Nagoya J. Med. Sci. 40: 13-23, 1978

POTENTIAL DEFLECTIONS AT THE TERMINAL OF THE FROG MUSCLE SPINDLE DURING STRETCH

FUMIO ITO and NAGAO YOKOYAMA

The Second Department of Physiology,
Nagoya University School of Medicine
(Director: Prof. Fumio Ito)

ABSTRACT

Dc-deflections were recorded with the paraffin gap method from the terminal of isolated single-type spindles in the frog sartorius muscle during ramp-and-hold stimulation. The amplitude of the dc-deflection over 60 μV was independent of the rate of afferent discharge and was little changed after inactivation of the axon terminal by irradiation with an ultraviolet light. The amplitude and the polarity of the dc-deflection could be altered with currents passing across the gap. A change in the longitudinal resistance of the axon in association with its lateral movement during stretch of the spindle may evoke a part of the dc-deflection by dividing a steady potential, although the dc-deflection below 60 μV in amplitude may be a genuine generator potential.

INTRODUCTION

Katz[1] has demonstrated that a depolarization (spindle potential) can be recorded from an isolated sensory axon at a point close to the terminal during stretch of the frog's fourth toe muscle which contains some spindle systems[2] or tandem-type spindles.[3] The spindle potential was satisfactorily explained by him as a transduction between mechanical input and the rhythmic output of impulses from the sense organ. Ottoson and Shepherd[4] showed spindle potentials of up to 4 mV or more in amplitude which were six times larger than those recorded by Katz,[1] and they considered them as a receptor potential in the frog's muscle spindle.

Previous experiments of the authors on single-type spindles in the frog sartorius muscle provide some paradoxical facts for dealing with the spindle potential as a generator potential as follows: 1) The amplitude of the spindle potential is dependent upon the diminution in number of extrafusal muscle fibers.[5] The result suggests that the spindle potential consists in part of unknown potentials which may be due to an injury current of muscle fibers spreading into the isolated spindle nerve. 2) Muscle stretch from a slack state often induces a positive spindle potential, during which some propagated spikes occur.[6] 3) The amplitude of the spindle potential depends upon a steady potential at the sensory nerve ending (usually $1-2$ mV positive in reference to the proximal portion of the axon); e.g., the spindle potential tends to become positive when the steady potential is driven negative by a catelectrotonic current,[7] by an excessive extracellular potassium concentration,[8] or by a hypertonic solution.[9] 4) Application of tetrametylammonium or EGTA (EDTA) gives rise to an increase or a decrease in the rate of discharge without discernible changes in the amplitude of dynamic and static components in the spindle

伊藤文雄，横山悠男

Received for Publication January 18, 1977

potential.[10] 5) The amplitude of the spindle potential decreases with increase in temperature,[11] while that of the receptor potential from Pacinian corpuscle parallels temperature change.[12]

It is the purpose of this paper to reevaluate the spindle potential and to verify the origin of the potential.

METHODS

The experiments were performed on twenty nine isolated muscle spindles in sartorius muscles of frogs (*Rana nigromaculata*) at room temperature, which varied from 24 to 27°C. The single parent axon of a spindle receptor was isolated along its intramuscular course until the capsule of the spindle receptor was cleared, but the capsule and its intrafusal muscle bundle were left attached to the remaining musculature.

The excised preparation was placed in a Ringer's pool in a perspex box, and the isolated nerve passed into another Ringer's pool through a liquid paraffin pool of 2 mm length. The paraffin pool was situated in a slit of 1 mm width at the center of a partition between the two Ringer's pools. The paraffin gap method was described in detail in an earlier paper.[13] A pair of calomel electrodes were inserted into subsidiary Ringer's pools, each of which was connected with the above two Ringer's pools perfusing the receptor and the parent axon by means of two Ringer's agar bridges. The responses at the sensory nerve terminal were displayed on the first beam of a dual beam oscilloscope through a high input-impedance amplifier. The second beam was used to display the tension of the muscle detected by a mechano-electric transducer (RCA 5734) during stretch of the muscle. The muscle was stretched by 1 or 2 mm at a velocity of 7 mm/sec from different initial lengths which were increments of 1 mm from an *in situ* length at which the muscle length was termed '+0 mm'. The term '+4 mm' means that the muscle was lengthened by 4 mm from the +0 mm.

In experiments for application of polarizing currents, a pair of calomel electrodes were placed into an arm of a bridge circuit,[7] through which potential differences across the paraffin gap were led and simultaneously polarizing currents were applied to the preparation. Direct currents of different intensities were delivered from an isolation unit of a stimulator. The bridge circuit was balanced with a direct current of subthreshold strength. The current applied to the preparation was monitored as a potential drop across a 10 KΩ resistor inserted between one of the electrodes and the input of current in the bridge.

In experiments for comparing responses recorded with the paraffin gap method and those with the unipolar recording which has been used to-date, a pair of non-polarizable electrodes of Ag-AgCl were employed. The nerve was lifted with one of the electrodes into paraffin oil covering the Ringer's solution perfusing the receptor. The other electrodes was inserted into the Ringer's solution.

In some experiments, the responses of isolated spindles were observed before and after inactivation of nonmyelinated terminal threads in the spindle capsule by irradiation with the ultraviolet light. The preparation was placed on a glass plate and covered gently with a quartz glass plate. The figure of myelinated branches in a spindle capsule was observed with an inverted microscope at a magnification of 200 X. The ultraviolet light was supplied by a mercury light source. The light was focused on a spot of approximately 1 mm in diameter on the object glass plate by means of quartz collective lenses situated at both entrance and exit diaphragms. The entrance diaphragm was set at 2 mm in diameter,

which was adequate to minimize the dispersion of the light. The exit diaphragm with an elliptical hole of 30 μ in diameter was moved over the quartz glass plate to a point along the nonmyelinated region. The microbeam of ultraviolet light irradiated from the top of the preparation through the exit diaphragm. It has been determined that the irradiation of ultraviolet light for 5 min duration suffices to inactivate the nonmyelinated terminal.[14] Following irradiation, the responses of the spindle terminal were recorded again under the same condition as in the control.

RESULTS

Relationship between the amplitude of muscle tension, the dc-deflection and the rate of afferent discharge

During the dynamic phase of stretch of the muscle by 1 mm at a velocity of 7 mm/sec from different initial lengths, the rate of afferent discharge increased from an initial value to peak at the end of the phase. In Fig. 1A, the initial and peak values are connected with a bar. The preparation responded with an increasing rate of discharge from 3 to 31 impulses/sec during muscle stretch of 1 mm from +0 mm initial length. The discharge rate decayed gradually during maintenance of the muscle length (+1 mm) and achieved a static level (12 impulses/sec in this case) approximately 6 sec after the peak. Keeping the muscle length (+1 mm), the preparation was displaced laterally by 1 mm in the Ringer's pool, because the isolated nerve must be moved laterally by approximately 0.5 mm in the paraffin gap during muscle stretch from +1 to +2 mm in analogy with the movement during stretch from +0 to +1 mm. The muscle stretch from +1 to +2 mm resulted in an increase of the discharge rate from 12 impulse/sec (the static rate at +1 mm) to 37.5 impulses/sec (the peak rate), as shown in Fig. 1A. Extension of the initial length increased the peak rate in parallel with the static rate.

Tension development recorded simultaneously during muscle stretch by 1 mm at 7 mm/sec in the same preparation as above was illustrated in Fig. 1B. The muscle stretch of 1 mm from +0 mm gave rise to a change in the tension from 0.05 to 0.2 g, which are connected by a bar in Fig. 1B. With extension from the initial muscle length, the amount of tension development during 1 mm stretching increased progressively and hence the length and the slope of the bar became longer and steeper.

The relationship between the tension development and the amplitude of the dc-deflection is shown as a function in Fig. 1C. The amplitude of the dc-deflection was measured

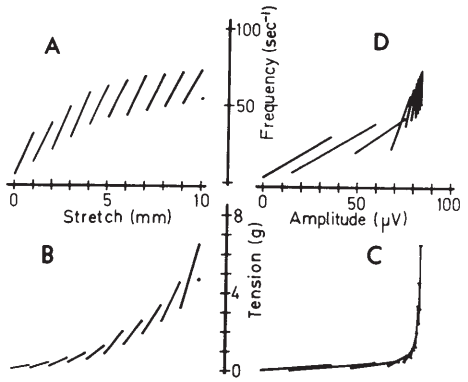

Fig. 1. Relationships between muscle stretch by 1 mm at 7 mm/sec from different initial lengths, the rate of afferent discharges, the tension development and the amplutide of the dc deflection. The data was obtained from an isolated spindle receptor. Each bar represents changes from the value of the initial state to the peak value at the end of the stretch.

from a base (at +0 mm being zero) to the peak or static deflections at the end of dynamic stretch or at a period 6 sec after the end. The amount of static amplitude during stretch of the muscle was also recognized as an initial amplitude for the next stage of the stretch. When the muscle was stretched from +1 to +2 mm, the tension changed from 0.1 to 0.4 g, while the amplitude of the dc-deflection increased from 15 to 60 μV. The amplitude of the dc-deflection increased almost linearly with increase of the tension until it attained 0.5 g, but in this case was saturated at a level of approximately 85 μV when the tension exceeded 0.5 g.

Fig. 1D represents a relationship between the amplitude of the dc-deflection and the rate of afferent discharge obtained from the same preparation as above. The muscle stretch from +0 to +1 mm induced a dc-deflection increase from 0 to 36 μV on which discharges increasing from 3 to 31 impulses/sec were superimposed, as shown by a bar in Fig. 1D. Bars with similar slope were also obtained when the muscle was stretched from +1 to +2 mm or from +2 to +3 mm. This implies that the changes in discharge rate during muscle stretch of 1 mm are proportional to the changes in the amplitude of the dc-deflection. That those bars do not form a line, however, suggests that the rate of discharge is not a function of the amplitude of the dc-deflection. This is supported by the fact that when the static amplitude of the dc-deflection exceeds 60 μV by extending the initial length over 3 mm the discharge rate can increase in spite of little increase in the amplitude of the dc-deflection during muscle stretch of 1 mm.

In Fig. 2, the peak rates of discharges are plotted versus the peak amplitudes of the dc-deflection in responses of a spindle receptor during muscle stretch by 1 or 2 mm at different velocities from initial lengths of +0 and +2 mm. In a range below 60 μV amplitude of the de-deflection, it was an approximately linear function of the discharge rate. However, with a dc-deflection larger than 60 μV the amplitude does not increase as the discharge rate increases. This result suggests that a dc-deflection below 60 μV in amplitude is possibly a receptor potential.

The dc-deflection before and after inactivation of the terminal by irradiation with ultraviolet light

Fig. 3 shows responses of a spindle receptor during stretch of the muscle by 2 mm

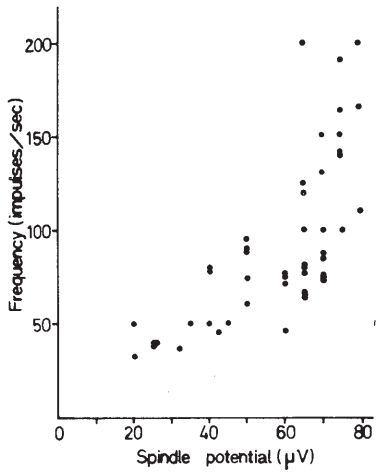

Fig. 2. Relationship between amplitude of the dc-deflection and the rate of afferent discharges in a spindle receptor, during muscle stretches of 1 to 2 mm at different velocities of stretch from the initial lengths of from +0 to +2 mm.

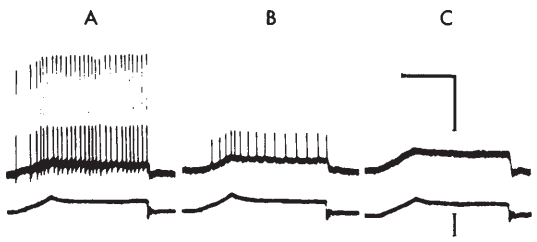

Fig. 3. Terminal responses of a spindle receptor (upper traces) before irradiation by an ultraviolet light (A), after inactivation of the first and second nodes of the subdivided myelinated branches in the spindle capsule by irradiation (B), and also after inactivation of all the terminals (C). Lower traces represent the tension developments during muscle stretch of 2 mm at 7 mm/sec from +0 mm initial length. Calibrations: 0.5 sec, 0.5 mV and 0.3 g.

from +0 mm at a velocity of 7 mm/sec, before (A) and after (B and C) inactivation of terminal branches. In Fig. 3A, propagated spikes occurred superimposed upon a dc-deflection which appeared almost in parallel with the tension development during the muscle stretch. Inactivation of nonmyelinated terminals and the subdividing node along subdivided branches in the same preparation as above left only propagated spikes with a small amplitude, because of a shunting effect in the inactivated subdividing node. During stretch of the preparation, the small propagated spikes occurred superimposed on a dc-deflection with an amplitude larger than normal (Fig. 3B). Further irradiation to the non-subdivided branches removed all spike responses without appreciable changes in the dc-deflection (Fig. 3C). Following inactivation of either or both the branches, no change in the dc-deflection was observed in 12 preparations out of 21, but a slight increase or decrease in the amplitude of the dc-deflection was detected in 6 or 3 preparations respectively.

These results suggest that the encoding process for initiation of an impulse may be blocked by the irradiation with ultraviolet light to the encoding site along the nonmyelinated threads, but the possibility that the activity of the transducer membrane may survive selectively cannot be excluded. However, it is considered likely that the dc-deflection may be an artifact and the irradiation of ultraviolet light may inactivate the transducer membrane together with the encoding site, because it has been observed in some preparations that the amplitude of dc-deflections increases with irradiation of the extrafusal muscle fibers.

Comparison of dc-deflections recorded by the paraffin gap method to those by a wire recording

A pair of nonpolarizable electrodes of Ag-AgCl were employed for recording the dc-deflection. One electrode was applied to the nerve which was lifted up in paraffin oil covering Ringer's solution, the other electrode being placed in the bath. When the distance between the spindle capsule and the Ringer-oil boundary was 3.5 mm or more, the axonal portion within 3 mm of the capsule moved laterally in Ringer's solution during the muscle stretch, but the proximal portions of the axon in the liquid paraffin did not move. Fig. 4A shows afferent discharges recorded in such a condition. The shape of each spike (Fig. 4Aa) did not differ largely from that recorded with the paraffin gap method (Fig. 4Ca) but the spike was neither followed by a positive after-potential nor superimposed on a dc-deflection (Fig. 4Ab and c). At the capsule-boundary distance of 1 mm, spike discharges occurred superimposed upon an appreciable dc-deflection during

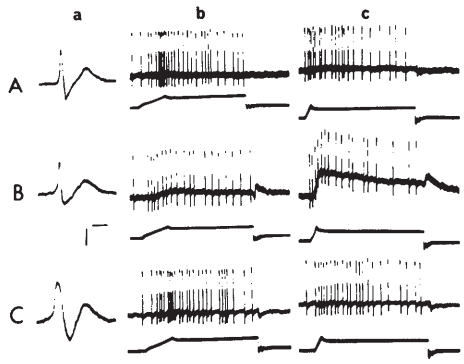

Fig. 4. Terminal responses of a spindle receptor recorded by a pair of Ag-AgCl electrodes (A and B) or by paraffin gap method (C), during muscle stretch of 2 mm at 7 mm/sec (b) or 35 mm/sec (c) from +0 mm initial length. The distance from the spindle capsule to the electrode was 3.5 mm in A and 1 mm in B. No significant differences in the shape of individual propagated spikes were detected in (a). Horizontal bar, 0.2 sec; vertical bar, 0.5 mV for upper traces and 0.3 g for lower traces.

the same amount of muscle stretch as mentioned above (Fig. 4Bb and c), while a transverse movement and some extensions of the axon along the Ringer-paraffin boundary could be detected under a binocular microscope. The amplitude of the dc-deflection was dependent upon the amount or the velocity of muscle stretch (Fig. 4Bb and c). There was no large difference in the shape of individual spikes recorded at the distance of 1 mm and those at 3.5 mm or more, except a prepotential in the rising foot of the spike (see Fig. 4Ba and Aa) which was distinguishable in the former but not in the latter.

The same preparation as mentioned above was also provided to record the terminal response by means of the paraffin gap method. When the gap of 2 mm in length was set up at an axonal portion 1 mm from the spindle capsule, spike discharges with a prepotential (see Fig. 4Ca) and a positive after-potential (see Fig. 4Cb and c) occurred superimposed on small dc-deflection during the same amount of muscle stretch, while the parent axon moved laterally by 0.4 mm at the center in the paraffin pool. The amount of transverse movement of the axon along the Ringer-paraffin boundary in the paraffin gap method should be analogous with that by the unipolar recording, because the capsule-boundary distance was equal in both recording methods. Consequently, the large amplitudes of the dc-deflections recorded by the wire recording method may be due to a movement of the wire electrode in the liquid paraffin or to an injury current from the central stump of the axon in the paraffin.

Dc-deflections modified by injury of the parent axon and by application of polarizing currents

Fig. 5 shows the response in an intact spindle receptor which has a positive steady potential of 1.2 mV at the terminal. Spike discharges occurred superimposed upon a negative dc-deflection of approximately 0.12 mV during muscle stretch by 2 mm from the *in situ* length. After crushing the second proximal node outside the capsule, which was located in a Ringer's pool distal to the paraffin gap, the steady potential shifted to 4.8 mV negative and all spike discharges were eliminated. In such a condition, the same amount of muscle stretch as mentioned above provoked a positive dc-deflection of 0.5 mV in amplitude (Fig. 5B). Decrease of the stretch was associated with decay in the amplitude of positive dc-deflection. Application of 0.4 % procain to the distal Ringer's pool abolished both the steady potential and the dc-deflection during muscle stretch, as shown in Fig. 5C. These results suggest that the polarity and the amplitude of the dc-deflection can be also dependent upon the injury current passing through the paraffin gap.

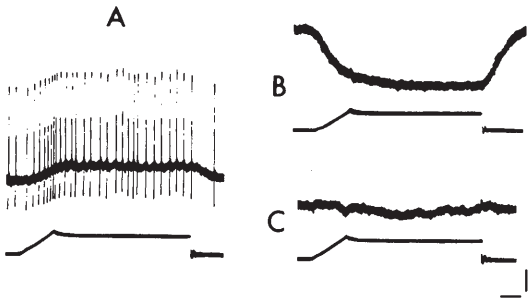

Fig. 5. Responses of a spindle receptor (upper traces) before (A) and after crushing the axon at the first node outside the capsule (B) and then after application of 0.4 % procain (C), and tension development (lower traces) during muscle stretch of 2 mm at a velocity of 7 mm/sec from +0 mm initial length. Horizontal bar, 0.2 sec; vertical bar, 0.2 mV for upper traces and 0.3 g for lower traces.

Fig. 6 shows effects of polarizations on the terminal responses of a spindle receptor during stretch of the muscle by 2 mm at 7 mm/sec from its *in situ* length. In a control in Fig. 6A, the muscle stretch gave rise to a transverse movement of the stem axon by 0.7 mm in the center of the paraffin pool while an acceleration of propagated spike discharges was superimposed upon an invisible dc-deflection. An anelectrotonic current of 2×10^{-8}A, which did not cause appreciable changes in the amplitude of discharge but suppressed slightly the rate of discharge,[7] was applied across the paraffin gap situated 0.5 mm from the spindle capsule. The same amount of muscle stretch as in Fig. 6A during the current passage resulted in a prominent negative dc-deflection of 0.4 mV in amplitude with spike discharges superimposed (Fig. 6B). The amplitude of the negative dc-deflection was dependent upon the amount of the anelectrotonic current. Some positive spikes which occur interposed between the negative propagated spikes may be initiated at a point along the axon in the proximal Ringer's pool and may conduct antidromically through the

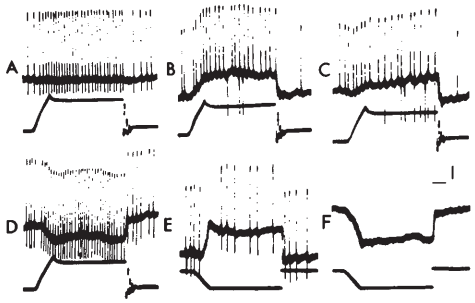

Fig. 6. Effects of anelectrotonic (B and C) or catelectrotonic currents (D) of 2×10^{-8}A applied on the terminal response of a spindle receptor (upper traces), in comparison with a normal response in absence of electrotonus (A), during muscle stretch of 2 mm at a velocity of 7 mm/sec from +0 mm initial length, which were represented by tension development (lower traces). E and F; responses (upper traces) of the same spindle receptor as that of A - D under catelectrotonic (E) or anelectrotonic current (F) of 5×10^{-8}A, during a transverse movement of 1 mm at 7 mm/sec, which was monitored by the driving voltage of the pen motor (lower traces). Horizontal bar, 0.2 sec; vertical bar, 0.2 mV for upper traces and 0.1 g for lower traces in A - D.

paraffin gap.[7]

Fig. 6D shows the response of the same spindle receptor as above when the muscle was stretched by 2 mm during application of catelectrotonic current of 2×10^{-8} A. The isolated axon was moved laterally by 0.7 mm around the center of the paraffin pool during the muscle stretch. A high rate of discharge of propagated spikes occurred superimposed on a positive dc-deflection of up to 0.3 mV. The amplitude of the positive dc-deflection was also a function of the amount of catelectrotonic currents.

In Fig. 7, the amplitude of the dc-deflection recorded from four preparations was plotted versus the steady potentials resulting from electrotonus. The marks with a small vertical bar in each preparation inducate the normal steady potential in the absence of electrotonic currents. These figures show that the amplitude of the dc-deflection is a linear function of the steady potential. Since the resistance across the paraffin gap is almost 2 MΩ in all of the preparations, it is calculated that the steady potential of 2 mV may be caused by a current of 1×10^{-9} A passing through the gap. The slope of the dc-deflection-steady potential relation curve indicates that the resistance across the gap may decay during the transverse movement of the axon at the center of the gap during muscle stretch, and also that the amount of reduction in the resistance may be approximately 0.2 MΩ.

When the axon was moved laterally by the same amount as mentioned above into a position distorted from the center of the gap, the amplitude of the dc-deflection was not always a linear function of the steady potential, and even the polarity of the dc-deflection was reversed. The record of Fig. 6C was obtained when the stem axon was moved laterally by 0.7 mm from the center toward a corner in the paraffin gap during the same amounts of muscle stretch as in Fig. 6B during anelectrotonic current of 2×10^{-8} A. Although the rate of propagated spike discharges increased almost in parallel with the tension enhancement during muscle stretch, no development of the dc-deflection in parallel with the tension change was observed (Fig. 6C).

One end of the muscle was detached from an anode pin of a transduce and the muscle was freely moved laterally by 1 mm at a slack state without extension in the Ringer's fluid, while the axon was moved laterally concurrently by 1 mm in the paraffin gap. During application of catelectrotonic current of 5×10^{-8} A, the movement of the axon from one corner to another in the paraffin gap produced a negative deflection like a spindle potential on which several spikes were superimposed, as shown in Fig. 6E. As the muscle was not fixed, the axon appeared to be stretched during the course of its transverse

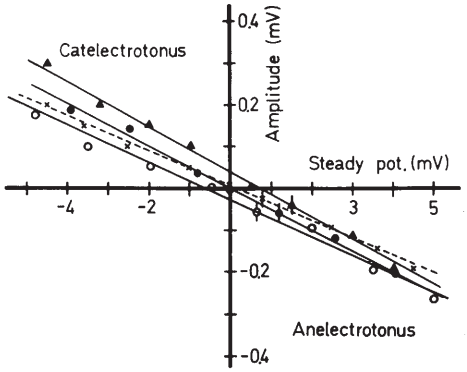

Fig. 7. Four examples of the linear relation between the amplitude of the dc-deflection and the steady potential which was altered by electrotonic currents. Marks with a small vertical bar represent the normal steady potential in absence of electrotonus.

movement. The rate of the propagated spikes during the negative deflection was not different from that without movement or was slightly lower. On the lower trace of the figure, a driving voltage for the stretcher was displayed instead of muscle tension because the muscle was not stretched. Application of anelectrotonic current of 5×10^{-8} A to the spindle terminal caused a positive deflection like a spindle potential but abolished spike discharges (Fig. 6F).

The above results suggest that the displacement of the axon in the paraffin gap may cause a change in the external longitudinal resistance of the axon. The reduction of the longitudinal resistance may result from a transverse movement of axon around the center in the paraffin gap, while an increase of the resistance may be due to a stretch of the axon during its movement in the corner of the gap.

DISCUSSION

The finding on a single-type spindle in the frog sartorius muscle that the amplitude of a dc-deflection of less than 60 μV is a function of the rate of afferent discharges is in a line with the result on a complex-type spindle in the frog toe muscle by Katz.[1] He demonstrated a proportional relationship between the rate of discharge and the amplitude of the spindle potential of less than 0.6 mV. Though dc-deflections of up to 150 μV in amplitude were sometimes recorded from the single-type spindle under normal conditions by the paraffin gap method, the amplitude of the dc-deflection appeared independent of the rate of afferent discharges (see Fig. 2), and also such a dc-deflection remain unchanged even after elimination of spike activities by irradiation with ultraviolet light of the terminal nonmyelinated filaments (see Fig. 3). These differences in the terminal responses between the single-type spindle and the complex-type spindle may be due to the difference in the number of ramifications of the myelinated axon in the spindle capsule or to the difference in the recording method. In the single-type spindle, the stem axon divides into two branches in the capsule, one of which subdivides into two short myelinated branches but the other does not, and their first nodes emit nonmyelinated filaments.[14] Ottoson and Shepherd[15] have drawn a schematic diagram of the terminal ramification of the axon in the complex-type spindle, in which the first 16 segments are myelinated.

The terminal response of the single-type spindle was recorded through a pair of calomel electrodes across a paraffin gap of 2 mm in length which was situated at a point along the stem axon within 0.5 mm from the spindle capsule. The proximal part of the sciatic nerve of approximately 20 mm in length was immersed in another Ringer's pool separated from that of the receptor side. The response of the complex-type spindle was recorded with calomel half-cell electrodes connected to the preparation, in which the dissected sensory axon of about 2 mm in length was lifted with an Ag-AgCl electrode into liquid paraffin covering the Ringer's solution.[4] In the present study, the terminal response in the single-type spindle recorded by means of a pair of nonpolarizable Ag-AgCl electrodes also consisted of a large deflection of a like spindle potential of up to 0.8 mV in amplitude (see Fig. 4Bc). This suggests that the large potential deflection may not be due to the difference in the preparations but be attributed to the difference in the recording method.

From the present experiment, it is possible to suppose that the external longitudinal resistance may be changed in association with changes in the length and thickness of a thin layer of Ringer's solution intervening between the axon and the liquid paraffin during stretch of the muscle. The resistance was reduced by approximately 10 % of its resting

value (nearly 2 MΩ) during transverse movement of the axon around the center of the paraffin gap, while it was increased when the axon was stretched. It seems likely that recording of terminal response by means of Ag-AgCl electrodes may provide a large resistance change between the two electrodes because the electrode appears to be often moved in the paraffin oil during muscle stretch. When the steady potential of a normal spindle receptor was cancelled by application of electrotonic current with an inversed polarity, no dc-deflection could be observed. It is concluded that the dc-deflection in the frog single-type spindle can be modified artificially by application of electrotonic currents or by injury current of the axon.

CONCLUSION

Dc-deflections recorded from isolated single-type spindles in the frog sartorius muscle were reevaluated with the paraffin gap method. The rate of afferent discharge increased proportionally with amplitude of the dc-deflection in a range below 60 μV, but appeared to be independent of amplitudes over 60 μV.

The amplitude of the dc-deflection was little changed after inactivation of the first node of the axon by irradiation with an ultraviolet light which removed all spike discharges.

The amplitude of the dc-deflection recorded from an axonal region near the capsule with a pair of Ag-AgCl electrodes was always larger than that with the paraffin gap method. The amplitude and the polarity of the dc-deflection could be altered with applications of different strengths of cat- or anelectrotonic currents or of injury currnet by crushing the axon and also with the position of the axon which was moved laterally in the paraffin gap during muscle stretch.

The measurement of resistance across the paraffin gap indicated a change of approximately 10 % in the longitudinal resistance of the axon in association with its transverse movement. The resistance change may evoke a potential deflection like the spindle potential by dividing a steady potential across the gap, with the possibility that the dc-deflection below 60 μV in amplitude may be a genuine generator potential.

ACKNOWLEDGEMENTS

A part of this work was supported by a Research Grant from the Ministry of Education 287014.

REFERENCES

1) Katz, B., Depolarization of sensory terminals and the initiation of impulses in the muscle spindle. *J. Physiol.*, **111**, 261–282, 1950.
2) Gray, E. G., The spindle and extrafusal innervation of a frog muscle. *Proc. Roy. Soc.*, **146**, 416–430, 1957.
3) Barker, D. & Cope, M., Tandem muscle-spindle in the frog. *J. Anat.*, **96**, 49–57, 1962.
4) Ottoson, D. & Shepherd, G. M., Transducer properties and integrative mechanisms in the frog's muscle spindle. In *Handbook of Sensory Physiology*, **1**, 442–499, Edited by Loewenstein, W. R. Berlin: Springer-Verlag. 1971.
5) Ito, F., Spindle potential of the frog muscle spindle depending upon the exclusion of extrafusal muscle fiber. *J. Physiol. Soc. Jap.*, **32**, 249–250, 1970.
6) Ito, F., Off-response from frog muscle spindles. *J. Physiol. Soc. Jap.*, **30**, 753–754, 1968.
7) Ito, F., Effects of polarizing currents on long lasting discharges in the frog muscle spindle. *Jap.*

J. Physiol., **20**, 697–710, 1970.
8) Ito, F., The effect of ions on the steady and generator potential of frog muscle spindles. *Proc. Jap. Acad.,* **45**, 409–412, 1969.
9) Ito, F., The behavior of frog muscle spindle in hyper- and hypotonic solutions. *Jap. J. Physiol.,* **20**, 394–407, 1970.
10) Ito, F. & Kuroda, H., Drug-induced modulation of the positive after-potential following the propagated impulses and of the discahrge pattern in the frog muscle spindle. *Jap. J. Pharmacol.,* **22**, 551–560, 1972.
11) Oyama, O., The effects of temperature on the dynamic and static sensitivities of the frog muscle spindle. *Nagoya J. Med. Sci.,* **33**, 13–25, 1970.
12) Loewenstein, W. R., Mechano-electric transduction in the Pacinian corpuscle. Initiation of sensory impulses in mechanoreceptors. In *Handbook of Sensory Physiology,* **1**, 269–290, Edited by Loewenstein, W. R. Berlin: Springer-Verlag. 1971.
13) Ito, F., Abortive spikes of the frog muscle spindle. *Jap. J. Physiol.,* **19**, 373–391, 1969.
14) Kuroda, H. & Ito, F., Functional differentiation of sensory terminal branches in the frog muscle spindle. *Proc. Jap. Acad.,* **48**, 206–209, 1972.
15) Ottoson, D. & Shepherd, G. M., Synchronization of activity in afferent nerve branches within the frog's muscle spindle. *Acta Physiol. Scand.,* **80**, 492–501, 1970.