ON THE ROLE OF THE SPINAL AFFERENT NEURON AS A GENERATOR OF EXTRACELLULAR CURRENT*

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The preceding paper (10) demonstrated that electrotonus in a stimulated dorsal root is the sum of two distinguishable although partially contemporaneous systems of current. One occludes strongly and develops a potential (DRβ) having the form and magnitude of the electrotonus appearing in a neighboring, unstimulated rootlet. The other system of current develops an additional negativity uniquely in an active root (DRα) and does not occlude.

Because DRα is non-occluding it was deduced, in agreement with the studies of Lloyd and McIntyre (14, 15), that it arises from a generator located within the primary afferent neuron. Further, it was demonstrated that DRα arises neither from injury currents at the end of the cut dorsal root nor from the effects of steady polarization arising centrally. These observations necessitated the proposal that DRα arises physiologically from a spatial change in the recovery cycle of membrane potential somewhere along the primary afferent neuron. The purpose of this paper is to investigate the precise region of origin of DRα within the primary cell.

Earlier work on this question by Lloyd and McIntyre led them to the conclusion that some or all of the potential which we have called DRα resulted from activity in presynaptic collaterals (14). With respect to this conclusion it may be noted that the form of DRα would be determined solely by a membrane potential fluctuation in the presynaptic collaterals only if the concurrent membrane potential variations in contiguous myelinated segments of the primary neuron were relatively small in size. If not, then root electrotonus would depend significantly upon events in the myelinated segments. In addition, if there are differences in the values of membrane potential between the myelinated segments themselves (root and column) during recovery from conduction, root electrotonus will appear as a result of this. Lloyd and McIntyre were aware of these propositions and in their analysis made what was at that time the reasonable assumption that the post-spike membrane potential

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fluctuations of both root and dorsal column segments of the primary afferent neuron were similar and negligible in size relative to those in the collaterals (14).

However, evidence has now been accumulated to suggest that there exist certain differences between the properties of dorsal root and dorsal column. Changes in microscopic anatomy at the root-cord junction have been described (23) as well as a gradient of increasing susceptibility to sodium lack along the dorsal root in approach to the spinal cord (18) and a differential response of root and column to potassium ion (9). Finally, evidence to be presented here, taken with that of preceding papers (21, 22), indicates that the form, as well as the magnitude, of after-potential and excitability cycles is dissimilar in these two structures. Consequently, it becomes necessary to examine the importance of this dissimilarity in the production of dorsal root electrotonus.

General Considerations

The hypothesis to be tested is that the root-column system constitutes an electromotive surface located somewhere near the anatomical root-cord junction and that its currents can account completely for the negativity unique to a stimulated root (DRα). To satisfy this hypothesis the extracellular currents of such a system must have the sign, form, duration, and magnitude of DRα. In addition, a special quantitative condition must be met. Electrodes on an active dorsal root will record only a fraction of the extracellular potential drop produced by the currents underlying DRα. Fig. 1 demonstrates the situation pictorially by treating the system as though it were entirely unidimensional, with root and column functioning essentially as two poles of a dipole. The longitudinal extracellular potential would then be given approximately by the heavy curve. Under the assumption that the dipole center lies very near the root-cord junction, only one-half the total extracellular RI (resistance current) drop generated by the electromotive surface would be recorded by conventionally placed root electrodes whose positions are indicated in Fig. 1 by the arrows. The rest of the RI drop would appear in the volume of the cord and beyond approach of an unidimensional analysis. Consequently, whatever voltage is experimentally assigned to the electromotive surface, it should be twice the value appearing as the conventionally recorded electrotonic deflection DRα.

In order to define the parameters of this dipole-like electromotive surface it is necessary to determine the intrinsic post-spike membrane potential variations occurring in the contiguous root and column segments of the primary afferent neuron. Until transmembrane electrodes can be employed with assurance to study the labile after-potentials of mammalian myelinated axons more indirect techniques must be relied upon. Two general methods have been used here. The first, which has the advantage of being the easiest to justify theoretically, depends upon the comparison of the monophasically recorded external action

potentials of both root and column. The weakness inherent in this approach lies in its reliance upon data which must be derived from preparations which have been manipulated and rendered monophasic. The normality of such preparations requires subsidiary justification. The second method is based upon comparison of the excitability changes associated with after-potentials and cannot be quantified as rigorously as the first; but it has the advantage of

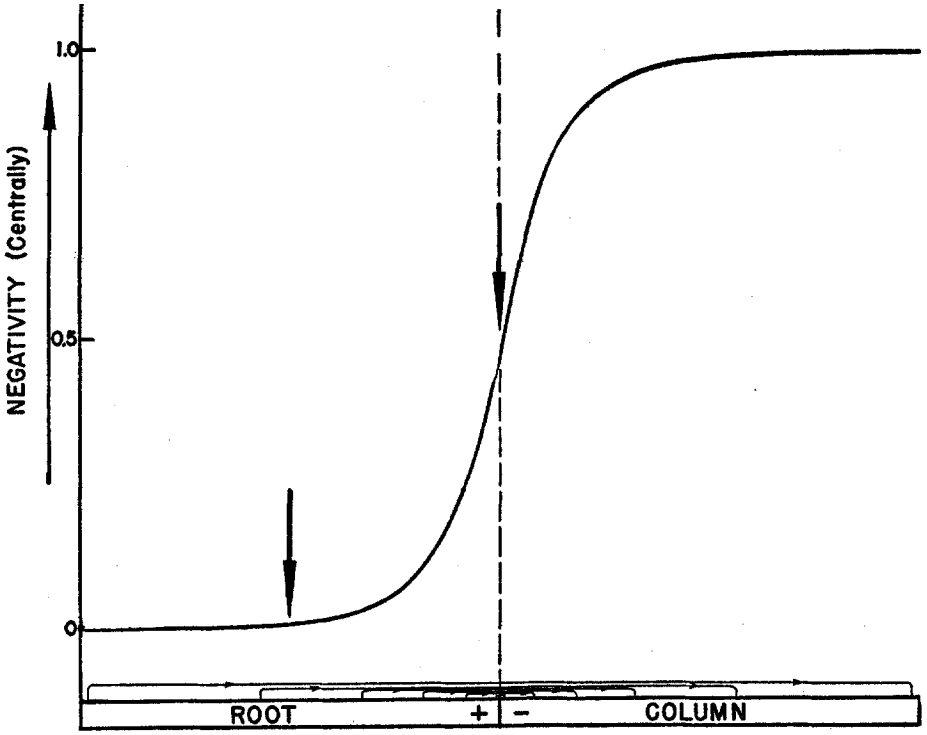

FIG. 1. Potential distribution of an hypothetical dipole at the root-column junction considered in one dimension. Arrows mark relative position of conventional root electrodes.

depending only upon data derived from intact, blood-perfused tissues *in situ*. Inasmuch as both methods lead to virtually identical conclusions they are mutually supporting.

The external action potentials and their excitability counterparts have been measured at distances sufficiently far from the proposed dipole to avoid their modulation by mutual interaction. However, it is necessary to assume for both analyses that the recovery cycle intrinsic to dorsal columns, which either *in vivo* or *in vitro* can be determined in pure form only in the thoracic region,

maintains itself with constancy into the lumbar region. That is, it is assumed that membrane properties per unit area remain constant even though the geometry of fibers may vary somewhat as they ascend in the dorsal columns. This assumption permits the values of the after-potential and excitability as determined in the thoracic cord (21, 22) to be extrapolated spatially into the immediate vicinity of the root-cord junction. This assumption is accepted because post-spike supernormality intrinsic to the dorsal columns has the same maximal value in both regions of the cord (21).

Evidence for a Root-Column Electromotive Surface Based on Potential Data.—

In order to use the external action potentials of root and column for comparison of their membrane action potentials it is necessary to assume that the externally recorded spike components of the action potential in both root and column represent equal changes in membrane potential. Thus, when the values of the externally recorded after-potentials of root and column are determined as percentages of their respective monophasic spikes, the after-potentials may then be considered to be related on an arbitrary but relative scale of membrane potential value. Stated otherwise, the spike potentials are taken as internal testing signals of known value which may be used to provide a control for the differences in geometry between column and root. Support for this proposition comes from the knowledge that the spike potential, as measured with internal electrodes, is very similar in a wide variety of vertebrate and invertebrate axons and consequently is not likely to be significantly different in two adjoined segments of a mammalian alpha fiber.

Therefore, the values of the after-potential of root and column were each measured monophasically as a fluctuation in the demarcation current relative to the magnitude of the fluctuation produced by the reference signal—the spike. It is then argued that if these after-potentials were measured against each other at the root-column junction, the maximum extracellular potential would equal the difference between the two conventionally recorded after-potentials (when all units are in per cent of spike height). Thus, if at any instant each tissue had an after-potential of equal size, no current would flow at the root-cord junction. But if the values differed, current would flow and develop an external potential field.[1]

[1] Ancillary assumptions are necessary for the quantitative aspects of this analysis since the external currents of the monophasic spike and after-potential are not drawn from the same segments of nerve. These are: (1) that possible core polarization is comparable in magnitude in the end segment of root and column, and (2) that the effective resistance of the membrane occupied by the spike is also similar (or else small compared to the total resistance of the local circuit) in root and column. These assumptions are reasonable and will be accepted for the present, but they have not been tested.

(*Footnote continued on following page*)

We will test this argument by recording the value of DRα as a per cent of accompanying monophasic spike at the root. If the hypothesis is admissable DRα should be approximately half of the predicted difference between the two after-potentials for all instances of time and must have appropriate sign.

The Action-Potential of Dorsal Root.—

In order to secure dorsal roots as normal as possible for study of their after-potentials, long L_7 and S_1 roots were cut distally (occasionally epidurally) beneath several centimeters of mineral oil at 37.5°C. through which 95 per cent O_2, 5 per cent CO_2 was bubbled rapidly and continuously.[2] In recording after-potentials, volleys (1/second) were initiated at the root near the cord junction and recorded after 3 to 5 mm. conduction, a distance over which temporal dispersion is not significant in roots. Monophasicity was usually accomplished by crushing although occasionally heat or cocaine was necessary. The proximal recording electrode was placed 10 to 15 mm. from the cord junction and 25 to 30 mm. from the cut end where the distal recording electrode was located. Cats prepared as in the preceding study (10) were used throughout the experiments.

Two criteria were established to ensure that manipulation of roots to this extent did not alter their behavior. First, current-calibrated excitability curves were taken from roots under three conditions of increasing manipulation: (*a*) entirely unmanipulated *in situ*, (b) intact but with pia removed, and finally (*c*) following section distally. In all cases the testing cathode was 1.0 to 1.5 cm. from the root-cord junction and in the third case also 2.5 to 3.0 cm. from the cut end. No significant differences were found between the first two cases or between these and the third case within 5 minutes after sectioning the root (prior to which time the after-potentials were always measured). Since excitability is a very sensitive index of the state of the after-potentials (17), the latter were judged to be normal at the time of study.

Beyond the study of excitability, the second criterion validating these after-

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In addition, the existence of significant fiber heterogeneity in the root-column system is made likely by the available evidence, but here we neglect this problem by dealing only with the average action arising in the intact system or its components when maximally activated. In this respect, it is of importance to mention that fibers generating more than 80 per cent of the total root spike potential area ascend at least 2 cm. in the dorsal columns (unpublished observations).

[2] It would have been desirable to avoid both cord electrotonus and the dorsal root reflex by cutting the roots at their junction with the cord. This was tried and found unsatisfactory because of the severe (and not easily controlled) spontaneous firing that arose from the cut end. Since such firing (which refers to that beyond the normal centripetal influx of sensory impulses) occurs with section of the root centrally but not distally it signifies yet another change in properties along the root in approach to the cord.

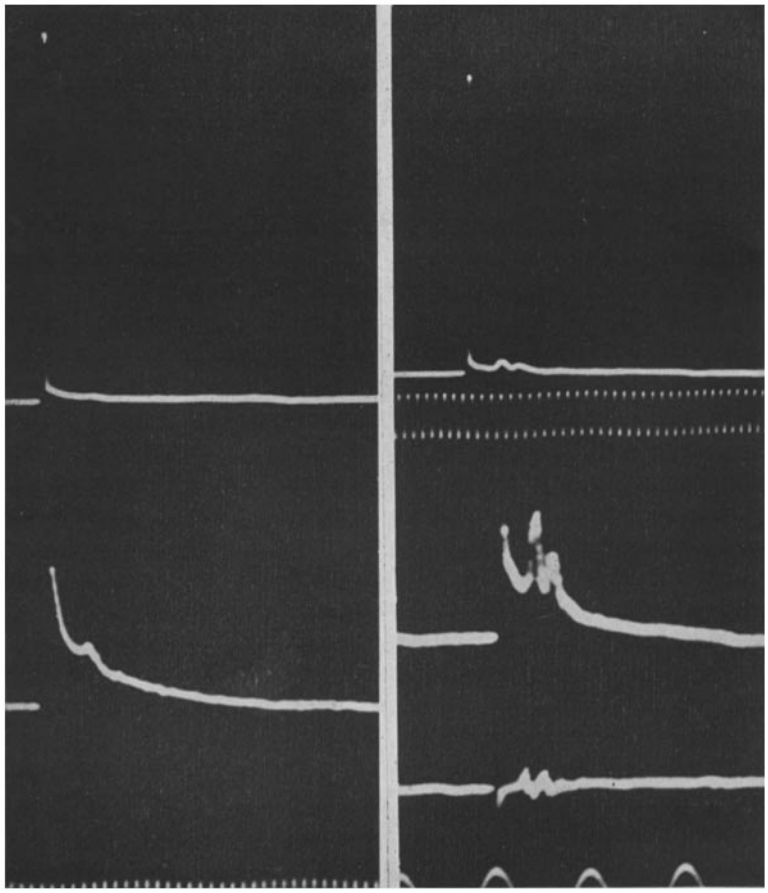

FIG. 2. *Left:* Upper trace shows spike and after-potentials of dorsal root, while lower trace presents the after-potentials at 10 times the amplification. Root 38 mm. long, cut and crushed distally 3 minutes before record. Stimulating anode at cord junction, cathode 6 mm. distally. Distal recording electrode on crush, central electrode 10 mm. from cord junction. Time in milliseconds. *Right:* Action potential of another dorsal root presented as above. In addition, the lowest trace shows at the same amplification as the after-potential record the potential between two guard electrodes 2.5 mm. on either side of the proximal recording lead. Time markers above, 1 millisecond; below, 10 milliseconds.

potential measurements consisted of placing a pair of "guard" electrodes on each side of the proximal recording electrode and 4 to 5 mm. from each other. Since over this distance the demarcation potential would be expected to fall by 70 per cent (see Fig. 3 of reference 10), the absence of any measurable potential difference between the guard electrodes demonstrated a lack of significant

longitudinal currents from either the distal cut end or centrally. The full recording of root after-potential in the absence of external polarization was thereby assured.

Typical results are shown in Fig. 2 (left) in which the complete action potential can be seen (upper) as well as the after-potential at higher gain (lower). A small dorsal root reflex (24) interrupts the negative after-potential. Since the reflex is recorded monophasically, interpolation of after-potential through it is readily accomplished. Another preparation is present in Fig. 2 (right). Its dorsal root reflex was considerably larger than average. The bottom record shows that there is no detectable potential difference between the guard electrodes other than that resulting from the dorsal root reflex.

Peak height of the negative after-potential has a mean value of 5.7 per cent ($\pm$ 0.2) of spike height (S.E. 42 roots). At 4.5 msecs. the value averages 2.0 per cent $\pm$ 0.06. These figures may now be contrasted with those found in dorsal column as reported previously (22). The peak value of dorsal column negative after-potential was 8.2 per cent $\pm$ 0.75 and at 4.5 msecs. averaged 4.2 $\pm$ 0.37 (S. E. 16 columns). These and the root experimental values each show a Gaussian distribution. Consequently the two tailed t test may be applied to the difference of the means found in root and column at both peak and at 4.5 msecs. The test shows that both pairs of means are significantly different at the 0.001 level.

A Reconstruction of Active Root Electrotonus.—

According to the theoretical considerations discussed earlier it is predicted that at 4.5 msecs. the value of the potential developed in a dorsal root by virtue of the difference between these after-potentials should be one-half of the difference between 4.2 and 2.0 per cent; *i.e.*, 1.1 per cent of the monophasic root spike. Further, the sign would be negative centrally like DRα, in view of the greater depolarization in the column segment. Since the magnitude of DRβ is negligible at 4.5 msecs., the total height of active root electrotonus at 4.5 msecs. may be taken as the height of DRα. This value has been measured in 22 preparations with the central electrode at the point of root entry and the distal electrode 10 to 15 mm. from the cord junction (again monitored with guard electrodes). The mean value was 1.1 per cent $\pm$ 0.05 (S.E.). The correspondence between the predicted value and the experimentally determined value is quite satisfactory and the sign is proper.

In order to carry out the reconstruction of active root electrotonus for all instances, compiled average curves are given in Fig. 3 for column and root negative after-potentials (inset) and neighboring root electrotonus (main figure, DRβ). The ordinate describes the height of these potentials in per cent of their monophasic spikes. One-half the difference between the two after-potentials (inset) has been transposed to the main figure and labelled DRα.

To it has then been added the average potential of a neighboring rootlet (omitting DRIV) again measured as a per cent of the monophasic spike. In the main part of Fig. 3 this has been labelled DRβ because of the prior demonstration (14, 21) of the equality of neighboring root potential and DRβ. The form of the reconstructed curve, obtained by adding DRα and DRβ, is in good agreement with typical recordings of active root electrotonus (see Figs. 1 and 2 of reference

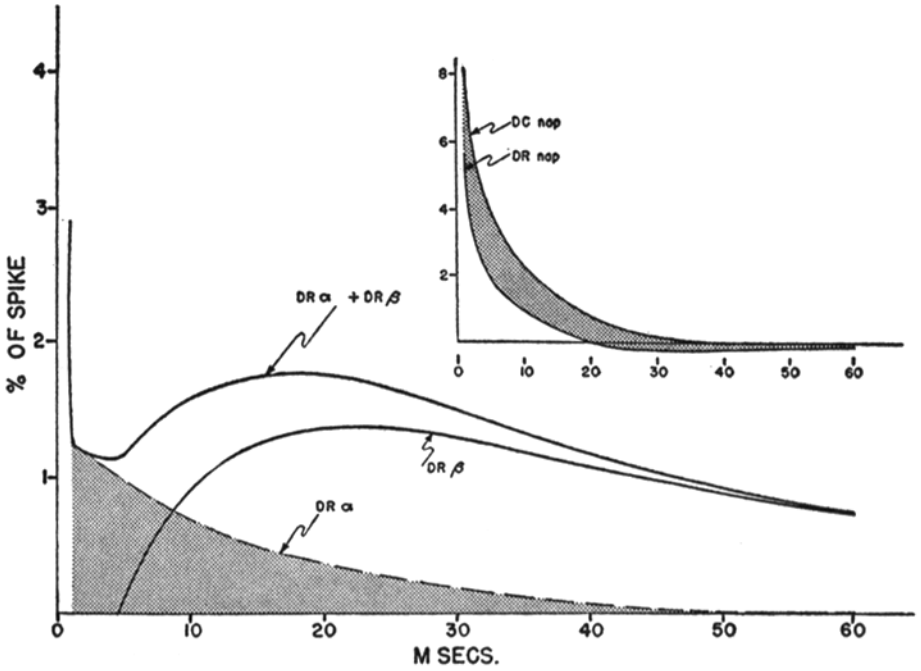

FIG. 3. Reconstruction of active root electrotonus from external action potentials of root and column. DCnap, DRnap equal dorsal column and dorsal root negative after-potentials respectively. (Description in text.)

10) and the duration of DRα calculated in this manner (see inset) is in keeping with the duration estimated independently from studies of occlusion (*cf.* Fig. 4 of reference 10).

Evidence for a Root-Column Electromotive Surface Based on Excitability Data.—

The Recovery of Excitability of Dorsal Root.—

Intact, blood-perfused dorsal roots were fitted with a pair of stimulating electrodes (cathode 1.0 to 1.5 cm. from the root-column junction) for conditioning and testing excitability. For recording the induced volleys, another pair of electrodes was placed more distally. After making certain that no post-spike

transmembrane currents flowed beneath the stimulating cathode the recovery of excitability following a single conditioning volley was determined by observing the variation of response to a constant submaximal stimulus. In a number of cases the assumption was checked that cathodal make excitation was being studied. In addition, supramaximal testing shocks following maximal conditioning volleys were shown to produce volleys of constant maximal height after refractoriness. This validated the use of constant submaximal stimuli as a measure of excitability change by demonstrating that changes in temporal

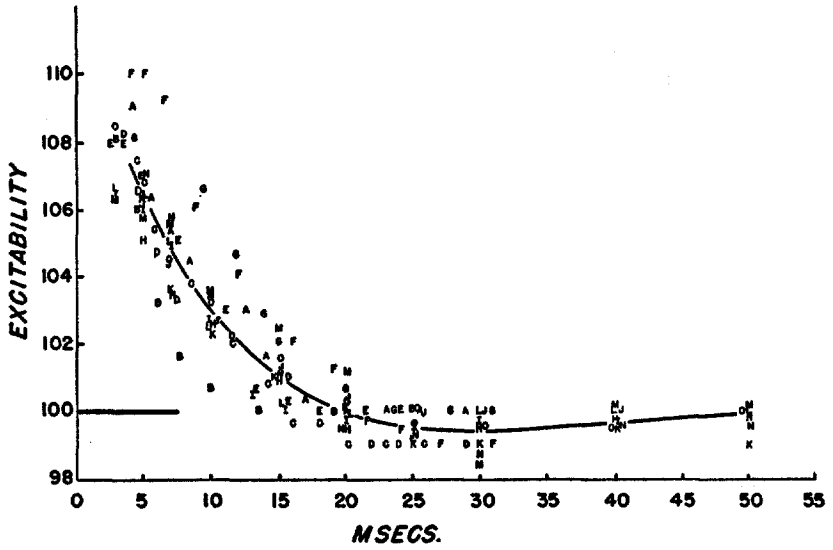

FIG. 4. Recovery curves of intact blood-perfused dorsal roots following conditioning by a single volley. The ordinate describes per cent excitability change in terms of equivalent current units in a manner standard for this study (100 per cent is resting value (12, 21)). The line gives an approximate average for the data.

dispersion and spike height per axon were not reflected in the testing volley. The changes in volley height were then transformed by appropriate calibration curves to the equivalent percentage change in stimulus strength required to produce the same variations in the absence of conditioning (12, 21).

Fig. 4 gives the recovery of excitability in intact roots. Its form closely mimics the after-potential. Both the duration (average 20 msecs.) and peak magnitude of supernormality (average 7.3 per cent) are about one-half the intrinsic excitability values found in intact dorsal columns (21). In addition, there is in root subnormality of significant degree. Fig. 5 plots together on a common percentage excitability scale the intrinsic dorsal root (curve DR) and intrinsic dorsal column recovery curves (curve DCTa), both derived *in situ*. The other two curves represent the total variation in excitability of active (DCLa) and passive

(DCLp) lumbar dorsal column fibers. All the curves are calculated averages of the data presented in Fig. 4 of this and in Fig. 2 of a preceding study (21).

It can be seen that the excitability data in Fig. 5 stand in the same relation as the potential data of Fig. 3. It can also be observed that if the data of Fig. 5 were handled like those of Fig. 3 they would yield a reconstruction of root electrotonus identical in all significant features. Thus, the excitability data derived from intact, blood-perfused tissues *in situ* support the potential reconstruction already presented.

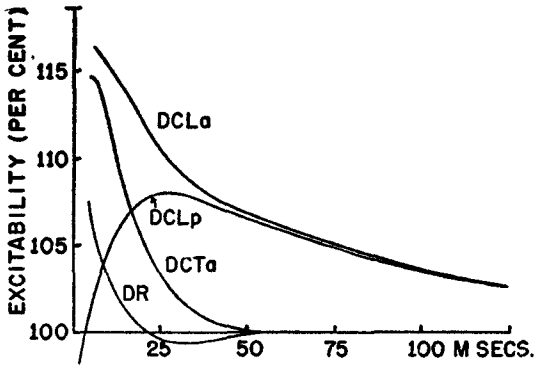

FIG. 5. Averages of compiled data representing per cent change in excitability at various loci along the dorsal root and dorsal column following a dorsal root volley. DR, intrinsic recovery cycle in dorsal root fibers taken from Fig. 4. DCTa, intrinsic recovery cycle in active thoracic dorsal column fibers; DCLa, excitability variation in active lumbar dorsal column fibers; DCLp, excitability variation in passive lumbar dorsal column fibers (averages of Fig. 2 of reference 21).

CONCLUSIONS

The evidence indicates that a powerful mechanism for generating extracellular current is present in the region of the root-column junction. This dipole-like generator would owe its existence to major changes which occur in certain membrane properties of the myelinated axon of the primary afferent neuron as it enters the central nervous system. The root currents of this generator satisfactorily account for DRα. Intramedullary current of such a root-column dipole could not, however, be the cause of any significant component of the intermediary cord potentials (11) because the longitudinal spatial distribution of the latter along the cord is quite pronounced. The cord potentials must arise from more extensive although not necessarily unrelated systems for generating currents in the substance of the spinal cord.

DISCUSSION

The preceding analysis, in so far as it attempts to establish that the primary cell electromotive surface responsible for DRα exists at the root-column junc-

tion, rests entirely upon circumstantial evidence. Final decision on this matter must await detailed knowledge of extracellular potential distribution at the junctional region analyzed with due regard for the effect of the marked non-uniformity in external resistance that exists there on the derivation of sources and sinks of current.

However, two facets of the present study require emphasis. (1) Even relatively minor differences in the recovery process of either L or Q fractions (17) at the root-column junction are quite capable of generating potentials of the magnitude of dorsal root potentials because such differences produce their steepest voltage gradients in the immediate vicinity of the recording electrodes. (2) Even if the existence of a root-column electromotive surface be granted, the analysis in this paper is not complete until conditions in nearby structures are defined, most importantly in the collaterals of the primary neuron. It is not possible at present to assess the role of collaterals in this respect. If their mean distance from the junction is short enough, the space constant of electrotonus adequately long in intervening tissue, and their post-spike membrane potential fluctuation sufficiently different from that in dorsal column tissue, then they could contribute to root electrotonus writing upon the currents of the proposed root-column dipole as calculated in this paper. Specifically, if the collaterals were more depolarized than the parent axons this would make some contribution to DRα in the sense of Lloyd and McIntyre (14) and make for a dual origin of DRα within the primary cell.[3] This problem lies in the domain of intramedullary evoked potentials and requires some comment.

A rather unlikely set of conditions must be met if one is to exclude the primary afferent neuron as a generator of intramedullary currents wherever collaterals arise from it over its longitudinal extent in the tracts. It would be necessary that myelinated and unmyelinated (collateral) portions of the tract fibers undergo similar intrinsic variations in membrane potential. This condition would be met only in the improbable event that the spike potential penetrates *in extenso* to the terminals of the primary cell and, having done so, that thereafter the recovery of membrane potential is essentially identical at all instants in greatly dissimilar regions of the neuron.

Because of the unlikelihood of these propositions it is worthwhile to consider what the intramedullary current-generating behavior of conducting tract cells would be if they were assigned only those events now known to occur in them as discussed in this and a preceding paper (21). Following the tract spike there persists in the myelinated segment prolonged and severe intrinsic depolarization (negative after-potential). Its action in the complete absence of

[3] It should be stated explicitly that this paper contributes nothing to our knowledge of DRIV and it would be expected that the proposed electromotive surfaces at the root-column junction would not significantly polarize adjacent rootlets (*e.g.*, myelinated fiber spike interaction leading to DRI, DRII, and DRIII occurs with an attentuation of about 300:1).

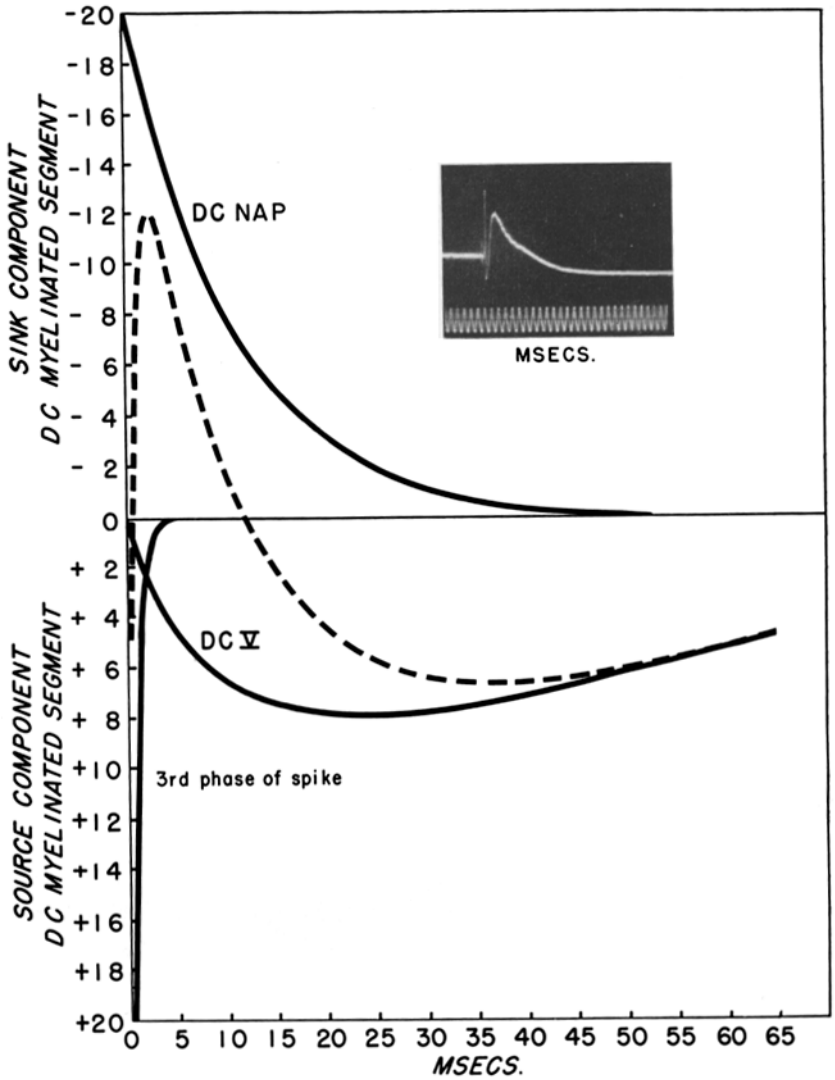

FIG. 6. Solid lines signify intrinsic and extrinsic membrane potential variations of myelinated fibers in the dorsal column. DCnap equals DCTa of Fig. 5, DCV equals DCLp of Fig. 5. Each is drawn to indicate the tendency of the depicted event to make the dorsal column a source (down) or a sink (up). The dotted line is their algebraic sum. It represents the net action of myelinated column cells as a source or a sink and therefore gives the approximate potential such a system would develop in its extracellular space. The ordinate is in units of current-calibrated excitability. For comparison the inset shows a record of the dorsal cord potential. Time in milliseconds.

other events would be to evolve a dorsally located sink of current in the white tracts proper by drawing current from a source in the unmyelinated segments ventrally in gray matter. But another, partially contemporaneous, and therefore interfering event must also be assigned to the tracts—their extrinsic depolarization, which in the absence of other events is of DRV form (21). Wherever the ultimate generator for this event may lie, either in collaterals of primary cells or in secondary cells, it succeeds in driving currents outward across the membranes of myelinated axons in the dorsal column. Thus the Laplacian of the transverse spinal cord extracellular potential would have some value other than zero in the columns and indicate a source of current there.[4] The contemporaneous nature of the two events outlined above demands that the net time course of column sources and sinks be their resultant. Since we are here dealing with linear subthreshold properties of nerve, the excitability change which each event induces in the tract fibers provides a crude scale for comparing the extracellular currents which accompany each event.

In Fig. 6 these statements are pictorially represented from the reference point of the dorsal columns as a source or a sink of current and as though the ensuing extracellular potential were recorded conventionally with an active electrode on the cord dorsum pitted against a distant one. There is striking resemblance to the dorsal cord potential itself (*cf.* inset) and the analysis predicts a 10 msec. column sink giving way to a more prolonged "source."

Two dimensional maps of the instantaneous potential distribution through transverse planes of the spinal cord following a dorsal root volley have been provided by Campbell (4). They were capable of resolving closed isopotential loops of great negative voltage lying within the dorsal column proper during the period of the classical negative intermediary dorsal cord potential. Thus at the time when these tracts are thought to be severely depolarized during their negative after-potential Campbell's data demonstrate a large potential minimum there.[5] Because of its great size this probably represents a sink of current

[4] If this generator of currents were really in dorsal horn interneurons, as seems likely, the column source would in fact be quite spurious in not lying at the surface of the responsible generator. It would be a consequence of the fact that the environment of active cells in the CNS is not homogeneous, specifically in the sense that it contains core-conductors. It may also be noted that proof of the existence of an extrinsic source in the columns, or the finding that excitability changes like DCV occur in columns after their own intrinsic activity is over, virtually excludes the possibility that we are here dealing with events arising as diffusion potentials a millimeter or so away in gray matter. Consequently it is proper to think in terms of electromotive surfaces and potential fields.

[5] Arguments to the contrary based on a one dimensional analysis of potential distribution (2) cannot possibly locate potential minima or maxima lying within two and three dimensions of space. Furthermore, the longitudinal spatial distribution of

residing in the dorsal column at the responsible generator. Following the potential minimum there appears a potential maximum in the columns and therefore a "source." The rough prediction of the spatial and temporal distribution of sources and sinks from knowledge of membrane potential events depicted in Fig. 6 is therefore consistent with those found by Campbell.[6]

The consistency of these several lines of approach suggests that some or all of the negative intermediary dorsal cord potential arises as a function of the negative after-potential in the myelinated segments of those cells which pass through spinal cord tracts, whether they be primary or appropriate secondary cells. Indeed, supraliminal activity of the kind proposed here, if it arises in myelinated secondary cells of convergent synaptic systems, necessitates some degree of occlusion in the resulting potential field like that exhibited by the negative intermediary potential. Lloyd and McIntyre (14), on the basis of asphyxial studies have proposed a dual origin for this potential. This would be consistent with the notion that both primary and secondary tract cells contribute to it by the mechanism outlined above.

The conclusions drawn here are first that some myelinated tract cells are involved in producing the negative intermediary potential, and second that it appears likely that both primary and secondary tract cells may be responsible. While the second conclusion is more tentative than the first its consequence is to place an electromotive surface at the column-collateral junction of the primary cell not far from the root-column system. Interaction between the two systems might distort somewhat the synthesis of root electrotonus proposed here but this cannot be established in the absence of knowledge about transmission properties along the system among other variables.

Certain observations made on potentials elsewhere in the central nervous system but which show reasonable phenomenological similarity with those in the spinal cord appear to be consistent with the above described origin of the negative intermediary potential. At the hippocampus (20), at the primary receiving areas (1, 3, 19), and following transcallosal activation (5, 7) potentials

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the sink, in so far as it might result from primary cell activity, would coincide with the distribution of collateralization of that cell. The fact that the distribution of the negative intermediary potential is skewed and may peak rostral to the active root entry zone (2) is therefore not of itself an observation ruling out a primary cell origin for this potential. It may also be noted, however, that knowledge of the extracellular potential distribution alone (4) is never sufficient to determine what the membrane potential variations are that produce it but only the difference in potential along the surface of the cell.

[6] In addition, the recent studies by Lettvin, McCulloch, Pitts, Wall, and Howland have resolved a large source in the white matter of the dorsal columns with its sink in the subjacent gray during the positive intermediary potential (*i.e.*, at 20 msecs. post-spike) (personal communication).

are evoked by synchronized volleys having a form similar to that of the dorsal cord potentials (*i.e.*, predominately biphasic but on occasion exhibiting subsequent phases). The initial phase of each evoked potential is shorter in duration (5 to 10 msecs.) than the second phase (40 to 100 msecs.). The durations of corresponding phases are roughly similar at the various sites. In addition, all the potentials are standing waves which show inversion of sign as an exploring electrode penetrates the tissue along an axis described by the path of the orthodromic volley. However, myelinated afferent tracts of the spinal cord penetrate gray matter by receding from the surface, while at the cortex the opposite situation prevails. Consistent with this inversion of geometry, the potential fields of each region are opposite in sign at comparable times when recorded from similar points relative to their surface boundaries. Thus, at the cortical surface the first two phases are consecutively positive and negative, while at the cord dorsum they are negative and positive.

It is in midcortical layers that the sign of the evoked potential inverts when recorded with a penetrating electrode. This is quite consistent with an afferent cell origin (but see Eccles (8)). A dipole existing about the discontinuity in membrane properties of the afferent cell should have a potential value which in the vicinity of the junctional region would be similar to that at a great distance (*i.e.*, at the reference electrode). Since, according to Lorente de Nó (16), thalamic afferents retain their myelin into midcortical layers (layer IV) before losing it to undergo terminal arborization, the depth at which potential inversion occurs should lie at the junctional region near layer IV. Experiments at the cortex agree with this thesis to within the limits of their resolution, and the findings in the spinal cord are similar. Other evidence has been provided to suggest that the surface positive cortical potential arises in the afferent neuron. In particular, Adrian specifically mentions the possibility of a tract after-potential as the underlying phenomenon (1), in part because following ablation of the cortex there was still recordable from the exposed white surface a positive potential of some 10 to 20 msecs. duration.[7] The findings of Hunt and Goldring (13) are consistent with this view. They have observed that while the cortical surface negative response is present early in the neonatal period, the cortical surface positive response does not develop until the afferent tracts become myelinated (*i.e.*, differentiated into myelinated and non-myelinated regions; but see alternative interpretation of Bishop and Clare (3)).[8]

[7] Just such a potential deflection would result from a variation in the demarcation current if a negative after-potential existed in the underlying white tracts as it would with cortex intact provided collaterals on the average showed a small post-spike variation in membrane potential.

[8] It should also be noted that Chang and Kaada (6) offer strong arguments for a purely cortical origin for the first phase of the evoked response of the optic cortex (their deflection 5).

For the several reasons given above it may be tentatively proposed that cord- and cortical-evoked potentials have homologous origins and that the first phase of each arises in part as a function of myelinated tract after-potentials. In the spinal cord this provisional characterization of primary cell currents may be visualized by considering the primary afferent cell as a tripolar structure composed of three contiguous segments (root-column-collateral) of differing intrinsic recovery sequences; for a period during the column negative after-potential the middle segment appears to serve as a stationary sink of current flanked by two sources, one in the root and the other in the collaterals.

Whether or not the hypothesis advanced here is valid, it serves to emphasize a fact of some significance: namely, that in the absence of specific knowledge about the uniformity of membrane properties in neurons which compose tracts of the central nervous system, caution must be exercised either in excluding or in accepting after-potential form as directly given by evoked potential.

The extracellular currents considered in this paper cannot now be judged with certainty for their role, if any, in transmission at *inter*neuronal junctions. But at those regions of change in membrane properties within a neuron which may be designated as *intra*neuronal junctions the currents which arise there must act to inhibit or facilitate repetitive conduction across such junctions as normally transmit with small margins of safety. The degree of invasion by successive spike potentials into presynaptic arborizations, frequently postulated to be incomplete, would be strongly influenced thereby.

SUMMARY

The prediction that a system of currents flows between the dorsal column and the dorsal root due to differences in their after-potentials was found to be consistent with the experimental findings. The form, magnitude, duration, and sign of the electrotonic component DRα fulfill the requirements of the postulated system.

A contribution of tract after-potentials to the evoked potential of intramedullary structures is indicated.

It is a conclusion of this and previous studies that profound changes occur in certain membrane properties of myelinated primary afferent axons as they penetrate the central nervous system. The working concept of abrupt intraaxonal junctional regions is therefore justifiable.

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