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CONTROL OF MOUTH OPENING AND PHARYNX PROTRUSION DURING FEEDING IN THE SEA ANEMONE *CALLIACTIS PARASITICA*

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SUMMARY

1. Activity in all three known conducting systems (the nerve net, SS1, and SS2) may accompany feeding in *Calliactis*. The most marked response is an increase in pulse frequency in the SS2 (the endodermal slow conducting system) during mouth opening and pharynx protrusion.

2. Electrical stimulation of the SS2 at a frequency of one shock every 5 s elicits mouth opening and pharynx protrusion in the absence of food.

3. A rise in SS2 pulse frequency is also evoked by food extracts, some amino acids, and in particular by the tripeptide reduced glutathione, which produces a response at a concentration of 10^{-5} M.

4. Although the SS2 is an endodermal system, the receptors involved in the response to food appear to be ectodermal.

5. The epithelium that lines the pharynx conducts SS1 pulses, but there is some evidence for polarization of conduction.

INTRODUCTION

All species of sea anemone so far examined have three separate, but sometimes interacting, conducting systems (McFarlane, 1969*a*, 1973*c*); these are the nerve net and two slowly-conducting systems (the SS1 and SS2). The nerve net runs throughout the endoderm and in some ectodermal regions. The SS1 and SS2 appear to lie in the ectoderm and endoderm respectively: they may be additional nerve nets but a non-nervous nature is likely, in view of their known properties and that neuroid systems are widespread in the Hydrozoa (Mackie, 1965; Spencer, 1974). Some behavioural outputs of these multiple conducting systems are known. The nerve net responds to mechanical stimulation (Passano & Pantin, 1955) and shows pacemaker activity (McFarlane, 1974*b*). It co-ordinates fast and slow symmetrical muscle contractions, the pulse frequency determining the contraction rate and also which muscle groups contract (Pantin, 1935; Batham & Pantin, 1954; McFarlane, 1974*b*). In the pre-feeding response of *Tealia felina*, SS1 pulses recorded when dissolved food substances contact the column cause oral disk expansion, apparently by inhibiting inherent activity of the oral disk ectodermal radial muscles (McFarlane, 1970; McFarlane & Lawn, 1972) – inherent is throughout used to mean occurring in the absence of recorded nerve-net pulses. The SS1 in *Calliactis parasitica* is activated when the tentacles of an anemone attached to a glass plate contact a *Buccinum* shell, the behavioural output is detachment of the pedal disk (McFarlane, 1969*b*).

To date the SS2 has not been shown to be directly involved in any behavioural

response although it is known to be spontaneously active in half-animal preparations of *C. parasitica* (McFarlane, 1973*a*) and to inhibit inherent contractions of endodermal circular and parietal muscles (McFarlane, 1974*a*). Apart from the pre-feeding response mentioned above, we have no information concerning electrical activity accompanying feeding in sea anemones, although Pantin & Pantin (1943) did show that electrical stimulation of the tentacles of *Anemonia sulcata* leads to mouth opening. The present work is the first direct demonstration of electrical activity during feeding in *C. parasitica* and shows that the endodermal slow conducting system, the SS2, co-ordinates mouth opening and pharynx protrusion.

MATERIALS AND METHODS

Specimens of *C. parasitica*, obtained from the Marine Laboratory, Plymouth, were kept in running sea water at 9–14 °C. They were fed mollusc flesh weekly: anemones described as starved had not been fed for at least 5 days. The food used in the experiments was pieces or extracts of *Mytilus edulis*. Polyethylene suction electrodes were used for recording and stimulation (McFarlane, 1969*a*). All recordings, unless otherwise stated, were from electrodes attached to tentacles of either intact animals or half-animal preparations (McFarlane, 1973*a*). The technique used to stimulate the SS2 selectively was to give 200 ms duration shocks to tentacles – under these conditions the SS2 has a lower threshold than the nerve net or SS1 (McFarlane, 1974*a*).

RESULTS

Feeding response

The feeding response of sea anemones has been described before (Parker, 1917; Pantin & Pantin, 1943; Reimer, 1973) and only a brief summary is given here. In *C. parasitica* the following phases are seen:

1. *Tentacle and oral disk activity.* Food is held on the tentacles by discharge of nematocysts and spirocysts. Tentacles in contact with the food shorten and bend towards the mouth. Simultaneously the oral disk radial muscles contract in the same sector, raising the margin of the oral disk and bringing the mouth towards the food.

2. *Mouth opening and pharynx protrusion.* These movements often start before the food reaches the mouth. The lips of the mouth pull apart, exposing the upper part of the pharynx. The pharynx protrudes and the food is brought into contact with the pharyngeal cilia that convey it down the pharynx.

3. *Ingestion.* The food is slowly moved down the pharynx, with no direct continuity occurring between the coelenteron contents and the external environment. The mouth slowly resumes its resting appearance.

4. *Digestion.* Food is retained for 1–2 days.

5. *Egestion.* The pharynx gapes producing continuity between the coelenteron and the outside sea water. The food remnants pass out in a mucus-covered ball.

C. parasitica also shows a pre-feeding response involving oral disk expansion in the presence of dissolved food substances (McFarlane, unpublished observations), though this is not as marked as in many other species.

Electrical activity during feeding

Intact anemones, starved for 7 days, show spontaneous electrical activity consisting of low-frequency SS2 pulses and occasional bursts of nerve-net pulses. Spontaneous events in half-animal preparations have been previously described (McFarlane, 1973*a*); the situation in whole, starved animals differs mainly in that the interburst interval for nerve-net bursts may be as long as 60 min compared with 10–20 min in the half-animal preparations. In the intact animal SS2 activity usually occurs at a low frequency (mean pulse interval about 60 s) and the anemone appears expanded with tentacles outstretched. Occasionally individuals show higher frequency SS2 activity (mean pulse interval about 15 s) and appear 'limp', with tentacles bent and pointing towards the base of the column. Electrical stimulation of the SS2 produces a similar appearance in intact animals (McFarlane, 1974*a*), presumably because the SS2 inhibits endodermal muscles.

Electrical activity in all three conducting systems may be recorded when food is placed on the tentacles or on the oral disk beside the mouth. When food is placed on tentacles close to the recording electrodes there is a maintained level of complex activity, possibly associated with contraction of the electrode-bearing tentacles. Quieter recordings are obtained from electrodes well removed from the point of application of the food. Fig. 1(*b*) shows nerve-net, SS1 and SS2 pulses recorded during the early stages of a feeding response. With multichannel recordings the approximate origins of the evoked pulses can be found by comparing the patterns of pulse arrival at electrodes attached to widely-separated tentacles, following electrical stimulation in various positions. Fig. 1(*c*) shows that in this instance the observed SS2 pulses may have originated at the point of application of the food, but the SS1 pulses clearly arose elsewhere, possibly at receptor sites on the column.

Nerve-net pulses are not always seen during feeding. They probably arise from mechanical stimulation of ectodermal mechanoreceptors in the tentacles and oral disk.

SS1 pulses frequently occur early in the feeding response and occasionally while the food is being moved down the pharynx. Some pulses may arise at the point of contact with the food but others originate in the column. As in *Tealia felina* (McFarlane & Lawn, 1972), SS1 chemoreceptors are clearly present in the column, but perhaps there are also receptors on other ectodermal regions such as the tentacles, oral disk and pharynx lining. Contact of tentacles with a *Buccinum* shell will, under certain circumstances, lead to SS1 activity (McFarlane, 1969*b*), but with both duration of response and pulse frequency greater than in feeding, suggesting the presence of two different SS1 receptor types in the tentacles. The SS1 pulse interval during feeding generally exceeds 10 s and the maximum number of evoked pulses is about 10. SS1 activity co-ordinates pedal disk detachment in *C. parasitica* (McFarlane, 1969*b*), but more than 30 pulses at a frequency exceeding 1 pulse every 10 s are required to produce complete freeing of the pedal disk. Hence the anemone does not normally detach during feeding. The action of the SS1 pulses elicited during feeding may be to cause the oral disk expansion observed following ingestion.

A further pulse type, occasionally resembling the SS1 pulse, was sometimes recorded from both feeding and non-feeding anemones. This pulse appears to be

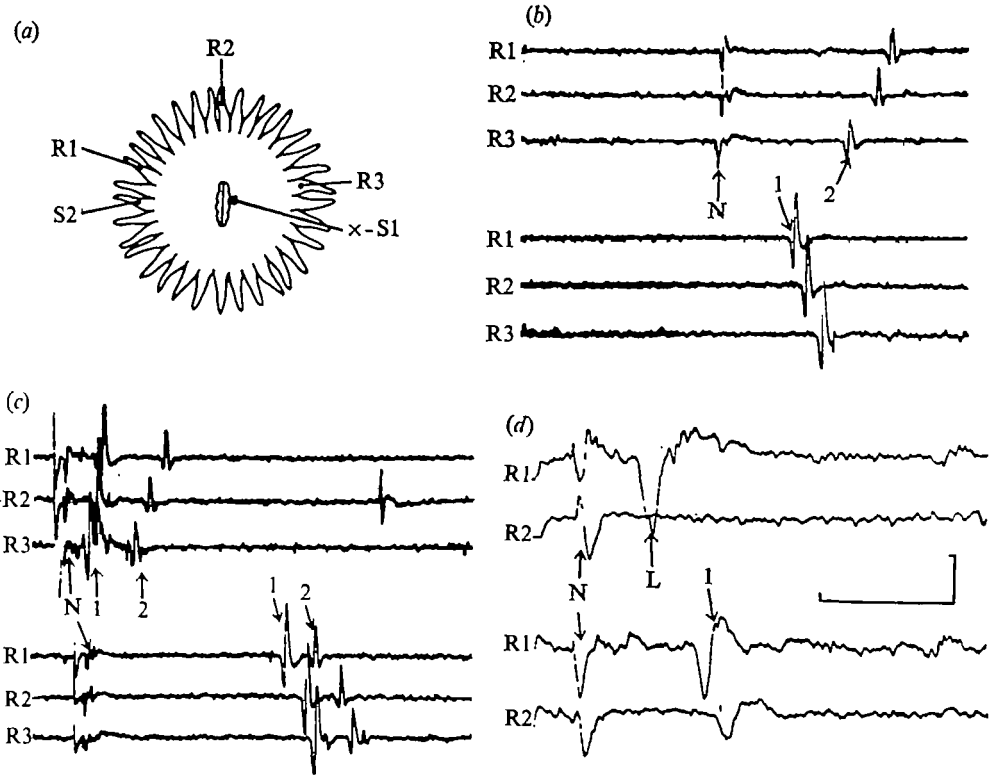

Fig. 1. Electrical activity recorded during feeding. (*a*) Arrangement of electrodes: R1, R2, R3: recording electrodes; S1, S2: stimulating electrodes. × : point of application of food. (*b*) Pulses in all three conducting systems may be recorded during feeding. This portion of a recording made during feeding shows nerve-net (N), SS1 (1), and SS2 (2) pulses from three recording electrodes attached to different tentacles, in response to food applied at ×. (*c*) Upper trace shows response to electrical stimulation at point of application of food (S1). SS2 pulses arrive at the electrodes in the same order as in feeding, suggesting that they arise as a sensory response. The SS1 pulses shown in 1 (*b*), however, clearly arose elsewhere. Lower trace shows that pulse arrival pattern of the SS1 pulse is closely reproduced by electrical stimulation at the base of the column. (*d*) Local pulse recorded from tentacle. Upper two traces show response to shock (at S2) above nerve-net threshold but below SS1 threshold. A local pulse (L) was recorded at an electrode close to the point of stimulation but not at an electrode further removed. Lower two traces show higher stimulus voltage that in this case failed to evoke the local pulse but did elicit an SS1 pulse, appearing at both recording electrodes. Time scale: *a*, *b* 500 ms, *C*, 250 ms. Amplitude scale: 10 μV.

associated with twitching of individual tentacles, a response which often spreads slowly around the tentacular crown. It is recorded only from a tentacle that twitches and not from neighbouring quiescent tentacles. The pulse sometimes follows electrical stimulation of tentacles (Fig. 1 *d*), normally at a stimulus intensity mid-way between the thresholds of the nerve net and the SS1. It differs from pulses in the known conducting system in that conduction spread is restricted, rarely extending to more than a 60° sector of the disk. Also, both pulse height and degree of spread increase as stimulus voltage is increased. The conduction velocity of the pulse is intermediate to that of the nerve net and SS1 (about 25 cm s^{-1} at 14 °C). It is presumably

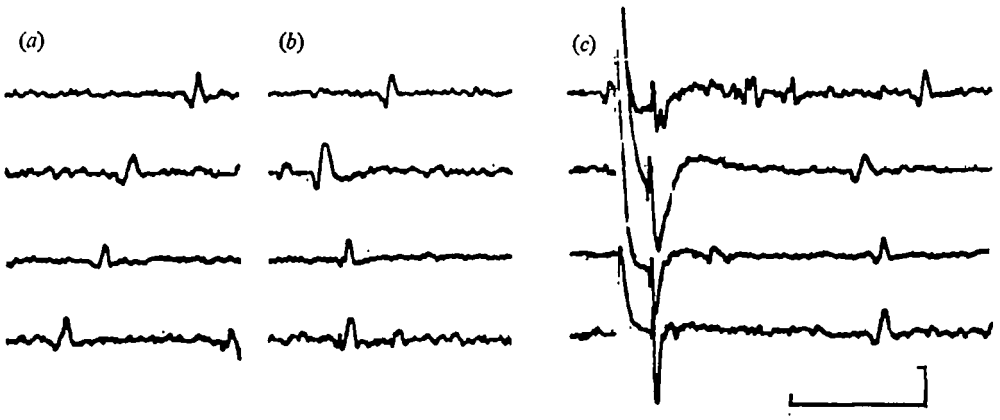

Fig. 2. Comparison of arrival times of spontaneous and food-evoked SS2 pulses at four recording electrodes attached to widely-separated tentacles. (*a*) Spontaneous SS2 pulse. 20 successive spontaneous pulses showed an identical pattern of arrival. (*b*) SS2 pulse during feeding. (*c*) Nerve-net and SS2 pulse following stimulation of upper pharynx near to point of application of food. Time scale: 500 ms. Amplitude scale: 10 μV.

potential accompanying contraction of the ectodermal longitudinal muscles of the tentacles, but the mode of propagation is not known.

The most noticeable feature of recordings made during feeding is a marked increase in SS2 activity, reaching a maximum when food contacts the mouth and during ingestion. This appears to be a sensory response, not simply an increase in the spontaneous activity of the SS2. Spontaneous SS2 pulses tend to show a fixed origin over long periods of monitoring (Fig. 2*a*), and shifts of origin, when they do occur, usually follow bursts of nerve-net activity (McFarlane, unpublished observations). Fig. 2(*b*) shows an SS2 pulse recorded from the same electrode positions as for Fig. 2(*a*) but during feeding the pulse origin is clearly different from that of the spontaneous pulse. A single shock applied at the position of the food (Fig. 2*c*) evoked an SS2 pulse that arrived at the recording electrodes in the same order as the pulse in Fig. 2(*b*), suggesting that this pulse originated at, or close to, the point of contact with the food.

Following ingestion, the SS2 pulse frequency falls but only slowly reaches its original level. In one case the mean SS2 pulses interval during a 30 min monitoring period was; 69 s prior to feeding, 33 s immediately after feeding, and 30 s 4 h later. Two days later (12 h after egestion) the mean SS2 pulse interval was 60 s. In all these experiments only small pieces of food were used; the effect of ingestion to satiation on the spontaneous SS2 pulse frequency has not been studied.

Control of mouth opening and pharynx protrusion

During mouth opening and pharynx protrusion there is a marked increase in the frequency of SS2 pulses (Fig. 3). Small pieces of food, however, may cause only slight mouth opening and pharynx protrusion with no or few associated SS2 pulses. It appears that the larger the food object the greater the increase in SS2 pulse frequency and the more this increase is prolonged.

Electrical stimulation of the SS2, in the absence of food, elicits mouth opening and

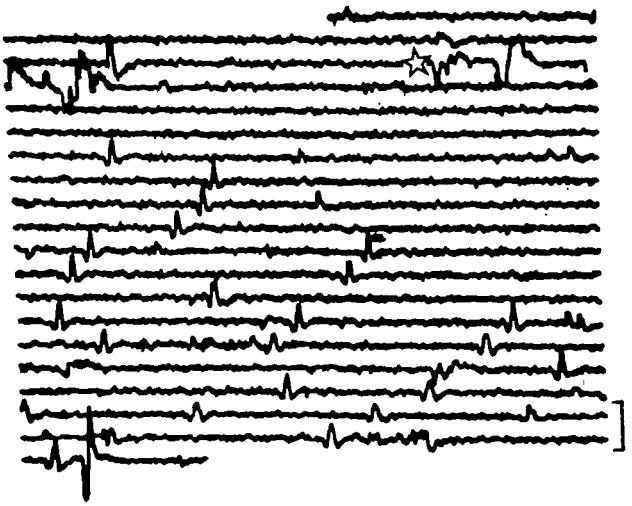

Fig. 3. SS2 pulses recorded from single electrode in response to placing food on a tentacle (at star). The 17 s delay before the SS2 pulses start was occupied largely by the movement of the food to the mouth. The record is continuous, from top left. One sweep lasts 5 s. Amplitude scale: 10 μV.

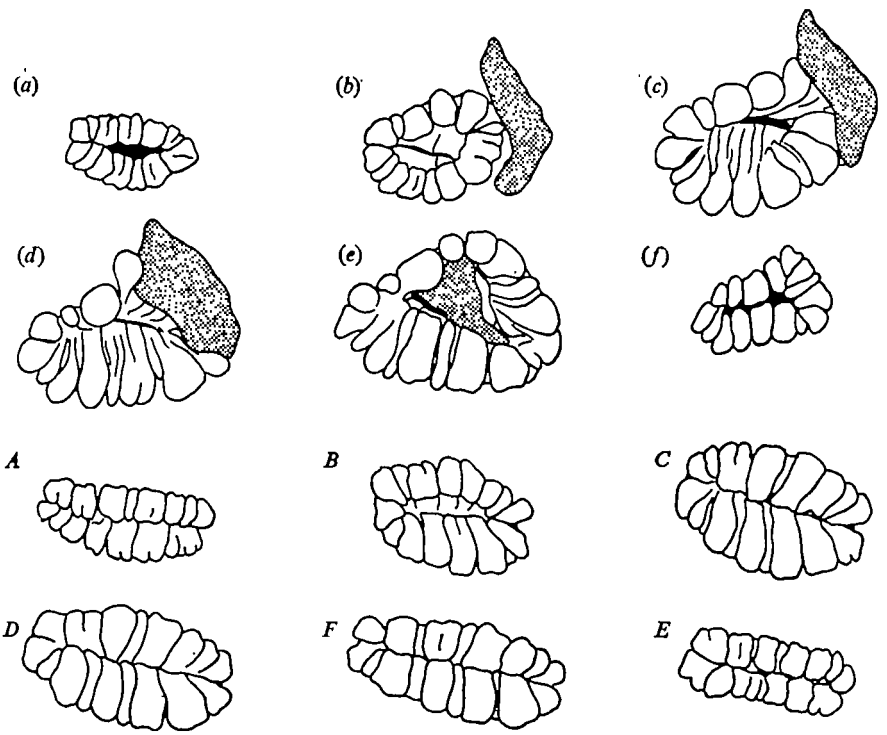

Fig. 4. Comparison of mouth opening and pharynx protrusion during response to food (*a–f*) and response to electrical stimulation of SS2 (*A–F*). Plan view of oral region drawn from photographs; feed is shaded. Time from application of food: *a*, 0 s; *b*, 10 s; *c*, 40 s; *d*, 70 s; *e*, 130 s; *f*, 190 s. Times from start of electrical stimulation: *A*, 0 s; *B*, 30 s; *C*, 60 s; *D*, 90 s; *E*, 150 s; *F*, 210 s.

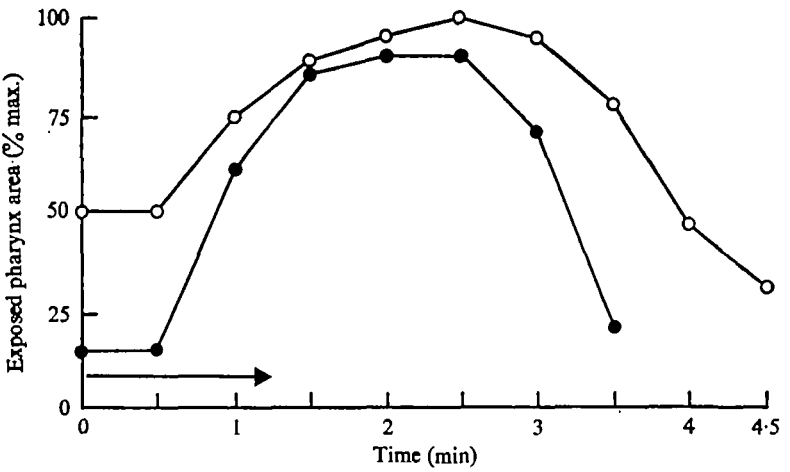

Fig. 5. Graph showing change in area of exposed pharynx with time for a normal response to food (●) and for the response to electrical stimulation of the SS2 in the absence of food (○), with SS2 pulse numbers and pulse intervals as recorded during the feeding response. Trials separated by 30 min. Arrow shows the duration of the increase in SS2 activity (here there were 12 pulses in 79 s). Measurements made from experiment shown in Fig. 4.

pharynx protrusion. Fig. 4 shows mouth and pharynx movements in a typical ingestion response. During this response 15 SS2 pulses were recorded, the mean pulse interval being 5·5 s and the minimum interval 3 s. After 30 min the SS2 was stimulated electrically with pulses, the same in number and interval as those recorded during feeding. The result of stimulation, a marked mouth opening and pharynx protrusion, is shown in Fig. 4. The main difference between the two cases appears to be that the response to electrical stimulation lacks the asymmetrical component obvious in the feeding response and which is probably due to local contraction of ectodermal radial muscles. A plot of the areas of exposed pharynx protrusion (measured from photographs) against time shows that the duration and time of onset of the response in feeding was similar to the electrically-elicited response (Fig. 5).

Mouth opening, pharynx protrusion, and the associated increase in SS2 pulse frequency are also evoked by food extracts and a number of chemical activators. These are most effective when squirted directly on to the mouth. Sea water controls produced no change in SS2 activity. 10^{-3} M solutions of arginine, serine and valine in sea water evoked mouth opening responses when squirted on to the mouth. The most dramatic responses, however, were obtained with proline and the tripeptide reduced glutathione, the latter giving a detectable effect at 10^{-5} M. 10^{-3} M glutamic acid did not elicit ingestion responses but caused wide mouth gaping, as seen during egestion. No other compounds were tested. Pantin & Pantin (1943) showed that mouth opening in *Anemonia sulcata* could be elicited by several chemicals. Reimer (1973) found that several amino acids and reduced glutathione caused pharynx responses in *C. polypus*, with proline being the most effective of the compounds tested.

The main problem arising during the course of this work is the location of the SS2 receptors, since the SS2 is endodermal (McFarlane, 1969*a*) yet the food was applied to the ectoderm. A possible method of activation is by the stimulating sub-

stances being taken into the coelenteron by the ciliary current in the siphonoglyphs, and there exciting endodermal receptors. However, direct injection of 10^{-3} M reduced glutathione or food extract into the coelenteron, via a cannula inserted through the body wall or through a tube inserted down the pharynx, failed to produce a significant alteration in SS2 pulse frequency. Application of the same solutions to the region around the mouth gave a marked increase in SS2 pulse frequency, accompanied by mouth opening and pharynx protrusion. Further support for a lack of SS2 chemoreceptors in the coelenteron comes from the observation that immediately after ingestion the SS2 pulse frequency falls back almost to its resting level. This is not due to adaptation of receptors since a fresh increase in SS2 activity can be evoked by placing food on the mouth.

There remain two possible interpretations of the observed increase in SS2 pulse frequency. Firstly, the food may excite ectodermal chemoreceptors that connect with ectodermal muscles – local contractions of the ectodermal muscles of tentacles and oral disk are obvious during feeding. These contractions will distort the underlying endodermal layer and may thus elicit SS2 pulses by mechanical stimulation. SS2 pulses were not, however, found to follow prodding of any region of *C. parasitica*. Secondly, there may be ectodermal receptors that connect directly with the SS2. It may be argued that the SS2 lies in the ectoderm of the oral disk and tentacles but this is unlikely since nerve-net and SS1 activities are abolished when a tentacle is lightly scraped, while the SS2 activity remains unaffected.

The conducting system of the pharynx lining

It is important to establish the nature of the epithelium that lines the pharynx for, if it is endoderm, some of the SS2 pulses observed during ingestion may arise from endodermal rather than ectodermal receptors. Stephenson (1928) states that the lining is usually regarded as modified ectoderm but that some evidence points to it being lined by modified endoderm. The physiological evidence given here, showing that the SS1 is present is this layer, supports an ectodermal origin. The SS1 here does, however, have some unusual properties.

The SS1 pulses recorded from the pharynx following electrical stimulation of the base of the column (Fig. 6*b*) are always smaller than SS1 pulses recorded from tentacles, but this may result from attenuation due to mucus on the pharynx. SS1 pulses are easily recorded from the upper part of the pharynx but become progressively more difficult to detect as the electrode is moved towards the base of the pharynx. Electrodes on the pharynx occasionally detect SS2 pulses, presumably recorded from the endoderm that forms the part of the pharynx lining the coelenteron. A single shock from a stimulating electrode attached to the upper 1/5th of the pharynx usually evokes an SS1 pulse that travels up the pharynx and then spreads out over the rest of the ectoderm. Stimulation of regions further down the pharynx, however, generally fails to elicit SS1 pulses, suggesting that there is polarization of conduction because SS1 pulses are recorded from the same region of the pharynx following column stimulation. One explanation for this may be that the SS1 in these parts of the pharynx has a very high threshold to stimulation. SS1 pulses often follow rather high intensity shocks but surprisingly, instead of showing the expected conduction delay of less than 1 s (based upon the rate of conduction down the pharynx), the dela

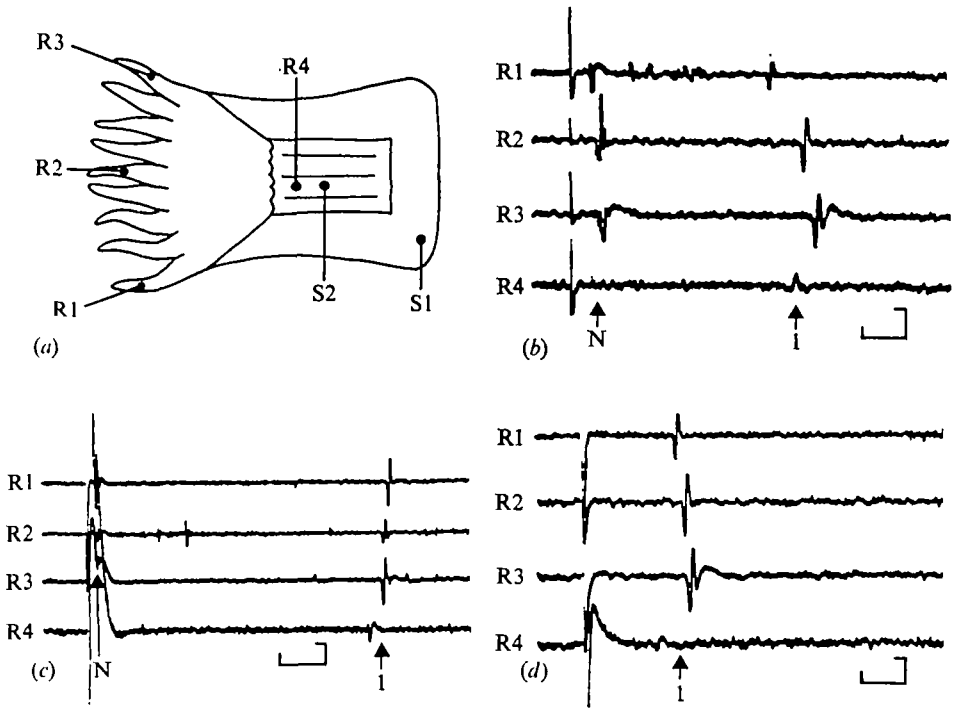

Fig. 6. SS1 conduction in the pharynx. (*a*) Half-animal preparation showing arrangement of electrodes. R1, R2, R3, R4; recording. S1, S2; stimulating. (*b*) Nerve-net (N) and SS1 (1) pulse following single shock to base of column at S1. The SS1 pulse was recorded at the electrode on the pharynx. (*c*) Response to single shock at S2. The nerve-net pulse follows with the normal delay but the SS1 pulse delay is extremely long. Note that R4, on the pharynx, also shows the delayed pulse, indicating that it arises through delayed initiation rather than an extremely low conduction rate up the pharynx. The large stimulus artefact at R4 is due to the high intensity stimulus required to elicit the delayed SS1 pulse. The nerve-net pulse can still be made out in the records from R1–R3. (*d*) Second of two shocks to S2, 1 s apart. Here an SS1 pulse follows the second shock with the 'short' delay expected for the known conduction velocity throughout the column SS1. Time scales: (*b*) (*d*) 200 ms, (*c*) 1 s. Amplitude scales: 10 μV.

between stimulus and response may be 3–8 s. This 'delayed' SS1 pulse (Fig. 6*c*) must arise as a result of delayed initiation rather than a very low rate of conduction, for the delay is large even when the recording and stimulating electrodes are closely adjacent. Here R4 and S2 were 3 mm apart. If the initiation delay is short, the conduction velocity between these electrodes must be 0·5 mm s^{-1}, compared with 8–12 cm s^{-1} for travel from R4 to the other recording electrodes. A low rate of conduction in the pharynx can be discounted because recordings made from two positions on the pharynx show that conduction between the electrodes is always 6–10 cm s^{-1}. It remains possible, however, that the SS1 pulse arises at a position remote from all electrodes: initiation must, however, still be delayed because an SS1 pulse will normally travel over the entire ectoderm within 1 s, yet delays here can be 3–8 s. At stimulation sites near the top of the pharynx, SS1 pulses are sometimes conducted normally but at other times are delayed. For example, in one position the SS1 pulse was recorded at the tentacles some 500 ms after stimulation. More generally,

however, stimulation in this position gave 'delayed' pulses; each of ten shocks at 1 min intervals being followed by SS1 pulses with a range of delays of 3·5–7·2 s (mean delay 4·75 s).

No explanation can be given as to the source of the above delay but one further feature is noted, repetitive stimulation appears to facilitate SS1 initiation (Fig. 6*d*). Although a single shock in many parts of the pharynx is not followed by an SS1 pulse, the second of two shocks about 1 s apart is often followed by an SS1 pulse initiated at the normal 'short' delay. This second shock is not followed by a nerve-net pulse because the nerve net in the pharynx has a long refractory period (McFarlane, 1973*b*).

DISCUSSION

Mechanism of mouth opening and pharynx protrusion

SS2 activity is known to inhibit inherent contractions of the body wall circular and parietal muscles (McFarlane, 1974*a*) but possible actions on other endodermal muscle groups, for example the circulars of the oral disk and pharynx, and the transverse and retractor muscles of the mesenteries, have yet to be established. Mouth opening and pharynx protrusion may result from inhibition of inherent contractions in the oral disk and pharynx circular muscles. The inherent activity of the ectodermal radial muscles of the oral disk (McFarlane & Lawn, 1972) would then pull the mouth open and the positive hydrostatic pressure in the coelenteron would cause the pharynx to protrude. The response may be restricted to the circular muscles in the mouth region because of a greater inhibitory action on the muscles concerned than on other endodermal muscles, or because the thin mesogloea at the mouth and pharynx will offer less resistance to expansion under hydrostatic pressure than the thicker mesogloea of the body wall.

Co-ordination of other components of feeding behaviour

The feeding behaviour of sea anemones contains an asymmetrical component, that is movements are directed towards the food. For example, although mouth opening and pharynx protrusion are in part symmetrical responses they often have a directional component (Fig. 4). Also, tentacular activity is restricted to tentacles either in contact with, or near to, the food. This is not readily explained in terms of the three known conducting systems, since all show unrestricted spread of activity. Neither Shelton's (1975*b*) model for restricted spread in corals, based on activity in an unrestricted-spread conducting system, nor Picken's (1969) 'pulse sorting' in a nerve net with regional differences in conduction velocity, appear to be applicable to the situation here. The available evidence (Josephson, 1966; McFarlane, 1973*b*) does not support Pantin's (1935) explanation wherein a requirement for interneural facilitation in the crown region leads to a limited spread of nervous pulses. A nerve-net pulse, elicited by a stimulating electrode attached to a tentacle, travels over the entire oral disk even when contraction is restricted to the area around the stimulating electrode. Perhaps there is another nerve net whose properties lead to local spread and whose activity is not detected or recognised using present recording methods, or alternatively the contractions may be conducted by mechanical or electrical spread through the ectodermal muscles of the oral disk and tentacles. The local potential

shown in Fig. 1(*c*) may be electrical activity accompanying local tentacle contraction but it reveals little about the mode of propagation, other than confirming lack of involvement of the three known conducting systems.

SS1 conduction in the pharynx

There is still no direct evidence as to the nature of the slow systems in sea anemones. In some respects the SS1 and SS2 share properties in common with some known or presumed non-nervous conducting systems: conduction velocity is low, pulse duration is long, and there is a marked increase in conduction delay and decrease in pulse size on repetitive stimulation (McFarlane, 1973*b*; Josephson, 1974; Spencer, 1974). The major difference between these slow systems and presumed neuroid systems in Hydrozoa is that of pulse size (Josephson, 1974), since suction electrodes record pulses measured in millivolts from hydrozoans but in only microvolts from anthozoans (see also Shelton, 1975*a*).

The apparent polarization of SS1 conduction in the pharynx could be regarded as a feature typical of nervous systems (involving one-way synapses), but although not present in any previously described neuroid system it is not a property that enables one to state definitely that the SS1 is nervous. A further major difference between the presumed neuroid systems in the Hydrozoa and the Anthozoa is that the hydrozoan systems are almost exclusively excitatory whereas the slow systems in sea anemones are inhibitory (McFarlane & Lawn, 1972; McFarlane, 1974*a*), but again we have no reason to regard inhibition as being a property restricted to nervous systems.

Origin of SS2 pulses during feeding

The available evidence points towards an ectodermal origin for the SS2 pulses seen during feeding. This suggests that this endodermal conducting system connects with ectodermal receptors. These are presumably independent of the SS1 chemoreceptors that respond to food since evoked SS1 and SS2 pulses do not show a 1:1 ratio of occurrence. The presence of ectodermal receptors connected to the SS2 is supported by work on the shell-climbing behaviour of *Stomphia coccinea* and *Calliactis parasitica* (Lawn & McFarlane; McFarlane, in preparation), where there again seems to be a sensory response in the SS2 but where one can rule out the possibility of diffusion of soluble activators to endodermal receptors.

It is of interest that the other slow conducting system, the ectodermal SS1, may have connexions with the endoderm. One result of SS1 stimulation in *C. parasitica* is extension of the column (McFarlane, 1974*a*), a response presumably brought about by contraction of endodermal circular muscles.

Two important points emerge from this and other recent studies on *Calliactis*: firstly, the known conducting systems can interact in complex ways, and secondly, as with the nerve net (Batham, 1965; Robson, 1965), both the ectodermal and endodermal slow systems may have transmesogloeal connexions with receptors and effectors or possibly even with other conducting systems.

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