THE CONDUCTION VELOCITY IN NARCOTIZED REGION AND IN DORSAL ROOT GANGLION OF NERVE

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It was previously shown by Verworn (1) and Lucas (2) that the excitation decreased in magnitude as it travelled through the narcotized region of the medullated nerve. Afterwards, Koike (3) and Pütter (4) obtained a conflicting result, and in keeping with it, Kato (5) introduced, after many experimental researches with his collaborators, the theory of decrementless conduction in narcotized region of nerve. But Ishikawa (6) and his coworkers carried out many experiments on the same problem and objected against the Kato's theory. It should not be underestimated that according to the Ishikawa's opinion the excitation of the adjacent region of the stimulated point behaves itself differently from that of the region far from the stimulated point. Subsequently, the discussions concerning the Kato's theory, however, threw the problem into confusion because of the discrepancy of the experimental results.

Important results have been obtained by Davis and his coworkers (7) i.e. they observed that the amplitude of the action potential wave decreased only at the boundary of the narcotized region when it propagated from the normal region into the narcotized region, and it propagated without decrement afterwards in the narcotized region. Furthermore, Tasaki (8), (9) found that in a single nerve fibre preparation the node of Ranvier has been narcotized almost instantaneously to the final state. In this way, however, the behaviour of conduction in narcotized region of nerve could not be sufficiently manifested.

In the present work the problem was examined from other point of view. Namely, the conduction velocity in every portion of nerve fibres was measured, in order to determine whether or not the action potential wave decreases in conduction velocity as it propagates through the narcotized region of nerve fibres.

In addition to the narcoysis experiment, the author has attempted to measure the time delay which was assumed by Erlanger et al. (10) to occur when the conducting impulse traverses the dorsal root ganglion.

METHOD

The "sciatic-tibial nerve preparation" or the "root-spinal nerve preparation" of a toad or a bullfrog was used. The latter consisted of the 9th dorsal or

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the 9th dorsal and ventral root and the 9th spinal nerve (sometimes incl. sciatic nerve). The spinal root of the Japanese toad is very long and convenient for the examination of conduction velocity in this region. The stimulation was effected by a breake shock from a coreless Porter coil using silver wire electrodes. Six or more pairs of knife-edge silver electrodes, which were made from thin silver plates of 0.2 mm. in thickness, were placed side by side at the interval of 1 cm. to lead off the action potential. At the recording a pair of leading-off electrodes was used in turn. The distance of a pair of electrodes was 2 mm. Each leading-off electrode was connected through a small mercury pool with a input terminal of the amplifier (fig. 1, A). The amplifier was a four stage condenser coupling one. The action potentials were recorded by means of a Yokogawa's oscillograph and the attached bromide paper camera. The moving velocity of bromide paper was 5-7 m/sec. (mm/msec.) and the least measurable length on the records was 0.2 mm. Accordingly, the error of the conduction time became 0.04 msec.

The narcosis was performed by immersing the preparation as a whole in the Ringer's fluid which contained a narcotic (urethane or ammonia) when the narcotization of the whole length of nerve preparation was desired. After the narcosis was effected sufficiently the preparation was brought in the more dilute narcotic solution or the Ringer's fluid, and after a while it was pulled out and laid on the stimulating and lead-off electrodes. In this state, the conduction velocity in each region stayed almost in a constant value throughout one series of experiment, because the recovery of the velocity is very slow in the recovery phase of narcosis. As the large fibres in nerve trunk are more resistant than the small fibres (Komizo (11), Erlanger and Blair (12)), the author perhaps examined the conduction of the fastest velocity fibres.

When the half length of the preparation was to be narcotized, a case divided into two chambers by the partition wall was used. The one was the narcosis

![Diagram A](image1.png)

**Fig. 1.** A) Arrangement for measurement of conduction velocity, B) Case for the narcosis experiment.

- **D**: partition wall.
- **E**: leading-off electrodes for the uninjured nerve surface.
- **I**: fixed leading-off electrode.
- **L**: chamber for the stimulating electrodes.
- **M**: mercury pool.
- **N**: nerve preparation.
- **R**: narcosis chamber.
- **S**: stimulating electrodes.
chamber and the other the chamber for the stimulating electrodes. As shown in fig. 1, $B$, six (or more) leading-off electrodes for the uninjured nerve surface were placed at distance of 3 mm. each and at the recording each of them was coupled in turn with another fixed leading-off electrode laid on the cut end of the preparation. In this case, after the narcosis was effected, the narcotic solution was exchanged for the Ringer's fluid, and after a while the Ringer's fluid in both chambers was sucked out. Thus the preparation was stimulated and the action potentials were recorded.

RESULTS

1) The conduction velocity in narcotized region

i) In the sciatic-tibial nerve preparation

Oscillograms obtained by the above described method are shown in fig. 2. The relation of the latent period (i.e. conduction time) and the length of nerve through which the action potential wave travels (i.e. conduction distance) is expressed as a curve in fig. 3. In this way the conduction velocity of the largest fibres ($\alpha$ or pre-$\alpha$ fibres) in each cm. of the preparation is clear at a glance as the gradient of the curve. The velocity before narcosis can be seen from fig. 3, a. In this case the curve is straight i.e. the velocity of the action potential traveling from peripheral to central part (centripetal conduction) is constant through the whole length. Furthermore, it is equal to that of the centrifugal conduction (not shown in fig. 3).

When the preparation was immersed in dilute narcotic solution, e.g. in 0.001% ammonia solution, the conduction velocity decreased a little, but it was constant through the whole length at both conductions. But in the case of stronger narcotic solution, e.g. 0.005% ammonia or 3% urethane solution, it was not constant in each part of the preparation. For example, the velocity in the recovery period from deeper narcosis is shown in fig. 3, b and c. At first the preparation was immersed in 0.1% ammonia solution for 10 minutes.

![Fig. 2. Oscillograms before and after narcotization. a: before narcotization, b and c: in the recovery period of narcosis by 0.1% ammonia solution. Each diagram includes six curves, which were obtained at different conduction distances. The direction of conduction is centripetal in a and c, and centrifugal in b. time: msec.]
FIG. 3. Nerve length: conduction time curve before and after narcotization by 0.1% ammonia solution.

a: before narcotization.
b: after narcotization, for centrifugal conduction.
c: after narcotization, for centripetal conduction.
†: stimulated point.

In this state the action potential had not been evoked by stimulation. Then it was immersed in 0.005% ammonia solution, and after almost stational state for this concentration was obtained, the measurement was performed. In fig. 3, b the velocity decreases abruptly from 12.5 m/sec. to 7.0 m/sec., the action potential wave travelling centrifugally from sciatic to tibial nerve. In the case of the centripetal conduction, as shown in fig. 3, c, it increases from 7.5 m/sec. to 12.0 m/sec. Both results indicate that the slowing and the quickning of the velocity is completely reversible and that each part of the preparation has a proper velocity. In many cases the velocity changed gradually and in a few cases abruptly at the tibial-fibular nerve branching or its central (1-2 cm.) neighbourhood. In short, the slowing which is shown in fig. 3, b is never caused by the decrement conduction in narcotized nerve but by the deepness gradient of narcosis.

In fig. 3, a, the straight line containing all measured points passes the stimulated point or its adjacent point, but in b and c the straight line which is drawn by combining four or two measured adjacent points crosses the ordinate at the more nearer point than the stimulated. The deeper the narcosis, the longer is the distance between the stimulated and the crossed point and the higher is the threshold. Hitherto this distance has been considered due to the spread of the stimulating current. But the supermaximal stimulation was always used in all cases. It is impossible to understand that only in b and c this current spread took place. It might be a plausible explanation that the limited length of excited part is required for conduction and this required length becomes longer in narcotized nerve than in physiological condition (Yamagiwa (13)). According to this assumption, the excitation starts from the stimulated point, and it would travel at decreased velocity at least in the adjacent region of the stimulated point. After all, it is difficult to determine whether the spread of the stimulating current actually occurs or not, and whether the decrement conduction exists or not at the adjacent region of the stimulated point.

ii) In the dorsal root–spinal nerve preparation

Now it is clear that the unequal deepness of narcosis depends not only upon the size of the fibres but also upon the parts of the preparation. Then the
author used the 9th dorsal root-9th spinal nerve preparation, the two parts of which were distinct in macroscopic appearance. Fortunately, the 9th spinal roots of the adult Japanese toad are longer than 4 cm. The dorsal root ganglion exists at the boundary of the two parts. The results are shown in fig. 4. When the preparation was immersed in 0.05% ammonia solution for 10 minutes, the velocity decreased from 20 m/sec. to 6.3–3.3 m/sec. in the dorsal root and to 10.0 m/sec. in the spinal nerve (fig. 4, c). Soon after the conductivity was lost in the dorsal root but maintained in the spinal nerve. In dorsal root the velocity decreases more rapidly at the central part than the peripheral, i.e. there is a gradient of sensitivity to narcotic solution. The results in recovery period are shown in fig. 4, d and e. The velocity in the dorsal root recovers faster but its absolute value is always smaller than that in the spinal nerve. In all cases the reversible slowing and quickening of the velocity were obvious. The velocity of each part at the centrifugal conduction was equal to that of the same part at the centripetal conduction.

In every case the velocity did not recover from deep narcosis to the initial value, i.e. that of the unnarcotized nerve. The cause of this incomplete recovery is probably the irreversible change occurring in the structure of the fibres due to the deep narcosis (Okada (14)). But from more dilute narcotic solution, e.g. 0.005% ammonia solution, the recovery was complete. Probably the effective concentration of the narcotic at the surface membrane of the axon may be considered to become not abruptly but gradually higher or lower, being hindered by the covering structures, the behaviour and the volume of which are different according to the parts of the preparation. Of course, one can not expect from these findings the explanation of the mechanism of narcotization. In the course of the narcotization the diffusion or the adsorption or the chemical process can play an important role.

II) The time delay in the dorsal root ganglion

The dorsal and ventral root-spinal nerve preparation was used. The relations between conduction distance \((d)\) and conduction time \((t)\) obtained by stimulating the ventral root and the dorsal root are shown in table 1, \(a\) and \(b\).

These two relations were plotted graphically (fig. 5). Each of them

![Fig. 4. Nerve length-conduction time curves on the dorsal root-spinal nerve preparation.](image)
TABLE 1

<table>
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<th>Ventral Root Was Stimulated</th>
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<td>(t) (msec.)</td>
<td>(d) (mm.)</td>
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FIG. 5. Conduction time-nerve length curves on the ventral (m) and dorsal (s) root-spinal nerve preparation.

\(a\): ventral root was stimulated.

\(b\): dorsal root was stimulated.

No time delay is found at the spinal ganglion \((D)\). Room temperature 10°C.

indicates a straight line. The straight line \(a\) and \(b\) correspond with the datum table 1, \(a\) and \(b\) respectively. In the straight line \(b\) the dorsal root ganglion \((D)\) is located. Namely, there is no time delay at \(D\). The conduction velocity of the ventral or dorsal root fibres is constant throughout the whole length of the preparation. By means of the least square method the equation of the line \(a\) \(d = 13.4 t + 0.28\) was obtained and for the line \(b\) the equation \(d = 15.2 t + 0.16\). So the velocity of the largest fibres in dorsal root (15.2 m/sec.) is larger than that of the largest fibres in ventral root (13.4 m/sec.).

This experiment performed repeatedly for thirty or more preparations but in all cases the time delay at the dorsal root ganglion could not be observed.

III) The change of the velocity at the boundary between normal and narcotized region

It can be seen in fig. 3 that the bending point of curve \(b\) is more peripheral than that of curve \(c\). To examine this hysteresis phenomenon exactly, the half length of the preparation was narcotized using the narcosis chamber. The results are shown in fig. 6. At first it was ascertained that the velocity was constant in both directions throughout the whole length of the preparation. The conduction in one direction is represented by a straight line \(a\) and the conduction in the opposite direction by a straight line \(a'\). After the conductivity
of the half length of the preparation was lost by immersing it in 0.1% ammonia solution, the narcotic solution was exchanged for the Ringer’s fluid, which was repeatedly replaced with new one. When the velocity of the narcotized part recovered appropriately and became stationary, the solution was sucked out and the nerve was stimulated. By this means the degree of the recovery varied scarcely during the experiment which required nearly 10 minutes. In fig. 6 the curve $b$ is the conduction when the action potential travels from normal to narcotized part, and the curve $b'$ is the conduction when it travels in the opposite direction. As in fig. 7 shown, the velocity calculated from the conduction time at every measured conducted distance was plotted against the conducted nerve length. Then $c$ and $c'$ in fig. 7 were obtained from $b$ and $b'$ in fig. 6 respectively. Now it is conspicuous that regarding the velocity the decrement is recognized in $c$ and the increment in $c'$. The decrement and the increment of the velocity

![Fig. 6. Conduction time-nerve length curves in recovery period of narcosis.](image)

The right half of the diagram is related to the narcotized part and the left half of it to the normal part.

$a$ and $a'$: before narcotization.

$b$ and $b'$: after narcotization.

toad, sciatic nerve, 21° C

![Fig. 7. The change of the conduction velocity at the boundary between normal and narcotized region of nerve.](image)

The curve $c$ and $c'$ were obtained from $b$ and $b'$ in fig. 6, respectively.

![Fig. 8. Nerve length-conduction time curve with the wave form of the action potential on sciatic nerve of bullfrog.](image)

$n$: narcotized region of 3 mm. length
The velocities on the both outsides of the narcotized region are equal, and diphasic wave is observed in both unnarcotized regions.
begin at the boundary between normal and narcotized region and continue further through the nerve length of 6-10 mm.

Finally it is shown in fig. 8 that the action potential travels slowly through the narcotized short region of nerve, but it is propagated at constant velocity on the both outsides of the narcotized region.

**DISCUSSION**

It is shown that the action potential wave in the sciatic-tibial nerve preparation propagated at a constant velocity through its whole length and the slowing of the conduction velocity occurring in narcosis is not caused by the decrement conduction but by the unequal deepness of the narcosis. In all previous studies the examination of the centripetal conduction was always excluded, because the twitch of the gastrocnemius muscle was observed in order to know the arrival of the excitation. The fact that the velocity at the centrifugal conduction is equal to that of the centripetal one in every part of the preparation verifies that the false decrement happens on account of the unequal deepness of the narcosis and not of the block of large fibres. It indicates rather the adequacy of the all-or-nothing law in each part of the nerve. So the theory of the decrementless conduction by Kato can not be abandoned.

Erlanger, Bishop and Gasser (10) reported that on the bullfrog the conduction time measured by observing the action potential (the sciatic nerve was stimulated) was shorter in the 8th ventral root than in the 8th dorsal root. They concluded that this time difference, which was about 0.14 msec., is due to the time delay at the dorsal root ganglion. But according to the previous study of Lenninger (15), this time difference could not be observed on the frog. The author observed in the 9th dorsal root of toad the greater velocity than that in the 9th ventral root. So from this fact and the constancy of the velocity in the dorsal root, it is evident that no time delay is detectable at the dorsal root ganglion. The velocity of the dorsal root in the 8th segment of the toad or the bullfrog was, however, in many cases larger than and often equal to the ventral root. In short, the time difference of both roots is due to the difference of the uniform conduction velocities.

The behaviour of the conduction velocity at the boundary between normal and narcotized region coincides with the results of Davis and his coworkers (7) who measured the amplitude of the action potential led off from nerve trunk. But it is entirely inadequate as an indicator of excitability or conductivity.

**SUMMARY**

On the sciatic-tibial nerve preparation or the root-spinal nerve preparation the conduction velocity of the action potential in each region was measured and the following results were obtained.

1. The conduction velocity in normal or lightly narcotized nerve is constant throughout the whole length of the preparation. The slowing which is often observed at the centrifugal conduction in deep narcosis is due to the unequal deepness of narcosis according to the parts of the preparation and not to the
decrement conduction, because at the centripetal conduction the quickning is observed.

2. No time delay is detectable when the action potential wave travels through the dorsal root ganglion.

3. At the boundary between normal and narcotized region the conduction velocity changes, namely it decreases when the action potential travels from normal to narcotized region and it increases when the action potential propagates in the opposite direction. The decrement and the increment of the conduction velocity begin at the boundary between normal and narcotized region and continue further through the nerve length of 6-10 mm.

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