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SOME FACTORS IN THE ACTIVATION OF CRAB MOVEMENT RECEPTORS

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INTRODUCTION

The elastic organ spanning the propodite-dactylopodite (PD) joint in the leg of the crab *Carcinus maenas* was first described by Burke (1954) and shown by him to have mechanoreceptive function. Text-fig. 1 is a diagram of the organ *in situ* showing its relation to the surrounding structures. The nerve cells of the organ are bipolar neurons, with ovoid somata, proximal running axons, and long distal processes which Burke was able to trace clearly for distances of 200 μ from the cells. The microanatomy of the PD neurons has been studied with the electron microscope by Whitear (1960). She describes scolopidial structures, each containing the distal processes of two cells, and states that this is the only type of end-organ structure visible in the preparations. She has also examined the carpopodite-propodite (CP_2) organ of *Carcinus* and finds that the scolopidia there generally contain only one ending of normal appearance (M. Whitear, personal communication). Text-fig. 2 is a composite schematic diagram of the structure of two such cells.

By the use of single-unit recording techniques Wiersma & Boettiger (1959) have shown that two distinct classes of mechanoreceptors are present in the PD organ: one class signals the position of the joint and the other responds to the movement of the joint. Single units of the latter class respond only to movement in one direction. Wiersma (1959) has shown that in the CP_2 organ of *Carcinus* most of the movement units respond to motion in the direction that causes shortening of the elastic strand with which the sensory cells are associated; but he did not propose a mechanism for stimulation.

The movement receptors are unique not only in their unidirectional sensitivity to movement but also in their discharge pattern. A very sensitive movement receptor shows a definite threshold with respect to the speed of the stimulating movement. At threshold it discharges spikes slowly and irregularly, and as the speed is increased the discharge becomes more rapid and regular. When the speed has been increased to the point where the cell is firing at about 40/sec. the discharge becomes quite regular, but a further increase in movement speed does not elicit a smooth additional increase in firing frequency. Indeed an increase of as much as tenfold in speed does not elicit a rise of the basic 40/sec. frequency. Instead, such vigorous driving causes occasional double or triple firings; but the doublets or triplets still recur at the 40/sec. rate.

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C. A. G. Wiersma (personal communication) has suggested that the low maximum discharge rate of the receptors might be due to an oscillatory change (with a frequency of 40/sec.) in the excitability of the mechano-transducer membrane. It does not seem to be a consequence of the refractoriness of the spiking mechanism, since the frequency within a doublet or triplet may be several hundred per second.

The present work is an investigation of the mechanical aspects of the activation of movement receptors, and of the mechanism limiting their discharge frequency.

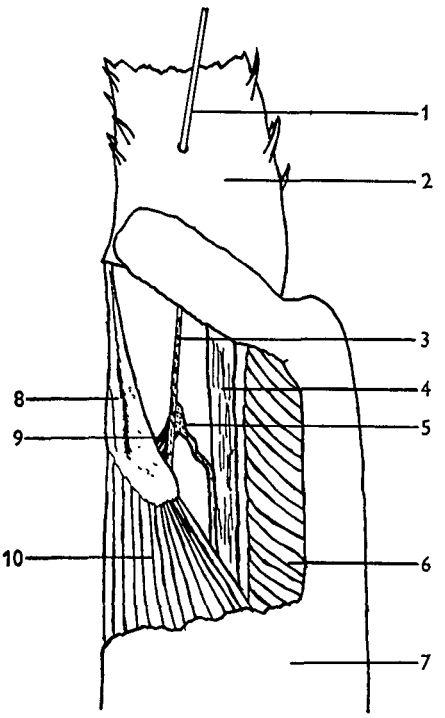


Fig. 1

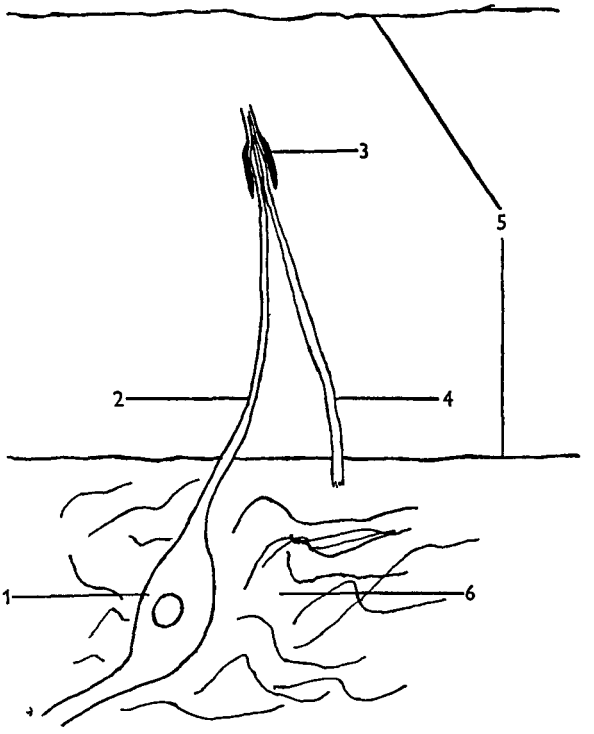


Fig. 2

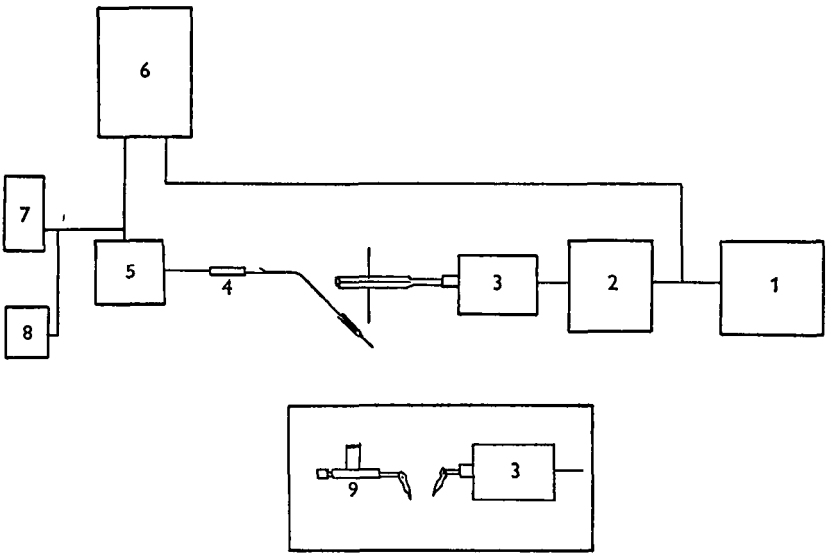
Text-fig. 1. Diagrammatic drawing of the PD organ *in situ*: 1, pin connecting transducer to dactyl (perpendicular to plane of drawing); 2, dactyl; 3, elastic strand; 4, nerve trunk from dactyl; 5, cell mass of PD organ; 6, opener muscle; 7, propodite; 8, closer tendon exposed by removal of distal fibres of closer muscle; 9, region where thin elastic fibres fan out from main elastic strand; 10, closer muscle.

Text-fig. 2. Composite diagram of the structure of a neuron of the PD organ; as reconstructed from the data of Burke (1954) and Whitear (1960); 1, cell soma; 2, distal process; 3, dense scolopidial tube; 4, other distal process entering same scolopidium; 5, borders of main elastic strand; 6, connective tissue mass binding cell bodies to strand.

MATERIALS AND METHODS

Experiments were performed on the PD organ of the shore crab *Pachygrapsus crassipes* Randall, obtained locally and renewed every 2 weeks when possible. The animals were kept in constantly running, filtered and refrigerated artificial sea water made up according to the formula of Tyler (1953) with distilled, de-ionized water. Crabs survived in this environment for as much as 2 months without overt signs of deterioration.

The arrangement for stimulation and recording is shown diagrammatically in Text-fig. 3. The stimulating system is based on an electromechanical transducer built to specification by a loudspeaker manufacturer. It is driven by a transistorized power amplifier to which appropriate waveforms are supplied by a Hewlett Packard low-frequency function generator. The response of the stimulating system extends from d.c. to several tens of cycles per second, with output excursion of over 1 cm. at the frequencies employed. The linearity of the excursion was checked photoelectrically and found to be excellent; the input waveform to the power amplifier as displayed on the lower beam of the oscilloscope is therefore a good measure of the speed of movement. A fine insect pin served to couple the transducer's motion to the dactylopodite



Text-fig. 3. Block diagram of apparatus: 1, waveform generator; 2, power amplifier; 3, transducer with arm and pin attached; 4, input probe of cathode follower; 5, cathode follower preamplifier; 6, main scope; 7, monitor scope; 8, audio system. The inset depicts the way the transducer is set up for stimulation of isolated organs: 3, transducer with spring clamp attached; 9, second spring clamp on micrometer drive mount.

of the leg at a point near the articulation. As a consequence of the large excursion of the transducer it was not necessary to employ any levers with their attendant 'play'. The recording system was entirely conventional. For recordings from axons a platinum hook electrode was employed, connected to the input of a high-gain a.c. preamplifier which drove two oscilloscopes (one for photography and one for visual monitoring) and an audio system. Responses from the PD nerve bundle and single units therein were obtained by the same method as used by Wiersma & Boettiger (1959).

For intracellular recording from PD receptor cells the organ was exposed where it lies in the propodite. The distal half of the shell of the propodite was removed on the ventral side only, leaving a bridge of shell on the dorsal side to support the articulation of the joint. The exposed fibres of the closer muscle were then carefully removed revealing the PD organ lying just ventral to the large nerve trunk emerging from the

dactylopodite. Text-fig. 1 depicts the preparation at this stage. The PD nerve bundle was separated from the large bundle, which was then removed, leaving the PD organ easily visible in transmitted light so that microelectrodes could be introduced into it. Since the cells of the PD organ must be free to move during stimulation, it was necessary to employ a microelectrode arrangement that could move with the impaled cell with a minimum of drag. For this reason an electrode of the Woodbury–Brady (1956) type was used: a 0·003 in. diameter platinum wire was inserted into the tip of a micropipette electrode filled with 3 M-KCl until it stuck in the tapered portion, the electrode was broken close to the tip, and the wire with the electrode tip affixed was slid out of the barrel of the pipette and attached to the input of a cathode follower preamplifier. This preamplifier was then connected to the oscilloscopes and audio system. The resistance of every electrode was measured and only those between 15 and 75 MΩ were used. Vibration was a severe problem since the system of elastic strand plus electrode suspension was highly compliant. It was necessary to perform these experiments in a very stable mechanical environment in order to obtain satisfactory recordings.

In order to ensure sufficiently long survival of the cells of exposed organs, Tyler's artificial sea water had to be made up with de-ionized water which had been glass-distilled. Unless this extra purification was performed all responses disappeared within 5–10 min after exposure of the organ.

To test the effect of nicotine on the movement receptors, a solution of this drug in artificial sea water was infiltrated into the region of the exposed organ with a medicine dropper drawn to a very fine tip. The responses were recorded in the meropodite. A discharge was usually noted at just the time of application of the solution but control application of artificial sea water elicited a similar discharge, which thus was judged to be a mechanical artifact due to agitation of the bathing solution. Nicotine concentrations are expressed in terms of millilitres of liquid alkaloid per millilitre of sea water and are the applied concentrations. No attempt was made to control or determine the final concentration of nicotine at the receptor.

RESULTS

Mechanical factors in the stimulation of movement receptors

In order to investigate what the mechanical conditions are which elicit responses to movement during opening and closing, a number of experiments were performed with various degrees of isolation of the elastic strand with which the receptor cells are associated. Burke (1954) had recorded from the isolated PD organ but did not comment on the movement response. The responses to passive opening and closing were first recorded and then the strand was shortened and lengthened directly using a pair of fine forceps to pull on it. This showed that the response to lengthening contained impulses from the same units that responded to closing and similarly for shortening and opening. Besides the change in the length of the elastic strand two other factors may play a part in the unidirectional response. Wiersma & Boettiger (1959) have pointed out that a group of short elastic fibrils cross the angle between main strand and closer tendon and have shown that the tension in these short fibrils is maximal when the tension in the main strand is minimal and vice versa. The

influence of these short fibrils was examined in two ways: the first was to cut these fibrils with the organ *in situ* and record the response of the organ to movement; the second was to remove the organ from the propodite and stretch and relax it directly under controlled conditions.

Cutting the fibrils produced no change in the response of PD units either to opening or closing. To achieve controlled stimulation of the organ in isolation the ends of the strand were placed in clamps, one of which was attached to the electromechanical transducer and the other to a micrometer drive. The speed and peak to peak amplitude of the stimulus were set with the electromechanical system and the average tension with the micrometer. The response was picked up from a length of the PD nerve bundle which was removed with the organ, and the micrometer was adjusted so that with no stimulus applied no position responses could be detected. According to Wiersma & Boettiger (1959) this lack of position discharge indicates that the strand is near its resting length. Despite the great mechanical stresses that must have been placed on the receptors during the mounting procedure, good responses were obtained when the strand was shortened and lengthened. An example is shown in Pl. 1, record A, of the response of such a preparation. Examination of the record shows that the movement receptors responded in a normal manner to stretch and relaxation of the elastic strand, both carried out at constant velocity. There are at least four units responding to extension of the strand and at least three others to shortening. Pl. 1, record B, shows the response of a single sensitive unit which responded to shortening of the strand. The cell fires regularly and responds only to shortening; the discharge ceases promptly when the movement reverses direction. The experiment described above leaves no doubt that the movement receptors do behave as usual if the PD organ is removed from the leg and stimulated under conditions that preclude tension changes in the short elastic fibrils.

The remaining factor that might influence the response of the movement receptors is rotational twisting of the organ about its long axis. It is possible that two groups of receptive units might have their endings running circumferentially around the strand in opposite directions so that rotation of the strand would stimulate one group or the other depending on its direction. That such rotation occurs in *Pachygrapsus* as it does in *Carcinus* (Wiersma & Boettiger, 1959) seems possible; even in isolation rotation could result from spiral orientation of the fibres in the strand. The following experiments were performed to test the effect of rotation on the responses of the movement receptors. An apparatus was constructed with a spring clamp mounted in a Teflon bearing in such a manner that the clamp could be rotated about its long axis. The distal end of the elastic strand was clamped while in its normal orientation and cut distal to the point of clamping. The end of the propodite along with the dactyl was then removed and the preparation was adjusted so that the axis of the clamp was parallel to the elastic strand and the length of the strand was changed as little as possible. The rotating clamp apparatus was mounted on a micrometer drive so that the length of the strand could also be changed to check the response of the organ to stretch and relaxation. Responses were recorded in the meropodite while the end of the organ was rotated in one direction or the other and while it was stretched and relaxed. Changes in length caused typical bursts of impulses from the movement receptors whereas rotation of the strand up to as much as two full turns gave no

response. The influence of rotation on responses to change in length was also tested. The end of the strand was rotated 360° in one direction and held there while it was stretched and relaxed, then turned back 720° and stretched and relaxed again. The responses in both these trials were identical to those obtained when the strand was not twisted. It is concluded that rotation alone will not stimulate the movement receptors nor will it affect their response to changes in the length of the strand.

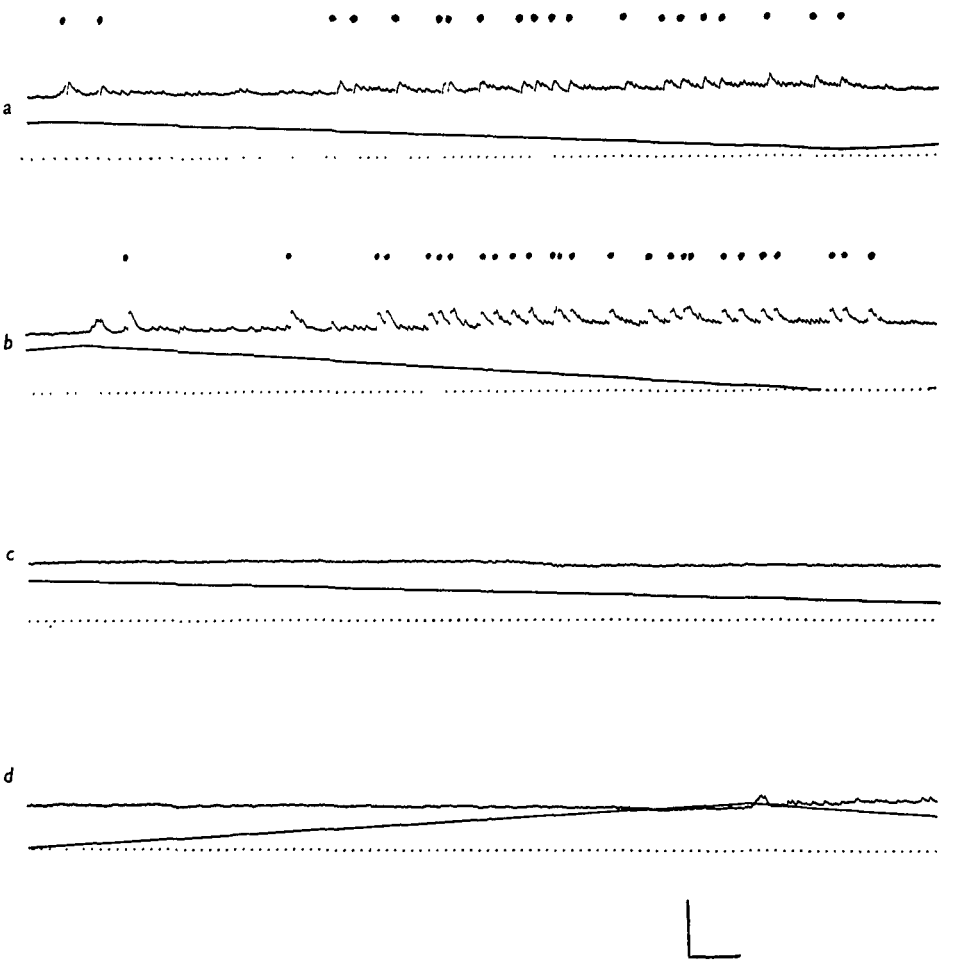
Processes responsible for the saturation effect

To investigate the possibility that some electrochemical process might limit the firing frequency of the movement receptors, intracellular recordings were made of the response to movement. Despite the use of the movable microelectrode it proved exceedingly difficult to maintain a penetration during stimulation of the organ. Of more than 150 attempts only some four gave any success. Pl. 1, records C–E, illustrate the result of one of them; the resting potential of this cell was approximately 65 mV. and the response is clearly from a movement receptor, the spike discharge occurring only during opening. The spike potentials are very small, not exceeding 10 mV. It would seem that they do not invade the cell soma into which the electrode was seen to be inserted (the satisfactory resting potential eliminates the possibility of incomplete penetration); the spikes must be blocked between the distal process and the cell soma. In all three records there is a maintained shift of the baseline potential in the depolarizing direction during opening. This might be taken to be the generator potential of the mechanotransducer membrane, but this seems unlikely for several reasons. In records D and E there is a maintained shift during closing as well as opening, yet no spikes are produced although the amplitude of the shift during closing is occasionally as great as it is at times during opening when spikes do appear. In record D the potential shift begins before the reversal of direction. In none of the records is the firing frequency a function of the magnitude of the potential shift. This maintained potential change thus fails to fulfil the criteria for a generator potential system and appears to be a recording artifact.

Text-fig. 4 is an intracellular record from another opening-sensitive movement receptor cell, taken at the same amplification as in Pl. 1, C–E, but at higher film speed. The spikes in this cell were full size, overshooting the zero potential and are thus lost in these records. Close scrutiny of the records shows that the location of each spike is indicated by a break in the trace and faint indications of the beginning and end of the spike potential making it possible to indicate where the spikes occur. This has been done with the row of dots at the top of each record. In this record the spikes take off rather abruptly, seemingly right from the baseline. This too indicates that the spikes are initiated at a distance from the soma and propagate through it. Here there is no maintained depolarization during the opening movement, which tends to confirm the conclusion that the maintained depolarizations in Pl. 1, C–E, are artifacts.

Each spike is associated with a depolarizing slow potential change of 20 msec. average total duration. The amplitudes of these slow potentials are variable (see Text-fig. 4) and although their mean amplitude in (*b*) is greater than in (*a*) it cannot be certainly stated that it is a function of movement speed since at higher speeds than that used in (*b*) it may decrease. Note that in record (*a*) the spike is complete

and has subsided to the baseline before the beginning of the slow potential, whereas in (*b*) the falling phase of the spike merges into the slow potential at a point 3–5 mV above the baseline. In records (*b*) and (*d*), taken at the same speed of movement,



Text-fig. 4. Intracellular recording from a sensitive opening unit of the PD organ. (*a*) and (*b*), Responses to opening at two different speeds; (*c*), response to movement at a speed subthreshold for spike production; (*d*), response to closing. The dots at the tops of the records indicate the positions of spikes which show only very faintly. Calibration: 20 mV., 100 msec.

there is a slow potential at the change-over from closing to opening. In each case the slow potential does not achieve the height of the other slow potentials nor does a spike occur. During opening many potentials are produced of variable, but generally low, amplitude. They last for 9–10 msec. and do summate. Nothing resembling even the smallest of these variable potentials is seen during either closing or subthreshold opening.

Effects of nicotine

In a further attempt to influence the behaviour of movement-receptor cells several drugs which affect crustacean nervous systems were applied to the PD organ; the one with the most notable effect on the movement receptors was nicotine. When applied to the PD organ in high concentration nicotine elicits a massive discharge in the PD nerve bundle. Single-unit recording showed that the frequency attained by a single nerve cell was quite high and much higher than the saturation frequency of the same cell when driven by movement. Pl. 2, record A shows the responses of two closing units of low sensitivity to movement. The shortest interspike interval exhibited by the unit with the smaller spike is 14·8 msec., corresponding to a frequency of 67 per sec.; this was the highest frequency to which this unit could be driven mechanically. Record B shows the effect of nicotine applied at a concentration 10^{-3}. The unit with the larger spike had been lost by the time of application of the drug and only the other one responded. The average interspike interval at the height of the discharge is 3·5 msec., corresponding to a frequency of 285 per sec. This fast part of the discharge lasts for almost 300 msec. Units more sensitive to movement behave in the same manner. Nicotine will elicit low-frequency spontaneous discharges in concentrations as low as 5×10^{-6}. To ensure that the nicotine was not stimulating the axons of the receptor cells directly, control experiments were performed in which the nerve was exposed to 10^{-3} and 10^{-2} nicotine without any of the drug reaching the PD organ. At these concentrations the drug never provoked a discharge from the axons nor did it affect the discharge evoked by movement.

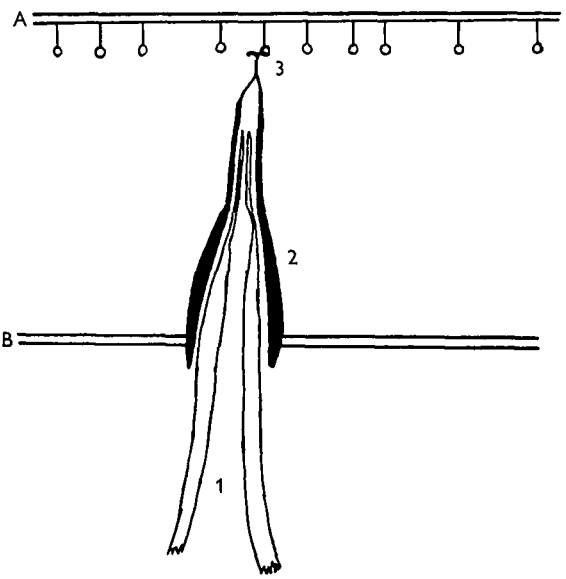
Nicotine also enhances the response of the movement-receptor cells to mechanical stimulation when applied in concentrations too low to provoke a discharge. Pl. 2, record C shows the response of a medium-sensitive closing unit during a just suprathreshold movement. The cell begins to discharge late in the course of the movement and only gives two spikes during the first closing and four during the second; this is typical behaviour for medium-sensitive units as shown in the work on *Carcinus* (Wiersma & Boettiger, 1959). After record C was made, nicotine, 10^{-6} was applied in the region of the exposed PD organ. In record D, taken 4·2 sec after this application, each closing movement produces many more spikes and the response begins earlier in the course of the movement; the effect is the same as obtained by increasing the movement speed, and is fully reversible as shown by record E, taken 6 sec. after washing out the preparation with fresh saline, completely replacing the bathing solution.

Nicotine was found never to block the effect of mechanical stimulation even in rather high concentrations.

DISCUSSION

The adequate stimulus for movement receptors is a change in length of the elastic strand of the joint organ. The most difficult problem posed by these unique units is that of response when the strand is shortened. Burke (1954) originally noted that position receptors of the PD responded to shortening of the strand but claimed that extension was much more effective in eliciting a discharge; however, Wiersma (1959) has stated that the favoured response-direction of a CP_2 organ is that in which

the strand shortens. The present work has shown beyond doubt that the opening units of the PD organ do indeed respond to shortening of the elastic strand and there is no significant difference in the response-evoking effectiveness of shortening and lengthening It may be seen in Pl. 1, record A, that four units respond to lengthening and three to shortening. In addition, the possibility that either twisting of the strand or tension on off-axis strands of elastic fibres may be responsible for the opening response has been eliminated. Whitear (1960) proposed that the two endings in a scolopidium belonged one to a closing unit and one to an opening unit and that activity in one silenced the other. In view of the correlation between anatomical and physiological data regarding the differences between PD and CP_2 organs it is reasonable that such may be the case; but some mechanism other than interaction must preserve the unidirectional response of the CP_2 units.



Text-fig. 5. Schema of the hypothetical mechanism of movement response. The structures labelled 1 are the distal processes entering a scolopidial tube 2. A and B are portions of the elastic strand that are assumed to be translated at different rates causing bending of the scolopidial tube whenever the 'catch mechanism' represented by 3 becomes bound to one of the points on A. It is not intended that the binding points on A should appear regularly arranged nor is it implied that they are spatially fixed in their locations.

Wiersma & Boettiger (1959) have shown that in *Carcinus* different parts of the PD organ are translated different distances when the organ is *in situ* and this was readily confirmed with the isolated PD organ of *P. crassipes*. If this condition applies to the two ends of a scolopidium, the latter must be bent into a curved shape during movement. Then if the distal processes lie at differing distances from the centre of curvature they will be stretched by different amounts; indeed if they lie in the plane of the curvature one will be stretched and the other shortened. Thus when the scolopidium is translated in one direction one distal process will be stretched and when it is translated in the opposite direction the other distal process will be stretched. Herein may lie the basis for unidirectional responses by single units. Whitear has

not reported on the orientation of the scolopidia and of their contained distal processes to the elastic strand, but a picture in Burke's paper shows distal processes entering the elastic strand at varying angles. Text-fig. 5 shows the essential features of this model. The elastic-tissue band labelled A is assumed to be transported at a different rate from that labelled B; bending of the scolopidium results.

It remains to characterize the mechanism that differentiates movement receptors and position receptors from each other. The foregoing hypothesis contains the germ of an explanation. If bending of a scolopidium is the adequate stimulus for movement receptors then the response will be cut off if the scolopidium is permitted to straighten out. A loose, easily ruptured mechanical coupling between the strand and the end of the scolopidium will suffice to explain movement sensitivity if it is assumed that the scolopidial structure is sufficiently rigid to straighten at the cessation of movement. The electronmicrographs (Whitear, 1960) indicate that the scolopidial tube is more dense than the surrounding material and may thus have sufficient rigidity. Such behaviour also sets up the adequate condition for response from the second ending in the scolopidium on the return movement; the scolopidium is straight at the start of the return movement and the response will begin immediately. If the same type of structural and functional entity is either more tightly coupled to the elastic strand, or of less rigid construction, then one may predict that it will respond to position rather than movement, since it will not tend to straighten out; and furthermore, structural intermediates would give the sort of intermediate position-affected movement response often encountered by Wiersma & Boettiger (1959) and Wiersma (1959). It is of prime significance here that Whitear only found one morphological type of end organ in any of the organs examined; although this datum must be discounted to the extent that staining for electronmicrography is still somewhat hit-or-miss, especially in the Crustacea.

With this reservation in mind, some predictions may be made regarding the behaviour of such a model system. The first is that a movement receptor should not produce an output whose frequency is as high as that of a position receptor. The model predicts that, for a given stress on the strand, the stress actually transmitted to the mechanoreceptive membrane of a movement receptor will be low and limited to the force necessary to rupture the coupling. Furthermore, those movement receptors that are least affected by position ought to have the lowest saturation frequencies, and intermediates should display intermediate frequency characteristics. This is indeed the case.

The high frequency displayed within doublets and triplets suggested that refractoriness of the spiking mechanism cannot be responsible for low output-frequencies in movement receptors. The results of nicotine application amply confirm that the spiking mechanism is capable of high-frequency firing. Nicotine is known to depolarize nerve-cell membranes (Goodman & Gillman, 1955) and since it has been shown that it does not evoke a discharge from (hence depolarize) the axon membrane but does add to mechanical drive, it must depolarize some portion of the distal process at a stage prior to spike initiation. The large depolarization that must be set up to produce a 285/sec. discharge does not desensitize the spike-producing locus for at least 300 msec., since that is the duration of the very fast discharge in Pl. 2, record B; therefore such an accommodative process occurring every 20 msec. or so cannot be

responsible for the 40/sec. saturation frequency. With refractoriness or accommodation of the spiking region ruled out as the basis for low frequency the only other stage beside mechanical coupling that could be responsible is the mechano-transducer mechanism *per se*. It is possible that movement receptors have a highly inefficient transducer mechanism in terms of depolarization for a given stress, but there is no way of testing this.

A second prediction is that stress would probably be applied to a movement receptor in a jerky manner. The distal tip of the scolopidium would not move through a uniform fluid but through a complexly structured medium. During the course of a movement the force on the ending would repeatedly build up to the point necessary to break the coupling of scolopidium to strand, whereupon the scolopidium would tend to straighten but then be caught up by a new set of linkages which would in turn be broken. One would expect this sort of process to produce an intermittent generator potential in contrast to the expectation of a reasonably smooth generator potential from a position receptor. A few intracellular recordings have been obtained from PD position receptors; they show fairly smooth generator potentials correlated with the position of the joint and the frequency of spike discharge; but not one of the recordings from movement receptors showed such generator potentials. Instead, the only potential changes, other than spikes, that are clearly correlated with movement in the appropriate direction are of an intermittent nature. In Text-fig. 4 it is clear that rapid, variable amplitude potentials arise only during supra-threshold movement. It is conceivable that these small potentials are aborted spikes produced in the distal process but the variations in their amplitudes and the fact that they are capable of summation argue strongly against such a conclusion.

The large, positive-going potentials which are associated with the action potentials appear at first glance to be afterpotentials produced by the spiking process. This view encounters some difficulty since the temporal relation of spike to slow potential change undergoes a shift from record (*a*) to record (*b*), both from the same cell and taken only a few minutes apart. It does not seem likely that a single event occurring in the membrane under the electrode would show such behaviour. Clearly the spike does occur in the soma membrane, so perhaps the slower potential change occurs elsewhere and spreads into the soma. It has already been pointed out that the action potential seems to originate in the distal process and propagate through the cell soma and I would propose that the slow positive potentials also arise in the distal process and are suprathreshold generator potentials. The rate of rise of these potentials increases from record (*a*), taken first, to record (*b*), which is to be expected if in the interval some damage due to the penetration of the electrode had healed. This would tend to increase the space constant of the ending and the rate of rise of an electrotonically conducted potential. The change in temporal relation of spike to slow potential may also be explained in this framework. Hodgkin & Rushton (1946) have measured electrotonic conduction in crustacean axons and their data show that at increasing distance from the source of a potential change the onset of the change becomes clearly measurable at increasing latency. Expressed in these terms it may be said that the speed of conduction of the onset of an electrotonic potential change was in the range of 2·28 m./sec., whereas spike propagation in crustacean nerve generally proceeds at 5–9 m./sec. Assuming that both the spike and the generator

potential must reach the soma from a distance of at least 200μ at velocities that differ by from 2:1 to 4:1, one is at liberty to conclude that the slow positive potentials are indeed generator potentials; and that the increase of space constant that changed their rate of rise also caused their apparent earlier arrival. When these potentials are longer than usual two spikes occur as may be seen once in record (*a*) and twice in record (*b*). The intermittent production of these suprathreshold generator potentials would thus also be in accord with the prediction made above.

It is worth making a point here that the evidence for spike initiation in the distal process was an unexpected dividend. Earlier work, particularly that of Case, Edwards, Gestland & Ottoson (1957) and Kuffler (1958) had shown that the action potential of crustacean stretch receptors is usually initiated in the axon or the proximal part of the soma; and this finding has led to an implicit belief that bipolar primary sensory neurons of Crustacea generally operate in this manner (Bullock, 1959). This would pose something of a problem in cells with very long distal processes such as those reported by Alexandrowicz & Whitear (1957) in certain proprioceptive organs in decapods; but the finding reported above disposes of such a difficulty. The plain fact that part of a crustacean neuron lies distal to its cell body need not indicate that it is incapable of spike initiation; the situation is comparable to that encountered in vertebrate sensory 'axons'.

Although further study of the crustacean movement receptors is required before they may be well understood, it appears that such understanding will shed considerable light on the adaptation of a general type of membrane to the many and varied requirements of sensing mechanical events. No special or exotic ionic mechanisms need be invoked to explain their behaviour, since merely placing the familiar type of mechanotransducer membrane in a specialized mechanical framework would appear to serve.

SUMMARY

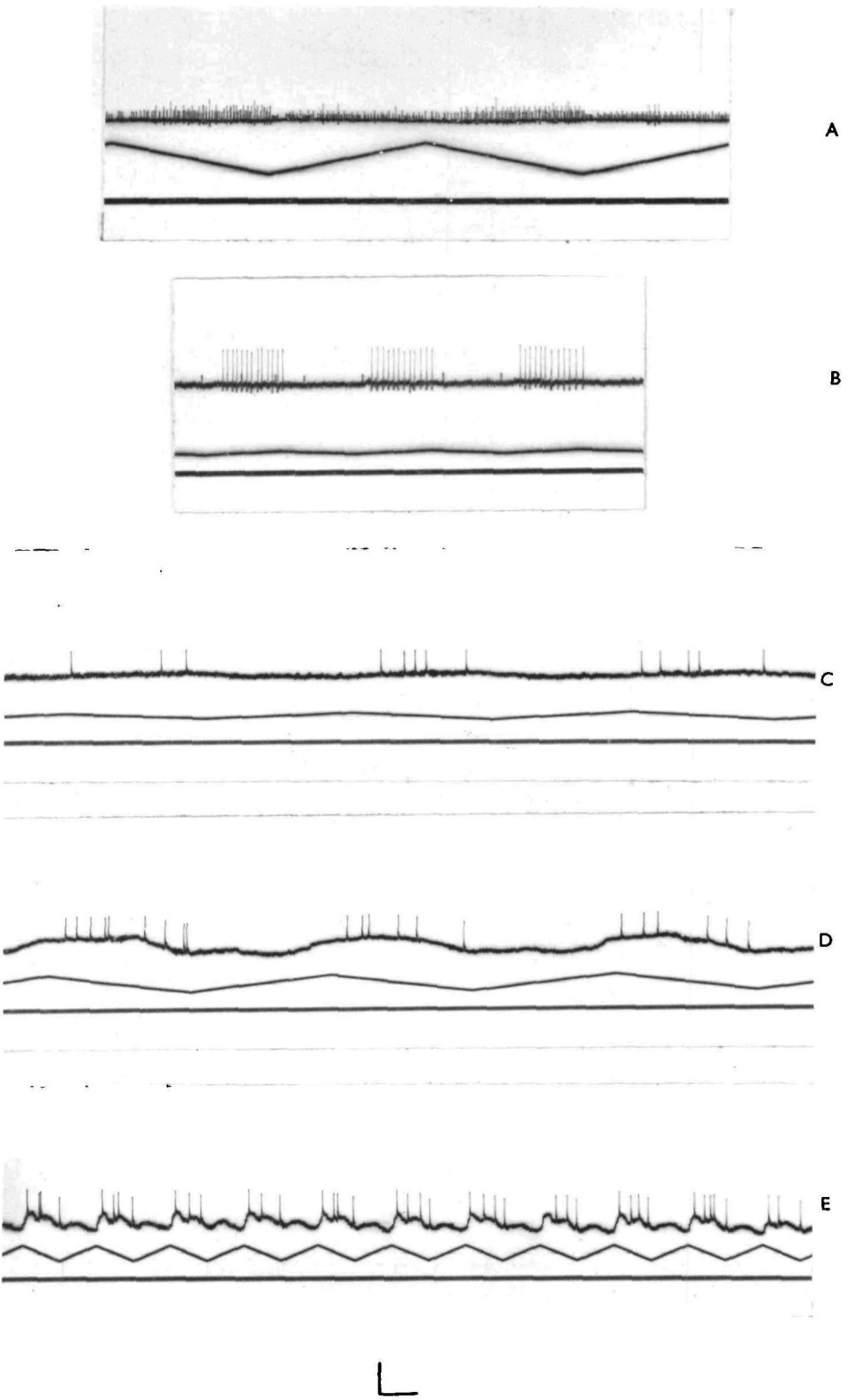
1. The activation of movement receptors in *Pachygrapsus crassipes* is examined.

2. Lengthening and shortening of the elastic strand of the propodite-dactylopodite (PD) organ are respectively the adequate stimuli for movement receptors responding uniquely either to closing or opening. These stimuli are also effective on the isolated PD organ. Twisting of the strand is without effect.

3. Intracellular records from the receptor cells show apparent intermittent generator potentials during effective stimulation. No electrical activity is observed at subthreshold speeds or during movement in the inappropriate direction. The action potentials are initiated in the distal process at a considerable distance from the soma.

4. Nicotine applied to the PD organ in high concentration elicits spike discharges of high frequency and long duration; it has no effect when applied to the axons. Applied to the PD organ in low concentration it potentiates the effect of mechanical stimulation without itself eliciting spikes.

5. These findings are discussed in relation to the structure of the receptor endings and a mechanism is tentatively suggested to account for the unidirectional sensitivity.



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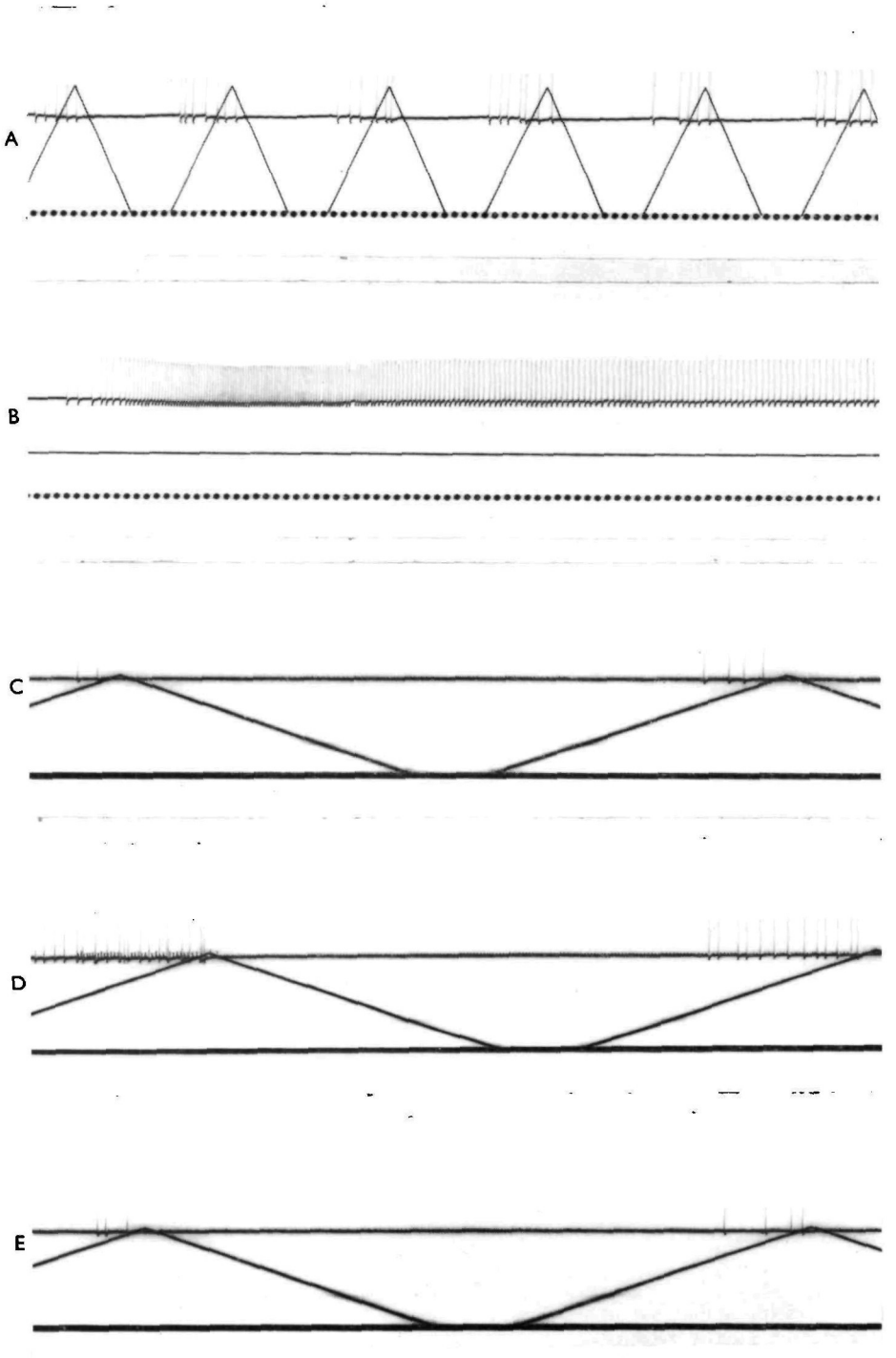

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EXPLANATION OF PLATES

PLATE 1

Records A and B. Responses of isolated PD organs to movement. (A) Bidirectional response of organ with several active units; (B) Response of a single opening-unit in the isolated organ. Records C–E. Intracellular recording from opening-unit in the PD organ. The same unit at three different speeds of movement. Calibration for records C–E: 10 mV., 200 msec.

PLATE 2

Action of nicotine on movement receptors. (A) Response of two closing-units to movement; (B) response of the unit with the smaller spike in (A) to nicotine, 10^{-8}; (C) response of another closing-unit to movement; (D) response of the unit in (C) after administration of nicotine, 10^{-6}; (E) the same unit as in (D) after the nicotine had been washed out.