Oesophageal sensors and their modulatory influence on oesophageal peristalsis in the lobster, *Homarus gammarus*

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[Plates 1–5]

The musculature and innervation of the oesophagus of *Homarus gammarus* are described as a prerequisite to studies on the mechanisms and control of food ingestion. Of particular interest are two paired sensors (the anterior and posterior oesophageal sensors) which are bilaterally situated at the oesophageal–cardiac sac valve. These are similar to contact chemoreceptors previously described in insects and are classified as such on morphological grounds and with indirect electrophysiological evidence.

Oesophageal peristalsis is effected by the coordinated contraction of the oesophageal musculature. This is controlled by rhythmical bursting neuronal activity, which can be recorded from the nerve trunks in the area. A characteristic burst recorded from the superior oesophageal nerve is used as an indication of oesophageal dilatation during peristalsis for studies on the feedback effects of the oesophageal sensors. Electrical and chemical stimulation of the posterior oesophageal sensors can initiate and increase the frequency of oesophageal peristalsis, while stimulation of the anterior oesophageal sensors can slow and terminate oesophageal peristalsis.

The results are discussed and a model presented of the role of the oesophageal sensors in feeding.

1. Introduction

Rhythmical motor activity plays an important part in the lives of most animals. It controls such diverse processes as respiration (see, for example, Wyse 1973; Young 1975), feeding (see, for example, Kater 1974; Siegler *et al.* 1974; Selverston *et al.* 1976), circulation (see, for example, Thompson & Stent 1976) and locomotion (see, for example, Bowerman 1977; Kristan *et al.* 1974a, b; Ort *et al.* 1974). In recent years, study of the neuronal mechanisms underlying such activity has increased (see, for example, Friesen & Stent 1977; Getting & Willows 1974; Perkel & Mulloney 1974; Russell & Hartline 1978). In particular, the foregut of decapod crustaceans has provided an excellent preparation for many studies on the neuronal control of rhythmic behaviour. Since the pioneer work of Maynard...

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(Maynard 1966, 1967; Maynard & Burke 1966; Maynard & Atwood 1969), the stomatogastric system has become a paradigm of a restricted neural network controlling well defined behavioural acts (see Selverston et al. 1976, for a review). Inherent in the study of the control of rhythmic behaviour is that of sensory modulation of the intrinsic action pattern. This paper describes the modulatory influence on oesophageal peristalsis of two bilateral oesophageal sense organs in a decapod crustacean (the lobster, Homarus gammarus).

The foregut of decapod crustaceans comprises a short oesophagus joining the mouth to an ectodermal, chitinized cardiac sac. This contains a complex gastric mill for trituration of food material (see, for example, Balss 1941). At its posterior limit is a pyloric press and filter which diverts small food particles into the hepatopancreatic ducts and larger food particles into the midgut (Vonk 1960). The anatomy of the cardiac sac, gastric mill and pylorus has been described in a wide variety of decapod crustaceans (Maynard & Dando 1974; Meiss & Norman 1977a, b; Fryer 1977), and that of the mandibles and labrum has also been described (Robertson & Laverack 1979; Wales et al. 1976). The oesophageal musculature typically consists of upper and lower anterior oesophageal dilators, lateral and posterior dilators and a complex oesophageal constrictor with longitudinal and circular fibres (Mocquard 1883; Paterson 1968; Pearson 1908; Yonge 1924). Observations on the oesophagus of Homarus gammarus showed that discrepancies exist between its anatomy and that reported for the oesophagus of other decapod crustaceans. This is particularly noticeable for the muscles at the oesophageal–cardiac sac valve. For these reasons, this paper first presents a detailed description of the oesophageal morphology and musculature of Homarus gammarus. Since the main nerves of the oesophageal system have been described by a number of authors (for a review, see Bullock & Horridge 1965), only a brief description of the general anatomy will appear below.

Dando & Maynard (1974) have presented a review of the sensory innervation of the foregut of decapod crustaceans, primarily based on the early work of Allen (1894), Ringel (1924) and Orlov (1926a, b), and on their own observations with Panulirus argus. Two bilaterally symmetrical groups of neurons on the anterior oesophageal wall were described in Homarus by Allen (1894), and redescribed in Astacus leptodactylus by Orlov (1926a). In this paper, these are referred to as the anterior oesophageal sensors (a.o.s.). Ringel (1924) described in Astacus innervated pegs in a series of sensory plates on the surface of the ventral cardiac gutter. In Homarus, similar structures can be found, although these occur on the posterior wall of the oesophagus, at the level of the oesophageal–cardiac sac valve. They will be referred to as the posterior oesophageal sensors (p.o.s.). The second part of this paper redescribes in more detail these oesophageal sensors (o.s. comprising the a.o.s. and p.o.s.) in Homarus gammarus.

Studies on labral movements during feeding indicated that labral swallowing activity (mirroring oesophageal peristalsis) is initiated only after some food material has been pushed into the oesophagus, and not by mechanical or chemical
stimulation of the mouthparts (Robertson 1978; Robertson & Laverack 1979). This implies that there is an internal mechanism for the initiation of peristalsis. The final part of this paper investigates the feedback effects of stimulation of the oesophageal sensors. In conclusion, a model is presented of the role of the oesophageal sensors in the control of oesophageal peristalsis in Homarus gammarus.

2. Materials and methods

Dissections were performed on fresh specimens in sea water. Details of the nervous system were obtained with the vital stain methylene blue. A stock solution of 2% (by mass) methylene blue in distilled water was added to the dissection dish, in sufficient quantity to colour the sea water light blue (approximately 15 drops of stock solution per 100 ml sea water). Staining and further dissection were alternated until maximal staining occurred. Sufficiently stained preparations were processed and mounted as permanent preparations.

Tissue for histological examination was fixed in sea water Bouin, embedded in paraffin wax, and sectioned at 10 μm to facilitate counting of sensory endings in the oesophageal cuticle. Serial sections were stained with either the Mallory triple stain or the Heidenhain Azan stain (Pantin 1964).

Pieces of tissue appropriate for examination with the scanning electron microscope were removed from an animal and fixed with 4% (by volume) formalin in sea water. After fixation they were washed in distilled water, dehydrated in an acetone series, critical-point-dried with CO₂, coated with gold–palladium, and viewed on a Cambridge S600 Stereoscan.

Conventional techniques were used to stimulate and record from selected nerves. The oesophageal sensors were chemically stimulated with an extract of Mytilus edulis; when one organ was stimulated the afferent axons of all other presumptive chemoreceptors in the area were cut.

3. Results

(a) Oesophageal anatomy

(i) Oesophageal morphology

The oesophagus is a short tube lined with thin, flexible cuticle, connecting the mouth with the cardiac sac. The anterior wall folds inwards to give the lumen a U shape in transverse section, with the arms of the U pointing anteriorly. This fold, and thus the anterior oesophageal wall, connect ventrally with the furcircular sclerite of the labrum (Robertson & Laverack 1979). The lateral walls of the oesophagus are continuous with the inner rims of the mandibles. Posteriorly, the oesophageal cuticle attaches to the metastomal plate of the ventral skeleton (Snodgrass 1952) and is associated with the paragnaths and the first maxillae. The dorsal limit of the oesophagus is defined by the oesophageal–cardiac sac valve (figure 1). This simple valve is composed of four lobes: one anterior lobe,
which is continuous with the anterior fold of the oesophagus; two lateral lobes (right and left); and one small posterior lobe, situated at the antero-ventral limit of the ventral gutter, between the ventral ends of ossicles Xa and XIII (Maynard & Dando 1974).

Closure of the opening between the oesophagus and the cardiac sac is performed mainly by the anterior and lateral lobes, which are invested with three pairs of extrinsic dilator muscles (OCSV 1, 2 and 3, see below).

Figure 1. Oesophageal–cardiac sac valve: (a) interior aspect from cardiac sac, (b) anterior aspect, (c) left lateral aspect; O5a, oesophageal dilator muscles; OCSV, muscles of the oesophageal–cardiac sac valve (hatched areas indicate the positions of their insertions); oss., ossicles Xa and XIII.

(ii) Oesophageal musculature

Movements and peristalsis of the oesophagus are controlled by four pairs of extrinsic muscles (the dilators) and one complex intrinsic muscle (the constrictor). The numbering starts with the most antero-ventral, and moves dorsally, laterally and then posteriorly. The oesophageal–cardiac sac valve (o.c.s.v.) is controlled by three pairs of extrinsic dilators and the upper limits of the posterior oesophageal dilator (O5a) and of the oesophageal constrictor (O4). The o.c.s.v. muscles are numbered from medial to lateral. The above muscles are portrayed in figure 2 (left lateral aspect), figure 3 (anterior aspect) and figure 4 (oesophagus split
ventrally and flattened). Included in the diagrams are the cardiac sac muscles C4, C5 and C6, the ventral cardiac muscle, CV1; and the cardio-pyloric valve muscles, CPV2a and CPV2b. The origins, insertions and routes of these muscles are virtually the same as those described for the homologous muscles in *Homarus americanus* (Maynard & Dando 1974).

Also shown (figure 4) is the paragnathal retractor, which originates from the medial, anterior cuticle of the ipsilateral mandible (Wales et al. 1976, in their figure 3).

O1 (O standing for oesophageal muscle) is the lower anterior oesophageal dilator. It runs from its origin on the median apodeme of the supra-labral ridge, underneath L6 and the oesophageal constrictor (O4), to a large diffuse insertion on the lower anterior wall of the oesophagus. Here the individual muscle fibres intermingle with those of L6. A small bundle (O1a) enters the anterior oesophageal fold and inserts on the furcicular sclerite of the labrum.

O2 is the upper anterior oesophageal dilator. This largish muscle originates on the median apodeme of the supra-labral ridge. Its posterior end passes between the fibres of O4 to insert on the anterior oesophageal wall dorsal to the insertion of O1.

O3 has its origin on the posterior margin of the epistomal plate. It is a large muscle and runs postero-medially to a broad insertion on the lateral oesophageal wall. The superior oesophageal nerve runs through the posterior end of O3, effectively separating its insertion into two heads (upper and lower). This is the lateral oesophageal dilator.

O4 is the intrinsic muscle of the oesophagus, the constrictor (figure 4). Although the majority of this muscle cannot be morphologically divided, there will be functional divisions depending on its innervation pattern. Its various attachments are as follows:

*Extrinsic.* (1) Two paired ventral non-muscular ligaments, anteriorly to the labral falciform sclerite and posteriorly to the metastomal plate. (2) Two paired ventral muscle branches, anteriorly to the labral furcicular sclerite (O1a) and posteriorly to the anterior cuticle of the paragnath. (3) A loose posterior connective tissue attachment to the first sternal apodeme of the endophragmal skeleton (the cephalic or head apodeme (Snodgrass 1952)).

*Intrinsic.* (1) One large, diffuse attachment on the anterior surface of the cardiac sac, on either side of the midline, and between the insertions of C5. (2) One paired dorso-lateral attachment to the cuticular thickening at the insertion of OCSV3a. (3) The passage of the fibres of the extrinsic oesophageal and o.c.s.v. muscles to their insertion on the cuticle through the fibres of O4 anchors O4 to the cuticle in three areas. These occur as well defined longitudinal bands anteriorly, laterally and posteriorly (hatched areas in figure 4). At these bands the fibres of the extrinsic dilators tend to run longitudinally up and down the oesophagus. It is possible that fibres from O4 also insert on the cuticle in these areas and thus contribute to effective anchorage.

O5 can be subdivided into upper (O5a) and lower (O5b) components. Both
parts have their origins on the cephalic apodeme and the first portion of the major mandibular abductor apodeme (M7 of Wales et al. 1976). O5a has a broad diffuse insertion on the dorsal portion of the posterior oesophageal cuticle. Its limit is defined by small branches at the level of the oesophageal–cardiac sac valve. O5b has insertions directly opposite the cephalic apodeme; it then becomes a broad flat muscle, which runs ventrally, to insert on the posterior ventral rim of the oesophagus. O5 is the posterior oesophageal dilator.

**Figure 2. Oesophageal musculature, left lateral aspect:** C, cardiac sac muscle; CPV, cardiodpyloric valve muscle; c. sac, cardiac sac; CV, ventral cardiac muscle; endo.sk., 1st sternal apodeme of endophragmal skeleton (cephalic apodeme); epi., epistoma; L, labral muscle; lig. st., ligamentous strap; mand., mandible; O, oesophageal muscle; OCSV, oesophageal–cardiac sac valve muscle; oss., ossicles Xa and XIII.

OCSV1 (OCSV standing for oesophageal–cardiac sac valve muscle) is broad in the dorso-ventral plane and narrows latero-medially. It arises midway along a narrow ligamentous strap which runs between the median apodeme of the supra-labral ridge and the exoskeleton, on the ipsilateral side of the cerebral ganglion. From this origin it passes posteriorly to its insertion in the anterior oesophageal fold, just below the anterior lobe of the oesophageal–cardiac sac valve. Contraction will open the valve and dilate the dorsal limit of the oesophagus.

OCSV2 originates on the supra-labral ridge lateral to the median apodeme. It is very narrow and runs postero-dorsally to its insertion on the oesophageal
cuticle at the lateral edge of the anterior lobe of the o.c.s.v. This muscle will act to open the valve by retracting the anterior lobe.

OCSV3 is divided into dorsal (OCSV3a) and ventral (OCSV3b) portions. They have a common origin on the posterior margin of the epistoma, lateral to the origin of O3 and medial to the common origin of CV1 and C4. OCSV3a inserts on a cuticular thickening in the dorso-lateral corner of the lateral lobe, and its action must open the valve by retracting this lobe. OCSV3b has a diffuse insertion on the ventral border of the lateral lobe and on the oesophagus, ventral to this. It can be described as an upper lateral oesophageal dilator.

Although closure of the valve will be passively induced by the relaxation of OCSV1, 2 and 3, and by the weight of food material in the cardiac sac, O4 will play a significant part. It can be seen that the dorsal limit of O4 consists of a band of muscle running around the oesophagus at the level of the valve and inserting anteriorly, laterally and posteriorly. Contraction will constrict the valve and bring about closure.
(iii) Oesophageal innervation

The oesophageal nervous system is shown diagrammatically in figures 5 and 6. The nomenclature used follows Maynard & Dando (1974), with one exception. They describe in *Callinectes sapidus, Homarus americanus* and *Panulirus argus*

![Diagram of oesophageal musculature](image)

*Figure 4. Oesophageal musculature. The oesophagus and the lower portion of the cardiac sac have been split along the posterior midline and flattened. The right side (left side of diagram) depicts the total musculature and the other side depicts the intrinsic musculature (O4 and C6). Note the attachments of O4: four paired ventral attachments consisting of two non-muscular ligaments (to the falciform sclerite of the labrum and to the metastomal plate of the ventral skeleton), two muscular attachments (to the fuscular sclerite of the labrum (O4a) and to the paragnathal cuticle), one median posterior attachment to the cephalic apodeme, one large median antero-dorsal insertion on the cardiac sac and one paired dorso-lateral insertion on the cuticular thickening at the insertion of OCSV3a. Hatching indicates where the insertions of the extrinsic oesophageal and OCSV muscles anchor O4 to the oesophageal cuticle. Abbreviations: a.l., anterior lobe of o.c.s.v.; conn. tiss., connective tissue attachment to cephalic apodeme; l.l., lateral lobe of o.c.s.v.; met., metastomal plate; ret. para., retractor paragnatha; Xa and XIII, ossicles of cardiac sac; others, as for figure 2.*

the postero-lateral nerve (p.l.n.) arising as a fusion of the dorsal–posterior oesophageal nerve (d.p.o.n.) and the ventral–posterior oesophageal nerve (v.p.o.n.) In *Homarus gammarus* there appears to be little, if any, fusion between the corresponding nerves, and so the p.l.n. is considered as arising directly from the
superior oesophageal nerve (s.o.n.), and a d.p.o.n. section is not described. The courses of the major nerves in this area have been well described in a number of animals (Allen 1894; Keim 1915; Mocquard 1883; Paterson 1968; Pearson 1908), but they are described here to provide the essential basis for subsequent electrophysiology.

**Figure 5.** Oesophageal innervation, left lateral aspect. The extrinsic muscles are indicated by the positions of their insertions. The arrow indicates the branch from the s.o.n. that innervates OCSV2 as it passes in front of this nerve. Abbreviations: a.o.s., anterior oesophageal sensor; C, cardiac muscle; c.c., circumoesophageal connective; co.g., commissural ganglion; CV, ventral cardiac muscle; i.l.n., inner labral nerve; i.o.n., inferior oesophageal nerve; i.v.n., inferior ventricular nerve; MPR1, mouth part receptor 1; O, oesophageal muscle; OCSV, oesophageal–cardiac sac valve muscle; o.g., oesophageal ganglion; o.l.n., outer labral nerve; o.n., oesophageal nerve; p.l.n., postero-lateral nerve; p.o.c., posterior oesophageal commissure; p.o.s., posterior oesophageal sensor; s.o.n., superior oesophageal nerve; st.n., stomatogastric nerve; v.p.o.n., ventral–posterior oesophageal nerve.

The motor innervation of the labral–oesophageal complex originates from the paired commissural ganglia (co.g.) and the unpaired oesophageal ganglion (o.g.). The commissural ganglia are situated ventrally on the circumoesophageal connectives (c.c.), which run between the cerebral ganglia (ce.g.) and the suboesophageal ganglion (s.g.), on either side of the oesophagus. Posterior to the oesophagus and anterior to the s.g. the connectives are joined by the post-oesophageal commissure (p.o.c.). The commissial ganglia give rise to three major nerve trunks: the inferior oesophageal nerves (i.o.n.), the s.o.n. and the v.p.o.n. The i.o.n. travel medially from the co.g. on the anterior surface of the oesophagus, to
meet at the oesophageal ganglion, which lies in the anterior midline. The s.o.n. travel in the same way, dorsal to the i.o.n., and meet in the anterior midline. From this junction there arises the short thick oesophageal nerve (o.n.), which connects ventrally with the i.o.n.–o.g. junction, and the stomatogastric nerve (st.n.), which travels dorsally to connect the oesophageal nervous system with the stomatogastric nervous system. The v.p.o.n. run dorsally from the co.g. and come out of the dorsal surface of the circumoesophageal connectives. From there they run dorsally and posteriorly on the surface of the oesophagus and terminate in sensory endings at the level of the o.c.s.v.

Issuing from the oesophageal ganglion are four major nerve trunks. The i.o.n. and o.n. are described above. From its antero-dorsal surface the o.g. produces a fine nerve, the inferior ventricular nerve (i.v.n.), which passes anteriorly, between the ligamentous straps, to the cerebral ganglia.
(b) Anatomy of the oesophageal sensors

(i) The anterior oesophageal sensor

The anterior oesophageal sensors (a.o.s.) are crescent-shaped organs, situated on the oesophagus, on either side of the anterior midline and at the level of the o.c.s.v. They occur at the lateral limits of the anterior lobe (a.l.) of the o.c.s.v. Their position in relation to the nervous system of the oesophagus is shown in figures 5 and 6.

![Diagram of the oesophageal sensors](image)

**Figure 7.** Right anterior oesophageal sensor, anterior aspect (diagrammatic). Note two groups of receptor material (a, b) comprising the organ and also a small group (two neurons shown) of large bipolar cells whose axons also run in the a.o.s.n. Inset, lateral view of the a.o.s. displayed in longitudinal section. Abbreviations: a.l., anterior lobe of oesophageal–cardiac sac valve; a.o.s., anterior oesophageal sensor; a.o.s.n., nerve innervating the a.o.s.; c.t., connective tissue; O4, oesophageal constrictor; OCSV2, dilator of oesophageal–cardiac sac valve; oes., oesophagus.

*Methylene blue staining.* Figure 7 and its inset show the a.o.s. from anterior and lateral aspects; this figure is based on information obtained by methylene blue permanent preparations (figure 8a, b, c, plate 1). Each sense organ is divided into two populations of receptor cells (a, b in figure 7, 8a). Group a (figure 8b) is composed of 250–300 small (15–20 \( \mu \)m long axis of cell body), bipolar neurons with short dendrites, situated in a narrow band at the lateral edge of a.l.

The second population, group b, consists of 50–60 larger structures, which
innervate a wide curving band along the dorso-lateral border of a.l. Closer investigation reveals each of these to be a bundle of 3–5 small (15–30 µm long axis of cell body), bipolar neurons. Thus, the total number of neurons in group b lies between 150 and 300. The neurons in each bundle are closely associated and tend to stain as a single structure. The underlying composition was revealed in preparations that had either stained poorly or destained during fixation and mounting. When the preparations are viewed with Nomarski interference contrast illumination, the dendrites of each bundle can be seen to be associated with discrete structures on the internal surface of the oesophagus. These were examined further with the scanning electron microscope. The axons of both group a and group b neurons travel ventrally in a large bundle (the a.o.s.n.) and join the superior oesophageal nerve.

The area around the a.l. is heavily invested with connective tissue in which the a.o.s. is embedded. This forms a bridge between the cardiac sac and the oesophagus, and effectively occludes the opening to the lumen of a.l. It also ramifies around the various muscle insertions, particularly those of OCSV2 and O4.

There is a further group of neurons whose axons also travel in a.o.s.n. to s.o.n., but which is not classified as being part of the anterior oesophageal sensor. The group consists of a small number (2–5) of large (60–80 µm long axis of cell body), bipolar neurons, found a short distance dorsal to the a.o.s. (figures 7, 8c). Their cell bodies are not always in close proximity with one another; their dendrites are long (several mm) and unbranched for as far as they could be traced. The dendrites travel over the surface of the cardiac sac and oesophagus in the region of the o.e.s.v.

Histology. Figure 9, plate 2, shows 10 µm transverse sections through the anterior wall of the oesophagus in the region of the a.o.s. Although it is not clear whether the epicuticle (ca. 5.0 µm thick) is penetrated, it is evident that the chitin layer (ca. 50 µm thick) is penetrated in two distinct ways, first, by pores between 1 and 3 µm in diameter, which contain stained filaments running from the epithelium and whose distal ends appear to be associated with small nodules on the epicuticular surface, and, secondly, by pores 5–8 µm in diameter, which occur in regions where the chitin layer has thinned considerably (down to ca. 20 µm). These large pores are associated with depressions (ca. 5.0 µm deep and ca. 20 µm in diameter) of the epicuticle and a concomitant thinning of the epicuticular layer. Several filaments of the type seen in the small pores can be seen entering each large pore, the underlying epithelium seems to be structured in a globular fashion.

The distribution and number of both types of pore were studied in serial sections (figure 10). There is a large number of small pores; these are confined to a sharply delineated band ca. 800–1000 µm long and ca. 100 µm wide. The larger pores are fewer in number and distributed with a concentration towards the dorsal end of the organ. The area to which they are confined is not as narrowly defined as that of the small pores; it has a width of approximately 600 µm.
Figure 8. Right anterior oesophageal sensor, methylene blue stained preparations. (a) Whole organ. Note two groups of receptor material: group a, numerous small bipolar cells; group b, several larger structures, which are bundles of 3–5 small cells. Scale mark 500 μm. (b) Enlargement of group a. Scale mark 300 μm. (c) Two bipolar cells whose dendrites travel over the surface of the oesophagus and cardiac sac and whose axons run in the a.o.s.n. Scale mark 300 μm.
**Figure 9.** Wax sections (10 μm) through the oesophagus in the region of the a.o.s., stained with the Mallory triple stain. Scale mark 50 μm. Upper pair, large pores (indicated by dots) through the chitin layer. These are associated with a thinning of the chitin and epicuticular layers and a depression of the epicuticle. Note also the globular structuring of the epithelium. Lower pair, small pores (indicated by dots) through the chitin layer. Stained filaments travelling through the pores from the epithelium are associated with nodules on the epicuticular surface.
Figure 11. Scanning electron micrographs of cuticular structures associated with the left (a) and right (b) a.o.s. Note numerous hillocks with small depressions over the lateral walls of the anterior lobe of the oesophageal–cardiac sac valve. Scale mark, 200 μm.
Figure 12. Scanning electron micrographs of cuticular structures associated with the a.o.s.: large hillocks with depressions on their raised surfaces. Each depression contains a small number (1–4) of small nodules clustered together. There is a sparse covering of bristles between the hillocks which are themselves devoid of setules. Arrowed in (b) is a depression in the centre of a nodule. This may indicate the presence of a pore or a region of thin cuticle. Scale marks: (a) 40 μm; (b) 10 μm.

Figure 13. Scanning electron micrographs of cuticular structures associated with the a.o.s.: small nodules (indicated by dots) on the surface of the cuticle. Arrowed in (b) is a depression in the centre of a nodule. This may indicate the presence of a pore or a region of thin cuticle. Scale marks: (a) 40 μm; (b) 10 μm.
Figure 16. Right posterior oesophageal sensor (methylene blue stained preparation). Scale mark, 200 μm.

Figure 17. Scanning electron micrograph of cuticular structure associated with the p.o.s.: one small group of the p.o.s. to show a scattering of approximately 50 depressions and a patchy covering of bristles in a well-defined area. Scale mark, 100 μm.
Scanning electron microscopy. The internal surface of the oesophagus in the region of the a.l. is heavily invested with a large number of cuticular setules, ranging in length from 200–500 µm and with a basal diameter of 8–10 µm. However, at each dorso-lateral corner of the a.l. is a crescent-shaped area that is devoid of these long setules. These areas correspond exactly with the positions of the a.o.s. as demonstrated with methylene blue.

Figure 10. The number and distribution of small (a) and large (b) pores in the right a.o.s. The inset (circled) shows the direction of sectioning, with regard to the whole organ, and the thickness of the sections (dors., dorsal).

Figure 11, plate 3, shows the left (a) and right (b) sides of the a.l. from a ventral aspect. Immediately obvious on the lateral walls of the a.l. are numerous small, rounded hillocks, whose basal diameters are approximately 50 µm and which appear to have small depressions in their centres.

Close examination of the whole area, moving laterally over the a.l. from the medial edge of the bare patch, reveals the following structures:

(1) Of the rounded hillocks mentioned above, 50–70. These commonly have depressions, 10–15 µm across, on their raised surfaces. Associated with each depression is a variable, small number (1–4) of small nodules (2–4 µm in diameter). Occasionally, tiny depressions can be seen in the centre of each nodule. Between the hillocks is a sparse covering of bristles, 7–10 µm long and 1–2 µm in diameter. The hillocks themselves are devoid of setules (see figure 12a, b, plate 4).
(2) Lateral to the hillocks is a long, narrow area which is also clear of setules. The epicuticle in this region is not obviously structured in any way, save for a profusion of small nodules, similar in size and shape to those described above. These, however, are present singly and do not form recognizable groups (see figure 13a, b, plate 4).

(3) Running alongside this is a narrow band (40–50 µm wide) with a dense covering of bristles (figure 13a). Among these can occasionally be seen a single line of pits, or pores, in the cuticle. These are typically 4–6 µm across.

The bristle band described above marks the lateral angle of the a.l., and the remaining area of the bald patch does not appear to be structured in any way. The above information is summarized in figure 14.

![Figure 14](image)

**Figure 14.** Ventro-lateral aspect of the left lateral wall of the anterior lobe (a.l.) of the oesophageal-cardiac sac valve to summarise information from scanning electron micrographs. (1) Large hillocks with groups of small nodules in depressions. (2) Narrow area devoid of setules but covered in small nodules. (3) Band of bristles at the lateral limit of the a.o.s., commonly having a single line of pores at its medial edge. Abbreviations: c.sac., cardiac sac; oes., oesophagus.

(ii) **The posterior oesophageal sensor**

The posterior oesophageal sensors are to be found on either side of the posterior midline of the oesophagus, at the entrance to the cardiac sac. They are present between the small posterior lobe and the lateral lobes of the o.c.s.v. and their positions are symmetrical about the midline.

*Methylen blue staining.* The right p.o.s. is portrayed diagrammatically in figure 15; photographs of methylene blue preparations are shown in figure 16, plate 5. Each sensor is composed of one large group of 150–200 sensory cells and two or three smaller groups, each containing 50–70 of these cells. Unlike the cell groups of the a.o.s., those of the p.o.s. do not appear to have a constant, uniform shape in different animals, and their positions are variable within a limited area.
The cells are small (10–15 μm long axis of cell body), bipolar and uniterminal, with short dendrites, that terminate at the epicuticle. Their sensory axons travel in the v.p.o.n. to the commissural ganglion.

Methylene blue preparations of the sensor, viewed by Nomarski interference contrast illumination, show that the dendritic endings are associated with distinct epicuticular structures that occur in an area devoid of large setules but invested with a patchy covering of bristles. These structures were examined by means of the scanning electron microscope.

Scanning electron microscopy. The cuticular lining of the oesophagus in the posterior region of the o.c.s.v. is similar to that in the anterior region in that there is a large number of large setules (200–500 μm long and 8–10 μm basal diameter). There are, however, no setules in the area innervated by the p.o.s. Within this area, distinct groups of superficial modifications can be seen. Figure 17, plate 5, shows one such group; two types of structure are noticeable: (a) collections of small bristles similar to those described at the a.o.s. epicuticular surface; (b) a scattering of small depressions of the epicuticle. The latter are about 10 μm in diameter; each contains a small nodule in its centre. These nodules are approximately 2 μm in diameter. The pattern of bristles and depressions does not form any recognizable configuration, save that the two are mutually exclusive.
(c) **Feedback effects of the oesophageal sensors**

Electrophysiological recording from the superior oesophageal nerve in a minimally dissected preparation reveals a complex, rhythmic burst which occurs at oesophageal dilatation during peristalsis (figure 18).

Using the s.o.n. dilator burst as an indicator of peristaltic frequency, the effect of electrical stimulation of the nerves carrying the sensory axons (v.p.o.n. and a.o.s.n.) and of chemical stimulation to the organs themselves (p.o.s. and a.o.s.) was studied.

![Figure 18. Rhythmical bursting associated with peristalsis recorded in the superior oesophageal nerve (s.o.n.) and the ventral–posterior oesophageal nerve (v.p.o.n.), (a) and (b) at different speeds. Open circles, small constrictor unit in the s.o.n., which fires on a 1:1 basis with a constrictor unit in the v.p.o.n.; closed circles, medium sized dilator unit; open triangles, very small unit in the v.p.o.n. corresponding 1:1 with the conspicuous large dilator unit in the s.o.n.](image)

(i) **The posterior oesophageal sensor**

Electrical stimulation of the v.p.o.n. could initiate bursting activity in the s.o.n. (figure 19). The experiments were performed on ageing preparations that had ceased spontaneous bursting. Three consecutive trains of pulses were delivered to the v.p.o.n. The first caused an increase in the background activity during stimulation, and bursting was initiated after cessation of stimulation. The second initiated bursting immediately after the onset of stimulation, and this continued after stimulation ceased. The third train initiated bursting immediately, but this ceased during stimulation. The burst frequency in each case was level and low at ca. 0.15 Hz. That the effect was short-lived was probably due to the age of the preparation.

Stimulation of the v.p.o.n. during spontaneous bursting activity of the s.o.n. caused an increase in the burst frequency (figure 20). This was variable during stimulation but subsequently settled down to a long-lived stable high frequency at ca. 0.3 Hz. The extent of the effect was dependent on the original level of activity.
No sensory activity could be recorded from the sensory axons of the p.o.s. in the v.p.o.n., but the effect of electrical stimulation could be mimicked by application of *Mytilus* extract directly on to the organ (figures 21, 22), i.e. application, of the extract onto the p.o.s. caused a rapid increase in the s.o.n. burst frequency to a level of ca. 0.3 Hz which was stable and maintained for several minutes.

![Graph](image)

**Figure 19.** Initiation of bursting activity in the superior oesophageal nerve. Graphs of the s.o.n. burst frequency and histograms of the number of spikes in successive 1 s bins during electrical stimulation of the v.p.o.n. Stippled bars mark the duration of each stimulus; (a) and (b) are continuous. In this and all following graphs, the instantaneous frequency of a burst is calculated as the reciprocal of the interburst interval, which is measured from the start of one burst to the start of the succeeding one.

(ii) *The anterior oesophageal sensor*

Electrical stimulation of the a.o.s.n. had two effects on the s.o.n. burst. First, the burst frequency was reduced from its initial level to approximately 0.1 Hz. Thus, the effect was more dramatic with a higher initial frequency. Secondly, the
Figure 20. Increase in the frequency of bursting in the s.o.n. on electrical stimulation of the v.p.o.n. Bars mark the duration of each stimulus; (a) and (b) are different experiments.

Figure 21. Trace to show the increase in the frequency of bursting in the s.o.n. and the v.p.o.n. on application of *Mytilus* extract to the p.o.s. Dots indicate artefacts associated with positioning of the pipette where some release of the extract may have occurred. The arrow indicates the time of release of the extract. Also apparent in the v.p.o.n. trace is the shortening of burst length and increase of spike frequency associated with an increase in the burst frequency.
number of spikes in each burst was reduced. Initially the number of spikes per burst varied around 30; during stimulation this dropped to a variation around 10. On cessation of stimulation the burst frequency and the number of spikes per burst increased, but without reaching their former levels (figure 23).

Attempts to record sensory activity in the a.o.s.n. came to naught. However, an application of Mytilus extract directly onto the a.o.s. mimicked the effect of electrical stimulation of the sensory axons, by reducing the burst frequency (figure 24), although there appeared to be no reduction in the number of spikes per burst. The burst frequency could, in fact, be reduced to zero with continued stimulation (figure 24a). To be effective, the extract had to be very closely and continuously applied to the a.o.s. From figure 24b it can be seen that the burst frequency increased towards its original level when the pipette containing the extract was removed without washing the organ.

![Graph](image1)

Figure 22. Increase in the frequency of bursting in the s.o.n. on application of Mytilus extract to the p.o.s. Closed triangle indicates the time of release of the extract.

![Graph](image2)

Figure 23. Decrease in the number of spikes per burst (a) and of the decrease in the frequency of bursting (b) in the s.o.n. on electrical stimulation of the a.o.s.n. Bars indicate duration of the stimulus.
4. Discussion

(a) Observations of the oesophagus

The general form and musculature of the oesophagus of Homarus differs little from that described in other decapod crustaceans: Astacus (Mocquard 1883); Cancer (Pearson 1908); Nephrops (Yonge 1924) and Jasus (Paterson 1968). Anterior, lateral and posterior oesophageal dilators are present in all and their courses are similar. However, it proved useful in this study to treat the oesophageal-cardiac sac valve as a separate entity with its own dilators. This concept arose after the fibres of these dilators had been followed through the oesophageal constrictor to their insertion on the cuticle. The anterior and lateral lobes of the o.c.s.v. are well developed and the muscles under consideration (OCSV1, 2, 3) were found to be associated with these lobes, rather than with either the oesophagus or the cardiac sac. Whether or not this morphological differentiation mirrors a functional division remains to be seen. A muscle, equivalent in size, shape and position to OCSV1, has been described as an oesophageal elevator (Mocquard 1883). While it will undoubtedly have this effect, its prime role must be to dilate the o.c.s.v. OCSV2 is a narrow, somewhat frail, muscle which has not yet been described in other animals. OCSV3 is probably represented by the antero-lateral dilators of the foregut in Cancer (Pearson 1908) and in Jasus (Paterson 1968). In these animals it is classified as a foregut muscle rather than an oesophageal muscle and it apparently inserts on the cardiac sac. It is also portrayed, unnamed, in figure 8 of Maynard & Dando (1974) for Homarus americanus, in which its course is the same as in H. gammarus.

The oesophageal constrictor is a complex muscle and little is known of its
detailed morphology. Its ligamentous and muscular attachments to the oesophagus and surrounding structures are reported in this study. The presence of a separate band of muscle at the dorsal edge of O4 lends credence to the concept of a separate muscular system at the valve, although the question of whether it is a functional division would have to be resolved electrophysiologically. This band has distinct attachments at the insertion of OCSV3a in the lateral lobe. The ventral muscular attachments to the paragnathal cuticle and to the furcule sclerite (O4a) may be significant in promotion of effective mouth closure. The movement of the paragnathals could prevent the loss of food material from the posterior part of the mouth, as has been suggested by Farmer (1974) for *Nephrops*. O4a will pull the labrum over the mouth as it closes. The ligamentous attachments are probably simple anchorage points that prevent the ventral rim of muscle O4 from riding up during contraction.

(b) Sensory systems of the oesophagus

Large multi-terminal neurons have been described as innervating the oesophagus of larval *Homarus* (Allen 1894) and *Astacus* (Allen 1894; Orlov 1926a, b). Dando & Maynard (1974) were unable to find large numbers of these cells (as described by Allen) in *Panulirus* or *Homarus*, but could confirm small numbers innervating the gut. In this paper a small group of neurons with a relatively constant position is described. They are innervated by the a.o.s.n. and their dendrites travel over the oesophagus and cardiac sac, in the region of the o.c.s.v. These cells are probably mechanoreceptors that respond to stretch and are suitably placed to monitor movements of the o.c.s.v.

The oesophageal sensors are present in a ring around the oesophagus at the level of the o.c.s.v. Although the organization of the a.o.s. is considerably more complex than that of the p.o.s., the individual receptor elements are the same in both organs. In group a of the a.o.s. and in the p.o.s., these elements occur singly; in group b of the a.o.s. they are organized into bundles of three to five. In the latter the nodules are grouped into the central depression of a relatively large hillock (50 μm basal diameter). Moulins (1968) gave five morphological criteria that may be used to classify organs as contact chemoreceptors. These are:

1. Similarity with previously described organs. Although similar organs have not been described in crustacean decapods, the o.s. are comparable with the epipharyngeal and hypopharyngeal organs of the cockroach, *Blaberus craniifer* (Moulins 1968, 1971), and with the A1 sensilla on the clypeo-labrum of *Locusta migratoria migratorioides* (Cook 1972). The former have indirectly been shown to respond to the application of chemicals (Moulins 1971). In these instances each ‘cone’ (equivalent in size and shape to the ‘nodules’ described for the o.s.) has been shown, by transmission electron microscopy (t.e.m.), to be innervated by four to five bipolar neurons. In the o.s. the number of sense cells stained with methylene blue approximates the number of nodules seen with the scanning
electron microscope, s.e.m. But methylene blue staining is known to be capricious and to stain only a proportion of the cells present. T.e.m. is necessary before a definite statement about the number of cells innervating each nodule can be given.

(2) Direct contact of the dendrite with the external medium. Whether or not there is a pore through the cuticle at the apex of the nodule is debatable. Cook (1972) could not identify pores in the A1 sensilla by t.e.m., but considered that small central depressions of the cones observed with the s.e.m. were indicative of pores. The nodules of the o.s. have similar depressions. However, it is possible that there are regions of very thin cuticle that have collapsed during the drying process. Other well described crustacean chemoreceptors, the antennular aesthetascs (Laverack & Ardill 1965), have no pores (Laverack 1975) and Ghiradella et al. (1968) claim that the permeability of the aesthetasc wall is sufficient to allow access of the stimulatory substance to the dendrite. This may be so for the o.s.

(3) A lack of modification of the peripheral processes. Mechanoreceptors tend to contain dense material surrounding numerous microtubules in the apical region (Thurm 1965, 1968). For the o.s. this would need to be confirmed with a t.e.m. study.

(4) A large number of sensory cells. Mechanoreceptors tend to have small numbers of neurons compared with chemoreceptors. The a.o.s. and p.o.s. comply with this criterion by having 400–600 and 250–350 neurons respectively.

(5) The position of the organ. The o.s. are admirably situated to sample food material in the oesophagus. The o.s. can thus be classified, on morphological grounds, as contact chemoreceptors.

An important point to note concerning the a.o.s. is that its endings are situated deep in the cleft between the anterior and lateral lobes of the o.c.s.v. In their normal position these lobes will completely occlude the organ, the bare patch of cuticle on the lateral lobe covering the cuticular structures of the anterior lobe (figure 14). The arrangement of the o.c.s.v. dilators (1, 2) is such that their contraction pulls the anterior lobe forward, as a whole, without collapsing it. This is not true for the lateral lobes, which have the insertions of OCSV3 (right and left) deep in their cavities. Thus, during normal opening and closing of the valve, the a.o.s. will not be available for stimulation and it will only become so when the cardiac sac is filled to capacity and the o.c.s.v. is stretched open.

(c) Feedback effects of the oesophageal sensors

(i) Initiation of oesophageal peristalsis

Intuitive reasoning leads to the conclusion that effective feeding behaviour is best elicited by chemical stimulation, the release of specific chemicals being a property of potential food material that differentiates it from non-nutritive matter. For example, Lee & Liegeois (1974) have categorized the chemosensory nerves that are important in food arousal for Pleurobranchaea californica (Mollusca, Opisthobranchia), feeding activity can be induced in Helisoma trivolis (Mollusca,
Pulmonata) by the application of crushed spinach leaves (Kater & Rowell 1973) and, in *Phormia regina* (Insecta, Diptera), there are three groups of chemoreceptors (tarsal ‘hairs’, labellar ‘hairs’ and inter-pseudotracheal papillae), which are sequentially stimulated to facilitate food ingestion (Gelperin 1972). Maynard & Dingle (1963) characterized the feeding responses of *Panulirus argus* (Crustacea, Decapoda) to chemical and chemotactile stimulation of the antennules and of the dactyls of the pereiopods. These responses are similar in *Homarus gammarus* (Crustacea, Decapoda). The limitation of behavioural studies of this sort is that oesophageal peristalsis cannot be observed in intact, free animals. Thus, one cannot determine whether chemical stimulation of the antennules, dactyls and mouthparts is sufficient to induce the total feeding repertory, or not. Observations of the movements of the labrum during feeding suggest that peristalsis is not initiated until some portion of the food has entered the oesophagus (Robertson & Laverack 1979). Thus, some internal mechanism, either chemosensory or mechanosensory, must be involved. The present study has shown that electrical stimulation of the v.p.o.n. can initiate bursting in the s.o.n. In addition, electrical stimulation of the v.p.o.n. and the application of a food extract to the p.o.s. can increase the frequency of the oesophageal rhythm. These factors are taken both as indirect evidence for a chemoreceptive function of the p.o.s. and as evidence that the potent stimulus for the initiation of oesophageal peristalsis is chemical stimulation of the p.o.s.

(ii) Termination of oesophageal peristalsis

Preventing of hyperphagia by some mechanism is essential for most animals. To date, relatively few such control mechanisms have been studied. Internal inhibitory feedback mediated by gut stretch receptors is documented for *Phormia regina* (Dethier & Gelperin 1967), *Locusta migratoria* (Insecta, Orthoptera) (Bernays & Chapman 1973), *Chortoicetes terminifera* (Insecta, Orthoptera) (Barton-Browne *et al.* 1975) and *Aplysia californica* (Mollusca, Opisthobranchia) (Susswein & Kupfermann 1975). In *Phormia*, input from abdominal stretch receptors augments the inhibition of feeding, mediated by foregut stretch receptors (Gelperin 1971). Some measure of control is also provided by the adaptation of chemoreceptors that excite feeding activity (Gelperin 1971, for *Phormia*; Barton-Browne *et al.* 1975, for *Chortoicetes*). In *Locusta*, as well as adaptation of maxillary palp chemoreceptors, which can be overcome by palpation (Blaney & Duckett 1975), a mechanism exists whereby the terminal pores of these chemoreceptor sensilla can be closed (Bernays *et al.* 1972). This effect is mediated by a nervous and hormonal pathway, of which foregut stretch receptors comprise the first element (Bernays & Chapman 1972). Long-term regulation of meal size can be brought about by negative feedback from increased blood osmotic pressure (Gelperin & Dethier 1967, for *Phormia*; Bernays & Chapman 1974, for *Locusta*). However, the normal meal size limit is usually set by foregut stretch receptors, irrespective of other conditions (Bernays & Chapman 1973, for *Locusta*). The
results reported here suggest that the system that may signal satiation in *Homarus gammarus* is different, being dependent on negative feedback mediated by chemoreceptor excitation.

Electrical stimulation of the a.o.s.n. results in a marked decrease in the frequency of the oesophageal rhythm. This may be due to an inhibitory influence of a small group of presumptive stretch receptors whose axons also can travel in the a.o.s.n. This could override excitatory chemosensory afference. However, chemical stimulation of the a.o.s. mimics the effect. This leads to the conclusion that the reduction in the frequency of the oesophageal rhythm is mediated by the a.o.s. Differences can be observed between the responses of electrical and of chemical stimulation. The number of spikes per burst is reduced with electrical, but not with chemical, stimulation and chemical stimulation can completely terminate the rhythm, while electrical stimulation has not been seen to do so. If one considers that the presumed mechanoreceptors may be acting in a similar way to those described as coordinating the feeding cycle of *Helisoma trivolvis* (Kater & Rowell 1973), then an explanation is possible. In *Helisoma*, phasic afferent activity from the mechanoreceptors inhibits the protractor motorneurons of the buccal mass to limit their spike output, and excites the retractor motorneurons to accentuate and regulate their burst. Thus, electrical stimulation of the chemosensory axons in the a.o.s.n. would reduce the frequency of the oesophageal rhythm, but the simultaneous stimulation of the mechanosensory axons could be both reducing the spike output of the dilator burst and ensuring that rhythmicity is maintained, albeit at a greatly reduced frequency.

The position of the a.o.s. is such that it can only be stimulated when the cardiac sac is filled to capacity and the o.c.s.v. is stretched. This observation corroborates the hypothesis that the a.o.s. mediates the termination of oesophageal peristalsis by signalling satiation. However, to be effective the response of the a.o.s. to continued stimulation would need to be very slow adapting; of this we have no evidence.

Russell (unpublished, cited in Selverston et al. 1976) has provided evidence that electrical stimulation of a chemoreceptor nerve on the anterior oesophageal wall of *Panulirus interruptus* provokes a two- to threefold increase in the frequency of the oesophageal and gastric rhythms. The organ innervated by this chemoreceptor nerve is possibly homologous to the a.o.s. of *Homarus gammarus*. The present report is contradictory and needs to be explained. Recently, Meiss & Norman (1977c) undertook a numerical taxonomical analysis of 11 species belonging to the five infraorders of the decapod Crustacea, using homologies between the muscles of the stomatogastric system. Reference to their figure 2 reveals that the average similarity between the Palinura and the rest of the Reptantia is relatively low at 61%. Further, Dando & Maynard (1974) have described the organ in *Panulirus argus* (in the Palinura with *Panulirus interruptus*) as being different from the organ in *Homarus americanus* (in the Astacura with *Homarus gammarus*). So there is no a priori reason to suppose that the functions of the two organs should be similar.
(d) **Model of the role of the oesophageal sensors in the control of oesophageal peristalsis**

This model (figure 25) is principally concerned with the role of the oesophageal sensors and no provision has been made to include the effects of other receptors (e.g. the presumptive stretch receptors also innervated by the a.o.s.n.). Thus it is understood that stimulation of the o.s. may not be the only means of producing the effects, although alone it is sufficient.

![Diagram of oesophageal peristalsis](image)

**Figure 25.** Model of the role of the oesophageal sensors in the control of oesophageal peristalsis. Explanation in text.

Food is broken down by the cooperative action of the mandibles and 3rd maxillipeds and presented to the mouth. This has the effect of mechanically stimulating the m.p.r. system and the labral mechanoreceptors to induce the mouth to open. Food is pushed into the oesophagus. As it comes into contact with the p.o.s. these are chemically stimulated to effect, by an unknown route, the initiation of oesophageal peristalsis. This transports food to the cardiac sac. From this point three ways to continue are possible.

1. If the cardiac sac is not full and food is still being presented, oesophageal peristalsis continues until the food runs out (2) or the sac is filled (3).

2. If the cardiac sac is not full and the food is finished, the p.o.s. adapt and oesophageal peristalsis terminates until such time as more food is obtained (A).

3. If the cardiac sac is full, then the o.c.s.v. will be stretched open and
stimulatory material allowed access to the a.o.s., and oesophageal peristalsis is inhibited. As the a.o.s. gradually adapt, oesophageal peristalsis is resumed. In this instance it is assumed that movement of the food once more restimulates the a.o.s. (similarly to locust maxillary palp palpation (Blaney & Duckett 1975)). This cycle continues to give irregular bouts of peristalsis until the cardiac sac is emptied by the action of the gastric mill and pyloric filter at which time the sequence returns to (1) or (2). Russell (unpublished, cited in Selverston et al. 1976) has shown that, in Panulirus interruptus, stimulation of the organ that is possibly homologous to the a.o.s. increases gastric mill and pyloric activity. It is interesting to speculate that the a.o.s. of Homarus may have a similar effect (dotted arrow), thus contributing to effective feeding. However, Russell further showed that stimulation of the organ in Panulirus also increases the oesophageal rhythm, and does not inhibit it as it does for the a.o.s.

Our principal conclusions are that oesophageal peristalsis can be initiated and maintained by p.o.s. stimulation and slowed and terminated by a.o.s. stimulation. The discovery that chemosensory afference can terminate a feeding rhythm is significant in its own right. However, a study of the mechanisms whereby such control is exerted would be valuable. In Palinurus vulgaris the neurons controlling the oesophageal rhythm are amenable to an intracellular electrophysiological analysis (Moulins & Vedel 1977). It is possible that this will prove to be so for Homarus gammarus. Thus, this system of the initial and final control of oesophageal peristalsis may afford a good opportunity to study the pathways and mechanisms by which sensory input can control the expression of rhythmical behaviour.

References

Allen, E. J. 1894 Studies on the nervous system of Crustacea. II. The stomatogastric system of Astacus and Homarus. Q. Jl microsc. Sci. 36, 483-496.


