

THE TRANSMISSION OF IMPULSES IN THE ECTODERMAL
SLOW CONDUCTION SYSTEM OF THE SEA ANEMONE
CALLIACTIS PARASITICA (COUCH)

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SUMMARY

1. The SS₁ fatigues in response to repetitive electrical stimulation. This fatigue is manifested by an increased conduction delay and a decreased SS₁ pulse amplitude.

2. Continued repetitive stimulation leads to the failure of the system. Recovery may take many seconds. Narrow strips of column fail more rapidly than wide strips.

3. The increased conduction delay is explained in terms of a decrease in the population of spiking cells.

4. A computer model is described and analysed. It suggests that conduction between electrically coupled ectoderm cells could be the basis for the SS₁. The SS₁ may have properties not so far experimentally demonstrated; for example, under certain conditions it could behave as a local system.

INTRODUCTION

Recent electrophysiological investigations into the control of behaviour in sea anemones (McFarlane, 1969*a, b*) have revealed the presence of two conduction systems in addition to the nerve net. These 'slow conduction' systems, the SS₁ and the SS₂ are situated in the ectoderm and endoderm respectively, though unequivocal evidence that the epithelial cells themselves are directly involved in the transmission of excitation has not yet been obtained. Neuroid systems have been positively identified in a number of Hydrozoans (Mackie, 1965; Mackie & Mackie, 1967; Mackie & Passano, 1968) and in the epithelia of developing toad tadpoles (Roberts, 1969; Roberts & Stirling, 1971). More recently, Spencer (1974) has suggested that the colonial pulse system in the Hydrozoan *Proboscidactyla flavicirrata* may also be neuroid. Mackie (1970) gives a review of known neuroid systems.

The sea anemone slow systems have been shown to have an important role in many aspects of the control of actinian behaviour. For example, the SS₁ is involved during pedal disc detachment in *Calliactis parasitica* (McFarlane, 1969*b*) and *Adamsia palliata* (Shelton & McFarlane, unpublished). In *Tealia felina* the SS₁ responds to dissolved food extract applied to the column of the anemone (McFarlane & Lawn, 1972) and is active during preparatory feeding behaviour, causing expansion of the oral disc and lowering of the oral disc margin (McFarlane, 1970). This effect is

brought about in part by the action of the SS₁ on the oral disc radial muscles (ectodermal) leading to inhibition of spontaneous contractions and an increase in length of the muscles (McFarlane & Lawn, 1972). The SS₂ can also have an inhibitory effect, in this case on certain endodermal muscles, and interacts with the nerve net in the control of circular and parietal muscle contraction cycles (McFarlane, 1974a).

A system showing many of the properties of the sea anemone slow systems has recently been identified in the Octocorallian *Pennatula phosphorea* (Shelton, 1975), and it would not be surprising to find that the majority of the Anthozoa possess similar systems. Despite the probable widespread occurrence of such control pathways in the Anthozoa and their fundamental importance to the regulation of behaviour in these animals, we know very little about the nature of 'slow conduction' or its morphological basis, although the suggestion has been made that it could be neuroid (McFarlane, 1969b).

This paper describes a series of experiments, carried out on the sea anemone *Calliactis parasitica* (Couch), designed to give some insight into the conduction of SS₁ pulses. A computer model based on the experimental evidence is described and analysed.

MATERIALS AND METHODS

Specimens of *Calliactis parasitica* were obtained from the Marine Laboratory, Plymouth and anemones with an expanded oral disc diameter of 3–5 cm were used. The animals were maintained in running sea water aquaria at 10–15 °C. Extracellular, polythene suction electrodes (after Josephson, 1965) with tip diameters of 100–300 μm were used to make recordings of electrical activity. Similar electrodes were employed to administer electrical stimuli. Recorded activity was amplified using differential pre-amplifiers and displayed on a Tektronix Type 564 storage oscilloscope.

Recordings were made from a variety of preparations – whole or half-animals and a range of strips of column of different widths. The strips were cut to include part of the pedal disc and part of the oral disc and pinned out flat (ectodermal surface uppermost) on a piece of cork submerged in the recording bath. Preparations were left overnight before use to recover from the operation. Although the largest SS₁ pulses may be recorded from the tentacles (McFarlane, 1969b) all recordings were made from the sphincter region of the column in order to ensure that the population of conducting cells was as uniform as possible. Stimulation of the SS₁ was made through a shallow flap cut in the column ectoderm. This ensured that only the SS₁ was excited by the stimuli (McFarlane, 1970) but repeat experiments stimulating the intact column showed that stimulation via a flap did not affect the rate or the manner in which the SS₁ fatigued.

RESULTS

The effect of repetitive stimulation of the SS₁ in *Calliactis* (McFarlane, 1973a) and of *Tealia felina* (McFarlane & Lawn, 1972) is an increase in the delay between the delivery of the stimulus and the arrival of the pulse at the recording electrode. This is coupled with a reduction in the amplitude of the evoked SS₁ pulse. A similar effect is observed following repetitive stimulation of the SS₂ in *Calliactis* (McFarlane, 1973a). The experiments were carried out by stimulating the intact column and recording from a tentacle and the effect was most pronounced at relatively high

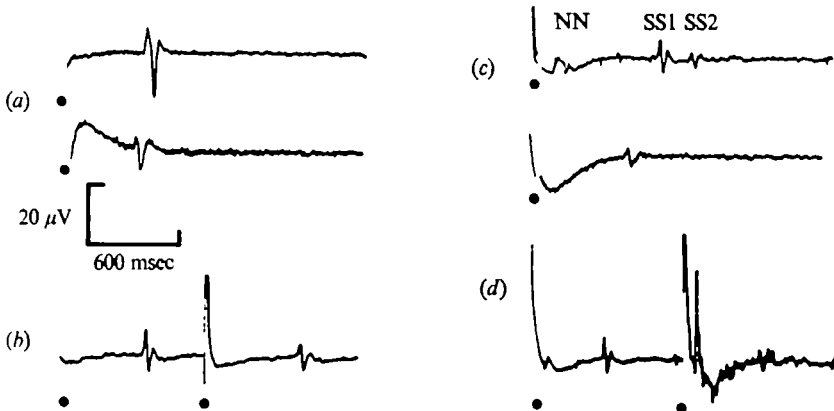


Fig. 1. Testing for activity in each of the conduction systems. Pulses evoked depend on the region stimulated; pulses recorded depend on the region stimulated *and* the recording site.

(a) Upper trace - recording from tentacle, lower trace - recording from sphincter region of the column. One electrical stimulus (●) applied to a flap cut in the ectoderm at the base of the column. Only SS 1 activity is evoked.

(b) Recording from tentacle. Effect of two flap stimuli. Note the increased delay and smaller amplitude of the second SS 1 pulse.

(c) Upper trace - recording from tentacle, lower trace - recording from sphincter. In response to a single electrical stimulus (●) to the intact column, a nerve net pulse (NN), an SS 1 pulse (SS 1) and an SS 2 pulse (SS 2) are recorded from the tentacle. Sphincter record shows nerve net and SS 1 pulses only.

(d) Recording from tentacle. Effect of two stimuli to the intact column. Facilitated muscle response to the second stimulus.

stimulus frequencies (once every three seconds). These results have been confirmed using flap stimulation and recording from the sphincter, though it should be noted that the SS 1 conducts more rapidly across the oral disc than up the column (McFarlane, 1969*b*). This may be related to the greater diameter of the oral disc ectoderm cells.

SS 1 fatigue in whole and half-animal preparations

Following repetitive electrical stimulation of an ectodermal flap, a marked increase in SS 1 pulse conduction delay occurred (Fig. 1 and Fig. 2*a*). The build up of fatigue in the system was marked by the failure of occasional SS 1 pulses to reach the recording electrode. Finally the whole system became refractory and would not conduct further SS 1 pulses until it had been allowed to recover. Full recovery took a surprisingly long time (Fig. 2*b*) (see also McFarlane, 1969*a*). At 12 °C, the effects of a single SS 1 pulse were detectable up to 40 sec afterwards. These effects were manifested by means of their delaying action on subsequent SS 1 pulses. The recovery occurred most rapidly, however, in the first 5 sec following the transmission of an SS 1 pulse. Recovery from total failure following a long train of stimuli could take several minutes. At lower temperatures, the delaying effect was even more long-lasting, and as a consequence refractoriness and failure occurred after fewer SS 1 pulses had been conducted at any given frequency of stimulation. The conduction delay of an SS 1 pulse was often doubled before the final failure of the system.

Concomitant with the reduction of conduction velocity of successive SS 1 pulses, there was also a reduction in the amplitude of recorded activity (Fig. 3*a*). Note the variability in the amplitude when the system was fatigued. Fig. 3*b* shows the

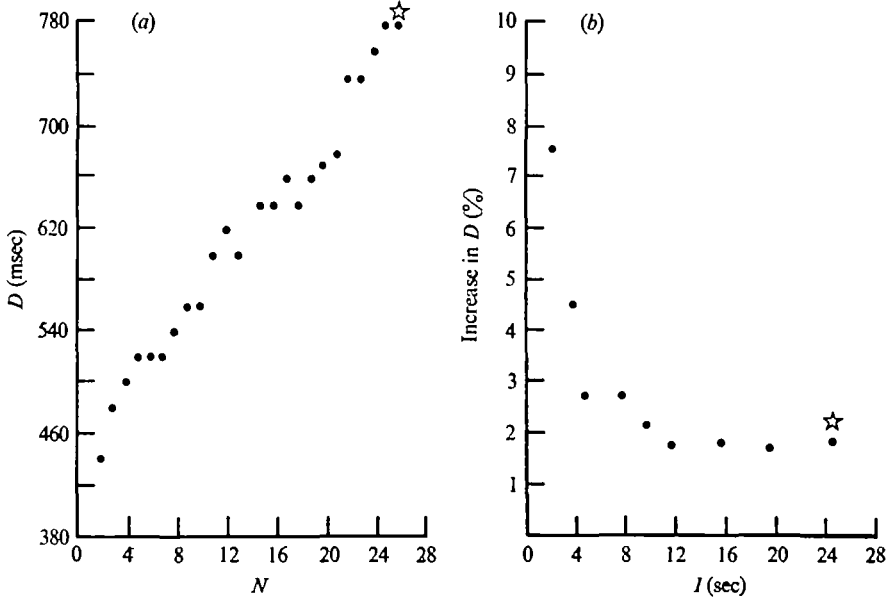


Fig. 2. Build-up of fatigue in the SS1.

(a) Graph of response delay (D) measured in msec, against stimulus number (N) for stimuli to the SS1 applied via a flap to a half-animal preparation at a frequency of 1 per 3.2 sec. In this and subsequent graphs, star shows point at which the system failed. Repeat experiments in which the intact column was stimulated showed that flap stimulation caused no significant change in the amount and build-up of fatigue in the SS1. Flap stimulation was preferable since no nerve net activity was evoked and thus no fast muscle contractions which could displace the electrodes.

(b) Percentage increase in delay of the second pulse compared with the first for paired stimuli to the SS1. I = interval (seconds) between the pairs of stimuli. This gives a measure of the 'recovery' rate of the SS1 following the transmission of a pulse.

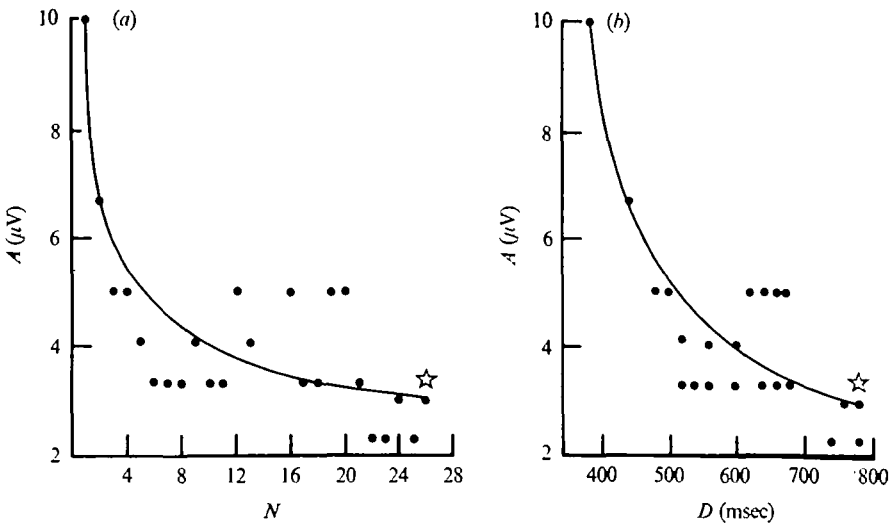


Fig. 3. Amplitude and conduction delay of SS1 pulses.

(a) Amplitude (A) in microvolts of evoked SS1 pulses following repetitive flap stimulation at 1 per 3.2 seconds. N = stimulus number.

(b) Graph of SS1 pulse amplitude versus delay (D) for repetitive stimuli as above. The lines are drawn in merely to indicate the trends of the graphs.

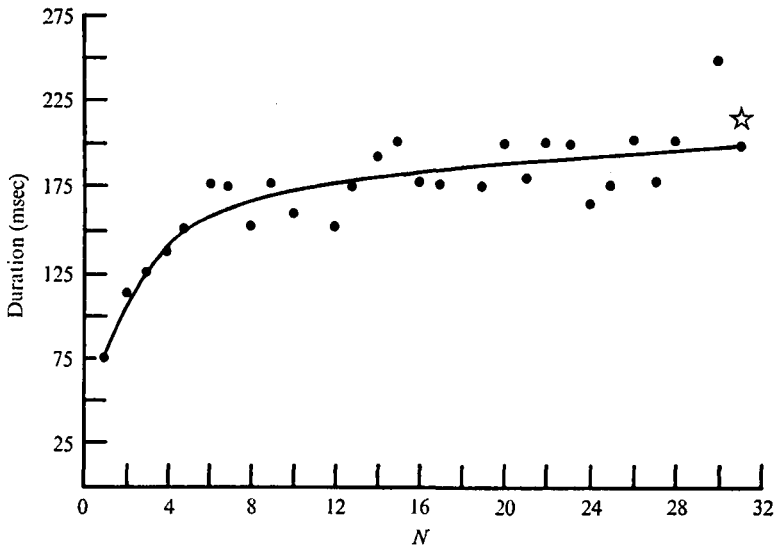


Fig. 4. Changes in duration of successive SS₁ pulses. Duration (msec) of SS₁ pulses is plotted against stimulus number (*N*). Note the initial increase in duration followed by variations as the system fatigued. Preparation as in Fig. 3.

relationship between the conduction delay and the size of the pulses recorded. With increasing fatigue, the duration of evoked SS₁ pulses became very variable (Fig. 4).

The use of two recording electrodes provided further data on the nature of fatigue in the SS₁. Fig. 5*a* shows an interesting and atypical case. In this experiment, there was a sudden jump in the conduction delay on channel 2 to a new and much higher value; after eleven pulses had conducted at this lower rate the conduction velocity returned to a value comparable to that of channel 1. This was quickly followed by total failure of the system.

Conduction of SS₁ pulses in column strips

The next series of experiments was designed to investigate the effect of the width of the conducting tissue on its ability to conduct long trains of SS₁ pulses. Every effort was made to ensure that the experimental conditions were as uniform as possible for every experiment. The temperature was maintained at 14 °C; the same electrodes were used in each case; all stimuli were applied at a frequency of 1 per 3.2 seconds; and the distance between stimulating and recording electrodes was kept as nearly uniform as possible for every preparation. Fig. 5*b* shows clearly that in a narrow strip, conduction failure occurred much more quickly than in a wider strip cut from the same animal. In this case a 33% reduction in width caused conduction failure after 27% fewer SS₁ pulses had been conducted. Note that the rate of increase in conduction delay was not greater in the narrow strip. The two curves are nearly superimposed upon one another until conduction failed in the narrow strip. Another point of interest is the upturn of the curve for delay in the final stages of fatigue. Compare Fig. 5*b* (curves for narrow strips) with Fig. 2*a* (curves for half-animal preparation).

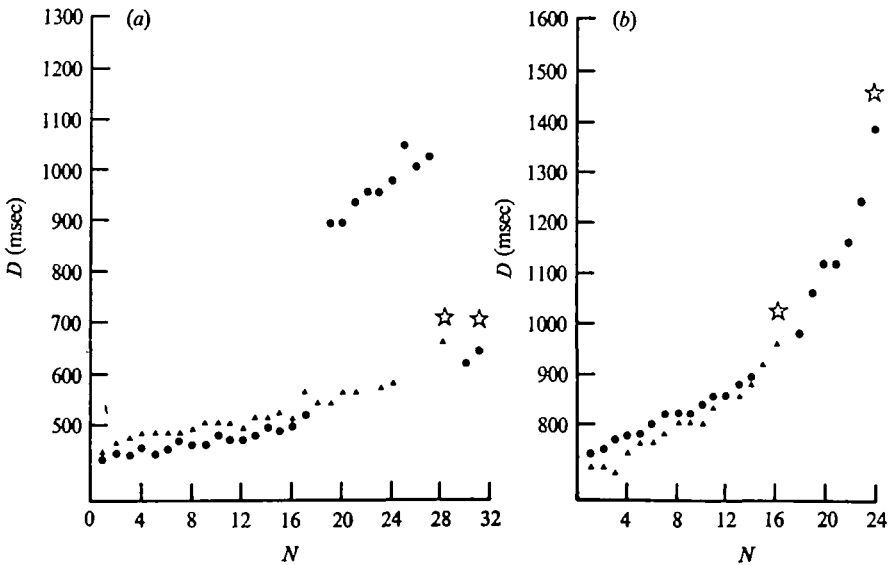


Fig. 5. (a) *Calliactis* half-animal. Data from two channel recording. Recording electrode 1 (●) 2.2 cm away from point of stimulation (a flap cut at the base of the column). Recording electrode 2 (▲) 2.5 cm away from point of stimulation. Both recording electrodes over the sphincter. Note the sudden 'jump' in the conduction delay to electrode 1. This may be explained as a local build-up of inexcitable cells necessitating conduction over a more circuitous route. Conduction delay was restored to a value comparable to that at electrode 2, 32 seconds later after the cells in that local region of fatigue had recovered a little. Total failure of the system occurred after the next three SS 1 pulses had been conducted.

(b) Fatigue in column strips. (▲) SS 1 pulses from 2.2 cm wide strip; (●) SS 1 pulses from 3 cm wide strip. Distance between stimulating and recording electrodes 3.5 cm and 4 cm respectively. Records from sphincter, stimuli via flaps in the base of the column. Conduction failure occurred in the narrower strip significantly earlier than in the wide strip, but there was not a greater increase in the rate of increment of conduction delay.

Ectodermal structure and conduction in the unfatigued SS 1

Electron microscopical evidence (McFarlane, 1969*a*; Shelton, unpublished) suggests that ectodermal cells in *Calliactis parasitica* are columnar structures up to several hundred μm long with a diameter of about 4–6 μm . Certain of the epithelial cells bear cilia and may have a sensory function. Desmosomal junctions (2–3 μm in length) join the cells together in a roughly hexagonal array to form a sheet of epithelium one cell thick. The external surface of the epithelium is covered with a border of microvilli. Half-desmosomes connect the epithelium with the collagenous matrix of the mesogloea. Membrane specializations have been observed between adjacent cells and these may be associated with the transmission of electrical excitation between ectoderm cells. Typically, these membrane specializations consist of a region (about 350 nm long) where the membranes are a fixed distance apart (about 25 nm). On each side of this, the intercellular space is occluded with electron-dense material. This may act as a seal to minimize current leakage.

In an unfatigued preparation, SS 1 pulses conduct up the column at about 6 cm sec⁻¹. During an experiment in which a half-animal was stimulated on a flap at the base of the column and a recording made 2.5 cm away over the sphincter muscle, there was a delay between stimulus and response of 440 msec.

By combining the electrophysiological and electron microscopical evidence given above, it is possible to calculate the amount of delay as an SS I pulse crosses each cell boundary, assuming that:

(a) the ectodermal cells truly represent the conducting units of the SS I;

(b) in the unfatigued SS I, conduction of excitation between the stimulating electrode and the recording electrode takes place along the shortest route;

(c) the delay in conducting across an ectodermal cell and the delay in generating the response beneath the stimulating electrode is negligible. This calculation was made and a value of 0.088–0.1 msec was obtained for the junctional delay. This compares with an average value for the delay of conduction across crayfish giant motor synapses (believed to be electrotonically coupled) of 0.12 msec (Furshpan & Potter, 1959).

DISCUSSION

This paper has provided data which enable a more detailed analysis of the process of slow conduction to be made. The small size of the ectodermal cells and the large amount of mucus secreted have so far prevented successful intracellular recording. Though even this would not be conclusive, it would be the best direct evidence for ectoderm cells as the basis for the SS I.

If the SS I is a neuroid system, the value obtained for the theoretical delay as SS I pulses conduct between ectoderm cells in an unfatigued system is consistent with there being electrical coupling between the cells. This is supported by the limited electron microscopical evidence showing possible low-resistance junctions between the cells. The alternative to a purely neuroid SS I is a system involving nervous elements in a net distinct from the well-documented nerve net described by Pantin (1935 *a, b, c, d*) and more recently by McFarlane (1969 *a, b*; 1973 *a, b, c*; 1974 *a, b*). In favour of the nervous nature of the SS I, it should be noted that there are distinct similarities between the properties of the nerve net and the slow system in certain octocorals (Shelton, 1975), but this may merely reflect similarities in the lay-out and mode of action of the two systems. Horridge (1956), using histological methods, showed that there were two nerve nets in ephyra larvae of *Aurelia*, and Passano (1965, 1973) has been able to demonstrate two different kinds of electrical activity from *Cassiopea*. As yet, however, there is no published evidence for the presence of nerve net cells in the column ectoderm of *Calliactis*, though this species has been intensively studied. The 'classical' nerve net (Pantin, 1935) is endodermal in the column. The weight of evidence so far accumulated tends to favour the idea that SS I pulses are conducted between ectoderm cells.

As a further test of the suggestion that the SS I is a neuroid system, it was decided to construct a computer model which displayed the experimentally observed properties in an attempt at gaining a greater insight into the conduction mechanism. The assumption was made that the system was made up of a flat sheet of discrete units each connected to six nearest neighbours in a hexagonal array. Increases in the conduction delay of pulses between two fixed points in the system might then be explained in one or both of two ways. Firstly, conduction might always be by the shortest route but with progressively increasing junctional delays as the system fatigued; secondly, it could be that in the fatigued system, the conduction path becomes longer and more

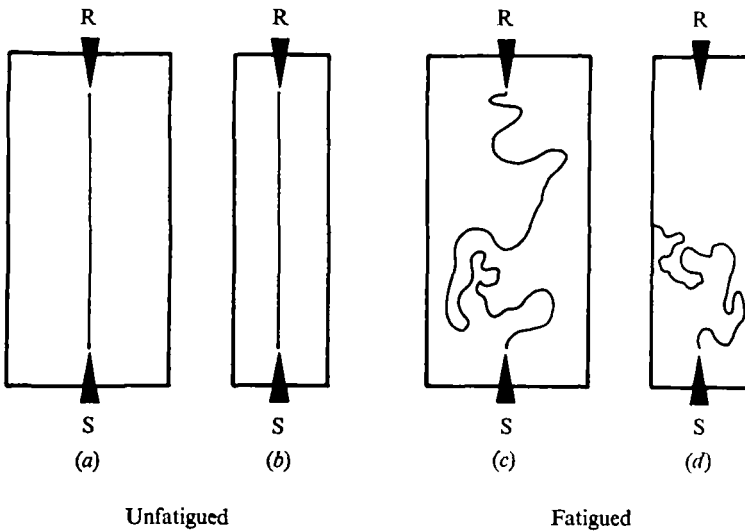


Fig. 6. Possible explanation for the premature failure of narrow column strips. S = stimulating electrode at base of column; R = recording electrode over sphincter.

(a, b) It is postulated that when the SS I is not fatigued, the fastest available conduction path between two points does not deviate markedly from a straight line. Both wide and narrow strips will conduct equally well and with similar conduction delays.

(c, d) When the SS I is fatigued, the rise in the population of non-spiking cells necessitates the transmission of excitation via more tortuous routes (one shown in each case). The chances of an SS I pulse traversing a particular distance become smaller with the narrow strip because of the increasing likelihood of the pulse being 'lost' over the edge of the strip.

tortuous and thus a pulse must cross more junctions (Shelton, unpublished thesis). The experiments with strips of column suggest that the latter explanation may be an important factor. While the system is unfatigued and conduction occurs via pathways which do not deviate markedly from straight lines, one would expect conduction delays in both wide and narrow strips to be closely similar. As fatigue built up, however, one would reach a point when the only pathways available are so winding that, with narrow strips, excitation is lost over the edge of the strip (Fig. 6).

It is postulated that there is a population of conducting units with a range of thresholds. In the unfatigued state, all the conducting units have a low threshold, but with repetitive stimulation the population of units with a higher threshold increases so that in any given area there will be a lower number of spiking cells. An unbroken chain of spiking units between any two points would thus be thinner and more tortuous than in the unfatigued state. The low density of spiking cells would further account for the observed reduction in the amplitude of the pulses recorded. Note that with a recording electrode tip of $200\ \mu\text{m}$ diameter, there will be about 1600 ectoderm cells beneath the recording electrode. The SS I pulses thus represent activity in a large number of cells and a reduction in the number of excitable cells would lead to a reduction in the amplitude of the pulse recorded.

The computer model sought to test the hypothesis outlined above. For the purposes of computation, the problem was reduced to its simplest form. An array of units (not necessarily single cells) each connected to its six nearest neighbours was set up. This corresponds to the observed packing of the ectoderm cells. A random population was

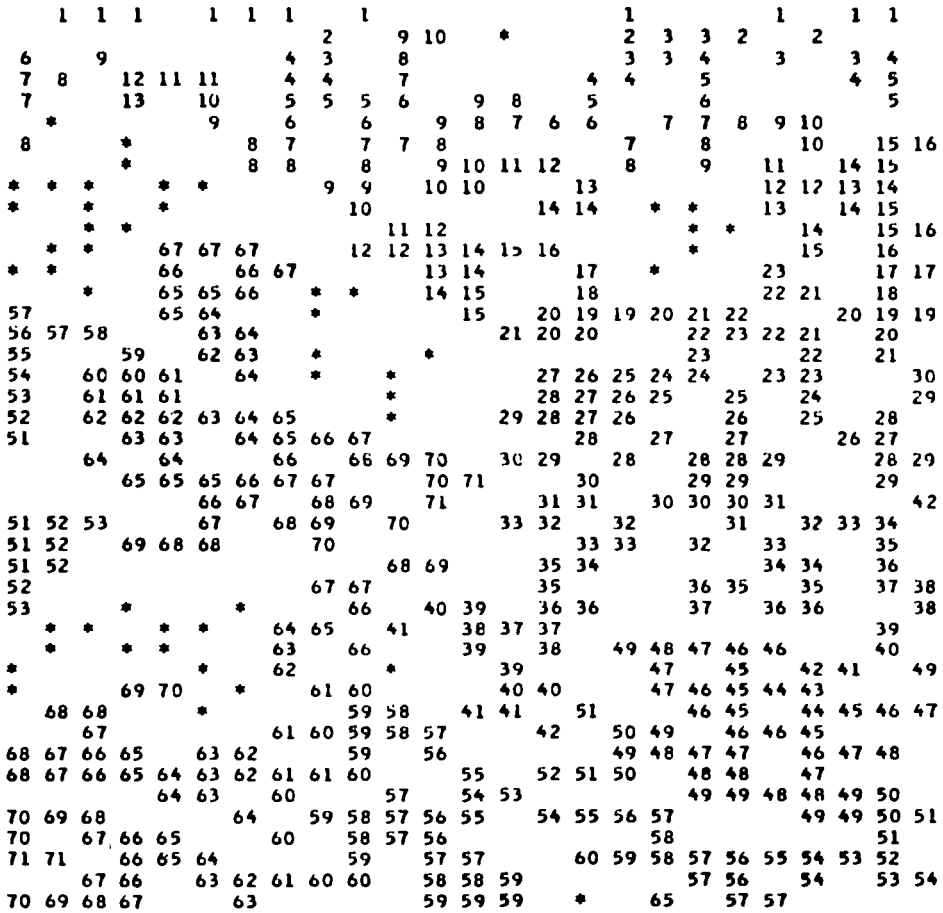


Fig. 7. A section of the print-out from the computer model. Each conducting unit was connected to six neighbouring units. In this example, a random 48% of the conducting units were given a 'low threshold' and could thus conduct spiking activity. Where spaces appear in the array, these represent units with a high threshold which cannot conduct spikes. Stars (*) represent units with a low threshold which were not directly connected to adjacent spiking units. All low threshold units along the top edge were stimulated and given the value 1. The numbers printed below represent the number of steps (a measure of time) taken to reach that point from the top edge by the shortest available route. Notice that the pathways of excitation were tortuous. Note, too, the juxtaposition of spiking units with widely different numbers. This is equivalent to SS1 pulse of long duration. The density of spiking units gave a measure of the amplitude of the SS1 pulse at that point. With higher numbers of low threshold units, pathways of excitation were more direct, the duration of the 'SS1' pulses was shorter and the amplitude greater. When the system was 'fatigued' (large number of high threshold units) the system failed to through-conduct and became a local system. This may have important consequences if this occurs in the real SS1 and is a possibility which should be investigated.

given a high threshold (inexcitable and therefore non-spiking) and the computer instructed to find the fastest pathways for the transmission of excitation through the maze of excitable and inexcitable units. The percentage of randomly 'fatigued' units could be changed to test whether a high population of such units led to a significantly larger number of steps being taken between any two points in the array. To test such a model by hand would have taken an extremely long time. The results of this opera-

tion on the model proved most interesting. There was only a very narrow range (between 50 and 60% excitable units) separating total rapid through-conduction from almost total failure to conduct with the size array which was used (80 units by 80 units). It would seem, therefore, that such a simple model is inadequate to explain the observed experimental data. Nevertheless, the model did show many of the right properties (Fig. 7). With a high proportion of inexcitable units, very long and tortuous pathways were formed and it was frequently observed that the number of steps taken to reach units close together in the array could be widely different. In other words, when the system was fatigued two things would happen:

(a) there would be a relatively small number of spiking units in any given area; (b) the duration of spiking activity in that area would be variable but longer than in the non-fatigued condition. In terms of recordable activity, this would lead to SS I pulses of small amplitude and long and variable duration. This corresponds with the observed response under such conditions. In the case of a high proportion of excitable cells, the model responded with a large number of spiking units in any given area, with all the spiking taking place over a much shorter time period, i.e. SS I pulses of larger amplitude and shorter, less variable duration. This again is confirmed by experimental data from the living animal. The experiments using strips of column of different widths suggest that, in the non-fatigued condition, pathways of excitation should be direct, and in the fatigued condition pathways of excitation should be long and winding. The model behaves like this. As it stands, however, it suffers from the fault that it will not give a smooth progression in the lengths of conduction paths in response to progressive increases in the number of excitable cells. It will only work in this manner over an extremely narrow range.

So far, only the spiking units have been considered, but it has been previously suggested in this paper that the ectodermal cells are all electrically coupled together via low-resistance junctions. If this is the case, electrotonic current flow will occur even between non-spiking cells. The amount of electrotonic spread required to depolarize a cell surrounded by high threshold cells is unknown; we have no information on the amount of current leakage which occurs between cells. Work on autonomic smooth muscle (Bennett, 1972, 1973) suggests that in order to evoke propagated spiking activity, an area of tissue (consisting of a number of cells) must be depolarized. The results reported here have shown that as the SS I fatigues, the properties of the system change from those of a sheet of conducting tissue to those of a reticular system. The model considered 'units' rather than cells and it may be that groups of cells make up each unit. Fig. 7 showed that under fatigued conditions some units with low thresholds were not spiking because there was no direct connexion to another spiking unit. Electrotonic current flow could induce spiking even though a low threshold unit was not directly coupled to another spiking unit. This would give the model the remaining property which it lacks, namely a smooth progression in the length of conducting pathways over a wider range of 'percentage of excitable units'.

Differences in threshold may be related to the time taken to repolarize following a spike. It has been shown that the effects of the transmission of an SS I pulse are still detectable many seconds after the passage of the pulse. In myoepithelial cells of the Siphonophore *Nanomia bijuga*, Spencer (1971) quotes a repolarization time of up to three seconds.

The model has been described in terms of a neuroid epithelium; it could have properties not so far experimentally demonstrated for the SS₁. For instance, under certain conditions it could show facilitation of the rate of conduction. The slow system in *Pennatula phosphorea* does exhibit this property (Shelton, 1975). It is interesting to note that when the model is 'fatigued' it behaves as a local system. This could have important consequences in the animal and may help to explain certain aspects of pedal disc detachment which is controlled by the SS₁ (McFarlane, 1969c). The model was designed to describe conduction in the SS₁; the SS₂ may well work in a similar way.

A type of fatigue similar to that reported here for the SS₁ has recently been shown in the colonial pulse system (possibly neuroid) of the Hydrozoan *Proboscoidactyla flavicirrata* (Spencer, 1974). There are also parallels in the fatigue shown by chick cardiac muscle (Lieberman *et al.* 1973).

McFarlane & Lawn (1972) have suggested that if the SS₁ is a neuroid system, the ionic changes associated with the transmission of a pulse could affect ectodermal muscles lying in close proximity to spiking ectodermal cells. Generally speaking, the changes in ion concentration associated with a spiking nerve are exceedingly small and localized, but it may be that, with a 'blanket' of depolarization such as that envisaged, changes in the molar concentrations of ions could be significant. There could also be an interaction, similar to the one they have suggested, between the SS₂ and endodermal muscles. In the oral disc of *Mimetricidium cryptum*, Batham (1965) has shown that the nerve net is ectodermal. The Hertwigs (1879, 1880) reported similar findings for *Sagartia (Calliactis) parasitica*. We know that the SS₂ can interact with the nerve net (McFarlane, 1974a, b) and it may be that the SS₁ and the nerve net are similarly linked perhaps in the region of the oral disc where the two conducting systems are in very close juxtaposition. This possibility should be investigated.

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