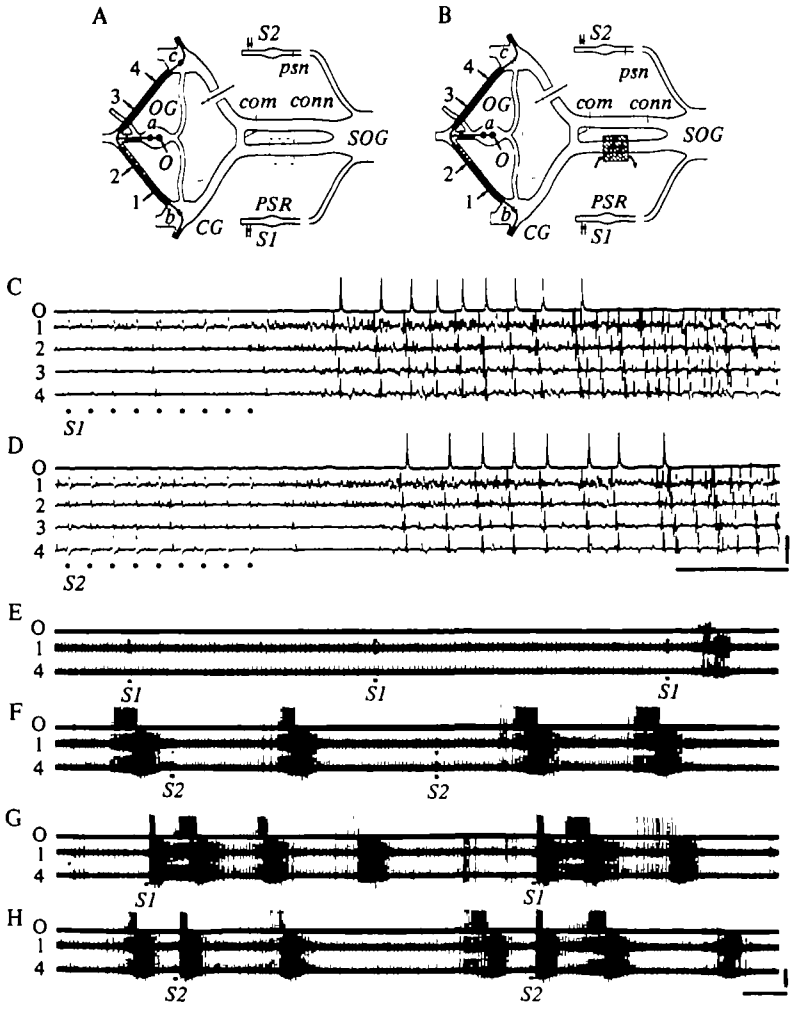


Fig. 6. Crossing of PSR inputs occurs in suboesophageal ganglion. (A, B) Diagram of the preparation. The right connective is cut between commissure (*com*) and right commissural ganglion. Impulse conduction along left connective is normal in (A), and blocked in (B) by means of a perfused isotonic solution of sucrose (striped square with arrows). For abbreviations see legend of Fig. 1. (C, D) With normal impulse conduction along left connective, an oesophageal burst is elicited in left commissural ganglion by stimulation of either left PSR (*S1* in C) or right PSR (*S2* in D). In C and D, extracellular spike chronology is 1–2–3–4, but D shows a longer delay characteristic of the crossed PSR input pathway (as in Fig. 5C). (E, F) When impulse conduction is blocked along left connective a stimulation of neither left PSR (*S1* in E), nor right PSR (*S2* in F) elicits an oesophageal burst, demonstrating that no crossing of sensory pathways occurs in the commissure. (G, H) Recovery of effects of *S1* and *S2* stimulations on left oesophageal oscillator after restoring normal impulse conduction along left connective tract, by washing out the sucrose solution. Note that, in E, OD1 shows a long period of silence. This sometimes occurs spontaneously, and is independent of conduction blockade along connectives (see Moulins & Nagy, 1980). Calibration: horizontal bars, 100 ms in (C, D), 2 s in (E–H); vertical bar, 20 mV.

experiment of Fig. 5B). The response latency is also 25–40% longer. The original response returns when normal conduction is restored (Fig. 5D). In each case the other oesophageal dilator units, which fire after OD1, originate in the same commissural ganglion as do the spikes of OD1. Therefore, even though each PSR excites only the ispilateral part of the oesophageal network, its inputs project on the oesophageal oscillators in both commissural ganglia via a direct and a crossed pathway; but the contralateral projection is normally ineffective.

It is possible to specify the pathways of the PSR inputs by checking for the existence of a possible crossing through the commissure (Fig. 6A, *com*). The right perioesophageal connective was cut between the commissure and the commissural ganglion (Fig. 6A). Under these conditions electrical stimulation of either PSR elicits an oesophageal burst originating in the left commissural ganglion. The burst resulting from stimulation of the right PSR, via the crossed pathway (Fig. 6D), shows a latency 25% longer than that resulting from stimulation of the left PSR, which travels via the direct pathway (Fig. 6C). If impulse conduction is then blocked along the left connective tract by a sucrose gap (Fig. 6B), stimulation of neither the left *psn* (Fig. 6E) nor the right *psn* (Fig. 6F) produces an oesophageal response, although the commissure remains intact. When impulse conduction is restored, the response returns (Fig. 6G, H). It must be concluded that the only site of crossing over of PSR information is in the suboesophageal ganglion.

(c) Nature and location of the control of the sensory pathways

The preceding results have shown that although each PSR projects bilaterally on to both oesophageal oscillators, only one of these projections is effective (i.e. the ispilateral one when both connectives are intact). Therefore it appears that the CNS controls the sensory pathways to ensure the unilaterality of their effects. Because the branching point between the direct and the crossed sensory pathways lies in the suboesophageal ganglion, at least two possibilities for the central control of the sensory inputs may be considered: firstly, this control may take place in the suboesophageal ganglion, using the samples of oesophageal activity which go to this ganglion (see preceding paper, Fig. 13); secondly, this control may be effected by the oesophageal motor network itself. To test these two possibilities, all of the direct connexions between the two parts of the oesophageal motor network were severed by cutting the *sons*, *ions* and the commissure (Fig. 7A). Each ganglion then produces oesophageal bursts with its own rhythm (Fig. 7C). Because the soma of OD1 has been separated from its two commissural spike initiating zones, no activity is recorded in the cell body. In such conditions, electrical stimulation of the left *psn* (Fig. 7A, *S1*) simultaneously elicits an oesophageal burst from both commissural ganglia (Fig. 7D). Thus the ipsi- and contralateral projections of a PSR on the oesophageal network in the commissural ganglia may both be expressed freely only when the two parts of the network are independent. This means that the central control of the sensory pathways from the PSR occurs at the level of the motor network, and not in a premotor centre. This control requires the integrity of the connexions between the elements of the distributed oesophageal network.

Cutting some of these connexions can also unmask a PSR excitation of OD1 in the oesophageal ganglion, which involves its somatofugal spike initiating site. If a

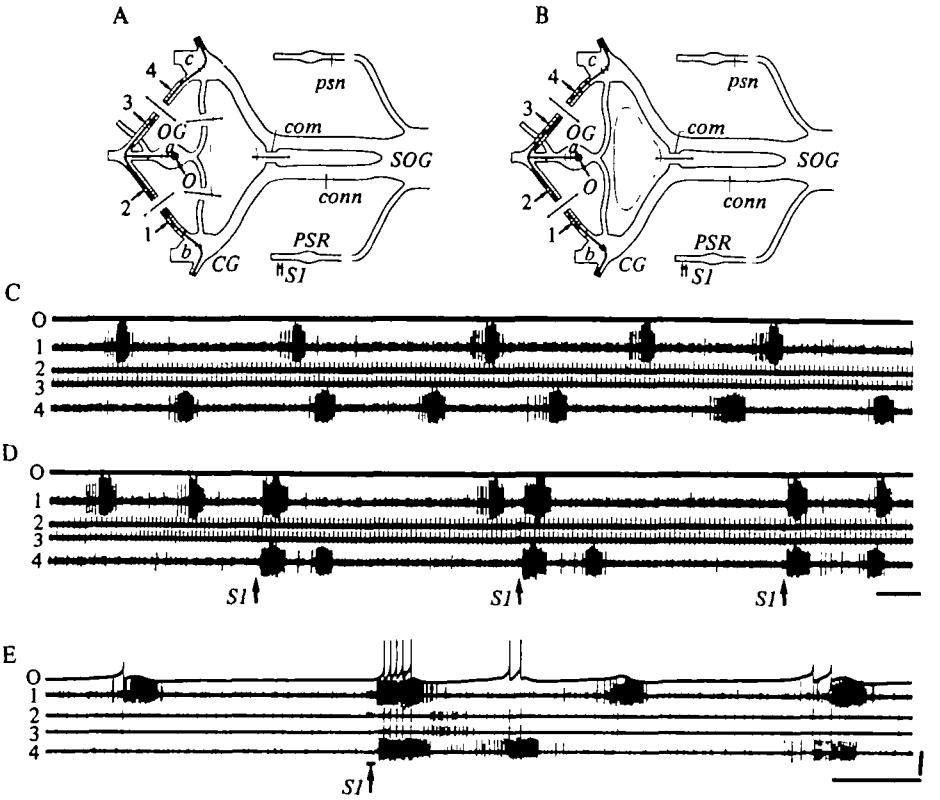


Fig. 7. The different projections of one PSR can be expressed when the connexions between the distributed parts of the oesophageal network are severed. (A) Diagram of the preparation used in (C) and (D). The two *sons*, the two *ions* and the commissure are cut. For abbreviations see legend of Fig. 1. (B) Diagram of the preparation used in (E). Both *sons* and the commissure are cut but the *ions* are kept intact. (C) When the two commissural ganglia are severed from each other, they generate oesophageal bursts with their own rhythms (compare 1 and 4). No activity occurs in the cell body of OD1 (O). (D) In this situation a PSR stimulation (*S1*) elicits both an ispilateral and a contralateral oesophageal burst. (E) When the *ions* are kept intact the same stimulation (*S1*) also elicits a burst from the oesophageal spike initiating zone of OD1. Note that each oesophageal burst, regardless of its origin (left or right commissural ganglion), depolarizes OD1 soma through *ions*. Calibration: horizontal bars, 2 s; vertical bar, 20 mV.

the nerves connecting the two commissural ganglia are cut except the *ions* (Fig. 7B), each oesophageal oscillator, bursting independently, stimulates the oesophageal spike-initiating zone *a* of OD1 through the *ions* (Fig. 7E). A small burst of OD1 somatofugal spikes is produced with each commissural burst. When the two oesophageal oscillators are induced to fire together by a PSR stimulation (Fig. 7E, *S1*), OD1 gives a stronger somatofugal burst. These somatofugal bursts of OD1 are usually not elicited by the stimulation of PSRs when the *sons* are intact and allow control of OD1 activity by the commissural ganglia.

(d) Is there a premotor step where PSRs' inputs are processed?

Since the central control of the sensory pathways from PSRs does not occur in the suboesophageal ganglion, it might be assumed that the PSR primary afferent fibres

project directly in the commissural ganglia. However, the following evidence suggests that the PSR projections on the oesophageal network are not direct. First, there is a difference of 50–80 ms between activation, by a *psn* stimulation, of the oesophageal network in the ipsilateral commissural ganglion compared with activation of the network in the contralateral ganglion (Figs. 5, 6C, D). Because the same commissural elements are involved in such activations, the difference must be due to the crossing in the suboesophageal ganglion. The mean rate of impulse conduction in primary sensory fibres, as measured on a *psn*, is about $1{\cdot}35\ m\ s^{-1}$. If these fibres passed through the suboesophageal ganglion without synapsing, impulses from the cell bodies of a PSR would reach a commissural ganglion after 70–80 ms. But a PSR stimulation elicits an oesophageal burst after a minimum delay of 200 ms (Figs. 4B, C; 5B, D; 6C, D). This delay may be due in part to synapses in the commissural ganglion, but it also suggests the existence of interneurones intercalated in the pathway at the level of the suboesophageal ganglion. It is thus highly probable that the inputs from the PSRs are integrated in the suboesophageal ganglion before projecting on to the different parts of the oesophageal network in the commissural ganglia. Nevertheless, as shown in the preceding paragraphs, this pre-motor integration step is not involved in the central control of the efficacy of the sensory pathways which occurs at the motor network itself.

Modulation of the oesophageal rhythm by rhythmical PSRs' inputs

The oesophageal activity is phasically influenced by stimulation of a PSR. But in intact animal, PSRs are rhythmically induced to fire by movements of the foregut (see Fig. 2). We can now ask to what extent the oesophageal rhythm is influenced by such rhythmical inputs.

(*a*) *The oesophageal network shows a periodically varying sensitivity to PSRs' inputs*

For studying the central control of the sensory pathways from PSRs, we used stimulations which are always delivered during the second half of the oesophageal period. In these conditions an oesophageal burst (as monitored by OD1 activity) is elicited after a delay of about 200 ms, and occurs before the expected time of occurence of the next spontaneous burst. In other words, such a PSR stimulation induces a positive phase shift of the oesophageal dilator burst.

Because a natural PSR stimulation can occur randomly in the oesophageal rhythm period, we examined the effects of electrically stimulating the PSRs at different times in the period of the oesophageal cycle. The PSR stimulated was always that ipsilateral to the active oesophageal oscillator. The stimulus used in these studies was a single pulse of current. Under these conditions, depending on the time of stimulus delivery within the oesophageal cycle, an oesophageal burst can be either positively or negatively phase-shifted by the stimulus (Fig. 8). If *psn* stimulation is given in the second half of the OD1 interburst interval, the next OD1 burst occurs sooner than it would have without stimulation. The oesophageal period is shortened and a positive phase shift of the oesophageal burst occurs (Fig. 8B). If stimulation is given near the end of an OD1 burst, the next burst is delayed and the oesophageal period is lengthened; a negative phase-shift occurs (Fig. 8C). A *psn* stimulation occurring a

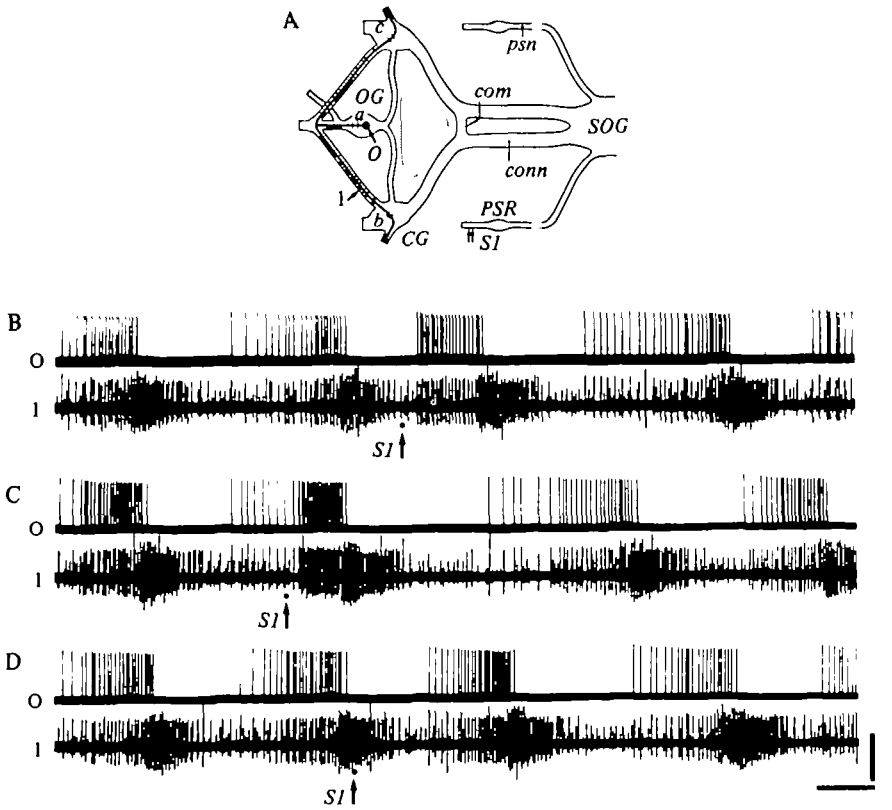


Fig. 8. OD1 (and the oesophageal network) shows a periodically varying sensitivity to PSR stimulation. (A) Diagram of the preparation. For abbreviations see legend of Fig. 1. (B) A PSR stimulation (*S1*), with a single electric pulse, occurring in the second half of OD1 interburst, advances the subsequent burst. (C) If the same stimulus occurs at the end of a burst, the next burst is delayed. (D) If stimulus is given in the beginning of OD1 interburst, OD1 period is not obviously altered. Note that other oesophageal units extracellularly recorded on a *son* (1) follow the shifts of OD1 bursts. Calibration: horizontal bar, 1 s; vertical bar, 20 mV.

the beginning of the OD1 interburst does not obviously modify the oesophageal period (Fig. 8D).

We have quantified these effects by plotting a phase–response curve (PRC) (Fig. 9B) in which the phase shift of the OD1 burst after a stimulation is expressed as a function of the stimulus phase. The phases here are defined relative to the oesophageal rhythm. As the free run period of this rhythm is somewhat variable, a reference period is defined as the average of the three unstimulated periods just prior to the stimulation (Fig. 9A, *Pm*) (each period is measured from the midpoint of a burst to midpoint of the next burst). The stimulus phase (Φ *stim*) is the ratio of the stimulus latency (Fig. 9A,*L*) to the reference period (Φ stim$=L/P_m$); the phase shift ($\Delta\Phi$) is the difference between the reference period (P_m) and the stimulated period (P_s) as a fraction of the reference period ($\Delta\Phi=(P_m-P_s)/P_m=1-(P_s/P_m)$). If the burst of

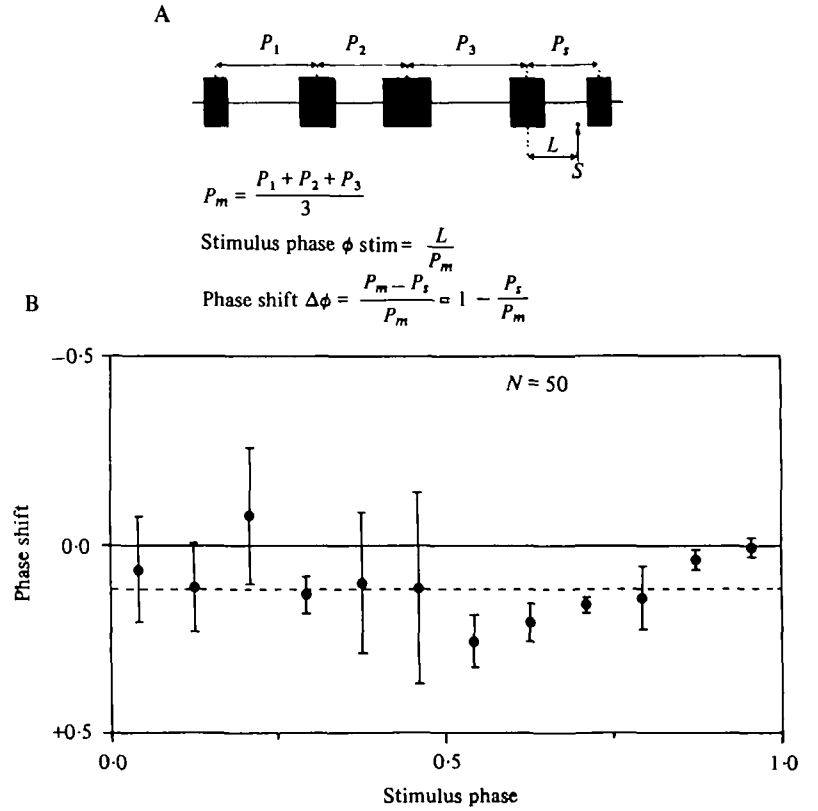


Fig. 9. The varying sensitivity of OD1 to PSR inputs is quantified by a phase-response curve (PRC). (A) Theoretical example to show parameters used: OD1 free-run period (P_m), stimulus phase (L/P_m), and phase shift of OD1 burst after stimulation ($\Delta\Phi$). Stimulation (S) is marked by vertical arrow; period is measured from midpoint of a burst to midpoint of the subsequent burst. For further details, see text. (B) Phase-response curve: the different values of stimulus phase are divided into 12 bins; each point represents the averaged phase shift of OD1 for a given bin; vertical bars represent the standard deviations. If burst of OD1 is advanced by stimulation, phase shift is positive and the PRC is below the horizontal solid line. If burst is delayed by stimulation, phase shift is negative and PRC is above the solid line. The dotted line represents the mean phase shift (+0·11).

OD1 following a stimulus is delayed, $P_s > P_m$ and $\Delta\Phi < 0$. If the burst following a stimulus is advanced, $P_s < P_m$ and $\Delta\Phi > 0$. As shown in Fig. 9B, the phase shift is negative for a stimulus phase of about 0·2 (stimulus occurring at the end of an OD1 burst), and the most positive for a stimulus phase of about 0·55 (stimulus occurring just after the middle of OD1 interburst). Thus, although the general effect of a PSR stimulation is to advance the occurrence of a subsequent OD1 burst (Fig. 9B, mean $\Delta\Phi = +0.11$), the occurrence of such a burst can also be delayed, depending upon the stimulus phase in the oesophageal cycle. The other oesophageal units recorded on a *son* (Fig. 8B, C, D, 1) follow the period variations of OD1. Therefore it may be concluded that each commissural network of oesophageal neurones shows a periodically varying sensitivity to PSR input.

(*b*) *Oesophageal rhythm entrainment by rhythmical PSRs' inputs*

It is known that if an oscillating system presents a periodically varying sensitivity to a rhythmical input, it can, between fixed limits, modify its own frequency to follow that of the input (Enright, 1965). The oscillator is thus entrained by the rhythmical input. Our results suggest that each of the two oesophageal oscillators has this necessary property for entrainment by the rhythmical PSR input. To demonstrate such an entrainment of each network, all the connexions between the two commissural ganglia are cut (see Fig. 7A), so that each commissural ganglion generates oesophageal bursts at its own frequency (Fig. 7C). With the *sons* cut the free-run period of each oscillator is rather variable (mean values are $5{\cdot}81 \pm 1{\cdot}50$ s for the left oscillator and $6{\cdot}20 \pm 1{\cdot}14$ s for the right one). A PSR is then stimulated with repetitive electrical pulses, with a given period near the free-run period of each oesophageal network. The effects of such stimulation on the two independent oesophageal rhythms are illustrated in the curves of Fig. 10A and B. A curve showing the phase of oesophageal bursts in the stimulus period, and plotted in real time, is drawn for each stimulus period. The theoretical example of Fig. 10C shows the measurements and calculations used in forming these curves (see legend). Each curve includes regions of absolute coordination (horizontal parts of the curves) where each electrical pulse is followed by an oesophageal burst at a fixed phase, separated by regions of phase drift, where the oesophageal oscillator temporarily escapes from PSR influence and follows its own rhythm. If the stimulus period is longer than the free-run oesophageal period, phase drifts are negative and the curve is falling. If the stimulus period is shorter, the phase drifts are positive and the curve is rising. The starting position of each curve is arbitrary. The free-run period of the right oscillator indicated by curves of Fig. 10B (between 5 and 6·6 s) fits well with the free-run period observed before stimulation ($6{\cdot}20 \pm 1{\cdot}14$ s). For the left oscillator, the free-run period indicated by curves of Fig. 10A (between 4 and 5 s) is somewhat different from the free-run period observed before stimulation ($5{\cdot}81 \pm 1{\cdot}50$ s). This difference is most probably due to the variability of the free-run period when both oesophageal oscillators are independent.

It appears that with PSR stimulus periods near the free-run period of the oesophageal rhythm, the oesophageal oscillator in each commissural ganglion exhibits relative coordination, which includes periods of phase drift, and absolute coordination with the stimulus cycle. Oesophageal rhythm entrainment by rhythmical PSR inputs can also be illustrated by phase histograms of oesophageal bursts in the stimulus period (Fig. 10D, E). For each stimulus period, the oesophageal bursts occur at a preferred phase in this stimulus period (0·22 in D, 0·12 in E). In Fig. 10D the preferred phase (0·22) in the stimulus period (4 s) corresponds to a phase shift of the oesophageal burst of $+0{\cdot}31$, relative to the free-run period (5·81 s). This value is around the maximum possible phase advance predicted by the PRC of Fig. 9 and is produced by a stimulus whose phase in the oesophageal rhythm is 0·54. This fits well with the stimulus phase (0·53) measured for the entrainment corresponding to curve 4 s in Fig. 10D. In Fig. 10E the preferred phase (0.12) in the stimulus period (5 s) corresponds to a phase shift of the oesophageal burst of $+0{\cdot}19$, relative to the free-run period (6·2 s). As seen in the PRC of Fig. 9 such a phase shift is produced

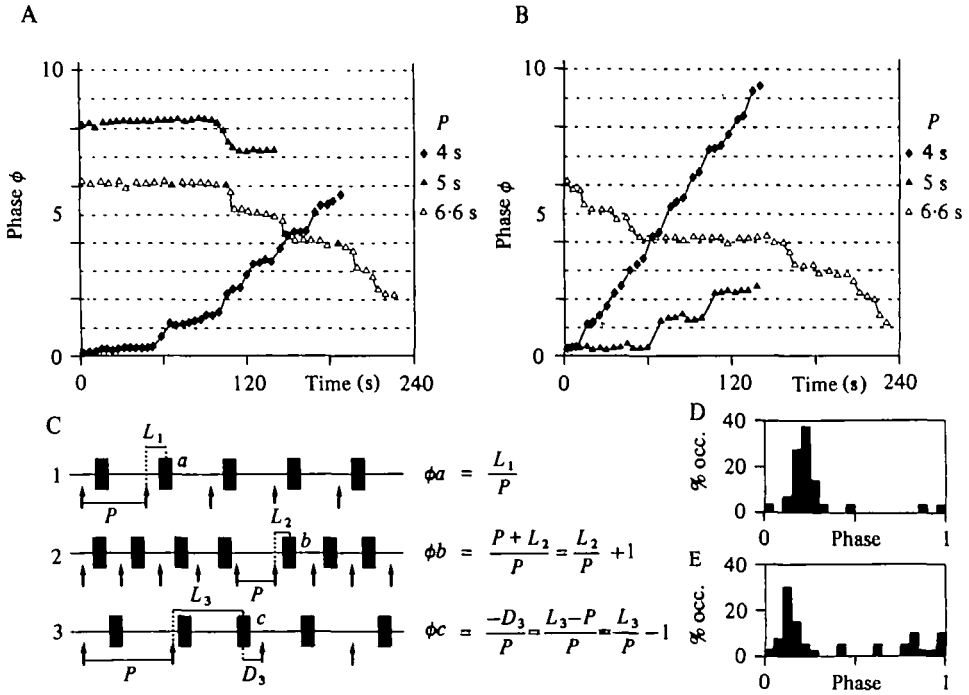


Fig. 10. The oesophageal rhythm is entrained by rhythmical PSR stimulation. Experimental procedure as in Fig. 7A; each commissural ganglion generates oesophageal bursts at its own frequency. (A) Evolution with time (in seconds) of the phase of the oesophageal bursts with three different PSR stimulation periods (4, 5 and 6·6 s) for the ipsilateral commissural ganglion. During absolute coordination (see C1) phase is constant (horizontal parts of the curves). Sometimes a drift of phase occurs (non-horizontal parts of the curves). A phase drift can occur in two situations: (1) when the stimulus period is shorter than oesophageal period (4 s), two successive stimulus pulses can occur between two successive oesophageal bursts. As indicated in C2, there is a positive phase drift. (2) When the stimulus period is longer than oesophageal period (5 and 6·6 s), two successive oesophageal bursts can occur between two successive stimulus pulses. As indicated in C3, there is a negative phase drift. Starting point of each curve is arbitrary. (B) Identical to A for contralateral commissural ganglion. (C) Theoretical example to show how phase of oesophageal bursts in the stimulus period is calculated to plot curves (A) and (B). 1, representation of absolute coordination; 2, representation of a positive phase drift; 3, representation of a negative phase drift; Φa, Φb, Φc, phases of a, b, c oesophageal bursts in stimulus period (P); *arrows*, stimulus electrical pulses. (D) and (E) Phase histograms of oesophageal bursts in the stimulus period for two stimulus periods (4 s in D; 5 s in E). The phase represented on abscissa is divided into 20 bins. Ordinate indicates percentage of occurrence of each of these 20 bins. For each stimulus period, oesophageal bursts occur at a preferred phase (0·22 in D, 0·12 in E) in the stimulus cycle. The small distributed values are due to phase drifts. Histogram D corresponds to curve 4 s in A, histogram E to curve 5 s in B.

by a stimulus whose phase in the oesophageal rhythm is 0·67. This again fits well with the stimulus phase (0·71) measured for the entrainment corresponding to curve 5 s in Fig. 10E. The smaller values of each histogram represent the phase drifts seen between the horizontal portions of the curves in Fig. 10A and B. The stimulation period which produces the best entrainment is similar for the two ganglia. Therefore the oesophageal network in both commissural ganglia can be entrained by rhythmical inputs from the PSRs and then can be phase and time-locked by other central neuronal networks whose activities drive PSRs discharges.

DISCUSSION

Existence of a premotor step in PSR's sensory pathways

The anatomy of the proximal parts of the posterior stomach nerves has until now been unclear (Dando, Chanussot & Nagy, 1974; Maynard & Dando, 1974). The *psn*'s were said to arise as a branch of the outer mandibular nerve in *Homarus* (Dando & Laverack, 1969; Wales, MacMillan & Laverack, 1976) and of the inner mandibular nerve in *Panulirus* and *Palinurus* (Dando & Maynard, 1974). Their sensory fibres were supposed to extend directly to the commissural ganglia in *Cancer* (Hermann & Dando, 1977). Our work on *Jasus lalandii* using a new isolated preparation including the stomatogastric nervous system and both *psns* (Nagy, 1977), has shown the following features:

(1) each *psn* joints with an outer mandibular nerve which enters the suboesophageal ganglion, and not directly the commissural ganglion;

(2) unlike what was reported to exist in *Cancer* (Hermann & Dando, 1977), a population of PSR primary sensory fibres synapses on suboesophageal interneurones. In other words, a pre-motor step in the sensory pathways of the PSRs exists in the suboesophageal ganglion;

(3) these suboesophageal interneurones project on to the oesophageal network in the commissural ganglia.

It is not possible to completely exclude the possibility of PSR primary sensory fibres, which pass through the suboesophageal ganglion without synapsing, and thus project directly on a commissural ganglion. However, the delay between PSR stimulation and the response of the ispilatral oesophageal network, as well as the differences in delay between responses of the ispi- and contralateral oesophageal networks, strongly suggests that PSR inputs are integrated in the suboesophageal ganglion before projecting on to the oesophageal network. The integration of PSR inputs in several centres fits well with the proposed action of the PSRs in coordinating the activities of several central neuronal networks, as will be discussed later.

Bilateral projections of inputs from each PSR

The oesophageal neuronal network is distributed in three nervous centers (Selverston *et al.* 1976; Moulins & Nagy, 1980). The data presented here show that the inputs from each PSR project simultaneously on to the different parts of the oesophageal network, (i.e. on the ipsi- and contralateral commissural ganglia and the oesophageal ganglion). A similar indirect bilateral projection occurs for sensory inputs from a superior laryngeal nerve to the two symmetrical pontic hemicentres which control swallowing in mammals (Jean, 1978). In this case the bilateral projection strengthens the synchrony of the discharge from the two hemicentres. This differs, however, from the situation in the lobster, in which only one of the multiple projections of the PSRs on to the oesophageal network is effective at a given time.

Central control of PSR's sensory pathways

We have shown that the inputs from a given PSR are usually effective only on the ipsilateral part of the oesophageal network. The unilaterality of the PSRs' effects is controlled by the oesophageal motor network itself, and requires the integrity of the

interconnexions between the different parts of this distributed network. Such a central control of sensory pathways has been described in several other sensory-motor systems. This control generally functions to prevent maladaptive self-stimulation during execution of a programmed motor task (for a review, see Stein, 1978). In our case the motor program involves at least two oesophageal oscillators (Selverston *et al.* 1976; Moulins, Vedel & Nagy, 1979). Complex central interactions, which can induce the two oscillators to alternate, allow the oesophageal network to produce a unique and well determined motor output (Moulins & Nagy, 1980). Central control of the variable efficacy of the different PSRs projections probably allows the PSRs to influence preferentially the part of the network which is actually determining the oesophageal activity at a given moment, without eliciting an inappropriate discharge from the contralateral oscillator. This observation might be of general interest, since numerous behaviours (such as locomotion and breathing) involve bilateral nervous control centres which are influenced by bilateral proprioceptors through direct and crossed pathways. These different pathways must be considered in terms of the responsiveness of their respective targets at a given time.

It should also be noted that PSR inputs are controlled not in the suboesophageal premotor step of the sensory pathway, but in the motor network itself. This is different from the central control of tactile receptors in the crayfish abdomen during the tailflip, whose mechanism is one of the few which are well understood. In the crayfish abdomen, the control is exerted on the synapses between primary afferent fibres and first-order interneurons (Bryan & Krasne, 1977).

Coordination of oesophageal rhythm with other foregut rhythms through PSR's proprioceptive loop

Rhythmical sensory inputs may have several functions in the execution of a central motor program: (1) to adapt expression of this program to environmental variations (Grillner, 1975; Bowerman, 1977; Clarac & Ayers, 1977); (2) to increase and stabilize the discharge frequency of an oscillatory system and to coordinate outputs of its follower neurones (Wendler, 1974; Kristan & Calabrese, 1976; Carlson, 1977); (3) to co-ordinate the activities of several oscillators (Pearson & Iles, 1973; Kristan & Stent, 1976; Bowerman, 1977). As oesophageal movements do not stimulate the PSRs it is unlikely that these last serve the first function mentioned above. This is probably done by mouthparts receptors, which monitor deformations of peribuccal areas (Moulins *et al.* 1969). The relationship between the PSRs and the oesophageal nervous system is, however, appropriate to the 2nd and 3rd functions.

For an input to modify the rhythm of an oscillator, the oscillator must show a periodically varying sensitivity to the input, i.e. a significant phase response curve (PRC) to this input (Enright, 1965; Pavlidis, 1973). This is true for endogenous oscillators (Ayers & Selverston, 1979; Benson, 1980; Pinsker, 1977 *a*) as well as network oscillators (Stein, 1976). If the input is itself driven by another oscillator, that last may govern the activity of the first (' magnet effect ' of Von Holst, 1973). We have plotted a PRC of oesophageal rhythm, as reflected in the activity of OD1, to PSR's input.

Theoretically such a curve may be used to predict the limits of stable entrainment (i.e. the range of input frequencies which elicit an oesophageal burst for each PSR stimulation), the lower and upper limits being respectively determined by maxim

possible phase delay and maximum possible phase advance (Pinsker, 1977*b*; Benson, 1980). The PRC shows that, in our conditions of simulation, a PSR rhythm between 9% slower and 27% faster than the oesophageal free-run rhythm would produce such an absolute co-ordination. But the oesophageal free-run period is variable, and when the oscillator rhythm is not rigorously constant, a fixed stimulus period induces only a relative co-ordination (Ayers & Selverston, 1979). Indeed in our experiments PSRs produce such relative co-ordination of oesophageal rhythm with intervals of absolute co-ordination separated by phase drifts.

In intact and semi-intact animals the PSRs are stimulated by gastric mill movements and discharge with a gastric rhythm (Dando *et al.* 1974; present report). Are the PSRs able to entrain the oesophageal oscillators to the gastric rhythm ? We have shown that, *in vitro*, each PSR can entrain the oesophageal rhythm when it is induced to fire rhythmically with a period near the oesophageal period. Therefore, for the PSRs to entrain the oesophageal rhythm to the gastric one, the gastric and oesophageal periods must not differ greatly. Indeed, it appears that, even in an isolated preparation, the oesophageal and the gastric periods are sometimes the same (Selverston *et al.* 1976). Thus, the PSRs may be able to entrain the oesophageal rhythm at the gastric frequency in the intact animal. Some coordination with ratios of gastric: oesophageal cycle of 1:2 and 1:3 has also been observed (Selverston *et al.* 1976). But it has been shown that an oscillator which is entrained by a periodic input firing at nearly its period, will also be entrained (with a ratio different from 1:1) by the same input firing with a period close to double or half that of the oscillator (Wendler, 1974; Stein, 1977). Thus in these conditions, PSRs might again be able to entrain the oesophageal rhythm at the gastric frequency. It has been reported that in deafferented preparations some gastric neurones receive trains of epsp's from the oesophageal neurones of the commissural ganglia (Selverston *et al.* 1976). Thus, central relations exist between the two rhythm generators. We propose that the PSRs pathway constitutes a peripheral loop through which the gastric and the oesophageal rhythm generators can be coupled, and that this loop can reinforce the central coupling which exists between them.

It appears that PSRs can also discharge with a cardiac sac rhythm (see Fig. 2), and complex central coupling between the oesophageal and cardiac sac rhythms has been described (Moulins & Vedel, 1977). Here again a peripheral mediated coupling through PSRs may reinforce the central one.

Complex behaviour can be understood as the addition of discrete behaviours, each one being organized by a given neuronal network (Carlson, 1977). The different networks involved in such a complex behaviour must be linked to insure a coordinated motor activity. Rhythmic activity of the oesophagus is an example of a discrete behaviour integrated into a complex one, the motor activity of the foregut, the final goal of which is to ensure progression and mechanical processing of the food. The other components of this behaviour are the movements of the cardiac sac, tritutation by the gastric mill teeth, and food filtering by the pyloric stomach. Each of these is governed by an identified neuronal network (Selverston *et al.* 1976; Moulins & Vedel, 1977).

Movements elicited by some of these networks (gastric and cardiac ones) rhythmically stimulate the PSRs (Dando *et al.* 1974; present work). PSRs could be also

stimulated by mouthparts movements during feeding, as some of their sensory c[illegible] probably monitor mandibular movements (Wales *et al.* 1976). The PSRs in turn influence the rhythmic discharge of the gastric, pyloric (Dando *et al.* 1974; Hermann & Dando, 1977; Nagy, 1977) and oesophageal networks (present work). Therefore the PSRs have the necessary characteristics to contribute to the coordination of complementary behavioural sequences (feeding, swallowing, food processing in stomach).

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