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CERVICAL BRANCHING OF LUMBAR VESTIBULOSPINAL AXONS

By C. ABZUG, M. MAEDA, B. W. PETERSON and V. J. WILSON

(with an Appendix by C. P. Bean)

From the Rockefeller University, New York, N.Y. 10021, U.S.A.

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SUMMARY

1. We have investigated the possibility that individual lateral vestibulospinal tract (LVST) axons branch so as to innervate more than one spinal cord level.

2. LVST cells in Deiters' nucleus were activated antidromically by means of electrical stimulation applied through fine metal electrodes inserted into the spinal cord. Both by directly measuring the spread of effect of stimulus current, and from theoretical considerations (Appendix), we determined that in most cases an estimate of spread of effect of stimulus current was 10 μm/μA. From the magnitude of the threshold stimulus and from the location of the stimulus point we could often exclude the possibility that the stimulus was spreading to the LVST instead of activating local branches.

3. Movable stimulating electrodes, or multi-electrode arrays placed in fixed position, were used to activate 115 LVST neurones antidromically by stimulation of local branches in the lower cervical or upper thoracic cord. Of these cells, 50 % were also fired antidromically by stimulation of the LVST at levels ranging from L1 to L4 (L_c cells). The remaining cells were not activated by the lumbar stimulus (C cells). An additional group of cells was only fired by the lumbar tract stimulus (L cells).

4. The distribution of locations of L_c cells within Deiters' nucleus more closely resembles that of L cells than that of C cells. In addition the median conduction velocity of L_c cells is similar to that of L cells, but higher than that of C cells.

5. Much of the information reaching the lower cervical level from neurones of the LVST is information that is also simultaneously being passed downward to the lumbar region. Such integration makes it possible for a single neurone to be used to co-ordinate widespread motor activity.

6. A theory is presented in a separate section (Appendix) to account for the spread of effect of stimulus current upon a myelinated axon submerged in an isotropic medium. The threshold for stimulation of a node by a nearby monopolar electrode is predicted to be proportional to the electrode-node spacing. The constant of proportionality is given in a closed form that depends on the electrical properties of both the neurone and the surrounding medium. The predictions of the theory are shown to be in good accord with the experimental results.

INTRODUCTION

There has been extensive investigation in the cat of the cells of Deiters' nucleus that project to the spinal cord in the lateral vestibulospinal tract (LVST). To some extent, the cells are arranged somatotopically: dorsocaudally in the nucleus there is a preponderance of cells projecting to the lumbosacral cord, while rostroventrally cells projecting to the cervical cord predominate (Brodal, Pompeiano & Walberg, 1962; Wilson, Kato, Peterson & Wylie, 1967). Afferent inputs to the nucleus are also distributed unevenly. For example, fibres from the labyrinth act mainly ventrally (Walberg, Bowsher & Brodal, 1958; Wilson *et al.* 1967) whereas fibres from the anterior lobe of the cerebellum act mainly dorsally (Brodal *et al.* 1962; Ito, Kawai & Udo, 1968). This pattern of input, coupled with the somatotopic distribution of LVST neurones, implies that different parts of Deiters' nucleus may serve different functions. On the other hand, the somatotopic organization of Deiters' nucleus is rather blurred: there is considerable intermingling of cells projecting to different levels of the spinal cord, as well as some overlap of inputs (Fanardjian, Sarkissian, Sargsian & Pakhlevanian, 1972; Wilson, 1972). There is another factor that should be considered. The cells in the 'hind limb area' of Deiters' nucleus have been tacitly assumed to influence only the lumbosacral spinal cord. The possibility exists, however, that axons extending that far caudally also branch more rostrally and therefore influence more than one level of the spinal cord. If so, then this branching of vestibulospinal axons would confuse the somatotopy still further. Such branching would be appropriate functionally, because some reflexes mediated by the vestibulospinal system involve combined activation of the musculature of the neck, trunk and limbs.

We have investigated the branching of vestibulospinal axons in the cat spinal cord by means of local antidromic stimulation. First, it was necessary to perform experiments to determine, for our stimulating conditions, how much current could be applied locally without activating axons in the tract itself. Using criteria resulting from these experiments, we will

show that many Deiters' cells projecting to the cervical cord also reach lumbar levels. Some of the results have been reported in a preliminary communication (Abzug, Maeda, Peterson & Wilson, 1973).

METHODS

Experiments were performed on adult cats prepared under pentobarbital anaesthesia (Nembutal; Abbott Laboratories), paralysed with gallamine triethiodide (Flaxedil; Davis & Geck) and respired artificially. A cannula was inserted into one of the femoral arteries, and mean arterial pressure was monitored by means of a mercury manometer. When necessary, metaraminol bitartrate (Aramine; Merck, Sharp & Dohme) was administered by I.V. infusion to maintain an arterial pressure of 80 mmHg or more.

The cerebellum was removed by suction, and micropipettes were inserted into Deiters' nucleus for recording extracellularly the action potentials of single neurones. The micropipettes were filled with 2M-NaCl saturated with Fast Green FCF and had resistances of 1–4 MΩ. Dye was ejected electrophoretically from the recording micropipettes at several appropriate positions and the dye marks were later used to determine the location of the electrode tip according to the method of Thomas & Wilson (1965). The animal was perfused with 10% formol saline, the brain stem was removed, and after further fixation 100 μm frozen transverse sections were cut in the plane of the electrode tracks. The sections were stained by the technique of Klüver & Barrera (1953), and histological reconstruction was used to determine the locations where cells were recorded. The positions of the tips of the metal electrodes used for stimulation in the spinal cord were marked by lesions made by passing 20 μA of cathodal current through each electrode for 20 sec. The locations of these lesions were subsequently determined in thionin-stained sections.

Stimulating procedures

Following dorsal laminectomies stimulating electrodes were inserted, as illustrated in Fig. 1, into the ventral white matter of the lumbar cord to activate the main body of the LVST and into the grey or white matter of the cervical enlargement to active branches of LVST axons.

Local stimulation: Spread of stimulus current. In order to measure the spread of stimulating current from the electrode tips, fine stimulating electrodes were moved through the spinal cord. The stimulating electrode used was one of two types: (i) a tungsten wire, electrolytically etched to a point and insulated with lacquer to the tip, with the insulation scraped off over a length of 50–200 μm from the tip; (ii) a glass-insulated tungsten electrode (Stoney, Thompson & Asanuma, 1968). Electrodes of both types had resistances of 100 kΩ or less. The stimulating electrode was first inserted stereotaxically into a selected portion of the spinal gray matter. A constant-current stimulator was used to apply cathodal pulses of 100 μsec duration at a rate of 5/sec through the stimulating electrode with respect to a distant indifferent electrode, and simultaneously a recording electrode was advanced through Deiters' nucleus. Recording tracks were made systematically throughout Deiters' nucleus in a checkerboard pattern, with a 300 μm rostro-caudal and mediolateral separation between tracks.

When unitary action potentials were recorded from a responding neurone it was then determined whether the neurone was responding antidromically as described previously (Abzug *et al.* 1973). In brief, most neurones encountered had a sharp threshold, responding with essentially constant latency to stimuli presented over

a range of intensity from near threshold to several times threshold, and all those that were tested were able to follow high-frequency repetitive stimulation. If a neurone fired spontaneously, then a check was made of the interval over which the occurrence of a spontaneous action potential blocked the appearance of a subsequent stimulus-evoked impulse, to see whether the cause of the blockage could be ascribed to collision of orthodromic and antidromic impulses (Darian-Smith, Phillips & Ryan, 1963). Almost all neurones responding to the spinal cord stimulus were found to be antidromically activated. After this was determined for each neurone, the stimulating electrode was moved up and down in its track, and the threshold of the neurone was measured at a series of points (Stoney *et al.* 1968; Jankowska & Roberts, 1972). Marking lesions were made at one or more locations in many tracks.

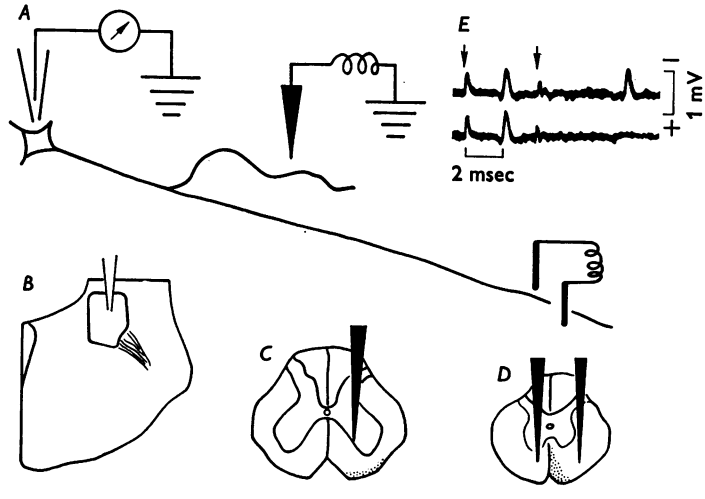

Fig. 1. *A*, schematic representation of the experimental arrangement. A vestibulospinal neurone of the type labelled L_c in the text is illustrated. A cervical branch of the neurone is antidromically activated by stimulation applied monopolarly at the cervical level, and the axon is also activated by means of a bipolar stimulus applied through the lumbar tract electrode situated at the upper lumbar region. *B*, *C* and *D* are schematic cross-sections through the recording site in Deiters' nucleus, and through the stimulus sites at the cervical and lumbar levels. The inset in *E* is the response of a representative L_c neurone to a pair of stimuli (arrows). The first stimulus was delivered monopolarly at the mid-cervical level at a strength of 1·5 times threshold, which was 27 μA. The second stimulus was applied bipolarly at the lumbar level. At a condition-test interval of 3·0 msec (upper trace) a response occurred to the lumbar stimulus, but when the interval was shortened to 2·9 msec (lower trace) the test response failed to appear.

Local stimulation through fixed electrodes. In some experiments stimulation was applied through electrodes that were placed in fixed positions in the spinal grey matter, while a search was made with a recording electrode in Deiters' nucleus to find as many antidromically activated neurones as possible. The electrodes were

arranged in two fixed linear parallel arrays, each containing three to five lacquer-insulated tungsten electrodes separated by a distance of 2·5 mm and oriented in a parasagittal plane. When a neurone was located in Deiters' nucleus that responded antidromically to the simultaneous application of current through all of these electrodes, then current was passed through one electrode at a time to identify the effective electrode(s) and determine threshold for each electrode.

Lumbar tract electrode. In all experiments we stimulated the LVST in the upper lumbar region to test whether Deiters' neurones activated antidromically from the cervical spinal cord also projected caudally to the lumbar cord. The lumbar tract electrode consisted of two tungsten wires insulated except for a distance of 0·3 to 1·0 mm from the tip. This electrode was inserted vertically into the spinal cord in the transverse plane at the level L1 to L4. One pole was just a little to the contralateral side of the midline from the recording electrode, and the other pole was 2 mm away on the ipsilateral side.

If a cell was fired by both the cervical and lumbar tract electrodes collision between the two impulses was studied to provide additional evidence that both were antidromic (Fig. 1*E* and Abzug *et al.* 1973).

RESULTS

Measurement of stimulus spread

Axons of the LVST pass through the ventrolateral region of the spinal cord at the cervical level while on their way down to lumbar levels, and therefore it was necessary to eliminate the possibility that an electrical stimulus might be effective in activating not a local terminal or collateral but instead an axon situated in the tract. Determination of the maximum safe values of stimulus current that could be used without spread to the tract was made by comparison of the results of three kinds of experiments. First, the stimulating electrode in the ventral horn grey matter was moved up and down, and the threshold for activation of a single vestibulospinal neurone was recorded as a function of distance in the dorsoventral direction (cf. Jankowska & Roberts, 1972; Jankowska & Smith, 1973; Roberts & Smith, 1973). Secondly, the threshold was compared among several locations in the horizontal plane determined from parallel vertical tracks of the stimulating electrode (*op. cit.*). And, finally, the current required for spread to LVST axons from fixed electrodes was measured directly.

Vertical tracking. Representative results for vertical movement of the stimulating electrode are shown in Fig. 2*A* for several vestibulospinal neurones. In order to minimize the effect of tissue drag, the electrode was inserted beyond the point of lowest threshold, and then the threshold was recorded during withdrawal. The data shown in Fig. 2*A* were treated in accordance with a theoretical formulation governing the spread of stimulus current that was developed by Charles P. Bean (Appendix). The result of this treatment is shown in Fig. 2*B*, in which the threshold of the neurones of Fig. 2*A* is replotted as a function of the calculated

distance from the stimulating electrode to the point of action of the stimulus.

Fig. 2 illustrates our finding that in most cases the spread of effect of stimulus current is of the order of 10 μm/μA. In the two instances where the stimulus current appears to spread farther than this (indicated by the open diamonds and filled circles) there may have been more than one low-threshold point within the range of the stimulating electrode, so that even though the electrode was moved progressively along its track the distance over which the stimulus current acted changed relatively little (cf. Appendix, Fig. 6).

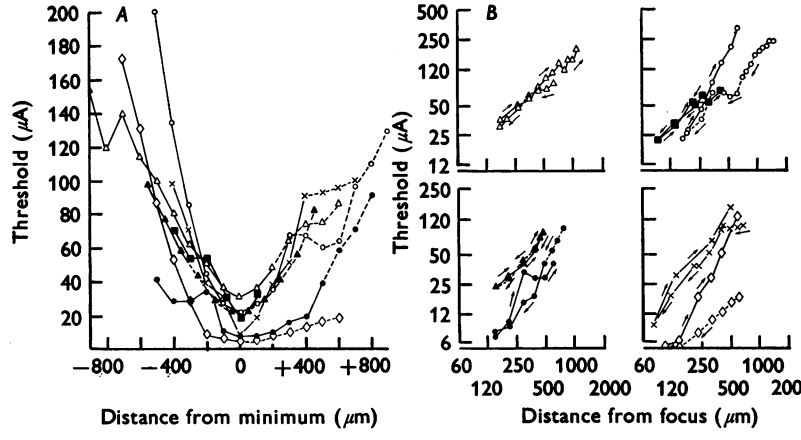

Fig. 2. Threshold (ordinate) as a function of distance (abscissa) during vertical tracking of the stimulating electrode. *A*, distance along the track of the electrode is plotted on the abscissa relative to the point of minimum threshold, which was assigned a value of zero. Positive values of distance indicate points ventral to the minimum, and negative values of distance indicate dorsal points. The portion of each curve indicated by a dashed line was obtained while tracking through the ventral or ventrolateral funiculus. Two of the tracks shown (× 's and open diamonds) were obtained for one neurone. *B*, data of *A* treated according to a theoretical model for spread of stimulus current (details in Appendix). The distance plotted on the abscissa is the calculated distance from the neural element being stimulated. Both abscissa and ordinate are in logarithmic co-ordinates. Arrows indicate the sequence of data collection from ventral to dorsal.

Low-threshold points in adjacent tracks. To supplement the results obtained with vertical tracking we also compared the lowest threshold achieved along each of several parallel tracks of the stimulating electrode. For each cell the lowest threshold in adjacent tracks occurred at about the same dorsoventral level, and therefore at least to a first approximation the set of lowest-threshold points for each neurone was situated in the

horizontal plane. The relationship between threshold and distance in the horizontal plane was not appreciably different from that observed during vertical tracking.

Direct measurement of spread of stimulus effect to the vestibulospinal tract. The results so far suggest that the spread of effect of stimulus current in most cases is less than 10 μm/μA. To confirm this estimate, a direct measure was made of the spread of effect of stimuli applied in the grey matter to axons of the LVST. For this purpose during several experiments a series of stimulating electrodes was placed in fixed position in the lumbar spinal grey matter. Two arrays of five lacquer-insulated tungsten stimulating electrodes were inserted into the lumbosacral region of the spinal cord in a parasagittal plane, both at a selected distance from the midline and at a selected depth calculated to place the tips near the extensor motoneurone nuclei. Then the threshold of each antidromically activated neurone was determined for stimulation through each of the ten electrodes.

The results for three representative cases are shown in Fig. 3. Note that for each neurone there is at least one electrode location where the threshold is less than 100 μA, presumably indicating stimulation in the ventral horn of a branch of an LVST axon. Two of the neurones selected for illustration have such thresholds from one of the more caudal electrode sites, thus indicating that the axon travels down at least as far caudally as this low-threshold region. Although the lowest caudal threshold for the neurone indicated by open circles in Fig. 3 is more than 100 μA, the axon must have descended at least to this region, because it is not possible that a threshold current of 200 μA can spread to activate an axon located more than 20 mm more rostrally.

Histological reconstruction was done to establish the placement of each of the stimulating electrode tips. The distance was then measured from each point of stimulation to the *farthest* region of the LVST at that rostrocaudal level, determined from the figures of Nyberg-Hansen & Mascitti (1964) and of Petras (1967). On the assumption that the activated axon was lying in this farthest region of the tract a maximum predicted value of threshold for each electrode was then calculated based on a spread of stimulus current of 10 μm/μA. These maximum predicted thresholds are also shown in Fig. 3. It is apparent that the actual values are often much higher than the maximum predicted values and that the spread of effect of stimulus current from points in the anterior horn grey matter to axons of the LVST occurs at a rate of less than 10 μm/μA.

Summary of stimulus spread

The results of the measurement of stimulus spread are summarized as follows: a value of 10 μm/μA was accepted as a conservative maximum estimate of the spread of effect of stimulus current to axons of the LVST. Therefore, in all of the cases presented below, where it was important to establish that stimulation was acting on a local branch or terminal of

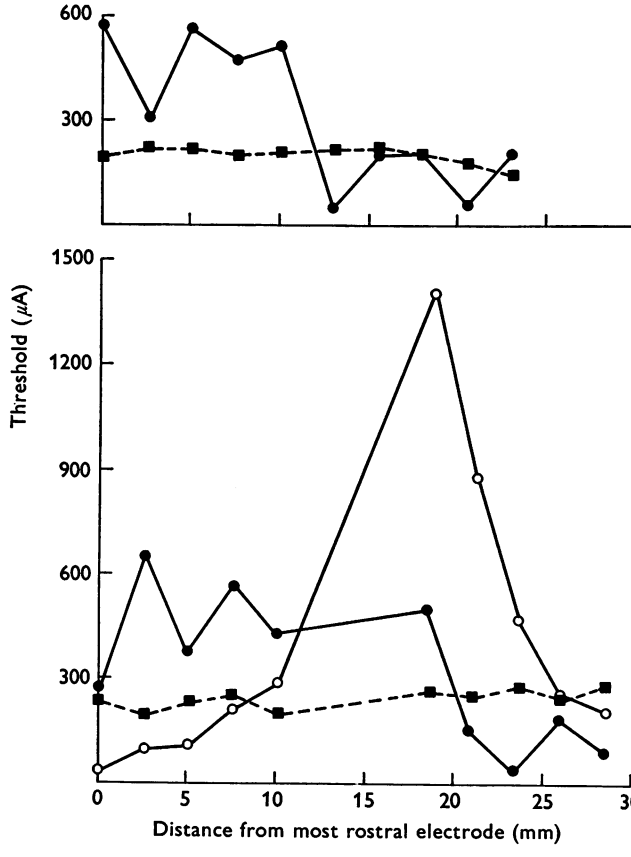

Fig. 3. Measurement of current spread to the LVST. Ten stimulating electrodes were positioned in the ventral horn as described in the text. Continuous lines in the graphs indicate thresholds for activation of three L_c cells from each electrode (ordinate). The abscissa indicates the distance of each electrode from the most rostral one. The values of the maximum predicted threshold from all electrode locations were calculated as explained in the text and are illustrated by filled squares connected by dashed lines. The cases illustrated here were taken from two experiments, and the data from each experiment appear on a separate plot along with the maximum predicted thresholds for that experiment.

a vestibulospinal neurone and not on the main vestibulospinal axon, measurement was made of the distance from the tip of the stimulating electrode to the nearest portion of the vestibulospinal tract as delineated by Nyberg-Hansen & Mascitti (1964) and by Petras (1967). If this distance was more than 10 μm/μA of threshold current then stimulus current was considered to have been activating a local terminal or collateral branch of the vestibulospinal neurone.

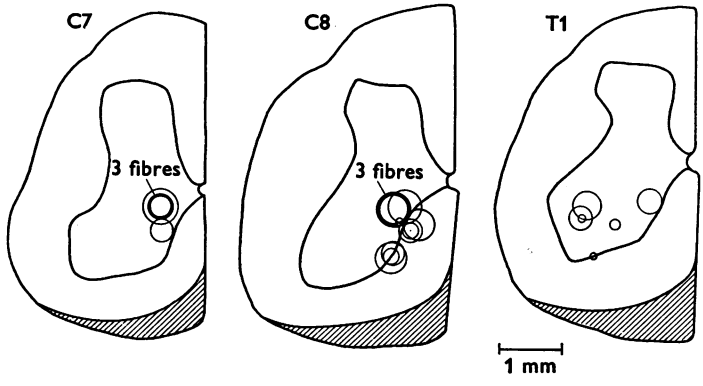

Fig. 4. Stimulus points from which axons of L_c cells could be excited with thresholds of 30 μA or less. Points from which 21 L_c cell axons could be excited have been superimposed on three schematic cross-sections of the cervicothoracic spinal cord at the levels indicated. (One neurone was excited from two different electrode locations and is therefore represented by two stimulus points.) Each point is represented by a circle with radius (in μm) equal to ten times the threshold current (in μA). The hatched area indicates the location of the lateral vestibulospinal tract. Where more than one L_c cell was excited from an individual electrode location, concentric circles have been drawn around the same centre point. Labels in the diagram indicate two cases where three fibres with very similar thresholds were activated from the same point.

Branching of vestibulospinal neurones

Many cells of the LVST that projected to the lumbar spinal cord were also activated from electrodes placed in the lower cervical and upper thoracic spinal cord, at levels ranging from C6 to T2. Earlier terminology classified cells projecting to the cervical or lumbar regions as C and L cells respectively (Wilson *et al.* 1967). In the present experiment we have called those L cells for which we found cervical collaterals L_c cells. We have retained the term L cells for those neurones projecting to the lumbar cord for which we did not find branches, and the term C cells for those neurones projecting to the cervical region which were not found to project to the lumbar cord.

In many experiments the stimulating electrode tips were positioned in the ventromedial part of the cervical or thoracic cord – either within the medial part of the ventral horn or in the ventromedial white matter just adjacent to the ventral horn, a region through which many LVST fibres pass on their way into the ventral grey matter (Nyberg-Hansen & Mascitti, 1964). Weak stimuli applied to electrodes in this region produced antidromic activation of numerous Deiters' neurones. Deiters' neurones were also sometimes activated antidromically from more lateral sites, some of which were within the motor neurone nuclei. For all these instances in which the stimuli were judged to be activating branches of LVST axons (see below) threshold ranged from 2·5 to 92 μA; 58 % of the fibres had thresholds less than 50 μA, and 4 % less than 10 μA.

The locations of stimulus points from which the twenty-one lowest-threshold (2·5–30 μA) axons of L_c cells were activated are shown in Fig. 4. Each stimulus location is represented by a circle with a radius (in μm) equal to ten times the stimulus threshold in μA, indicating the maximum extent of stimulus spread from each point calculated according to the measurements of stimulus spread described above. The extent of spread makes it extremely unlikely that the fibres being activated were located within the LVST, whose approximate location, estimated from the results of Nyberg-Hansen & Mascitti (1964) and of Petras (1967), is indicated by the hatched areas in the Figure.

Further evidence that stimuli were not spreading to the LVST was obtained for several neurones. In these cases vertical movement of the stimulating electrode tips indicated that the threshold for exciting an individual axon was lowest in the ventral grey matter and increased markedly as the electrode tip was advanced deeper into the ventral white matter (see for example Fig. 2*A*).

Although we have concentrated on the hazard of stimulus spread to the LVST, the possibility of spread to the medial vestibulospinal tract (MVST) also exists. We believe that such spread is not a significant factor in our experiments for several reasons. First, although electrophysiological evidence suggests that in the cat a few vestibular neurones projecting into the MVST may reach lumbar levels (Precht, Grippo & Wagner, 1967), anatomical evidence restricts the tract to the rostral half of the cord (Nyberg-Hansen, 1964; Petras, 1967). Therefore, it is highly unlikely that any of our L_c cells were activated antidromically by spread to the MVST. Secondly in the cervical cord the MST is located along the anterior median fissure in the dorsal three-fourths of the ventral funiculus (Nyberg-Hansen, 1964). This location was usually sufficiently far removed from our stimulating positions to avoid the possibility of spread. Thirdly, and most important, is the location in Deiters' nucleus of all the cells that we studied. Although there has been general agreement that in the cat MVST cells are located in the medial and descending vestibular nuclei (see Wilson, 1972), Akaike (1973) has recently suggested that some MVST cells are in Deiters' nucleus. They are, however, apparently situated caudally and ventrally in the nucleus, in areas close to the descending and medial nuclei (Fig. 1 in

Akaike, 1973). Only a small part of our sample is to be found in areas that might contain MVST neurones. Taking all of these considerations together, we conclude that most if not all of our neurones are LVST cells.

A measure of the likelihood that a given stimulus might be activating a fibre within the LVST can be obtained by dividing the distance, D, of the stimulating electrode tip from the nearest estimated location of the LVST (in μm) by the stimulus strength, T, at threshold (in μA). Our measurements of current spread indicate that if this D–T ratio is more than 10, the stimulus is too weak to spread to the LVST. D–T ratios for the twenty-one stimulus sites shown in Fig. 4 ranged from 36 to 240. In seven experiments a total of 115 Deiters' neurones were found that could be driven antidromically from the cervical spinal cord by stimuli weaker than 100 μA and with D–T ratios greater than 10. Of these 115 neurones fifty-eight were also activated antidromically from the lumbar cord (L_c cells). The remaining fifty-seven accepted cells were not activated from the lumbar cord (C cells). Examination of the D–T ratios for activation of L_c and C cells from the cervico-thoracic spinal cord shows that 63 % of C, and 70 % of L_c, fibres had ratios of 20 or more, and 25 % both of C and of L_c fibres had ratios of 40 or more. Thus, the relative numbers of L_c and C cells would remain the same even if the criterion D–T ratio were raised from 10:1 to 20:1, or even to 40:1. In the same seven experiments many neurones were encountered that were activated from the lumbar cord but could not be activated by cervical stimuli of 100 μA (L cells). We did not record from such cells systematically, but we did study the properties of some of these L cells from comparison with properties of C and of L_c cells. It of course is possible that neurones classified as L cells may have sent collaterals to regions of the cervical enlargement that lay outside the range of the stimuli used.

TABLE 1. Classification of cell populations according to segmental position of the stimulating electrodes

Cell types	L1–2	L3–4
L_c	25 (64 %)	33 (43 %)
C	14 (36 %)	43 (57 %)
Total	39 (100 %)	76 (100 %)

The population of L_c, L and C neurones just described was obtained in three experiments where the lumbar tract electrode was located within the first or second lumbar segments, and in four experiments where these electrodes were located within the third or fourth lumbar segments. Table 1 breaks down the C and L_c cell populations according to the segmental level of the lumbar tract electrode used. The proportion of

neurones identified as L_c cells was considerably higher in experiments with the lumbar tract electrode at L1–2 than in experiments where this electrode was at L3–4. The χ^2 test indicated that this difference was statistically significant ($P = 0{\cdot}05$). Thus, our results indicate that a relatively large fraction of neurones that supply branches to the cervical enlargement leave the LVST between L1 and L4. Not all L_c cells end in the upper lumbar cord, however, since we found some that could be activated by microstimulating electrodes in the upper sacral spinal cord.

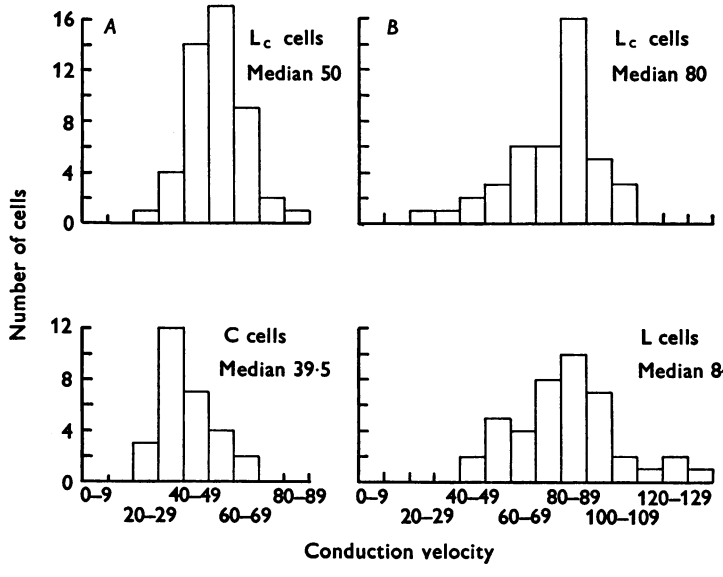

Fig. 5. Conduction velocities of vestibulospinal neurones. *A*, conduction velocities of L_c cells (upper) and of C cells (lower) calculated from cervical latencies and distances from cervical stimulation sites to Deiters' nucleus. *B*, conduction velocities of L_c cells (upper) and of L cells (lower) calculated from lumbar latencies and distances from lumbar LVST electrodes to Deiters' nucleus.

Locations and conduction velocities of different types of vestibulospinal neurones

By reference to extracellular dye marks (see Methods) we reconstructed the locations of 135 antidromically activated vestibulospinal neurones, thirty-two of them L_c cells, within Deiters' nucleus. As has been described previously (Wilson *et al.* 1967; Peterson, 1970) there are more L than C cells dorsally in the nucleus, while more ventrally C cells outnumber L cells. The thirty-two L_c cells were scattered throughout the nucleus, but their dorsoventral distribution more closely resembled that of L cells than of

C cells. When the nucleus was divided dorsoventrally into thirds, only 19% of L_c cells and 26% of L cells were located in the ventral third, which contained 43% of the C cells.

Conduction velocities of the axons of C, L and L_c cells are shown in Fig. 5. For comparison of conduction velocities of L_c and C cells, the velocities of the L_c cells as well as of the C cells were calculated by dividing the distance from the stimulus point in the cervicothoracic cord by the latency of the neurone's antidromic response to stimulation at that point. A similar calculation was made for L_c and L cells, using the lumbar tract electrode as the stimulus point.

As illustrated by Fig. 5 the conduction velocities of axons of L_c cells obtained from cervical latencies were smaller than the conduction velocities obtained for the same axons when lumbar latencies were used. As noted earlier (Ito, Hongo, Yoshida, Okada & Obata, 1964) there may be a decrease in conduction velocity as the impulse approaches the soma, as well as an increase in conduction distance (relative to straight line measurement) of LVST axons within the brain stem. These factors would be relatively more important with short conduction distances, thereby accounting for our observed difference in conduction velocity.

As shown in Fig. 5*A*, the median conduction velocity of L_c cells was 50·0 m/sec whereas the median conduction velocity of C cells was only 39·5 m/sec. The Mann–Whitney U test showed that the conduction velocities of L_c cells were significantly greater than the conduction velocities of C cells ($P < 0{\cdot}01$). Fig. 5*B* shows that, although the distribution of L_c cell conduction velocities was skewed toward lower values while the distribution of L cells was skewed toward higher values, the median conduction velocities were quite similar (80 m/sec for L_c, 85 m/sec for L cells), and the U test failed to reveal a significant difference between the two distributions ($P = 0{\cdot}15$).

Thus our findings on location and conduction velocity show that the properties of L_c cells are similar to those of L cells but different than those of C cells.

DISCUSSION

By stimulating within the cervical grey or white matter we activated a number of axons of Deiters' cells at thresholds ranging from 2·5 to 92 μA. Except for the lowest, these values are considerably higher than the thresholds recently determined for other myelinated axons: as little as 0·1 μA for axons of Ia interneurones (Jankowska & Roberts, 1972) and comparable values, ranging up to 3 μA, for axons of Renshaw cells (Jankowska & Smith, 1973). It is not surprising that our values are higher, because in most cases we stimulated from fixed positions and therefore made no attempt to find the point of minimum threshold. Furthermore, our electrodes usually had considerably larger tips and lower

resistances than the glass pipettes used for stimulation by the other investigators, with a resulting lower current density at the tip: the threshold could therefore be expected to be higher even in those cases in which we approached the fibres. Considering the magnitude of our stimuli, it is important to be sure that we did indeed stimulate collaterals of the LVST rather than spread to the tract itself.

Both from the data that have been presented and from theoretical considerations (see Appendix), we conclude that over a considerable range threshold current for activation of an LVST axon is approximately proportional to the distance of the activated node from the electrode tip, with a slope of 10 μm/μA. Therefore, a fibre is unlikely to be further away from the stimulus point than a distance (in μm) equal to 10 times the threshold (in μA). This estimate of stimulus spread is comparable to that of BeMent & Ranck (1969*a*), but lower than the estimate that emerges from other studies, in which the distance–threshold relation was not linear. For example, in a careful study of dorsal spinocerebellar tract fibre thresholds Roberts & Smith (1973) found that for sites transverse to the stimulated node a 10 μA current spread 250 to 400 μm (25–40 μm/μA). Comparable, and even greater spread was seen by Jankowska & Roberts (1972) in their study of Ia interneurones. It is difficult to account for the discrepancy between our estimates of spread and those of these other investigators, although the type of axon and the geometry and resistivity of the area of spinal cord being stimulated could be a factor. Nevertheless, even if our data are interpreted in terms of their estimate of spread rather than ours, by rejecting neurones with *D–T* ratios less than 20, or even 40, many of the fibres we have classified as cervical collaterals rather than axons of LVST fibres will remain in that category. There is therefore no question that even with the most conservative criteria a substantial part of our fibre population consists of cervical branches of LVST axons. For the rest of the discussion we will assume that all fibres with *D–T* ratios of 10 or more are in fact collaterals.

In our experiments 58/115 of those Deiters' cells whose axons branched within the lower cervical or upper thoracic cord were also activated antidromically from the lumbar tract electrode. The fact that we found more L_C cells when stimulating at L1–2 than at L3–4 suggests that our fraction of L_C cells would have been greater had we always stimulated at the more rostral level. Many axons leave the LVST between L1 and L4 (Fanardjian *et al.* 1972), and it may be that these axons supplying the more rostral lumbar segments are more likely to branch rostrally in the cord than are the LVST axons that descend to lower levels (see below). Finally, it is possible that many C cells that cannot be activated from the lumbar cord may have axons that reach thoracic levels. Whatever percentage of

Deiters' cells does branch to supply more than one level of the spinal cord, it is clear that many cells do so, and this branching must be taken into account in considering the somatotopic arrangement of the nucleus.

It is possible that some L_c neurones project to the cervical and lumbar enlargements and influence both forelimb and hind limb motoneurones. On the other hand, it may be that L_c cells are more concerned with control of the axial, particularly the back, musculaure. The presence of a higher fraction of L_c cells with stimulation at L1–2 as opposed to stimulation at L3–4 supports this notion, although the fibres may have terminated at levels other than those at which we stimulated them, since Nyberg-Hansen & Mascitti (1964) have shown that after entering the grey matter LVST axons may course for a considerable distance caudally before terminating. Whatever the precise function of L_c cells they provide an example, in a descending tract, of a case where activity of a single neuron may be used to coordinate widespread muscular activity. In this respect L_c cells may bear some resemblance to invertebrate command interneurones (Wiersma, 1952). More specifically, our data indicate that the lower cervical and upper thoracic areas of the spinal cord receive at least as much information from neurones that are simultaneously conveying the same information to the lumbosacral region as they receive from neurones that are targeted exclusively to them. Because there are many L_c cells, such integration at the level of the single cell is an important one in the lateral vestibulospinal system.

Recently pyramidal tract cells have been shown to branch widely to motor and sensory subcortical structures (Endo, Araki & Yagi, 1973) but it is not known whether the cells branch and supply different spinal levels. Perhaps multiple branching is likely to exist not only in the vestibulospinal tract but also in other tracts that mainly influence axial and proximal muscles, for example the reticulospinal system (Lawrence & Kuypers, 1968). A search for the presence of branching in a variety of descending pathways may provide some insight into the functional role of branching axons.

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APPENDIX

A THEORY OF MICROSTIMULATION OF MYELINATED FIBRES

By C. P. Bean

From the General Electric Research and Development Center, Schenectady, N.Y. 12301, U.S.A.

The argument of this paper relies on a knowledge of the relationship between threshold current and axonal distance for excitation of myelinated fibres using microstimulation electrodes. This relationship can, as shown in the body of the paper, be illuminated through tracking experiments. The measurements from such experiments suffice to make the principal point of this communication but they may be usefully supplemented by a theory of microstimulation. Such a theory serves to identify the important parameters and functional dependences of this mode of excitation. This Appendix gives a brief account of one such theory that follows from a number of simplifying assumptions. This approach suggests a specific mode of analysis of the tracking experiments – a mode that is applied to representative data of this paper. Lastly, a brief comparison with other experiments is made. Owing to limitations of space only a sketch of the derivation can be given. A more complete work, in progress, will be a detailed treatment with an analysis of the basic assumptions and more extensive caveats concerning the range of application of these ideas.

The assumptions of the calculations are those usually employed (BeMent & Ranck, 1969*b*). The neurone is presumed to be infinitely long, to be myelinated in a regular fashion, and to have excitable nodes that may be approximated as points. The stimulating electrode is presumed to be spherical and to be situated a distance d from the nearest node. Excitation is assumed to occur when any node has its transmembrane potential difference depolarized by a critical amount, ΔV_c. The electrode and axon are taken to be located within an infinite, electrically isotropic medium of resistivity ρ. The electrical properties of the axon, before the act of excitation, are presumed to be linear. Consequently, for steady state or rheobase conditions, the axon is electrically described by the transmembrane resistance (R_m) of the node and by the internal axonal internode resistance (R_a). As indicated below, there is a further simplification in that these resistances always occur as a quotient in the final results of calculations.

The calculation of the nodal depolarization has two elements. First is

the calculation of the external potential at each node while the second is the calculation of the internal potential. For the first, as is well known (e.g. Jeans, 1911, p. 352), the assumption of current conservation and Ohm's law in an isotropic medium of resistivity ρ, yields, for a spherical electrode carrying a steady current I,

$$V_{ext} = \rho I/4\pi r, \tag{1}$$

where r is the distance from the centre of the electrode. If the electrode is presumed to be a distance d from the nearest node which is given an index O then the external potential at this node is

$$V_{ext}(O) = \rho I/4\pi d. \tag{2}$$

This expression ignores any distortion of the current paths from the electrode by the fibre itself. This assumption both implies that the diameter of the fibre is small compared to relevant dimensions of the problem and also that the nodal current density is small compared to the external current density. The internal potential presents greater difficulties in calculation. If the electrical properties of the axon and node are presumed linear, the application of Ohm's law and conservation of currents gives a set of difference equations

$$(R_m/R_a)\,[V_{int}(n-1)+V_{int}(n+1)-2V_{int}(n)] = V_{int}(n)-V_r-V_{ext}(n). \tag{3}$$

In this expression, the subscripts int and ext stand for internal and external, respectively, while V_r is the resting potential and n is the index of a node. This system of equations is equivalent to that customarily derived as, for instance, by Taylor (1952), Lussier & Rushton (1952) or BeMent & Ranck (1969*b*). Eqn. (3) demonstrates the earlier assertion that only the quotient of the nodal membrane and axonal internodal resistances has significance in the approximations of this calculation. This significance may be made plain by considering the solution of (3) for the case wherein $V_{ext}(n)$ is identically zero. This case is mathematically and conceptually identical to that of a telegraph wire with regular faults as pointed out by Tasaki (1953) and emphasized by Taylor (1963) and Cole (1968). The general solution (Jeans, 1911, p. 319) is

$$V_{int}(n) = V_r + B_1 \exp(-nL/\lambda) + B_2 \exp(nL/\lambda). \tag{4}$$

In this expression, B_1 and B_2 are constants fixed by the boundary conditions, G is the internodal spacing and λ is an electrotonic coupling distance or space constant that is defined by the equation

$$\exp(L/\lambda)+\exp(-L/\lambda) = 2+R_a/R_m. \tag{5}$$

Eqn. (5) shows that, as in the case of non-myelinated fibre (e.g. Cole, 1968), the space constant is determined by the ratio of membrane to axonal

resistance. Indeed, for most cases of interest, λ/L is greater than unity and is closely given by $(R_m/R_a)^{\frac{1}{2}}$.

The solution given in (4) can be used to derive an expression for the threshold–distance relationship for the case of an electrode close to the zeroth node. In this approximation the external potentials are vanishing small at all other nodes, hence (4) applies. For the positive half space, with a boundary condition of $V_{int} \to V_r$ at infinity, the specific solution is

$$\underset{n \geqslant 1}{V_{int}}(n) = (V_{int}(1) - V_r) \exp(-(n-1)L/\lambda) + V_r. \tag{6}$$

The consequence of this solution is that the depolarization of the nearby zeroth node is given by

$$\underset{d \ll L}{\Delta V(O)} = -2V_{ext}(O)/(1+\exp(L/\lambda)). \tag{7}$$

The subscript condition emphasizes that this result applies only for an electrode near to a node. If this last expression is set equal to ΔV_c and (2) is used to give an explicit form for $V_{ext}(O)$, then with suitable rearrangement the threshold electrode current for excitation, I_{th}, is given by

$$\underset{d \ll L}{I_{th}} = -(4\pi d \Delta V_c/\rho)(1+\exp(L/\lambda))/2 = -Ad. \tag{8}$$

The negative sign indicates that a cathodal current is required to trigger a nearby node. The relationship given in (8) is the central result of the simple theory in that the threshold close to a node is predicted to be proportional to distance from the node. The constant of proportionality, A, has a specific dependence on the parameters of the axon and surrounding medium. The explicit form of this result extends a previous formulation (Bean, 1973) to arbitrary values of λ/L.

It is possible to solve numerically the equations of current continuity, Ohm's law, and external potentials for a specific set of constants and thus overcome the restrictions of the analytical approximation given above. This is done by assuming an axon of a finite number of nodes, say twenty, and assuming the axonal current to be zero at one end. For one choice of internal potential at the end in question, the difference equations allow successive inference of potentials at succeeding nodes. In general these solutions will be strongly divergent at the other end. By successive approximations one can search for the initial choice of internal potential at one end that will just bring the axonal current at the far terminus to zero. From the consequent outward current at the central node we obtain the depolarization at that node and the consequent electrode current threshold. Repeated calculations show the results to be quite insensitive to the initial choice of axonal length. A set of computer calculations is

given in Fig. 6. For the sake of illustration, specific values of the relevant parameters have been assumed as listed in the Figure caption. The parameters are usual (BeMent & Ranck, 1969*b*) and might be expected to be correct within a factor of two. The Figure shows numerical results for four tracks making different angles with the axon. Each track is made to pass arbitrarily close to a node. Several points may be noted from these results. First, as required by (8) above, the threshold–distance relationship is linear for small displacements from the origin. (This computer

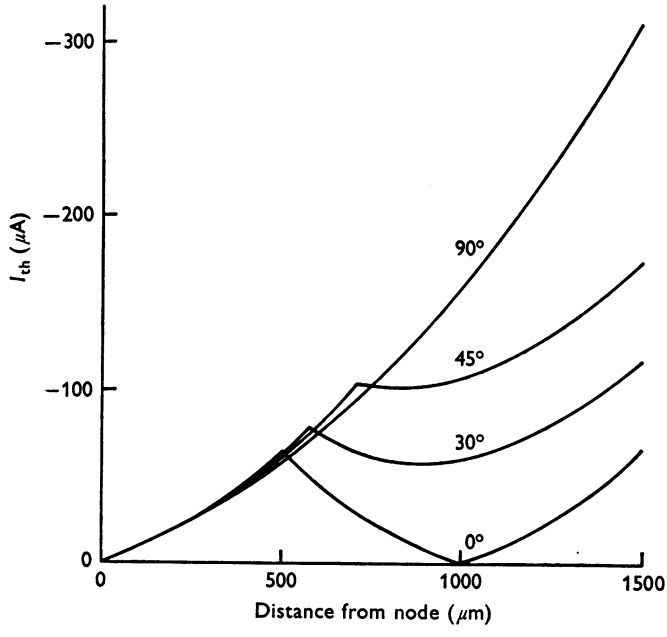

Fig. 6. Threshold–distance curves computed for various tracks. The calculations assume a long myelinated fibre with a 1 mm internodal spacing immersed in an isotropic medium of 300 Ω cm resistivity. Each node is presumed to have a critical depolarization potential of 15 mV. The space constant is taken as twice the internodal spacing. The cathodal current source is a point and assumed to be on long enough that steady state or rheobase conditions apply. Tracks are taken to be parallel to the fibre, at 30°, 45° and perpendicular as indicated. Each track is assumed to pass through a node and distance along the track is measured from that node.

solution gives a limiting slope identical within round-off error to that predicted by (8).) For larger displacements the relationship becomes somewhat superlinear. (As derived in the pioneering work of BeMent & Ranck (1969*b*), the threshold for the perpendicular track approaches a cubic dependence at large distances. Any approximation to a square law in this theory arises in the transition from the linear to cubic dependence.)

A second point is that for small displacements – say less than half the internodal spacing – the threshold–distance relationship is isotropic. This conclusion, of course, is vitally linked to the assumption that the axon is immersed in a medium of isotropic resistivity. For tracks that are not perpendicular to the axon, there are distinctive cusps and non-monotonic features to the threshold–distance relationship. A cusp occurs as the identity of the first excited node is shifted along the axon. In particular, a track exactly parallel to the axon is periodic, as shown in the lowest curve. With the constants assumed in this calculation, a 50 μA rheobase pulse could not excite a node 500 μm or more away. This result, suitably adjusted for finite pulse length, is in accord with the major part of the data reported in the body of this paper. A last point, not illustrated in the Figure, is that an anodic pulse is predicted to require a higher threshold by a factor of from five to nine depending on the path of the track and position in the track. This arises from the fact that the closest node is hyperpolarized by an anodic pulse and, hence, critical depolarization occurs in a node one or two internodal lengths away where the driving force is weaker. For the specific case of an electrode close to a node, the approximations that led to (8) give for the ratio of anodal to cathodal thresholds, $-2/(1-\exp(-L/\lambda))$. This assertion extends the validity of an earlier result that applied for $\lambda/L \gg 1$ (Bean, 1973). This relationship may afford a useful method to infer λ/L in specific instances.

The analysis of experimental tracks is not completely straightforward owing to the fact that, except in rare instances, they do not graze a node. Hence, a minimum threshold corresponds to an unknown distance, d_o, of closest approach to a node. If one assumes, as a first approximation, the generalized linear law of (8) and if z measures the distance along the track with a minimum threshold at z_{min}, then by application of the pythagorean theorem

$$d^2 = d_o^2 + (z - z_{min})^2 \tag{9}$$

and, hence

$$I_{th}^2 = A^2(d_o^2 + (z - z_{min})^2). \tag{10}$$

This equation suggests the mode of plotting experimental data shown in Fig. 7*A*. The intercept, $-d_o^2$, on the horizontal axis corresponds, in this instance, to a distance of closest approach of 165 μm while the slope A is $0{\cdot}12_2$ μA/μm. Fig. 7*B* shows the best fit of the equation (8) as a solid curve while the filled circles are the experimental measurements. Under this interpretation, the experimental points would be almost coincident with the 45° track of Fig. 6. There may even be a cusp visible in the leftmost points of Fig. 7*B*. The close numerical agreement between these results and the predictions of Fig. 6 is not as significant as it may appear in that the experimental points are not extrapolated to the rheobase

condition. This extrapolation would lower them by about a factor of two. Fig. 2 of the main text illustrates the range of curves obtained in the present experiments, and shows the general linear threshold–distance relationship found through experiment.

The deduction or assertion that the limiting threshold–distance relationship is linear rather than a square law is somewhat controversial. On the

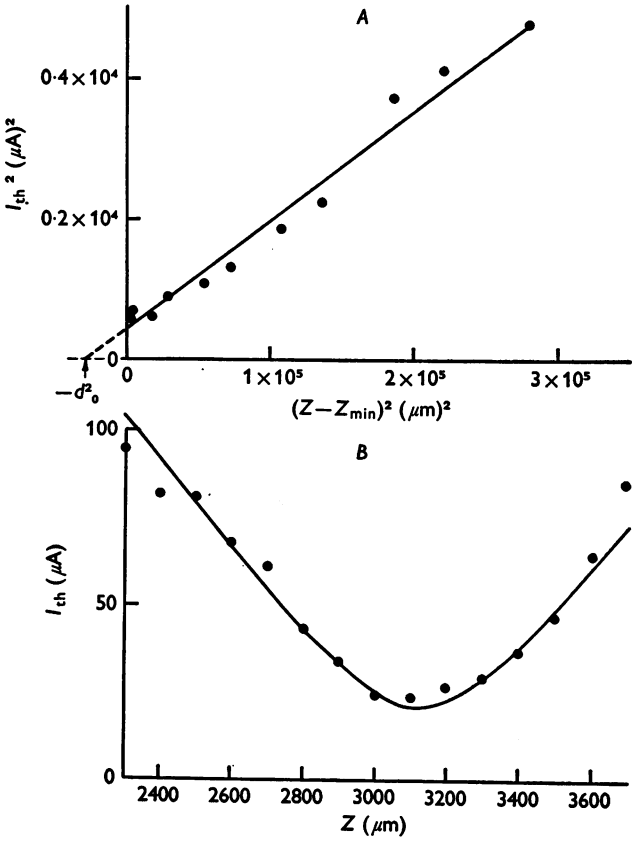

Fig. 7. An example of the method of analysis using the linear approximation. The experimental track through gray matter of the cervical enlargement is typical of data reported in the body of this paper.

A, the square of the threshold current as a function of the squared displacement from the track minimum (ascending track). The z co-ordinate measures distance from the dorsal surface of the spinal cord. The fitted curve implies a distance of closest approach of 165 μm and a linear distance–threshold relationship of 8·2 μm/μA.

B, fit of the linear threshold–distance approximation to experimental data. The constants derived from Fig. 7*A* are used to generate the continuous line. The continuous points are experimental thresholds for a 0·1 msec cathodal pulse.

one hand, both the linear law and the isotropy of the threshold–distance relationship have been shown by Tasaki (1950) to hold for isolated toad peripheral motoneurones in Ringer solution. The magnitudes are also in good accord with the numerical predictions using the specific parameters assumed in the treatment above – with the substitution of a value of $\sim 90\ \Omega$ cm for the resistivity of Ringer as compared to the rather arbitrary $300\ \Omega$ cm taken for grey matter. On the other hand, a number of publications from the Göteborg school give data for excitation in the spinal cord of the cat that are at some variance with the ideas presented here. Threshold tracking curves for excitation of the axons of Ia inhibitory interneurones (Jankowska & Roberts, 1972), axons of Renshaw cells (Jankowska & Smith, 1973) as well as axons in the spinocerebellar tract (Roberts & Smith, 1973) have all been interpreted as requiring a parabolic threshold–distance relationship close to the node of an axon. In addition, the absolute magnitudes of the thresholds at a given distance – say 100 μm – from the focus are lower by factors of 3–20 than those obtained in the experiments of this paper or expected on the basis of the parameters assumed in Fig. 6. Within the framework of the ideas presented here one would have to postulate either a very high local resistivity or a very low threshold. It is of some importance to ascertain whether either of these conditions applies or if the theory outlined here is, for some reason, not applicable to the Göteborg results. Very recently, Merrill (1974) has published data on the excitation of axons of respiratory neurones. He, in common with the Göteborg group, interprets his results in terms of a parabolic threshold–distance relationship.

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