J. Physiol. (1956) 133, 54–67

THE SITE OF INITIATION OF IMPULSES IN PACINIAN CORPUSCLES

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(*Received* 10 *February* 1956)

After mechanical stimulation of a Pacinian corpuscle, a sequence of potential changes can be recorded from the axon at the point where it leaves the capsule of the receptor (Gray & Sato, 1953). These potential changes can be divided into three distinct components: first the receptor potential, secondly, a component of all-or-nothing nerve impulse activity occurring peripheral to the site of recording, and thirdly another phase of all-or-nothing activity central to the recording region. Recently, Quilliam & Sato (1955) have investigated, by histological methods, the distribution of myelin on the axons from Pacinian corpuscles; they described the regular occurrence of a node of Ranvier at the point at which the axon leaves the corpuscle and another half-way between this node and the terminal (Fig. 1). Their work shows that there were two nodes of Ranvier between the recording region used by Gray & Sato and the non-myelinated terminal. The peak of the second phase of activity must therefore be attributed to the all-or-nothing activity at the node of Ranvier lying at the point where the axon leaves the corpuscle. The main purpose of the work described in this paper has been to identify the phases of all-or-nothing activity attributable to the nodes described by Quilliam & Sato and, in particular, to see whether all-or-nothing activity occurs in the non-myelinated terminal after mechanical stimulation of the receptor.

METHODS

Preparations of Pacinian corpuscles and their axons were made by the technique described by Gray & Sato (1953). The preparations were mounted in a manner similar to that used by them, but the recording length of axon was mounted, not in air, but in petroleum jelly as described by Gray & Ritchie (1954).

The stimulating and recording techniques used were also the same as those used by Gray & Sato, except that two large Ag–AgCl–NaCl agar electrodes were allowed to dip into each pool.

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One pair was used for recording and the other for passing current (Fig. 1). The constant current was obtained by connecting a calibrated potentiometer, having coarse and fine-stepped controls, through a 42 MΩ series resistor to the electrodes; dry batteries totalling from 126 to 504 V were used across the potentiometer. In most experiments the fresh preparation was examined with a polarizing microscope, and at the end of the experiment it was fixed in osmic acid and mounted in the manner described by Quilliam & Sato (1955).

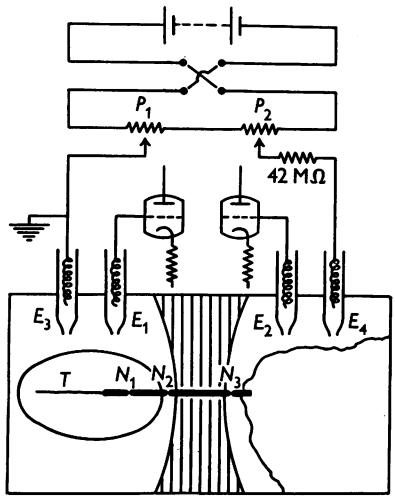

Fig. 1. Diagram of the preparation with the recording and polarizing circuits. T, 'non-myelinated' axon terminal: N_1, N_2, N_3, nodes of Ranvier; E_1, E_2, recording electrodes; E_3, E_4, electrodes for polarizing currents; P_1, P_2, coarse and fine-stepped potentiometers.

RESULTS

Effects of constant currents

Pacinian corpuscles were stimulated mechanically and records, having the general form described by Gray & Sato (1953) (see Fig. 2*a*), were obtained from electrodes E_1 and E_2 (Fig. 1); the preparations were then polarized by passing a current between electrodes E_3 and E_4 (Fig. 1). The procedure normally adopted was to record, on a single photograph, groups of about ten responses every 5 sec. Immediately after photographing each group, an event which took from 0·2 to 0·5 sec, the polarizing current was switched to the next value; at each setting of the potentiometer a record was made with both ascending and descending currents and with the current switched off. Satisfactory results were obtained from twelve preparations.

Normally the current was increased in small steps, and it was found that the response recorded without current remained constant even after the corpuscle had been exposed to currents sufficient to block one node, i.e. node 2, (Fig. 1). When, however, the currents were further increased a point was usually reached at which a subsequent response to mechanical stimulation,

in the absence of current, consisted only of a receptor potential. It seems probable that, with these large currents, the density of inward current across the membrane of the node nearest the barrier was sufficient to cause damage, resulting in an abnormally low membrane potential when the current was switched off. All-or-nothing activity often returned if the preparation was allowed to rest and could always be brought back by a small anodal polarization of the corpuscle. Another finding, consistent with the view that large currents caused damage near the barrier, was that above a certain level increases in current had little or no effect on events at the nerve terminal;

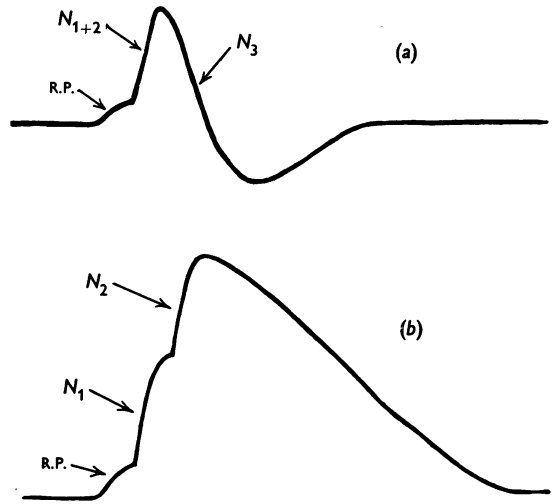

Fig. 2. Diagram of recorded potential shapes. *a*, without polarizing current; *b*, with a current of intensity sufficient to reveal all the phases. R.P., receptor potential; N_1, N_2, N_3, and N_{1+2}, phases of all-or-nothing activity—for attributions see text.

this could best be explained by assuming that the current was being short-circuited through a damaged node near the barrier. The currents used were larger than those required to have similar effects on single nerve fibres dissected from nerve bundles and mounted on an air gap, but in the Pacinian corpuscle preparations a smaller part of the applied current passed through the axon because of the greater amount of tissue left round the fibre.

Anodal polarization

Changes in impulse form. These experiments all show, in whole or in part, that certain changes occurred in the recorded potential when the corpuscle was anodally polarized. Without current the record of the potential change had the same shape as that described by Gray & Sato (1953) (Fig. 2*a*, Fig. 3, 0μA). Applying a current so that the corpuscle was positive increased the amplitude and the rate of rise of the all-or-nothing activity peripheral to the

barrier (N_{1+2} in Fig. 2*a*), while having the reverse effect central to the barrier (N_3 in Fig. 2*a*) as in cathodal polarization. This change can be seen in Fig. 3 in the frames taken with currents of +0·3, +0·6, +0·9 and +1·2μA. In the last of these frames, i.e. at +1·2μA, a clear step can be seen in the rising phase; in the next frame, that at +1·5μA, the activity above the step has

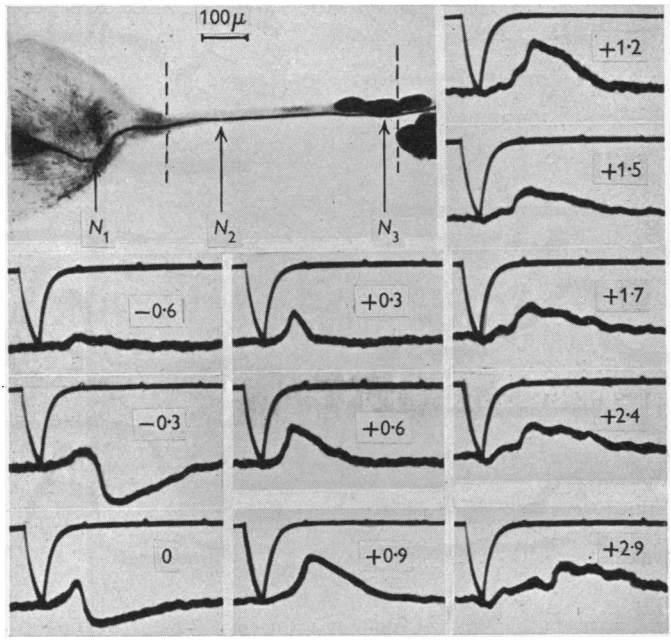

Fig. 3. Effects of polarizing currents on the recorded potentials. Top left, photomicrograph (retouched) of the preparation used to obtain the records in the rest of the figure; N_1, N_2 and N_3 are nodes of Ranvier, the dotted lines represent the maximum extent of the petroleum jelly barrier. Top beam signals time (1 msec) and indicates amplitude and time course of mechanical stimulus, which is identical in all records; bottom beam, superimposed records of potential changes—the waves in the last few frames are an artifact caused by the stimulus during the flow of large currents; the figures on each frame indicate the current strength in μA, the sign indicating the polarity of the corpuscle.

gone entirely. Fig. 4*a* illustrates a result obtained when the current was just critical; the last phase of the potential appeared only in about half of the traces. The disappearance of this phase of activity (N_2 in Fig. 2*b*) left the receptor potential (R.P. in Fig. 2) and a phase of all-or-nothing activity (N_1 in Fig. 2*b*). Changes of stimulus strength showed that the phase N_1 (Fig. 2*b*) arose in an all-or-nothing manner from the receptor potential; Fig. 4*b* is a record taken with the mechanical stimulus strength at its threshold value and shows the N_1 phase arising from the receptor potential. A further increase in the current strength raised the threshold for the appearance of the N_1

phase of all-or-nothing activity; this can be seen in Fig. 3 at current strengths +1·5, +1·7, +2·4 and +2·9μA as an increase in latency (see Gray & Malcolm, 1950), and in Fig. 5 in which an increase in current between *a* and *b* resulted in a proportion of the stimuli failing to set up an impulse; an increase of mechanical stimulus strength, as in Fig. 5*c*, was able to compensate for the effects of the polarizing current.

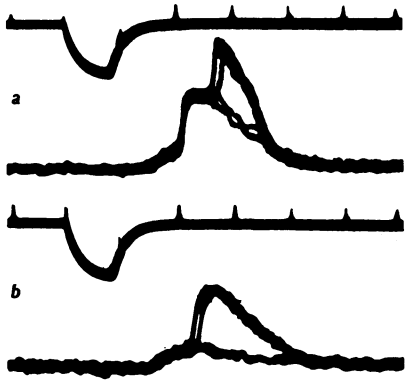

Fig. 4

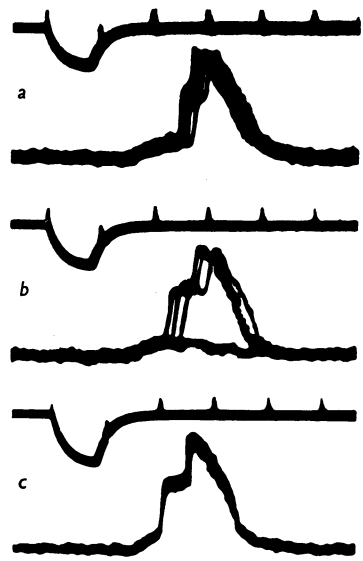

Fig. 5

Fig. 4. Records obtained under 'critical' conditions. Top beam signals time (1 msec) and indicates mechanical stimulus; bottom beam, superimposed records of the potential change. *a*, with the current strength critical for block of the N_2 phase; *b*, the N_2 phase has gone, stimulus strength critical for the N_1 phase arising from the receptor potential.

Fig. 5. Increase in threshold with anodal polarization. Top beam signals time (1 msec) and indicates mechanical stimulus; bottom beam, superimposed records of the potential change. *a*, record at given current and mechanical stimulus strength; *b*, current increased, mechanical stimulus as in *a*; *c*, mechanical stimulus increased, current as in *b*.

The point at which the N_2 phase takes off from the N_1 phase can be seen clearly in Fig. 3 at 1·2μA; even with smaller currents a discontinuity corresponding to the start of N_2 can be seen. In the original records of all experiments this discontinuity has been found, even with small currents. It was thus possible to measure the time from the beginning of the receptor potential to the beginning of phase N_1 and also the time from the beginning of phase N_1 to the beginning of phase N_2. These times are plotted in Fig. 6. It is hoped to demonstrate in this paper that there is considerable evidence for the assumption that the phase N_1 is due to the activity of node 1 (Fig. 1), and phase N_2 to the activity of node 2. If this is so then Fig. 6*a* represents a

latency curve for the excitation of node 2 by the activity of node 1 at various intensities of current; Fig. 6*b* is then the corresponding curve for the excitation of node 1 by the receptor potential.

These experiments have revealed two phases of all-or-nothing activity in addition to the receptor potential. Quilliam & Sato (1955), in an investigation of the myelination of the nerve fibres to Pacinian corpuscles, showed that a node of Ranvier occurs regularly near the point at which the nerve fibre leaves the corpuscle and another node occurs inside the corpuscle halfway between this point and the beginning of the terminal segment. These are illustrated in Fig. 1 and in Fig. 3, which includes a photograph of the

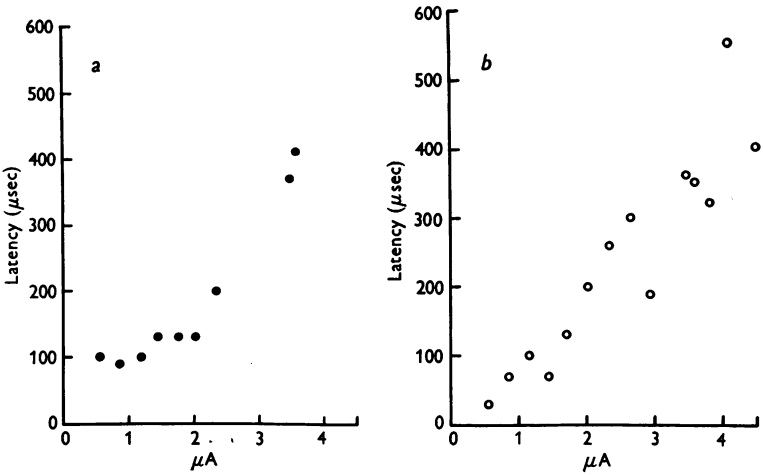

Fig. 6. Increase in latency of activation with increasing anodal polarization. Abscissa: current in μA, corpuscle positive. Ordinate: *a*, time (μsec) from beginning of N_1 phase to beginning of N_2 phase; *b*, time (μsec) from beginning of receptor potential to beginning of N_1 phase.

actual preparation used to provide the records illustrated in the rest of the figure; in Fig. 1 these nodes have been numbered 2 and 1 respectively. The positions of the nodes of Ranvier were determined histologically in each experiment, and their relations to the position of the barrier were noted; there was, however, always some doubt about the exact position of the barrier margins, but in every experiment the barrier was *effectively* between nodes 2 and 3. There were, however, minor variations in the relation between the nodes and the barrier, and these variations could be related to certain differences in the recorded potentials. Occasionally node 3 lay in the barrier and a small component, due to its activity, could be seen on top of the record. Sometimes node 2 was relatively far from the corpuscle (e.g. Fig. 3) and lay just inside the barrier; in these circumstances the N_2 phase of regenerative activity was relatively small while the N_1 phase and the receptor potential

were correspondingly large. In all experiments the peak of the N_2 phase of regenerative activity was clearly due to the activity of node 2, which always lay near the peripheral margin of the barrier. As the density of polarizing current leaving the fibre must diminish at each successive node away from the barrier, it is reasonable to assume that activity at the node nearest the barrier would be blocked at smaller values of total current than would the activity of the next node, in this instance node 1; it can be assumed, therefore, that the N_2 phase of regenerative activity was wholly due to the activity of node 2. The manner in which the N_2 phase disappeared was typical of that seen on blocking a single node (Tasaki, 1953).

When the polarizing current was increased to its maximum effective value, no further block or even discontinuity in the rising phase of the potential resulted. This suggests two possibilities: (1) that the whole of the N_1 phase is due to activity at node 1 and that no all-or-nothing impulse occurs in the non-myelinated terminal after mechanical stimulation; or (2) that the receptor potential does generate an all-or-nothing impulse in the non-myelinated terminal, but that there was no detectable discontinuity between this potential in the terminal and that of node 1, because the anodal polarization was inadequate. The polarizing currents increased the threshold for all-or-nothing activity of the receptor; this effect must have occurred at the site of the earliest all-or-nothing activity, *wherever that was.* A change of threshold at the terminal would almost certainly be less than that at node 1 (see discussion); thus a lower limit can be set to the maximum change of threshold at node 1 by measuring the maximum change of threshold for the N_1 phase of the potential; these measurements are described in the next section. In the discussion it will be argued that the threshold of node 1 would have been raised by an amount adequate to produce a discontinuity in the N_1 phase of the potential, if node 1 were excited by an all-or-nothing impulse in the terminal.

In one experiment the same technique was used to analyse the activity occurring central to the barrier on the arrival of an antidromically conducted impulse; by anodal polarization of the central side of the barrier it proved possible to block successively the activity of two nodes, and to produce a marked discontinuity between the activity of the third and fourth nodes.

Measurement of threshold. The threshold for all-or-nothing activity, arising in the receptor, was measured with various intensities of polarizing current, the corpuscle being anodal. The changes of threshold that could be produced were limited (see Fig. 7), probably because there was breakdown of the axon membrane at node 2 with the large currents, and for the reasons already given it was the maximum change of threshold with which we were primarily concerned.

The thresholds were measured by recording, superimposed, the responses to groups of identical stimuli. The threshold was taken as that stimulus strength at which approximately half the responses included all-or-nothing activity. The threshold was measured without polarizing current and then with different values of current; repeat measurements without current were made at frequent intervals. When the polarizing current reached levels at which damage began to appear and no all-or-nothing activity could be obtained without some anodal polarization, then, instead of measuring the threshold without current as a reference point, the threshold at some suitable small value of anodal polarization was used. At each point both the stimulus strength and the size of receptor potential were measured. The receptor potential is the immediate stimulus for the impulse, but the size of the recorded receptor potential depends on the space constant of the fibre between the terminal and the point of recording; over the greater part of the working range the space constant would have been increased by the anodal polarization and, consequently, the receptor potential would have appeared larger. The largest currents, however, appeared to cause some damage which resulted in a reduction of space constant and of the amplitude of all recorded potentials. On the other hand, stimulus strength is not linearly related to the receptor potential;

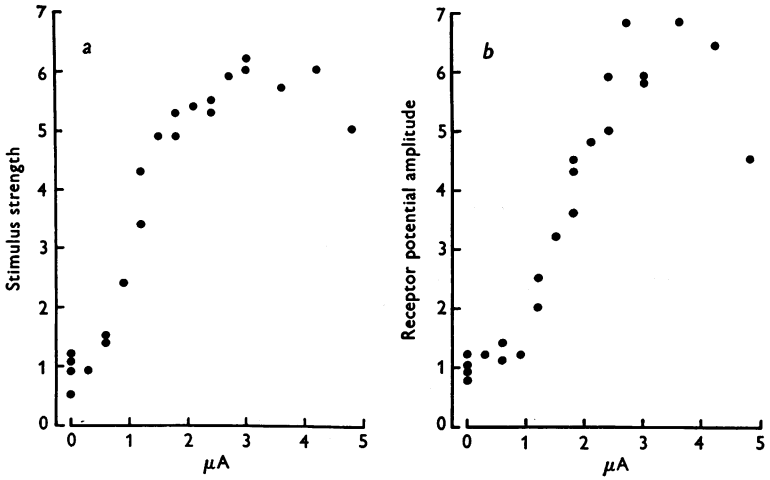

Fig. 7. Increase in threshold with increasing anodal polarization. Abscissa: current in μA, corpuscle positive. Ordinate: *a*, amplitude of threshold stimulus in multiples of threshold value in the absence of current; *b*, amplitude of receptor potential at threshold in multiples of threshold value in the absence of current.

over the working range the relation is not far from linear, but with big stimuli the departure from linearity is great (Gray & Sato, 1953). Also, anodal polarization may have caused the receptor potentials resulting from given stimuli to be greater than they would have been without the current. Thus, over most of the working range, stimulus strength measurements are likely to have underestimated the threshold change in these experiments, while measurements of receptor potential would have tended to overestimate it. Since the results obtained with both measures were similar (Fig. 7*a*, *b*) it seems improbable that the errors were large. Technically, stimulus strength measurements were more accurate and, as they probably underestimate threshold changes with increasing polarization, these are the values quoted.

Consistent results were obtained in two experiments, specially designed to make these measurements, and confirmatory results were obtained from several others; the curves in Fig. 7 are typical. The threshold was raised to

a maximum of 5 and 6 times in the two experiments. In one of these experiments the threshold with a 3μA current was compared directly with the threshold with no current; observations were made as quickly as possible with currents as follows, 0–3–0–3–0μA, and consistent results were obtained. The current increased the threshold for regenerative activity, as measured by the stimulus strength, by 7·4 times; as measured by receptor potential amplitude the ratio was 10.

Cathodal polarization

Comparatively small currents abolished all all-or-nothing activity in the preparation when the cathode was applied to the pool containing the Pacinian corpuscle (Fig. 3, 0·6μA); these currents were usually less than half those of the opposite polarity required to block node 2. With smaller currents all-or-nothing activity could be seen, but it became smaller in amplitude and rate of rise as the currents increased (Fig. 3, 0·3μA). In the penultimate stage all-or-nothing activity could only be detected as small steps arising from the receptor potential at threshold; with larger stimuli these potentials merged into a single graded response.

Effects of current on the receptor potential

Anodal polarization of the corpuscle increased the recorded amplitude of the receptor potential, and cathodal polarization decreased it. Attempts were made to follow this effect in preparations whose impulse activity had been abolished with procaine. The currents used, however, must also have altered the recording conditions, and with anodal polarization it proved difficult to prevent impulse activity, which obscured the receptor potential, from appearing. For these reasons no quantitative observations of the effects of polarizing currents on the receptor potential were made.

Effects of procaine

When 0·1% procaine was placed in the pool containing the Pacinian corpuscle the impulse gradually disappeared, in a manner similar to that found with increasing cathodal polarization; that is to say, the amplitude and rate of rise declined steadily and became more and more dependent on stimulus strength, until all signs of all-or-nothing activity ceased. The end effect of the action of procaine differed in one respect from that of cathodal polarization: appropriate doses of procaine did not affect the recorded amplitude of the receptor potential, which was always diminished by cathodal polarization. This last result was probably partly due to the facts that procaine is unlikely to have affected the space constant of the fibre, and so the recording conditions, and that diffusion of procaine into the corpuscle may be slow (cf. Gray & Sato, 1955).

It was expected that procaine placed in the peripheral pool would reach any node outside the corpuscle much more rapidly than any inside, and that it would thus be possible to distinguish the activity of node 1 from that of node 2; this was not successful, since no step such as that seen with anodal polarization appeared. Some experiments were done in which anodal polarization was used to distinguish the activity of individual nodes during the application of procaine to the corpuscle. These experiments gave uncertain results, but it seemed probable that the time interval between the onset of the observed effects of procaine at the two nodes was not long.

Size of potentials

Gray & Sato (1953) quote figures for the maximum amplitude of the receptor potential as compared with that of the all-or-nothing activity arising on either side of the recording barrier. The maximum receptor potential was always relatively large and, in the absence of histological evidence, it was assumed that the first phase of the impulse arose in the non-myelinated terminal. The demonstration by Quilliam & Sato (1955) of two nodes of Ranvier between the terminal and the recording barrier made these values of amplitude improbable; their observation also means that no satisfactory measurements of the amplitude of the receptor potential can be made with this method, since they would be dependent on the state of the intervening nodes. None the less, six experiments were performed under conditions expected to reduce some of the errors known to have been present in the earlier work; for example, artifacts were reduced and the time required for all observations was shortened as far as possible. First the record was made monophasic by procainizing the central pool, then by careful procainizing of the peripheral pool impulse activity was abolished leaving the receptor potential little altered in size (see Gray & Sato, 1953). The amplitude of the maximum receptor potential, in terms of the amplitude of the impulse on the peripheral side of the barrier, was 59% ($n^*=6$, S.D. = 14%). In terms of the impulse central to the barrier—obtained by subtracting the monophasic from the diphasic potential—the result was 38% ($n^*=5$, S.D. = 17%).

DISCUSSION

The results obtained with increasing anodal polarization of a Pacinian corpuscle show that the all-or-nothing activity arising in, or very near, the corpuscle can be divided into two distinct phases. The later, and greater, of these two phases must arise from the node of Ranvier immediately peripheral to the recording barrier. Since all preparations used in the electrical experiments have been examined histologically, it is possible to assign with confidence this phase of activity to node 2, the node Quilliam & Sato describe as

lying immediately outside the corpuscle. The other phase of all-or-nothing activity, which immediately precedes that of node 2, must arise largely from node 1, the node in the convoluted segment of the fibre.

The main question posed in this paper, however, is whether or not an all-or-nothing impulse occurs in the non-myelinated terminal after adequate mechanical stimulation. The N_1 phase could not be divided by increasing polarization, nor could a discontinuity be produced on its rising phase. There is therefore no evidence that there is all-or-nothing activity in the non-myelinated terminal; to argue that such activity does not occur, we must show that the currents used would have been sufficient to raise the threshold of node 1 enough for a discontinuity to appear on the rising phase of the N_1 potential. Measurements have shown that the threshold for all-or-nothing activity in the receptor was raised up to 7·4 times by the polarizing current; if all-or-nothing activity does occur in the terminal this figure will refer to the change of threshold in the terminal membrane. If it is now assumed that the change of threshold produced by the current at node 1 is at least as great as, and probably greater than, that produced at the non-myelinated terminal, the increase in threshold at node 1 will have been at least 7·4 times. But normally an all-or-nothing response is less than 10 times the change of membrane potential required to excite (Hodgkin, Huxley & Katz, 1949; Brock, Coombs & Eccles, 1952; Castillo & Katz, 1955), and the safety factor for one node exciting the next in frog myelinated nerve is 5 to 7 (Tasaki, 1953). Even if the attenuation of the activity of the terminal by the last internode is ignored these figures would suggest that node 1 must have been nearly blocked. In these experiments a discontinuity between the N_1 and N_2 phases has usually been clear with a polarizing current about half that required to block the N_2 phase; that is in the range when threshold was related to current in an approximately linear manner. It therefore seems that the failure of the currents used to produce a discontinuity in the rising phase of the N_1 potential is best explained by an absence of all-or-nothing activity in the non-myelinated terminal after excitation by a mechanical pulse.

It has been assumed that the change of threshold produced by the current at node 1 is not less than that produced at the terminal. Since all polarizing current must pass through the barrier, that entering the non-myelinated terminal must all pass down the last internode; the extra-axonal fluid in the pool may be regarded as having a uniform potential and it therefore follows that the potential, due to the current, across any part of the membrane of the non-myelinated terminal must be less than that across node 1. The threshold for an all-or-nothing response in the terminal may be assumed to vary with changes of membrane potential in the same way as that of the nodes of Ranvier. Therefore changes of threshold due to anodal polarization of the corpuscle must have been greater at node 1 than at the terminal.

The absence of all-or-nothing activity in the terminal, when the corpuscle is stimulated mechanically, might be due either to an inability of the terminal membrane to undergo an all-or-nothing change in any circumstances, or simply to its inability to show all-or-nothing activity in the presence of the receptor potential. It has not been possible to distinguish between these by means of antidromic impulses. These results do not exclude the possibility that there is graded activity in the non-myelinated terminal involving a specific change in sodium permeability.

The failure to distinguish distinct phases of activity attributable to each node of Ranvier when using procaine may have been due to the smallness of the interval between the action of the drug at the two nodes. There may, however, have been another factor. Gray & Sato (1953) showed that impulse activity arising peripheral to the barrier often exhibited grading of its amplitude near threshold. The amplitude of this potential must depend on the activity of node 2, and gradation of activity here must mean that there is even more grading in the activity which excites it, the impulse activity of node 1. In these experiments the grading of the impulse was usually associated with an impulse of small amplitude and rate of rise, and disappeared on anodal polarization. It seems likely that the axon terminal had a low membrane potential. This may well be due to the fact that these were preparations isolated from the body and kept at room temperature, but there is some evidence that the terminal may always have a rather low membrane potential. The threshold and rate of adaptation of the isolated and the circulated preparations are similar (Gray & Sato, 1953); the impulse near the corpuscle is also probably smaller than that further from the ending, even in preparations in the intact animal. The records obtained from Pacinian corpuscles (Gray, 1947), which show that those conducted orthodromically are different in shape from those conducted antidromically, have been analysed; the monophasic activity under each electrode has been calculated, and that calculated as occurring near the corpuscle had a slower rate of rise and smaller amplitude than that under the other electrode. A low membrane potential may then possibly exist at the terminal, even in preparations with natural circulation.

The measurements of maximum receptor potential size have given a lower figure than those found by Gray & Sato (1953). If it is assumed that the impulse peripheral to the barrier is much below full size, it is probably better to relate receptor potential size to that of the impulse central to the barrier. If this is done the mean value is 38%, with a big margin of error. The longitudinal current due to the receptor potential diminishes by an unknown factor along each successive internode; the amplitude of the record will thus depend on the number of intervening nodes and the size of the attenuation factor. This factor for myelinated nerve fibres from the frog sciatic is

about 0·5 (Tasaki, 1953). Since there are two nodes between the axon terminal and the site of recording in our preparations, a similar attenuation factor would mean that the receptor potential is about 50% greater in amplitude than the impulse at node 3. The errors in the absolute size of the impulse used as a reference, in the estimate of attenuation factor and in the measurements themselves, are all so great, however, that the results can only indicate an order of magnitude. They do suggest that Gray & Sato were correct in saying that the receptor potential was probably of the same order of magnitude as the resting and action potential, even if their reasons for saying so have proved wrong.

Since the manuscript of this paper was completed, Eyzaguirre & Kuffler (1955*a*, *b*) have published an account of the activity of a stretch receptor in the crayfish. They conclude that no all-or-nothing activity occurs in the receptor terminal with either ortho- or antidromic activation.

SUMMARY

1. Records have been obtained of the action currents flowing along the internode between the second and third nodes on the axon from a Pacinian corpuscle. Polarizing currents have been passed through the terminal parts of the axon.

2. The activity due to each node has been distinguished by means of differential blocking.

3. Evidence is presented that when a Pacinian corpuscle is mechanically stimulated no all-or-nothing impulse activity occurs in the non-myelinated terminal, which therefore appears to be concerned solely with the production of the receptor potential during orthodromic activation.

4. The absolute value of the maximum receptor potential has been considered. The evidence is inaccurate, but indicates that the receptor potential amplitude is of the same order of magnitude as the resting and action potentials.

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