RESPONSE OF THE NON-MYELINATED NERVE TERMINAL IN PACINIAN CORPUSCLES TO MECHANICAL AND ANTIDROMIC STIMULATION AND THE EFFECT OF PROCAINE, CHOLINE AND COOLING

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In Pacinian corpuscles, Gray & Satō recorded, from the axon at its emergence from the corpuscle, the graded response or the receptor potential in response to mechanical stimuli. Diamond, Gray & Satō, from the results on experiments in which anodal currents were passed through the terminal, have come to the conclusion that the receptor potential is generated at the nerve terminal and the all-or-nothing response is initiated from the first node of Ranvier lying inside the corpuscle. Later Loewenstein & Rathkamp have shown that the nerve terminal is still capable of responding to mechanical stimuli after removal of the lamellae and that, if the first node is compressed, every sign of all-or-nothing activity disappears. These results led them to believe that the impulse is initiated from the first node. On the other hand, Hunt & Takeuchi, by recording potential changes from Pacinian corpuscles, in which the outer lamellae had been removed to the inner core, have demonstrated that the non-myelinated terminal can initiate and conduct impulses. This last observation has recently been confirmed by Ozeki & Satō who recorded extracellularly potential changes to antidromic and mechanical stimuli from the non-myelinated nerve terminal in Pacinian corpuscles with a glass microelectrode; the potential changes produced by antidromic stimulation are triphasic and of all-or-nothing nature, and those produced mechanically are always preceded by a graded negativity or 'generator current' resulting from mechanical deformation of the membrane.

In the present experiment, in which the electrical response from the non-myelinated terminal of Pacinian corpuscles to mechanical or antidromic stimuli is recorded across an oil-saline interface placed close to or at the terminal, additional evidence for the impulse initiation at the terminal is given. Effects

Received for publication August 4, 1963.

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of procaine, choline chloride and cooling on the terminal and the depression of
the receptor potential by preceding impulses have also been investigated.

METHODS

Preparation. Cats were anaesthetized with sodium 5-ethyl-5 isoamylbarbiturate
(amobarbital sodium), and a piece of mesentery containing a number of Pacinian
corpuscles was removed. The axon outside a corpuscle was freed from the connect-
tive tissue, cleaned and cut about 3-4 cm from the corpuscle. The outer lamellae and
a part of the inner core surrounding the axon were removed with needles under a
binocular microscope and the axon from its entrance to the corpuscle to the central
core was cleaned. The nerve terminal, removed of nearly almost all the lamellae,
kept responding to mechanical stimuli for more than 12 hours without showing any
sign of deterioration.

Phase contrast micrographs in Fig. 1 show a normal corpuscle (A) and the nerve
terminal inside the corpuscle, in which lamellae had been removed to the central core
(B). The myelinated axon inside the corpuscle and a part of the non-myelinated
terminal are shown in C of Fig. 1. It is seen here that the axon, immediately enter-
ing the corpuscle, shows a tortuous course, is irregular in outline and has one node
of Ranvier. Inside the inner core the axon runs straight to the central core and no
nodes of Ranvier are found.

Recording. The mesentery including the corpuscle was immersed in a thin layer
of Ringer-Locke's solution (NaCl 154 mM, KCl 5.6 mM, CaCl₂ 2.16 mM, NaHCO₃ 2.40 mM)
and the latter was covered with liquid paraffin, in which the axon was suspended on
three Ag-AgCl electrodes. The electrode arrangement is shown schematically in Fig.
2. The proximal pair of electrodes (1 and 2 in Fig. 2) was used for the antidromic
stimulation of the terminal, and potential changes were recorded with both distalmost
electrode in paraffin (3) and an Ag-AgCl electrode (4) made contact with the mesen-
tery in Ringer-Locke's solution. The proximal recording electrode (3) was located at
the axon about 1 mm from the proximal part of the non-myelinated terminal. The
oil-saline interface was adjusted close to or at the proximal part of the terminal.
Potential changes across the oil-saline interface were led to a capacity-coupled am-
plifier having a time constant of 1.4 sec and were displayed on one beam of a dual-
beam oscilloscope.

In several preparations the axon adjacent to the non-myelinated terminal was
placed in saline while the terminal and proximal part of the axon were suspended in
oil. The connective tissue attached to the distal end of the terminal was mounted
on an Ag-AgCl wire, which was connected to the grid of the amplifier. The potential
changes to mechanical and antidromic stimulation of the terminal, recorded by this
method, showed nearly the same features as those by the method described above.

Stimulation. Mechanical stimuli were applied to the distal part of the non-myelini-
ated terminal with a fine glass stylus having a diameter of about 50 μ, which was
attached to one end of a Rochelle salt crystal driven by an electric pulse of 0.5-1.0
msec from a square pulse generator. The movement of the tip of the stylus, cali-
brated under a microscope, had a sensitivity of 0.4 μV.

Application of procaine and choline chloride. When procaine was used to abolish
the impulse, a drop of 0.2-0.5% procaine was added to the Ringer-Locke's fluid near
the terminal. Immediately after the impulse was abolished, the fluid was sucked
with a fine pipette and new Ringer-Locke's solution was added keeping the original
oil-saline interface practically unchanged. In using choline chloride to block the
impulse, the Ringer-Locke's fluid immersing the nerve terminal was sucked first and choline chloride, which had been prepared by replacing all NaCl by isotonic choline chloride, was added to the saline bath. The procedure was repeated two or three times until complete block of the impulse was obtained.

_Temperature._ Experiments were carried out generally at room temperatures of 18-24°C. In a few experiments the temperature of the bath was changed using ice bags to cool the metal plate, on which the oil-saline bath containing the terminal was mounted. This method enabled us to lower the bath temperature to as low as 11°C.

**RESULTS**

*Potential changes generated at the non-myelinated terminal and the first node.* Potential changes, generated in response to mechanical stimuli and recorded from the oil-saline interface placed near or at the terminal, were graded in amplitude and rate of rise, when stimulus strength was weak, while with stimuli stronger than a critical intensity a diphasic response of all-or-nothing
type was observed. A typical example is shown in Fig. 3, in which negativity of the terminal relative to the axon is shown upward. In A a mechanical stimulus of just subthreshold strength was applied to the terminal and in B that of just suprathreshold, while a mechanical stimulus of about 3 times the threshold strength was applied in C. The graded response to mechanical

![Fig. 3. Response of the nerve terminal to mechanical and antidromic stimuli. A and B, response to mechanical stimuli of just subthreshold strength and just suprathreshold, respectively; C, response to mechanical stimulus of 3 times the threshold. In A, B and C several sweeps were superimposed. In D, E and F, response to mechanical stimulus and that to antidromic stimulus followed by mechanical one are superimposed, but in F procaine had been applied to the terminal. In this and subsequent figures the upper trace indicates potential changes across an oil-saline interface, negativity of the terminal being shown upward, and the lower trace electric pulses applied to the terminal. Left-hand time scale applies to A-E, and righthand one to F. Corpuscle No. 40.](image)
stimuli shown in A and B is the receptor potential originated from the non-myelinated terminal. The all-or-nothing negative phase of the diphasic response should also be generated from the terminal since there is no possibility of the presence of a node of Ranvier distal to the oil-saline interface. The positive phase of the all-or-nothing response can be attributed to nodes of Ranvier proximal to the interface, particularly to the first node.

The diphasic potential of all-or-nothing nature was also recorded when the proximal part of the axon was stimulated electrically, as shown in Fig. 3D and E. The initial downward deflection in these figures can be attributed to the first node, while the succeeding upward deflection to the terminal.

Response of the terminal to mechanical stimuli following antidromically or mechanically produced impulse. The all-or-nothing response from the terminal can be blocked if mechanical stimuli are applied to the terminal within several msec after an antidromic impulse, leaving the graded response alone. In Fig. 3D and E the response to mechanical stimulus alone and the response to mechanical stimulus preceded by an antidromic impulse were superimposed, and in Fig. 4 the mechanical stimulus of a fixed intensity was applied to the terminal at varying intervals after an antidromic impulse. The failure of the all-or-nothing response to mechanical stimuli immediately after a preceding antidromic impulse indicates the presence of a refractory period in the terminal. The interval of the antidromic impulse and the subsequent impulse by mechanical stimulation, when the latter is critically blocked, differs according to stimulus strength; it is shorter with greater strength of mechanical stimuli. This can be seen in Fig. 5, where mechanical stimuli of four different strengths

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FIG. 5. Depression of mechanically produced response of the terminal by preceding antidromically elicited impulse. Strength of mechanical stimuli (voltage applied to the crystal) is 13.6 V for ○ and ●, 9.1 V for × and ⊗, 6.4 V for □ and ■, and 3.5 V for + and ⊕. ○, ×, □, and +, receptor potentials; ●, ⊗, ■, and ⊕, all-or-nothing responses. Ordinate, amplitude of mechanically produced response; abscissa, interval between peak of antidromic impulse and that of mechanically produced response. Corpuscle No. 30.

were applied to the terminal at varying intervals after an antidromic impulse. FIG. 5 shows two additional facts. First, transition from the impulse to the receptor potential is abrupt, when the interval between the conditioning impulse and test response becomes small, and therefore the two potentials can be separated distinctly during the refractory period. The second fact shown in Fig. 5 is that the amplitude of the receptor potential becomes smaller with decreasing interval after the antidromic impulse. Similar phenomenon was observed by DIAMOND, GRAY & INMAN 7) and was called 'depression'. The depression could also be observed, when two mechanical stimuli of suprathreshold strength were applied to the terminal in succession. The depression of the receptor potential to the second mechanical stimulus produced by a
preceding mechanically produced impulse was found to increase with an increase in the strength of the conditioning mechanical stimulus, and the depression by an impulse produced with a mechanical stimulus of just threshold strength was found to be nearly the same as that produced by an antidromic impulse.

Effect of procaine and choline chloride. After applying procaine and choline chloride to the non-myelinated terminal, the antidromic impulse becomes monophasic, indicating that impulse conduction from the first node to the terminal is blocked, whereas the response to strong mechanical stimuli is still diphasic (Fig. 6C). However, the latter finally becomes monophasic, showing the receptor potential alone (Fig. 6D). Response of the nodes lying in the oil is hardly affected by either procaine or choline, when it is tested antidromically. However, after applying procaine or choline, the amplitude of the antidromic impulse becomes larger by about 10–20% than that recorded before (compare Fig. 3E with F and Fig. 6B with C). This may result from the fact that in the untreated terminal the all-or-nothing response is generated before the first node activity reaches its maximum. However, the possibility that change in the recording position or in the resistance by application of procaine or choline

![Graphical representation of the effect of choline chloride on mechanically and antidromically produced responses of the terminal.](image-url)
may affect the potential amplitude, can not be overlooked.

After abolishment of the all-or-nothing response from the terminal by procaine or choline the antidromic impulse produces little effect on the succeeding receptor potential, as shown in Figs. 3F and 6D. This can be more

**Fig. 7.** Effect of antidromic stimulation on succeeding receptor potentials from the terminal, where the all-or-nothing response was blocked by choline chloride. Several sweeps were superimposed by changing the interval between antidromic and mechanical stimuli, the mechanical stimulus strength being constant. Corpuscle No. 34.

**Fig. 8.** Depression of mechanically produced response by preceding antidromic stimulation of the terminal. ○ (receptor potential) and ● (all-or-nothing impulse), before abolishing impulse initiation at the terminal; +, after the abolishment by choline chloride. Strength of mechanical stimulus is the same in both experiments. Abscissa, interval between peaks of antidromic and orthodromic responses; ordinate, amplitude of orthodromic responses. Corpuscle No. 34.
clearly seen in Figs. 7 and 8, where the interval between antidromic and mechanical stimuli was varied without changing the strength of mechanical stimulus.

*Time course of the receptor potential.* The receptor potential, obtained by abolishing the impulse activity by applying antidromic and mechanical stimuli successively (A+M procedure) or mechanical plus mechanical stimuli (M+M procedure), showed a rise time of about 1 msec, and a falling phase of exponential decay having a time constant of 0.9 msec in average at bath temperature of 20-21°C. Values of the time constant obtained with five preparations are shown in Table 1. The time constant measured after block of the impulse by choline chloride shows a greater value than that obtained by blocking the impulse with either A+M or M+M procedure, while the time constant measured after procanizing the ending shows no significant difference from that before procanization (Table 1). Prolongation of the time constant by choline may be attributed to the possibility that the membrane resistance of the terminal is increased by choline.

<table>
<thead>
<tr>
<th>Corpuscle No.</th>
<th>30</th>
<th>31</th>
<th>33</th>
<th>34</th>
<th>35</th>
<th>Mean</th>
</tr>
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<tbody>
<tr>
<td>Bath temperature (°C)</td>
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<td>21</td>
<td>21</td>
<td>21</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Time constant</td>
<td>0.92</td>
<td>1.1</td>
<td>0.93</td>
<td>0.85</td>
<td>0.80</td>
<td>0.92</td>
</tr>
</tbody>
</table>
| *Room temperature.*

Effect of cooling. When the Ringer bath containing the non-myelinated terminal was cooled, the time course of both the receptor potential and the all-or-nothing response was prolonged, and below 14-15°C initiation and conduction of the impulse at the terminal were abolished. The temperature at which initiation and conduction of impulses are blocked is nearly the same as that reported previously on intact Pacinian corpuscles. The effect of cooling the terminal is shown in Fig. 9; at 16°C no sign of conduction block is seen, but at 11°C the antidromic impulse became monophasic and mechanical stimuli did not elicit the all-or-nothing impulse. During the course of this experiment it was generally observed that after abolishment of the impulse initiation from the terminal at 14-15°C the receptor potential produced by strong stimuli was
still capable to initiate an impulse from the first node. It may suggest that there is a difference in susceptibility of cooling between the terminal and the first node.

When invasion of the non-myelinated terminal by an antidromic impulse was blocked by cooling, antidromic impulses did not affect subsequent receptor potentials, as was observed on procaine- or choline-treated terminals (Fig. 9E). This is shown in Fig. 10 more quantitatively; in this figure depression of the receptor potential is noticed at 21°C and 16°C, but no appreciable change in the amplitude of receptor potentials following antidromic impulses is observed at 11°C, where impulse initiation was blocked.

Changes affected in various parameters by a change in temperature are shown in Table 2. It was observed that the amplitude of receptor potential was more affected by temperature change than was that of the action potential of the node, but that reduction in the receptor potential amplitude by a fall in temperature was dependent on stimulus strength employed8,9). This may explain the discrepancy shown in Table 2, in which the ratio of the receptor potential amplitude to the amplitude of the antidromic impulse of the first node became smaller with a fall in temperature in No. 34, while it became larger in No. 31. The time constant of decay of the falling phase of the
RESPONSE OF SENSORY NERVE TERMINAL

Fig. 10. Depression of mechanically produced responses by preceding antidromic stimulation of the terminal at three different temperatures. Bath temperature is 20.5°C for ○ and ●; 16°C for □ and ■; 11°C for +. ○, □ and +, receptor potential; ● and ■, all-or-nothing response. Abscissa, interval between peaks of antidromic and mechanical responses; ordinate, amplitude of mechanical response. Corpuscle No. 34.

Table 2.

<table>
<thead>
<tr>
<th>Corpuscle No.</th>
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</tr>
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<tr>
<td>Bath temperature (°C)</td>
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<td>21</td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>20</td>
</tr>
<tr>
<td>AAP (μV)</td>
<td>151</td>
<td>119</td>
</tr>
<tr>
<td></td>
<td>82</td>
<td>45</td>
</tr>
<tr>
<td>MRP (μV)</td>
<td>65.5</td>
<td>35.6</td>
</tr>
<tr>
<td></td>
<td>41</td>
<td>35</td>
</tr>
<tr>
<td>Ratio of MRP/AAP</td>
<td>0.43</td>
<td>0.30</td>
</tr>
<tr>
<td></td>
<td>0.50</td>
<td>0.78</td>
</tr>
<tr>
<td>Time constant (msec)</td>
<td>0.88</td>
<td>1.74</td>
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<tr>
<td></td>
<td>1.1</td>
<td>1.35</td>
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</table>

The maximum receptor potential at room temperature was measured after blocking the impulse by applying conditioning antidromic stimuli.

Receptor potential was generally prolonged by a fall in temperature; it becomes nearly doubled when the temperature is changed from 21°C to 11°C. This is consistent with Q_{10} value of −1.4, obtained on intact Pacinian corpuscles⁸, because the time constant changes logarithmically with a change in temperature.
Fig. 11. Relationship between amplitude of mechanically produced response and stimulus strength. •, response of the terminal to mechanical stimulus; ○, response of the terminal to mechanical stimulus preceded by antidromic impulse (receptor potential); □, response of the first node following the terminal activity. □ at zero stimulus strength indicates average response amplitude of the first node to antidromic stimuli, horizontal bars showing range of its variation. Corpuscle No. 35.
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Relationship between stimulus strength and amplitudes of receptor and all-or-nothing potentials. By applying mechanical stimuli of varying intensities after the antidromic stimulus with an interval just sufficient to block the all-or-nothing response, the relationship between the amplitude of the receptor and all-or-nothing potentials and the stimulus strength was obtained. An example of the measurement is shown in Fig. 11. As can be expected, the receptor potential is gradually increased in amplitude with an increase in stimulus strength up to a certain maximum value (empty circles in Fig. 11), while the amplitude of the all-or-nothing response from the terminal (filled circles) is increased suddenly to a constant value at the stimulus strength of 3 V. Empty squares indicate the amplitude of the impulse of the first node produced by mechanical stimuli, while a filled square at zero stimulus intensity represents the average amplitude of the antidromically produced impulse of the node. The maximum value of the receptor potential relative to the amplitude of the all-or-nothing impulse of the first node, elicited antidromically, is about 60% in this figure.

Relative amplitude of the receptor potential and all-or-nothing potentials. The ratio of the maximum amplitude of the receptor potential to the antidromically produced impulse of the node was measured in several preparations. The ratio in four preparations is shown in Table 3, which varies from 0.40 to 0.64. The average ratio for six preparations is 0.49. Although antidromic impulses depress subsequent receptor potentials, the values measured by A+M or M+M procedure and listed in Table 3 suffered very little depressant effect by antidromic stimulation since the latter was delivered to the terminal so as to block the all-or-nothing response but not to affect the receptor potential appreciably.

The relative value of the maximum receptor potential amplitude to the antidromic impulse of the first node obtained immediately after abolishing the all-or-nothing response by procaine or choline is 0.48 in average as shown in Table 4. This figure is identical with that measured by the A+M procedure.

The amplitude of the all-or-nothing response from the terminal tended to decrease when strength of mechanical stimuli was increased, although this tendency can not be seen in Fig. 11. It is noteworthy in Fig. 11 that the all-or-nothing response from the terminal is smaller in amplitude than the maximum receptor potential and is much smaller than the one elicited antidromically at the first node. The amplitude ratio of these potentials is about 0.2-0.6, the average value of four preparations in Table 3 being 0.36. The ratio can be changed by changing the position of the oil-saline interface. For example, by slacking the suspended axon a little so that the interface could be placed more proximally i.e. nearer the first node, the ratio changed from 0.4 to 0.26 in one preparation and from 0.48 to 0.32 in another.
TABLE 3.

Amplitude of the maximum receptor potential, all-or-nothing response of the terminal and that from the first node in response to mechanical stimuli. Values were expressed relative to the amplitude of the antidromically produced impulse.

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<thead>
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<th>34</th>
<th>35</th>
<th>36</th>
<th>mean</th>
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<tbody>
<tr>
<td>Bath temperature (°C)</td>
<td>21</td>
<td>18.5</td>
<td>20</td>
<td>19</td>
<td></td>
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<tr>
<td>Max. receptor potential</td>
<td>0.48</td>
<td>0.43</td>
<td>0.64</td>
<td>0.40</td>
<td>0.49</td>
</tr>
<tr>
<td>All-or-nothing response from the terminal</td>
<td>0.37</td>
<td>0.31</td>
<td>0.54</td>
<td>0.23</td>
<td>0.36</td>
</tr>
<tr>
<td>All-or-nothing response from 1st node</td>
<td>0.78</td>
<td>0.81</td>
<td>0.93</td>
<td>0.895</td>
<td>0.85</td>
</tr>
</tbody>
</table>

In all experiments the maximum receptor potential was obtained by abolishing the impulse critically by the preceding antidromic impulse.

TABLE 4.

Relative value of the maximum receptor potential (MRP) to the antidromically produced impulse (AAP), measured immediately after the impulse from the nerve terminal being abolished by procaine and choline chloride.

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</tr>
<tr>
<td>Ratio of MRP/AAP</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>By ( A+M ) procedure</td>
<td>0.52</td>
<td>0.48</td>
<td>0.50</td>
<td>0.47</td>
<td></td>
</tr>
<tr>
<td>After choline</td>
<td>0.45</td>
<td>0.35</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>After procaine</td>
<td></td>
<td></td>
<td>0.65</td>
<td>0.47</td>
<td></td>
</tr>
</tbody>
</table>

* Room temperature.

The figure of 0.36 for the ratio of the all-or-nothing response at the terminal to the first node impulse is smaller than that of the maximum receptor potential to the first node impulse. This is unlikely. A possible explanation for this is that the first node impulse is probably initiated at the rising phase of the terminal activity, and therefore smaller values were obtained for the amplitude of the impulse at the terminal, as they were measured with abolishing the first node activity. The evidence for this argument can be seen in Fig. 8, where the all-or-nothing response to test mechanical stimulus (at room temperature) becomes greater by more than 10% with decreasing interval between the conditioning and test stimuli until the test response becomes monophasic. This suggests that the amplitude of the all-or-nothing response at the terminal is nearly the same as that of the maximum receptor potential and is half of the first node impulse.

From Fig. 11 and Table 4 it will be seen that the amplitude of the orthodromically elicited impulse at the node is smaller than that of the antidromically produced one, the former being about 0.8-0.9 of the latter, and that the former amplitude is gradually reduced with an increase in the stimulus strength. This has been noted by Hunt & Takeuchi\(^5\), who attributed it to
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a decrease in the potential difference between the terminal and the first node by the depolarization of the terminal membrane resulting from mechanical stimulation.

The relative amplitude ratio of the maximum receptor potential, the all-or-nothing impulse at the terminal and the orthodromically elicited impulse at the first node to the antidromically produced impulse from the node is obtained from Tables 3 and 4 presented above. The average ratio is 0.5:0.36:0.85:1, but, if we assume the amplitude of the all-or-nothing impulse at the terminal to be almost equal to the amplitude of the maximum receptor potential, the ratio would be 0.5:0.5:0.85:1.

DISCUSSION

Hunt & Takeuchi\(^5\) and Ozeki & Sato\(^6\) have recently shown that the non-myelinated terminal in Pacinian corpuscles initiates impulses to both antidromic and mechanical stimuli. The present investigation confirms these results, and presents additional evidence indicating that the impulse is initiated at the terminal. The evidence presented in the Results can be summarized as follows: (i) By recording potential change to mechanical stimuli across an oil-saline interface placed close to or at the non-myelinated terminal, three component potentials were observed; the graded response, the all-or-nothing response generated at the terminal and the first node impulse. (ii) During the refractory period following the all-or-nothing response of the terminal only the receptor potential can be observed. (iii) Procaine, choline and cooling applied to the terminal abolish first of all the all-or-nothing response of the terminal and later the receptor potential. (iv) Antidromic stimulation produces a diphasic response indicating invasion of the terminal, and the invasion is blocked by procaine, choline and cooling. (v) When the terminal is made inexcitable by procaine, choline and cooling, antidromic stimulation does not produce depression of subsequent receptor potentials. (vi) The amplitude of the maximum receptor potential is nearly equal to or rather greater than that of the all-or-nothing impulse at the terminal. If the all-or-nothing response were initiated from the first node and if in the present experiment the first node had been peripheral to the oil-saline interface, the maximum receptor potential should have been appreciably smaller than the impulse, because the former is generated at a more distant site from the recording position than the latter.

Diamond et al.\(^3\) recorded the receptor potential across an air gap between the 2nd and 3rd nodes and showed two potential steps at the rising phase of the potential by suprathreshold mechanical pulses when they passed anodal polarizing currents through the corpuscle. These were attributed to the activity of the first and second nodes and it was concluded that no all-or-nothing impulse
activity occurs in the non-myelinated terminal. The results in the present experiment seem to give clues to understanding the discrepancy between the results by DIAMOND et al.\(^2\) and those by HUNT & TAKEUCHI\(^3\) and by OZEKI & SATO\(^6\). In the present experiment it has been found that the relative amplitude of the maximum receptor potential, all-or-nothing response of the terminal and mechanically and antidromically produced impulses of the first node is, in average, 0.49:0.36:0.85:1. In addition, the all-or-nothing response from the terminal has been shown to decrease in amplitude with an increase in stimulus strength. These facts together with the capacitance existing between the terminal and the second node would contribute to show the compound potential of the graded and all-or-nothing responses from the terminal as if it were a single graded response, when it is recorded between the 2nd and 3rd nodes.

It has been shown by DIAMOND, INMAN & GRAY\(^10\) that perfusion of Pacinian corpuscles with a Na-free solution abolished the nervous impulse in less than 2 minutes, but that the receptor potential was reduced in 11-30 minutes to 10\% of the initial value. In the present experiment it was also noticed that the all-or-nothing impulse was abolished much more quickly than was the receptor potential. The difference in the time necessary to abolish the potential was taken by DIAMOND et al.\(^10\) as an evidence to indicate that impulses and receptor potentials do not occur at the same site. However, from the conclusion, reached in the present experiment, that both responses are generated at the non-myelinated nerve terminal the time difference in abolishing the responses probably indicates that the mechanism for impulse initiation is more susceptible to Na-free solution than is that for generator response. Similar explanation may be applied to selective abolition of impulses by cooling the terminal below 14-15\(^\circ\)C.

The receptor potential recorded in this experiment has a rise time of about 1 msec and a time constant of 0.9 msec for the exponential decay of the falling phase at 18-21\(^\circ\)C. The time constant of the falling phase of the receptor potential, recorded between the 2nd and 3rd nodes of the corpuscle, is 1.7 msec\(^7\), 1.10 msec for 24.8\(^\circ\)C\(^6\) and 1.8 msec for 20\(^\circ\)C\(^6\). The large values of the time constant obtained by these investigators compared with the present ones may be attributed to the effect of capacitance and resistance of the nodes and myelin sheath existing between the non-myelinated terminal and the second node, because the decay of the falling phase of the receptor potential results from a passive recharging of the nerve terminal and axon with a time constant determined by their resistance and capacitance. In agreement with this idea, the time course of the generator current recorded from a point of the non-myelinated terminal has been shown to have a much faster time course, a rise time of 1 msec and a decay time of 1 msec\(^6\)

GRAY & SATO\(^1\) and DIAMOND et al.\(^7\) have shown that, when two sub-
threshold mechanical stimuli were delivered to the corpuscle with an interval of less than 10 msec, the receptor potential to the second stimulus is reduced in amplitude and rate of rise. The latter authors have further found that the receptor potential is depressed by a preceding antidromically or mechanically produced impulse and that the depression by an antidromic impulse is never more than that produced by an mechanically produced impulse. Our findings have confirmed these results, and, in addition, have presented a new finding regarding depression; antidromic stimulation of the terminal can produce depression, but the depression of the subsequent receptor potentials cannot be observed when the terminal is affected by procaine, choline and cooling. This fact indicates, on one hand, that an antidromic impulse should invade the terminal, and, on the other hand, it points out that a change of membrane potential in the non-myelinated terminal should cause the depression. If a change of membrane potential in the non-myelinated terminal were a principal causal factor, the depression by an antidromic impulse must be as great or greater than that produced by a maximum receptor potential. Unfortunately, this has not been investigated in the present experiment. As has been observed by DIAMOND et al. and confirmed in the present experiment, increased depression results from the response to a large mechanical stimulus and antidromically conducted impulses have never caused more depression than mechanically excited ones. Therefore, if increasing the mechanical stimulus above threshold can increase depression, it must add something other than further depolarization. It may be said that the depression is a phenomenon resulting from a change of membrane potential in the terminal, but a change in the membrane properties due to mechanical deformation of the terminal can contribute to the amount of depression.

SUMMARY

1. Potential changes in response to antidromic and mechanical stimuli were recorded across an oil-saline interface placed close to or at the non-myelinated terminal of the Pacinian corpuscle, of which the lamellae had been removed up to the central core.

2. An antidromic stimulus produced a diphasic potential indicating that an antidromic impulse can invade the terminal, while a mechanical stimulus generated a diphasic response of an all-or-nothing nature superimposed on a graded response, or on the receptor potential. This all-or-nothing response to mechanical stimuli can be abolished and the antidromic impulse becomes monophasic when procaine, choline or cooling is applied to the terminal.

3. When a mechanical stimulus was delivered within a few msec after an antidromic impulse, the all-or-nothing response to mechanical stimuli was abolished, and only the receptor potential was observed during the refractory period of
the terminal. The amplitude of the receptor potential became smaller as the interval between the mechanical and antidromic stimuli was shortened. The depression of the receptor potential could not be observed, when the terminal was made inexcitable by procaine, choline and cooling so that antidromic impulses could not invade the terminal.

4: The relative amplitude of the maximum receptor potential, the all-or-nothing impulse at the terminal and the mechanically and antidromically elicited impulses at the first node of Ranvier was measured; their average ratio being 0.49:0.36:0.85:1.

5. The receptor potential has a rise time of about 1 msec and the time constant of decay of its exponential falling phase is 0.9 msec, which is smaller than that obtained in intact Pacinian corpuscles.

The authors are indebted to Professors H. Hayashi and T. Takeuchi and their colleagues for helping us to take phase-contrast micrographs, and they are also grateful to Professors J. A. B. Gray and C. C. Hunt for their criticism and valuable discussion.

REFERENCES


