NON-HOMOGENEOUS CONDUCTION IN GIANT AXONS OF THE NERVE CORD OF PERIPLANETA AMERICANA

BY I. PARNAS,* M. E. SPIRA,* R. WERMAN† AND F. BERGMANN‡

* Department of Zoology, The Hebrew University, † Department of Pharmacology, The Hebrew University—Hadassah Medical School, Jerusalem, Israel, and ‡ Institute of Psychiatric Research, Indiana University Medical Center, Indianapolis, Indiana

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INTRODUCTION

Earlier microelectrode studies of insect giant axons were directed towards the elucidation of membrane phenomena (cf. Pichon & Boistel, 1967). Recently, Callec & Boistel (1966a, b, 1967) used microelectrodes to determine the organization of pathways in the cockroach nervous system, with special attention being paid to the sixth abdominal ganglion.

In previous papers of this series (Spira, Parnas & Bergmann, 1969a, b) evidence was presented that in the cockroach giant axons run continuously from the sixth abdominal ganglion to the suboesophageal ganglion. Histological studies (Spira et al. 1969b) revealed that both in the abdominal and thoracic ganglia the giant fibres form isthmuses. In addition, there is progressive tapering of the axons in the thoracic cord. In the present experiments we further support our previous findings by intracellular recordings from single giant axons. Evidence will be presented here that conduction of impulses by giant axons is non-homogeneous in the thoracic cord due to variations in axonal diameters. Furthermore, it will be shown that the short conduction delay of 0.6–0.7 msec at each thoracic ganglion and the great sensitivity of these structures to block by nicotine can be explained on the basis of these anatomical features.

MATERIALS AND METHODS

The procedure for isolation of the complete nerve cord from cerci to head has already been described (Spira et al. 1969a). The preparation was bathed in the solution described by Yamasaki & Narahashi (1960).

To facilitate microelectrode penetration the cord sheath was softened by treatment with 1% pronase for 2 min. (Willows, 1967). Glass microelectrodes were filled with 3M-KCl and had a resistance of 30–40 MΩ. Conventional techniques for intracellular recording were used. Methods of extracellular stimulation and recording have been previously described (Spira et al. 1969a).

RESULTS

Responses of giant fibres recorded with extracellular and intracellular electrodes

The ascending and descending responses of giant fibres were recorded with extracellular electrodes as previously described (Spira et al. 1969a). At the same time
intracellular records from single axons were made. As soon as the microelectrode impaled a fibre, a steady membrane potential of $-50$ to $-60$ mV. was observed and this persisted for up to 6 hr.

Figure 1 shows extracellular responses at $A_8-A_4$ on the upper beam of each pair of sweeps in response to stimuli applied to $S_0-T_1$. The intracellular spike, recorded at $A_8-A_4$, is shown on the lower beam. With low stimulation intensities (Fig. 1A) only a single spike appeared in the upper beam, while no transient was registered by the intracellular electrode. However, with a slight increase in stimulus strength (Fig. 1B) a spike was recorded by the microelectrode while a second spike appeared in the extracellular record. Further increase of stimulus strength produced recruitment of more giant axons (Fig. 1C and D, upper sweep).

![Figure 1. Giant fibre responses recorded with intracellular and extracellular electrodes. Descending spikes elicited from $S_0-T_1$, extracellular recording at $A_8-A_4$ (upper beam) and intracellular recording at $A_5-A_4$ (lower beam). From A to D, gradual increase in stimulus strength. E, Ascending response elicited from the cercal nerve. Calibration: microelectrode, 50 mV., macroelectrode, 0.25 mV., time scale 10 msec. Negativity of rostral extracellular electrode downwards; of microelectrode upwards.](image)

When the cercal nerve was stimulated electrically, a number of action potentials appeared in the upper beam, while again only a single spike was recorded by the microelectrode. In Fig. 1E the intracellular spike precedes some of the giant potentials in the upper beam, while in Fig. 1A-D it always followed the extracellular potentials. This is a consequence of the microelectrode being placed caudally to the extracellular electrode.

Figure 2 illustrates the interaction between an ascending impulse, evoked by electrical stimulation of the cercal nerves, followed by a descending stimulus, applied to the connective $S_0-T_1$. When the interval between the two stimuli was more than 13 msec, the extracellular electrode registered the complete, separate sets of ascending and descending spikes (Fig. 2A); likewise, the microelectrode recorded a single spike in response to each stimulus (lower beam of Fig. 2A). When the interval was gradually shortened, more and more of the descending giant potentials were eliminated and only the slower and smaller components of this complex survived (Fig. 2B–D). The
descending spike recorded by the microelectrode (lower beam) was already blocked at a delay of 12.5 msec. (Fig. 2B). In contrast, the descending giant spikes recorded on the upper beam were not completely blocked until the delay was reduced to 9.2 msec. (Fig. 2D). Calculations reveal that collision in this experiment occurred rostrally to the microelectrode, just below the stimulating electrode at S₀-T₁.

When the cercal nerves were stimulated with a weaker pulse, so that only the extracellular electrode but not the microelectrode recorded ascending responses, then the intracellular descending spike was not blocked by the preceding ascending response at any delay (Fig. 2E–H). This result indicates that in the same axon no interaction other than collision takes place.

Fig. 2. Ascending/descending interaction. Upper beam, extracellular recording at A₁–A₄; lower beam, intracellular recording at A₅–A₆. Stimulus at the cercal nerve (Asc) precedes the one at S₀–T₁ (Des). A–D, gradual decrease in time interval between ascending and descending stimuli. Note that at a delay of 12.5 msec. the intracellular descending spike (80 mV.) was blocked (B). Only with further shortening of the delay, complete block of descending giant responses also ensued (C–D). E–H, Shock strength for ascending volley was reduced below the threshold for producing a spike in the axon penetrated by the microelectrode; descending intracellular spike was not blocked at any delay. Scale: microelectrode, 50 mV.; macroelectrode, 0.25 mV.; time scale, 10 msec. Intracellular positivity downwards. Negativity of rostral extracellular electrode downwards.

Ipsilateral–contralateral recordings

The two S₀–T₁ connectives were stimulated together and the evoked responses were recorded by extracellular electrodes from both connectives between A₅–A₆ and by an intracellular electrode in an A₅–A₆ connective. Ascending responses were evoked by stimulation of the cercal nerve contralateral to the connective impaled by

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the microelectrode (Fig. 3, inset). Figure 3A shows that the microelectrode recorded a spike only in response to the descending stimulus. No ascending spike was observed when the contralateral cercal nerve was stimulated. The extracellular electrodes recorded an ascending response, probably from the connective ipsilateral to the cercal nerve stimulated, but some crossed cercal fibres may be involved (Roeder, 1948; Callec & Boistel, 1967). When the delay between ascending and descending pulses was gradually reduced, no block of the intracellular descending spike was observed, while part of the extracellular descending responses were sometimes blocked. We have no evidence whether these blocked responses result from depression of the action potentials in the ipsilateral connective or involved some of the crossed cercal fibres at $A_9$, demonstrated by Roeder (1948) and Callec & Boistel (1967).

Fig. 3. Ipsi/contralateral interaction. Scheme: experimental arrangement. Note that the cercal nerve (St.) is stimulated contralaterally to the connective impaled by the microelectrode ($R_2$). Upper beam: extracellular recording at $A_9-A_9$ ($R_2$). Ascending (Asc) pulse precedes descending one (Des). A: control; Note that only a descending spike (80 mV.) was recorded by the intracellular microelectrode. B--F: gradual reduction of the time interval between the ascending and descending stimuli; note that the intracellular spike was not blocked. Scale: 10 msec. Microelectrode, 50 mV.; macroelectrode, 0.25 mV.

**Non-uniform conduction**

Although the evidence accumulated so far (Spira et al. 1969a, b) indicates that the ascending giant fibres are continuous up to the $S_0$ ganglion, some of our earlier results, such as ganglionic delay in the thorax and block of conduction by nicotine, still had to be explained. Therefore it was decided to study the descending responses induced by stimulation at $S_0$-$T_1$, $T_3$-$T_2$ and $T_2$-$T_3$. 
Three approaches were used in the following experiments to elucidate whether synapses or areas of low safety factor for axonal conduction (Coombs, Curtis & Eccles, 1955; Eyzaguirre & Kuffler, 1955) are present in the thoracic ganglia. First, the connectives at each level were stimulated by twin pulses with a variable delay between the first and second pulse. Secondly, the giant axons were fatigued by stimulation with long, high-frequency (50–100/sec.) trains of pulses. Thirdly, the effect of low doses of nicotine on ganglionic conduction was studied. In all of the following experiments both ascending and descending responses were obtained when the microelectrode impaled an abdominal axon.

**Twin pulses**

In the experiment of Fig. 4 the microelectrode was inserted at A₁–A₂. In Fig. 4A the responses to twin pulses at S₀–T₁ are shown. At a delay of 4 msec. between the two pulses there was an abrupt drop of the second spike response. This result indicates that the region of refractoriness was far from the recording microelectrode; otherwise a briefer refractory period would have been observed. One of the explanations could be a region of low safety factor. In such a case we can calculate the position of such a region. The recording microelectrode could easily discriminate potentials as low as 5 mV. propagated decrementally. Assuming a space constant of about 1 mm. (0.86 mm.—Yamasaki & Narahashi, 1959, and Narahashi & Yamasaki, 1960; 1.3 mm. —Boistel & Coraboeuf, 1954, and Boistel, 1959), the region of low safety factor must have been at least 2 mm. rostral to the recording microelectrode.

The same general behaviour was observed for the responses evoked from T₁–T₂ (Fig. 4B). The second response was blocked in one step at a delay of 4 msec. However, the responses elicited from the T₂–T₃ connectives (Fig. 4C) were blocked at a much shorter delay, namely 2 msec. Almost the same delay of 2 msec. was observed...
for block of the second response of the ascending twin pulses given at A$_4$-A$_8$ (Fig. 4D). It is obvious that the delays between the first and second pulses are divided into two groups. For stimuli given above the mesothoracic ganglion (T$_2$) the delays were approximately 4 msec., and for stimulation below T$_2$ only 2 msec. However, when a cercal nerve was stimulated, a very long delay, 10 msec., between the twin pulses was necessary to produce a second spike (Fig. 4E), suggesting a qualitatively different mechanism, i.e. the known synapse at A$_8$. For the S$_0$-T$_1$ and the T$_1$-T$_2$ connectives, we argue that the long delays resulted from the presence of a specialized region of low safety factor. In the impaled axon, such a region existed above ganglion T$_3$, i.e. at T$_2$ and perhaps at T$_1$ as well.

In all cases, recovery from cercal nerve stimulation was at least twice as long as that from thoracic stimulation.

Repetitive stimulation

The foregoing experiment suggested a region of low safety factor at T$_2$. On repetitive stimulation such a region should be fatigued and then blocked. Figure 5A shows the spike in the same axon as the one in Fig. 4, elicited by 50/sec. stimulation at S$_0$-T$_1$ (superimposed sweeps). This response indeed was blocked in less than 30 sec. (Fig. 5B). After the block had taken place, stimulation was switched to the T$_1$-T$_2$ electrodes. As can be seen in Fig. 5C, the response was still blocked, indicating that the region with the lowest safety factor was below T$_1$. On the other hand, with further switching of the stimulation to T$_2$-T$_3$, spikes could again be evoked and these were not blocked even when stimulation continued for many minutes (Fig. 5D). The responses induced at A$_4$-A$_8$ also showed no fatigue (Fig. 5E). In this axon, therefore, a region of low safety factor for descending impulses existed at T$_2$. Again, it cannot be excluded that

Fig. 5. Repetitive stimulation. Conduction failure induced by repetitive stimulation of the same axon as that of Fig. 4. Superimposed sweeps in all cases. Recording at A$_4$-A$_8$. A, 50/sec. stimulation at S$_0$-T$_1$. B, Block of the responses from S$_0$-T$_1$ took place after stimulation continued for 30 sec. C, Stimulation at T$_1$-T$_2$ after block at S$_0$-T$_1$. The response was blocked here also. D, Response to stimulation at T$_2$-T$_3$; note that there was no block of the spike responses even after stimulation continued for a few minutes. E, 50/sec. stimulus at A$_4$-A$_8$. No fatigue even after a few minutes. Scale: 5 msec., 50 mV.
a second, less effective site of conduction blockage was present at $T_1$. On the other hand, repetitive stimulation of the cercal nerve resulted in extremely rapid fatigue. The fatiguability of $T_2$ in this case is thus intermediate between that of an axon and that of a synapse.

**Effect of nicotine**

After a period of rest, stimulation of the $S_0-T_1$ connectives (Fig. 6A) again elicited a spike in the same axon used in the foregoing experiments (Figs. 4, 5). Nicotine in a concentration of 10 µg./ml. was applied only to the thorax, causing a rapid block...
(1 min.) of the responses to $S_0-T_1$ and $T_1-T_2$ stimulation (Fig. 6B, D). On the other hand, the response elicited from $T_2-T_3$ (Fig. 6E) was not blocked by the alkaloid even after 5 min. of incubation. A much longer period would be needed to block conduction at $T_3$ (Spira et al. 1969a). Likewise, the responses elicited from $A_4-A_5$ (Fig. 6F) or from the cercal nerves were not blocked. However, as soon as nicotine ($10 \mu g./ml.$) was applied to the whole preparation, the response from the cercal nerves was also blocked (Fig. 6G–I), while responses to stimulation at $T_2-T_3$ or $A_4-A_5$ persisted (Fig. 6K, L). The three sets of experiments, carried out on the same axon, are thus complementary and confirm the presence of at least one region of low safety factor at $T_2$, a region which is both rapidly fatigued and sensitive to nicotine.

In the same manner it was shown in other cases that the region of lowest safety factor may be situated at $T_1$, $T_2$ or $T_3$. In most cases, only one such region could be demonstrated in each axon. However, by the use of different techniques (see Fig. 7) two sensitive zones were sometimes found.

Recordings near thoracic ganglia

The twin-pulse experiment of Fig. 5 led to the conclusion that the site of low safety margin was at least 3 mm. rostral to the abdominal microelectrode at $A_1-A_2$. The results with twin pulses, repetitive stimulation and the application of nicotine indicate that the thoracic ganglia are the sites of greatest vulnerability. It was therefore decided to record near the thoracic ganglia. Since the space constant of abdominal giant axons is approximately 1 mm, an intracellular electrode at the caudal edge of a ganglion (i.e. about 1 mm. from the centre of the ganglion) should still be close enough to the zone of low safety factor to observe potentials spreading electrotonically.

Electrodes were inserted only at the base of $T_2$ and $T_3$, since histological observation showed that the size of the axons at these levels ($30-50 \mu$) makes possible impalement of the fibre, while the small diameter ($10 \mu$) at $T_1$ greatly reduces the likelihood of successful penetrations. Moreover, experiments were performed only using axons that responded to stimulation of the cercal nerve as well of $S_0-T_1$. In many cases thoracic axons were found that responded only to $S_0-T_1$ stimulation, but not to impulses ascending from the cercal nerve. This is due to the fact that many more giant fibres are present in the thoracic cord than in the abdomen (Spira et al. 1969b). On the other hand, an axon that responded to stimulation of the cercal nerve could always be activated by stimulation of the $S_0-T_1$ connectives as well.

Recording at the base of $T_3$

Figure 7 shows intracellular recordings from the caudal base of ganglion $T_3$, when stimulation was applied at $S_0-T_1$ (Fig. 7A), $T_1-T_2$ (Fig. 7E) and $T_2-T_3$ (Fig. 7I). The rising phase of the spikes induced at the different levels was sharp. When the connectives were stimulated repetitively at 50/sec. the response elicited from $S_0-T_1$ showed first an increase in latency (Fig. 7B, C), then a prolonged depolarizing after-potential (Fig. 7C) and, finally, a complete block in less than 30 sec. (Fig. 7D). The spike elicited from $T_1-T_2$ behaved differently (Fig. 7E–H). Again, with repetitive stimulation, the latency increased. However, after repetitive stimulation another phenomenon preceded the block: the rising phase of the spike was changed so that a slower-rising pre-potential became manifest. Again the spike was followed by a depolarizing after-
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potential (Fig. 7G). With further stimulation the spike failed to invade the zone of recording, and only a brief, small but persistent depolarization of 12 mV. was recorded. This response probably represents a spike propagating electrotonically from the point of block. The brief time-course, i.e. the fact that the response was similar in duration to the spike, and the constancy of the electrotonic response which showed neither fatigue nor facilitation, both speak against the possibility that the small transient of Fig. 7H represents a synaptic potential. Figure 7F and G (arrow), shows that the threshold for the all-or-none spike is well defined. This finding and the presence of a local response (7G) can best be explained if the spike—after electrotonic decrement through an area of low safety factor or of electrical inexcitability—was sometimes re-initiated very close to the recording microelectrode.

The small response shown in Fig. 7H persisted for long periods of stimulation at high frequencies; after a period of rest a full spike was again recorded at the micro-electrode. The same general pattern followed stimulation at T2–T3 (Fig. 7I–L). The size of the electrotonically propagated responses initiated by stimulation at either T1–T2 or at T2–T3 was the same (cf. Fig. 7H with L). From the size of the persistent depolarization and the known values of the space constant, it is probable that the block began approximately two space constants rostral to the recording microelectrode, i.e. at the rostral border of ganglion T3. Furthermore, the clear local responses indicate

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Fig. 7. Multiple sites of low safety factor. Responses to stimulation at S0–T1 (A–D) and T1–T2 (E–H) recorded at the caudal base of ganglion T2. Note that A, E and F are at low gain. A, Control response to stimulation at S0–T1. B–D, Progressive changes during repetitive stimulation at 50/sec. E, Stimulation at T1–T2. F, After repetitive stimulation at 50/sec. note slowing of initial rise time with spike appearing at a threshold of 12 mV. (arrow). G–H, Superimposed sweeps at higher magnification during repetitive stimulation of T1–T2. I–L, 50/sec. repetitive stimulation of T1–T2. Scale: 5 msc.; A, E, F, I, 25 mV.; others 12.5 mV. See text for discussion.
almost successful attempts at re-initiation of the spike within a space constant of
the microelectrode, i.e. caudal to the zone of low safety margin, in the caudal half of
the ganglion. Otherwise, local responses would not have been recorded by the micro-
electrode.

The results presented in Fig. 7 indicate the presence of a second zone of low safety
factor in addition to that at T3. Since no potential was recorded at T3–Ax associated
with block of the S0–T1 stimulus (Fig. 7C, D), an additional, more remote site of
conductivity vulnerability than that at T3 must be postulated. Thus, in this axon there
must exist a second region of low safety factor above T2, probably at T1.

Fig. 8. Mid-thoracic recording. Intracellular recording at connective T5–T4 of spikes elicited
from the cercal nerves (A–C), from A1–A4 (D–E) and from T1–T4 (F–I). A, Twin pulses
delivered to the cercal nerves; block occurred at a delay of 5 msec. B–C, Repetitive stimula-
tion of the cercal nerves produces an increase in latency (B) and block (C). D, Responses
stimulation (50/sec) at T1–T4 caused slowing in rise time (G) and block of the spike leaving
only a local response (G–I). Scale: A–G, 10 msec.; H–I, 5 msec.; A–G, 33 mV.; H–I, 20 mV.
See text for discussion.
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Recording at $T_3-T_3$

A microelectrode was inserted between ganglia $T_2$ and $T_3$, penetrating an axon that responded to stimulation of both the cercal nerves (Fig. 8A) and the $T_1-T_2$ connectives (Fig. 8F). The axon also responded to $S_9-T_1$ stimulation (not shown). When twin pulses were given to the cercal nerve, an all-or-none block of the second response occurred at a delay of 5 msec. (Fig. 8A). With repetitive stimulation of the cercal nerve, the latency was increased (Fig. 8B) and complete blockage was obtained after 5 sec. (Fig. 8C). The block occurred at a point remote from the recording micro-electrode, probably at the vulnerable synapses located at $A_3$.

When ascending spikes were evoked from $A_1-A_2$, i.e. close to the microelectrode, a different pattern was observed. With twin pulses a gradual block of the second response occurred (Fig. 8D), indicating a relative refractory period of 8 msec. With repetitive stimulation block did not occur, but an after-depolarization appeared (Fig. 8E). This result indicates that no region of increased vulnerability to axonal conduction existed between the stimulating electrodes of $A_1-A_2$ and the microelectrode. It is important to note the lack of a synapse at $T_3$ or even a region of low safety factor, in conflict with Roeder's postulation of synapses at $T_3$ for all ascending giant fibres.

Stimulation at $T_1-T_2$ revealed that an area of low safety factor existed at ganglion $T_2$, i.e. rostral to the microelectrode. With high-frequency stimulation, the full spike was blocked after an increase in latency, and only a small residual potential of 11 mV. was recorded (Fig. 8H, I). This potential remained constant over many minutes. After rest, normal spikes were again obtained. In no case was there found evidence of block of an ascending stimulus in the thoracic ganglia on repetitive stimulation or use of twin pulses.

DISCUSSION

Intracellular recordings from the giant axons confirm that there are continuous giant fibres from ganglion $A_4$ to the suboesophageal ganglion, contradicting the earlier observations of Roeder (1948) and Hess (1958). In our experiments the micro-electrodes were inserted from different angles and from all sides of the nerve cord, precluding selection of a specially oriented giant-fibre population. In all cases of recording from abdominal giants, spikes were elicited both from $S_9-T_1$ and from the cercal nerves, and by proper timing, descending spikes could be occluded by ascending spikes at all levels of the axon as far rostrally as the suboesophageal ganglion. We therefore conclude that most, if not all, of the abdominal giant axons reach the suboesophageal ganglion. Furthermore, recordings made close to thoracic ganglia support the scheme suggested previously (Spira et al. 1969b) that in the $A_4-S_9$ pathway the abdominal giant axons are electrically continuous throughout the thorax.

The histological studies of Pipa et al. (1959) traced the abdominal ventral giant group into the thorax. In degeneration experiments in which the abdominal nerve cord was severed (Spira et al. 1969b), we clearly identified the collapsed ventral giant group in the thoracic connectives and some evidence for degeneration of dorsal axons was also obtained. The present microelectrode study is consistent with the previous evidence that the abdominal dorsal giant axons narrow sharply in one step at ganglion $T_3$, while the ventral giant group tapers gradually after ganglion $T_3$ (Spira et al. 1969b).
In addition, the giant axons change their diameter along their course, forming isthmuses in each of the thoracic ganglia. Three kinds of experiments, namely twin pulses, fatigue, and sensitivity to nicotine, gave evidence for the presence, in each of the giant axons, of one or more areas of low safety factor. When a microelectrode was inserted into axons near ganglion T₂ or T₃, and stimuli were applied to the thoracic connectives, the spike evoked at the thorax failed to invade the zone of recording and small potentials of only 10–15 mV. were recorded. These responses are distinguished from synaptic potentials by their brief time course and by their constancy in size over a large range of stimulation frequencies.

However, there is one property of the areas of low safety factor which in the absence of other observations could be interpreted as indicating a synapse, namely polarization of conduction. Thus, it is interesting to note the experiment of Fig. 8, where an axon was impaled at the connective T₂-T₃, i.e. between the isthmus in ganglion T₃ and another in ganglion T₂. Here, however, only the isthmus of ganglion T₂, but not that in T₃, behaved as a region of low safety factor. In no case was evidence of a low safety zone found for ascending impulses. It seems therefore that the properties of the isthmuses are not symmetrical, i.e. they behave as areas of potential vulnerability to axonal conduction only in one direction.

There are two possible ways of explaining the undirectional vulnerability of conduction in the absence of synapses. Moreover, both of these explanations take into account the evidence against synapses: the bi-directional conduction of single stimuli; the brief delays at the ganglia; the brief and constant-amplitude potentials, seen after repetitive stimulation. The first possibility is that of a polarized electrical junction as demonstrated by Furshpan & Potter (1959) at the crayfish lateral giant/giant motor junction. A more attractive explanation, consistent with our histological results, is based on the changes in the diameter of the axon. It should be remembered that there is a progressive tapering of the giant fibres in a rostral direction throughout the thoracic cord. Superimposed on the tapering are more drastic narrowings, i.e. the isthmuses (Spira et al. 1969b). Thus, the change in diameter from isthmus to connective is always substantially greater at the caudal end of the isthmus. Conduction from the zone of transition from a small diameter axon into a sudden enlargement is always vulnerable. Failure of conduction in such situations has been demonstrated in many nerves and is well characterized by the detailed studies of initial segment (I-S) spike invasion into the soma of spinal motoneurons (Coombs, Curtis & Eccles, 1957). Another relevant example of such vulnerability is the antidromic invasion of the crustacean stretch receptor. Here, Eyzaguirre & Kuffler (1955) demonstrated that repetitive stimulation leads to increased latency, an initial slowing of the rising phase of the action potential and sustained after-depolarization. It is of interest to note that Bullock & Turner (1950) reported one-way block of conduction in Lumbricus giant axons in the rostro-caudal direction, similar to the present findings.

Thus the thoracic isthmuses present a special anatomical arrangement whose properties could be confused with those of a synapse. The delay of 0.6–0.7 msec, the liability to fatigue and the sensitivity to nicotine led Roeder to postulate a synapse at each thoracic ganglion, but even at that time he noted the atypical behaviour of these ‘synapses’. In the light of the present studies, we may conclude that the criteria used by Roeder were not sufficient to positively identify a synapse in the pathway.
Both his results and those of the present experiments are better explained by the occurrence of thoracic isthmuses.

It is interesting to note the difference between a thoracic isthmus and an abdominal one. While the first shows fatigue and sensitivity to nicotine, the abdominal isthmuses are not affected under the same experimental conditions. Although the giant axons in the abdominal ganglion may narrow to as little as 10 μ, they do not behave as areas of low safety factor. The reason for this difference may be the greater length of the isthmuses in the thoracic ganglia, corresponding to the much greater size of the thoracic ganglia themselves. This would be especially important if the isthmus were electrically inexcitable.

It has been established that giant axons which are excited by stimulation at or below A₄ conduct impulses through the abdominal and thoracic cord to the suboesophageal ganglion. The action potentials evoked by stimulation at S₀–T₁ and recorded at abdominal connectives may be propagated antidromically in these axons; however, the possibility should be considered that they represent physiological orthodromic activity as well, i.e. these axons may normally be innervated synaptically in the thorax or more rostrally. Preliminary experiments show that some of the abdominal giant axons respond to electrical stimuli given to the supra-oesophageal ganglion, or to stimulation of the antennae. In this connection, it should be recalled that Maynard (1956) elicited responses of thoracic giant fibres in the cockroach by stimulation of the antennae. Furthermore, Hughes (1965) recorded 'spontaneous' descending giant spikes from abdominal connectives in this animal.

It is thus not unlikely that the giant axons in the cockroach serve as a bi-directional pathway in physiological conditions. Hughes (1965) has suggested that in an ancestral form there was bi-directional conduction, but that in the cockroach giant descending and ascending pathways have become separated. In view of our present results, it is possible that the primordial condition of bi-directional pathways persists in the central nervous system of the cockroach. The function of a rapidly conducted unpolarized signal might be to clear all ganglionic stations by inhibition of ongoing motor activity in preparation for a slower set of signals which indicates the direction and nature of a noxious or threatening stimulus and thereby initiates the appropriate defensive reaction.

In conclusion we would like to suggest the following scheme (Fig. 9) for the arrange-
ment of the giant axons in the nerve cord of the cockroach. Both dorsal ($D$) and ventral ($V$) giant fibres are innervated by cercal sensory axons through a synapse at $A_4$ and possibly by sensory inputs in the thorax as well (question marks in figure). Both groups show short isthmuses in the abdominal ganglia but behave differently in the thorax. Axons of the dorsal group narrow precipitously at $T_3$ but continue as fine fibres throughout the thoracic cord. The ventral group, on the other hand, shows progressive tapering in the thorax, interrupted by elongated narrowings of each axon at each thoracic ganglion, while extending rostrally at least to the suboesophageal ganglion. Finally, the outputs ($a$) of the giants to motor axons of the leg (Roeder, 1948) in the thorax may be either direct or through interneurons.

**SUMMARY**

1. Studies with intracellular electrodes show that the abdominal giant axons of the cockroach give ascending responses to stimulation of cercal nerves and descending responses to stimulation of $S_0-T_3$ connectives.

2. In the thoracic region one or more areas of low safety factors occur for descending conduction.

3. These areas, which are considered not to be synapses, are blocked by low doses of nicotine ($2-5 \mu g./ml.$) fatigued by repetitive stimulation and show conduction delays of $0.6-0.7$ msec.

4. It is concluded that the abdominal giant axons extend continuously from $A_4$ to suboesophageal ganglion.

5. The possibility of bi-directional conduction under physiological conditions is discussed.

**REFERENCES**


Cockroach giant fibres


