THE CLASSIFICATION OF NERVE FIBERS

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As more is learned about the mechanism of operation of the central nervous system, the characteristic of the mechanism that becomes the most impressive is the exquisite timing of the events. For example, if an impulse of subliminal effectiveness impinges upon a synapse, it leaves a trace which lasts only a few tenths of a millisecond. If within that time another impulse, likewise of subliminal effectiveness, arrives at the same region on the surface of the neuron, the two impulses together will raise excitation to the threshold, and a relayed message will be sent upon its way. Stated in other words, if the two impulses arrive at the synapse at an interval of separation longer than a few tenths of a millisecond, there is no transmission; and the local disturbance that each impulse produces dies out promptly, to leave the junctional area restored to its antecedent state ready for any new impulses that may be coming on. Transmission at any point thus depends upon the arrival of excitation from the various controlling centers in exactly the right time relations. Thereby the flow of impulses is always properly directed toward the end reaction appropriate to the conditioning data picked up by the body receptors.

The timing of impulses starts in the beginning of all reactions—in the sensory messages that report changes in the state of the body and in the character of the environment. At least no other interpretation explains as satisfactorily why sensory messages are carried at so wide a range of velocities. For some reason not at all well understood it is necessary for some impulses to shoot out ahead to prepare the central nervous system for other impulses that arrive later. In as simple an event as pricking the skin of the finger with a needle there is first a short burst of impulses carried in fibers conducting at 90 meters or more per second (20). Then there follow impulses at other velocities, many at about twenty meters per second; and finally there is a trail of impulses at about one meter per second. Before any of the last mentioned group arrives at the centers, a complex neurological event has taken place—the withdrawal of the hand; and the sensation of pricking has been appreciated. The sensation aroused by the slow fibers is still, however, that of pain indistinguishable qualitatively from the pain already felt. But in its setting it has a stinging, irritating character. It can be relieved by rubbing the spot, that is, it may be inhibited by sending into the central nervous system a flood of impulses carried in rapidly conducting fibers.

It is not my intention in the present paper to follow up this argument, but to let the observations stand as an introductory background to the main theme, which is the basis of the numerous velocities in nerve fibers. Visualization of the wide range of velocities can best be accom-
plished by showing what happens when a sensory nerve is artificially stimulated with an electrical shock of short duration. All the impulses start out together from the locus of the stimulus, but the fast impulses forge ahead. Thus, if a lead be taken at some distance from the point of stimulation, at the beginning of the action potential there are initially recorded the first impulses to arrive; and the other impulses are recorded later, at times inversely proportional to their velocities. As all the velocities are not equally represented among the fibers, the result is an action potential with distinctive elevations. The contour here presented (Fig. 1), taken from the saphenous nerve of the cat, is characteristic of all sensory nerves. It starts with a high elevation contributed by fibers conducting at velocities ranging approximately between 95 and 35 m.p.s. The high elevation is succeeded by a very small one in which velocities between 35 and 22 m.p.s. are represented; and this in turn is followed by a somewhat larger one representing velocities between 22 and 15 m.p.s. In the wake of the latter there are still a few impulses at a velocity slower than 15 m.p.s.

Figure 1 (left). Action potential of the saphenous nerve of the cat as recorded after 4 cm. of conduction from the stimulating cathode. The first disturbance in the line marks the moment of stimulation, and the interval between this point and the start of the first high elevation measures the conduction time of the fastest fibers. The potentials contributed by the other fibers follow in succession.

Figure 2 (right). Action potential of the saphenous nerve of the rabbit as recorded after 4 cm. of conduction.

The first elevation is actually a fusion of two elevations, as can be shown in leads at long conduction distances, or more easily in the nerves of young animals (Hrush), or in the saphenous nerve of the rabbit (Fig. 2). For the sake of ease of reference, the elevations have been given names. The terminology has undergone a series of revisions in the course of its historical evolution, and it is not entirely satisfactory. Subject, however, to the definition here given, it seems well to adhere to the original designations in terms of the Greek alphabet; and the elevations are accordingly called alpha, beta, gamma, and delta. Collectively the fibers so designated are spoken of, for reasons that will appear later, as the A group. The group takes in all the medullated fibers of somatic nerves. The remaining, unmedullated fibers are included in the group known as C. In order to visualize the potentials in C fibers it is necessary, because of their small size, to resort to much higher amplification.
than is necessary for the A fibers; and because of their slow velocities, to much lower sweep speeds, (Fig. 3).

After this preparation we are ready to penetrate further into the velocity problem. At the outset it may be stated that the size of the fibers is to play a large part in the argument. The first association of velocity with fiber size was made by Göthlin in 1907, long before the advent of the modern vacuum tube era in electrophysiology, and at a time at which knowledge about velocities was in a highly fragmentary state. Göthlin’s contribution is to be recalled merely because of its inherent fundamental idea, as his treatment was a theoretical one based upon the structure of axons and the Thomson cable equation, and his deductions are not in accord with the facts about conduction as they are now known. The association of nerve function and fiber size again

![Figure 3. Drawing of the action potential of the saphenous nerve of the cat, constructed from two records made at different amplifications and sweep speeds. Activity of all the fibers is shown. The A complex depicts activity in the same group of fibers represented in Figure 1 (but from a different nerve). The interval between the A and C elevations is filled in by the negative after-potentials of the A fibers.](image)

appeared independently four years later in the work of Lapicque and Legendre. What these investigators actually did was to measure chronaxies and correlate them with the axon size, and their statement that the velocity in the fibers was in direct relationship with the size was purely an inference. As it has turned out, it has been proven that the inference was correct, but that the chronaxie-size relationship had to be abandoned. Blair and Erlanger in 1933 demonstrated that the chronaxie of frog fibers is almost constant in the velocity range between 7 and 30 m.p.s.; and Lapicque, as the result of some experiments performed with Pèzard, has now modified his view and gives prominence to his older idea that chronaxie is a parameter of the protoplasm of the fibers.
In 1924 it was my good fortune to be working in Prof. Lapicque's laboratory at the Sorbonne. At that time the observation that Erlanger and I had made of the temporal dispersion in the action potential of nerves was very recent, and when I showed the records to Prof. Lapicque he immediately said that the velocity variation at the root of the dispersion must be attributable to fiber-size variation. Thereupon we proceeded to test the hypothesis (14). Because of the supposition that chronaxie might be related to the cross-sectional area of the axons, which was incorrect although we did not know it, we tried first to correlate velocity and area, but with no success whatever. Then we tried the first power of the diameter and the result was much more promising. At this time the work was interrupted by my return to St. Louis, and it was there taken up again with Dr. Erlanger.

Before going further it will be necessary to say a word about the method employed. The first step was to record the action potential at a known distance of conduction. Then the nerve was sectioned and a distribution map of the fiber diameters prepared. The velocity of the fastest fiber, which could be read from the record, was assigned to the largest fiber and the velocities of the other fibers were calculated therefrom on the basis of the diameter ratios. There was experimental evidence to show that the spikes all had the same duration and it was calculated that the recorded heights of the spikes should be as their cross-sections. Accordingly, the further procedure was to calculate the potential that fibers of each size should contribute to the compound action potential, plot the result as spikes at the conduction times determined by the velocities, add the spikes together, and compare the sum with the action potential as actually recorded. The resemblance of the reconstructed and the recorded potentials was remarkably close, and it was felt that at least a first approximation to the size rule had been found.

But, whatever there was of satisfaction was only that which the ignorant can enjoy. And it was short-lived. The situation may be compared to the knowledge of geography before America was discovered. Our fiber world ended at gamma, with an uncertain and nebulous delta in sight beyond it. As soon as high gain amplifiers were built it was possible for us—that is, for Dr. Erlanger and myself—to see that there were two well-defined elevations that had not previously been recognized, those now labeled delta and C. With the finding of these elevations the fiber problem became wide open again. Reconstructions made under the rules gave so inadequate a fit with the recorded action potential as to indicate that something was wrong. Indeed, it was even doubtful whether the method was applicable at all, as it could be expected to work only within a homogeneous group of fibers. A new question was raised: How many kinds of fibers are involved? The natural inference was that there were three kinds. But there was difficulty in connection with the second elevation, which was then labeled B and is now labeled delta in mammalian nerves. The fibers in the B elevation in frog somatic nerves were obviously not homologous with those in the mammal. A solution of the difficulty came through a parallel study of visceral nerves made by Bishop and Heinbecker.
These investigators delimited the B elevation in visceral nerves and showed through estimations of the spike durations and the refractory periods that the constituent fibers are different from those in the C elevation and in the alpha portion of the A elevation. In the frog the B fibers pass through the grey rami into somatic nerves, but they do not do so in the mammal. The fibers which produce the elevation, then labeled B, in the action potential of mammalian somatic nerves were shown to have properties much closer to those of the fast A fibers than to those of the B fibers of visceral nerves. Thus, if they really were different from A fibers, a separate, fourth class had to be defined.

Without important additions or changes the formulation remained as here stated until 1933, when Blair and Erlanger introduced a radically new idea. On the basis of data obtained upon single frog-axons,

![Figure 4](image)

Figure 4. Course of the whole potential cycle in a unit mammalian A fiber, drawn to scale from two records made at different amplications and sweep speeds. It can be seen from the almost invisible size of the positive after-potential in this drawing that much higher amplification is needed to reveal the after-potentials than is needed for the recording of the spike.

which indicated that from the fastest fibers down to those conducting at one meter per second there is a progressive increase in the duration of the spikes and the refractory periods, these investigators proposed that the fibers make up a continuously varying series; and they felt that the division into groups was unjustifiable.

In the past few years our laboratory, for two reasons, has taken a renewed interest in nerve fibers. The first and foremost reason was that there was a need for more precisely determined constants of mammalian fibers as equipment for the interpretation of the events that take place in the central nervous system. The second reason was to prepare the way for a return to the old problem of velocities. With the new information there came new tests for homogeneity. Previously the classification of fibers had depended largely upon the properties of the spike. But the spike is only part of the action potential. The whole action potential is
made up of three parts: first the spike, then a negative after-potential, followed by a positive after-potential (Fig. 4). The after-potentials are even more typical of the fibers than the spikes.

Every precaution was taken to keep the nerves in the best possible physiological condition. Unlike the spikes, which are very stable, the after-potentials are extremely labile. Environmental changes alter both their form and duration; therefore, they are very sensitive indicators of the state of the nerve. With this lability it may perhaps be asked how one can know what the normal is like. The answer is the

Figure 5 (left). Action potentials in the sphenous nerve of the cat recorded at the distances indicated on the ordinates. Because of crowding in the figure, only the projection of the events on the base line is shown at 2 cm. of conduction. The time interval subtended by the converging lines at the axis of abscissas measures the duration of the alpha and delta spikes. (Gasser and Grundfest, 1939.)

Figure 6 (right). Unitary axon spikes arranged according to velocity (simultaneous responses in 2 fibers in record 4). The velocities are marked at the left in meters per second. (A spike of a 90 m.p.s. fiber is shown in Figure 8.) All records have the same time scale. (Gasser and Grundfest, 1939.)

following. The after-potentials determine a cycle of excitability, and if the form of the after-potential corresponds exactly to the excitability as determined on the same nerve in situ under its natural perfusion of blood, there is every reason to believe that the fibers are normal.

Alpha spikes recorded from single axons in spinal roots last 0.4 to 0.43 msec. (Fig. 8). In nerve fibers they may appear at this duration or a few hundredths of a millisecond longer. But the last few hundredths of a millisecond cannot be considered as being significant, as the recorded duration depends in part upon the material from which the spike is led—that is, upon the distance beyond the active point at which the potential is picked up. Now let us inquire into the range of the spikes to which
this duration applies. Does it extend to the delta elevation, for example? The quickest way to become oriented as to the answer is to make an extrapolation of the duration of the elevation, as recorded after conduction, back to zero distance, where there is no dispersion of the axon potentials. In this way one obtains information forthwith about the whole group of five hundred or more fibers. The result of the procedure is apparent in Figure 5. At zero distance the duration of the delta group is the same as that of the alpha-beta group, about 0.5 msec. One could not let the matter rest, however, without recording the spike duration in single axons. When thus recorded the durations all fall between 0.4 and 0.5 msec, without systematic difference with respect to velocity (Fig. 6). Therefore, within the limits of the accuracy of measurement, in fibers in which the velocities range between 10 and 115 m.p.s., the spike durations may be considered as being constant.

The refractory periods, on the other hand, vary in a continuous manner, as described by Blair and Erlanger for frog fibers. This observation was the occasion for considerable surprise, as no previous examples had been found of exceptions to Adrian's original finding that the absolutely refractory phase ends at the base of the spike. The variation of the refractory period is one of the legs of support of the contention that fibers vary to make a continuous series; therefore it demands special consideration. It is at this point that the usefulness of the after-potentials with their accompanying excitability cycles enters. For the refractory period has no greater significance in the characterization of fibers than have the after-potentials. And it may be said at once that the excitability cycles—and the after-potentials, where they can be recorded—are identical throughout the series. Figure 7 shows the

![Graph showing after-potentials and excitability cycle of delta fibers.](image-url)
after-potentials and the excitability cycle in the delta fibers of the cervical sympathetic nerve of the cat, and the excitability cycle in the delta fibers in the saphenous nerve. They resemble their counterparts in alpha fibers. Some hitherto unsuspected variable must enter into the determination of the refractory period, probably the fiber size, as it also enters into the determination of the resting threshold of excitation. Support for this view is also found in Hursh’s measurements on immature alpha fibers. In young animals, in which the alpha fibers are small, the refractory period is longer than it is in the adult. Taking into consideration all the evidence, the medullated fibers of mammalian somatic nerves may be regarded as constituting a homogeneous series.

Figure 8 (left). Spikes recorded from unit axons of the A, B, and C groups. (See references 10 and 11.)

Figure 9 (right). After-potentials of the A, B, and C groups. The records are from multiple fiber preparations. (See references 10 and 11.)

In B fibers the spike, lasting about 1.2 msec., is longer than in A fibers (Fig. 8); and the after-potentials are in even greater contrast. Whereas the A fibers have a negative after-potential lasting about 15 msec., there is normally no visible negative after-potential in B fibers (Fig. 9), although one may be developed by special procedures. And whereas the positive after-potential in A fibers lasts only until about 70 msec. and in size amounts to only the equivalent of 0.1 to 0.4 per cent of the spike potential value, the B positive after-potential lasts 100 to 300 msec. and at its maximum has a size 1.5 to 4.0 per cent of that of the spike. The velocities found in B fibers range between 15 and 3 m.p.s. Thus, inasmuch as the slowest A fibers in the cat conduct impulses at about 10 m.p.s., there is an overlapping of velocities between
the two groups. Likewise there is an overlapping in the thresholds of excitation. The break between the two groups comes in the durations of the spikes and the characteristics of the after-potential; and it is obvious that neither of these features is tied to the velocity. In this connection it is interesting to recall that the delta fibers in the saphenous nerve of the rabbit, which are A in quality, have velocities in the range usually associated with B.

In the C fibers, which conduct impulses at velocities between 2 and 0.6 m.p.s., another sharp break in the properties occurs. The C spike lasts about 2 msec. (Fig. 8) and is followed by a negative after-potential lasting 50 to 80 msec. (Fig. 9). The latter in turn is succeeded by a positive after-potential traceable for 1 to 2 sec. Wherever the C fibers have been properly studied they have been found to be unmyelinated. And they are set off from other fibers in other ways, as for example in their high resistance to asphyxia.

Recovery of excitability after a single response

![Recovery of excitability after a single response](image)

Figure 10. Course of recovery of excitability following single responses of A, B, and C fibers to excitation.

As a final point in the differentiation of fibers the course of their recovery of excitability following excitation must be mentioned. The excitability curves are quite characteristic and follow the configurations of the after-potentials (Fig. 10).

When all the data are tabulated it is apparent that while all velocities are present between the fastest and the slowest—between 115 and 0.6 m.p.s.—there is no corresponding variation in the properties. The fibers fall into three distinct groups with sharp boundaries. The classification of fibers made in this way, however, cannot be taken as the end of all classification. A number of observers have shown that there are large differences in the acetylcholine content of fibers—in the ventral roots as opposed to the dorsal roots, and in preganglionic fibers as opposed to postganglionic fibers. But these differences in metabolism do not reflect themselves in observations made with electrophysiological methods and they are part of another story.
With the confirmation of the validity of the division of fibers into groups, the way now became clear for the reinvestigation of the size-velocity relationship. Before entering upon a description of the final experimental drive made by Dr. Grundfest and myself, it will be necessary to mention two interim developments in order to fill in the picture of the background against which we were working. After observing a linear relationship between the spike size and conduction velocity in frog fibers, Blair and Erlanger reasoned that if the spike size is proportional to the axon area, as had always been held, the velocity must vary as the square of the diameter. Pumphrey and Young, on the other hand, concluded from observations made on velocities in relation to size in squid fibers that the velocity varies more nearly as the square root of the diameter. Even after due consideration is given to the difference between the two forms studied, the divergence of opinion inherent in these formulations is enormous. The correlating factor in one case is the fourth power of that in the other.

![Figure 11](image_url)

**Figure 11.** Velocities of conduction in the most rapidly conducting fibers of selected nerves, compared with the diameters of the largest fibers in those nerves. (Hursh, 1939.)

Before Pumphrey and Young's paper was published, Hursh in our laboratory was already making correlations between the maximal velocity and the size of the largest axon in mammalian nerves selected to yield collectively a wide range in diameters. When the data were completed they represented velocities from 8 to 117 m.p.s., and the points connecting velocity to fiber diameter all fell about a straight line (Fig. 11). Not only did the data confirm the originally proposed diameter-velocity relationship, but they demonstrated its applicability at a range of velocities at which it had not previously been shown to apply. It may be asked, then, why the matter was not allowed to rest there and why we went on to the laborious reconstructions. The reason, as will soon become apparent, is that the reconstruction method affords a much more sensitive test of a velocity hypothesis.
Most of the observations were made on the saphenous nerve, usually of the cat, because of the well-marked elevations in the action potential with which the fibers may be fitted. After the action potential had been recorded and a map prepared of the distribution of the fibers according to sizes, it was possible from mere inspection of the map to make straight-away certain predictions about how the fibers would enter into the compound action potential. It can be seen in Figure 13 that the fibers fall into two main piles which should center on the alpha and the delta elevations. The delta elevation should start with fibers 3 to 4 μ in diameter; and, as the largest fiber in the nerve has a diameter of 14 μ and the factor relating the velocity of the fastest alpha fibers to that of the fastest delta fibers is approximately four, it follows that the delta elevation would be in the right place in the reconstruction.

After all the favorable preliminary indications it was surprising to see how poor were the first reconstructions made under the old rules. The delta elevation was about in the right place, but far too small. Numerous as were the small fibers, they did not supply nearly enough area to the end of the action potential. Another fault was that instead of a clean separation between alpha and delta, alpha crowded over and filled up the intervening space.

The most serious fault was the lack of potential area in the delta region—so serious that it revealed the fact that the assumption of the spike height varying as the cross-section of the axon must be incorrect. Almost every other part of the original rules had been questioned, rightly or wrongly. But it had occurred to no one to question this one, probably because it was hardly recognized that it was an assumption. It was based on the sound physical fact that the potential drop across a resistance, through which a current is flowing from a source that would yield the potential, E, on open circuit, depends upon the internal resistance of the source. As the resistance of the axons would vary as their cross-sections, this dimension was taken to be the controlling one in the calculation of the spike heights used for the first reconstructions; and, with the apparent success of the reconstruction of the early part of the action potential there was no reason for doubting its validity.

When once the difficulty was recognized, it was easy to see what the correct relationship must be. Blair and Erlanger, it will be recalled, had shown that the spike height in frog fibers varies directly with the velocity; and Zotterman had confirmed the relationship in mammalian fibers. Taken together with Hursh's demonstration that the velocity varies with the axon diameter, the correct interpretation of the observation of Blair and Erlanger is that the spike height varies as the diameter. Thus the sole support for the viewpoint that the velocity varies as the cross-section was removed. What had been neglected was the fact that the resistance of a nerve fiber depends not only upon its cross-section, but also upon a length. It has previously been pointed out that the spike durations are constant; therefore the wave length varies with the velocity, that is, with the axon diameter. Thus the proper statement about the internal resistance controlling the height of the spike is that the resistance varies directly as the diameter—because of the wave length—and inversely as the square of the diameter—
because of the effect of the cross-sectional area. The net variation, therefore, is inversely as the first power of the diameter. I will not bother you with the calculation of the spike heights when this variable is introduced. Suffice it to say that under the conditions obtaining in nerves, the spikes vary approximately as the axon diameters, as the experimental data indicate. It is the experimental fact, as illustrated in Figure 12, that I want to stress rather than the calculation.

With the needed potential area supplied, attention could be directed to other faults in the reconstructions. The alpha elevation was too broad and the alpha crest came too late. This fact meant too much temporal dispersion. Not only was all possibility that the velocity might vary as the square of the diameter removed, but linear variation itself appeared to be too great. And one thought of Pumphrey and Young's observations on squid fibers.

Figure 12. Comparison of the duration and heights of spikes in single axons conducting at different velocities in a branch of the saphenous nerve of the cat. The two groups are from different preparations. Left, velocity_s/velocity_s^2 = 4.2; height_s/height_s^2 = 4.3. Right, velocity_s/velocity_s^2 = 3.3; height_s/height_s^2 = 3.8. (Gasser and Grundfest, 1939.)

By now the correspondence between the recorded and the reconstructed potentials was so close that it was decided to change the plan of attack, in order to bring out wherein the discrepancies lay. It was decided to locate the fibers in the reconstruction in the right places, to cause them to make their proper contributions to the potential form, and then to calculate the velocities that would give them those positions. Thereby empirical velocity-diameter curves would be derived. In each instance excellent fits were obtained, as Figures 13 to 18 will show. The extreme sensitivity of the method will be noted. An error of a fraction of a micron in locating the slower fibers would throw their potentials grossly out of position. In each instance the same pattern emerges in the size-velocity curves. While their ends touch upon a straight line, the intermediate course is serpentine.

The next step was to account for the features of the size-velocity curves. The first question asked was whether any part of the curve was traceable to the fact that the outside diameters instead of the axon diameters had been used in the reconstructions. Use of the outside diameter is valid only in so far as its ratio to the axon diameter is
Figures 13-18. Distributions of fibers according to size and reconstructions of the action potentials from these distributions, shown for three different nerve preparations (Gasser and Grundfest, 1939). In the reconstruction curves the contour of the action potential as actually recorded is represented by a solid line. Where the reconstruction deviates from the record, the deviation is shown by lines made up of dashes or dots. Insets, corresponding size-velocity curves. Each dot in the curve represents a triangle in the reconstruction. Figs. 13 and 14 (upper). Saphenous nerve of the cat. Action potential record and reconstruction made for 4 cm. of conduction. Figs. 15 and 16 (center). Bundle of the saphenous nerve of the rabbit. Action potential record and reconstruction made for 4 cm. of conduction. Figs. 17 and 18 (lower). Saphenous nerve of the cat. Action potential and reconstruction made for 6 cm. of conduction. In Figure 18 there may be seen the positions of the spike potentials in relation to the sizes of the fibers contributing them.
A constant ratio was claimed by Donaldson and Hoke; but the more recent measurements of Schmitt and Bear and of Arnell are not in accord with their description. Arnell's data show that the myelin is relatively thicker on fixed mammalian fibers smaller than 8 micra. As the slopes of the velocity-diameter curves are greater below 8 µ, the observation appeared to be significant to our problem. If the fibers below that diameter have relatively thicker myelin sheaths, the fiber velocities determined by the axon would be slower than those indicated by the outside diameter and cause a depression in the outside diameter velocity curve.

We fortunately had in our possession a preparation of a fasciculus of the saphenous nerve containing 675 fibers, on which both the inside and the outside diameters had been measured. The ratios of the diameters were calculated. Above 8 µ the ratio was practically constant at 0.69, a value corresponding with Donaldson and Hoke's figure of 0.71. Below 8 µ the axon diameter outside diameter ratio fell off progressively.

![Figure 19. A, fiber diameter-velocity curves from Figures 14, 16, and 18 calculated in terms of the axon diameter and represented respectively in curves 1, 3, and 2. B, curves in A brought together by multipliers. (Gasser and Grundfest, 1939.)](image)

With the aid of this curve, the size-velocity curves were recalculated in terms of the axon diameters, with the result that in two of the derivative curves the initial upward concavity disappeared, and in the third it was nearly obliterated (Fig. 19A). The three curves were then brought together by multipliers, in order to visualize the form of the axon-diameter velocity curve when freed from idiosyncrasies of the individual curves. While superficially the composite curve is linear, a close inspection reveals the fact that the points fall along a line having a slightly downward curvature (Fig. 19B). Whether or not this residual curvature is real cannot be stated. Some of the curvature can be accounted for on the basis of technical difficulties in measuring diameters, which I will not go into. And at times one gets perfect reconstructions of alpha spikes on the basis of a rigidly linear relationship between size and velocity. On the other hand, there is no theoretical reason why the graph should be a straight line, and in all probability it is a very flat curve approaching a straight line. Whatever deviation from linearity is
present causes the velocity to change more slowly than it would in strict
proportionality to the diameter of the axons. But in any case, the
deviation is negligible compared with that in the root functions proposed
by Pumphrey and Young for squid fibers.

As the facts now stand, the velocity of conduction may be considered
to be approximately in linear relationship to the diameter of nerve axons.
The correlation is an empirical one and would apply equally well to any
property of the nerve fibers which varies in the same manner as the axon
diameter. With this statement we have the last entry in the log of the
course taken in the solution of what at the outset appeared to be a
simple problem.

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