THE ELECTRICAL REACTION OF MUSCLES BEFORE AND AFTER NERVE INJURY.

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(Continued from p. 65).

Case 7.—This is the case of a patient aged 28, who had an attack of acute poliomyelitis on October 8, 1915. The paralysis remained in the trunk, right arm, and legs. He was examined on December 27, 1915. At this state the left tibialis anticus had moderate voluntary power, but did not react to faradism applied in

![Diagram](Fig. 8)

the region of the motor point unless a very strong current was used. It reacted more readily when the current was applied lower down the leg. The right tibialis anticus had no voluntary power at all and did not react to the strongest faradic currents which could be
used. Fig. 8 shows the curves for both legs with the cathode applied high up near the motor point. That for the left tibialis anticus is drawn as an interrupted, and that for the right as an unbroken line. The right is a continuous curve, but the left shows a discontinuity when the strength is increased to 34 and the duration reduced to 0.0056 second. With smaller durations the strength remains constant and does not rise again until the duration is in the neighbourhood of 0.0004 second. Fig. 9 shows the curve for the left tibialis anticus with the electrode at a lower level. The curve rises gradually until the duration is reduced to 0.064 second and then remains constant at a strength of 14 until the duration is in the neighbourhood of 0.0004 second, after which it rises steeply.

Case 8.—The patient had an attack of toxic polyneuritis of unknown origin in August, 1915. To begin with both arms and legs were paralysed, but when he was examined he was recovering rapidly, had fair voluntary power in the arms and could walk for a short distance unaided. In spite of this the leg muscles showed no response to strong faradic currents, and gave the typical reaction of degeneration with galvanic currents.

The curve for the tibialis anticus on each side is shown in fig. 10. The curve for the right leg is an unbroken line, and that for the left is interrupted. Both show a discontinuity, that on the left occurring when the strength is 20, and that on the right when the strength is 31.
Case 9.—The patient was shot through the right thigh in May, 1915. Immediately after the injury the leg was completely paralysed below the knee, and there was complete loss of sensation over the area supplied by the sciatic. There was some return of voluntary power in the calf muscles in September, but the tibialis anticus was still paralysed. The reaction to faradism was not determined. The strength duration curve was determined on September 24 and again on October 7. At the first determination the current strength was not increased above 20; below this strength the curve is continuous and the chronaxie is 0.004 second. Fig. 11 shows the curve on October 7. There is a discontinuity at a strength of 24 and a duration of 0.0024 second. The lower part of the curve agrees in all respects with that determined on September 24. A study of figs. 6 to 11 shows that there are certain common features in the strength-duration curves of all the muscles which gave evidence of recovery. In every case the curve is discontinuous and appears to be composed of two curves of the same form but with very different time constants. The lower curve rises gradually at long durations; the upper starts from a higher base line and does not begin to rise until the duration is very short. The point of intersection of the two curves varies widely, but it would appear from figs. 6 and 7 that it occurs at weaker strengths and longer durations as the condition of the paralysed muscle improves. If the
time constants of these curves are determined it is clear that the upper curve has a short chronaxie which approximates closely with that for healthy muscle with intact nerve supply. In fact, the

only point of difference between the curve for healthy muscle and the upper portion of these complex curves lies in the greater current strength needed in the case of the incompletely recovered muscle.
Again the lower half of the complex curve is very much like the curves for completely denervated muscle. The chronaxie of this part of the curve is certainly much more variable than the chronaxie after division of the sciatic, but the figures are of the same order in both cases. A good example of this agreement is shown in fig. 12. In this figure the curve marked a is that for the right tibialis anticus in Case 6 (fig. 7) on a larger scale, b is a typical curve for denervated muscle (Case 1, fig. 4), and c is a curve for muscle with intact nerve supply. If the curve a could be made to rise from a higher base line it is easy to see how the complex curve might be produced by a combination of the curve for intact with that for denervated muscle. There is certainly no sign of any gradual transformation of the curve for denervated muscle into that for healthy muscle, and this holds good for every case investigated.

### Table III.

**Commencing Recovery after Paralysis**

<table>
<thead>
<tr>
<th>Case</th>
<th>Chronaxie of lower curve</th>
<th>Chronaxie of upper curve</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>November 27</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Right 0.011 second</td>
<td>0.00008 second</td>
</tr>
<tr>
<td></td>
<td>Left 0.010 second</td>
<td>0.00010 second</td>
</tr>
<tr>
<td></td>
<td>December 8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Right 0.008</td>
<td>0.00016 second</td>
</tr>
<tr>
<td></td>
<td>Left 0.010</td>
<td>0.00012 second</td>
</tr>
<tr>
<td></td>
<td>26</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Right 0.0095</td>
<td>0.00016 second</td>
</tr>
<tr>
<td>7</td>
<td>Right 0.015</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Left 0.020</td>
<td>0.00010 second</td>
</tr>
<tr>
<td></td>
<td>0 (b) 0.020 (?)</td>
<td>0.00012 second</td>
</tr>
<tr>
<td>8</td>
<td>Right 0.020</td>
<td>0.00024 second</td>
</tr>
<tr>
<td></td>
<td>Left 0.0005</td>
<td>0.00040 second</td>
</tr>
<tr>
<td>9</td>
<td>September 24</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.004</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>October 7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.004</td>
<td>0.00012 second</td>
</tr>
</tbody>
</table>

Extreme values 0.020 Mean, 0.011 sec. Extreme values 0.0004 Mean, 0.00016 sec.

For denervated muscle (Table II) the corresponding values are:

Extremes 0.013 Mean, 0.011 sec.

For muscle with intact nerve supply (Table I) the values are:

Extremes 0.0025 Mean, 0.00016 sec.

The time constants of all the double curves are shown in Table III. The chronaxie of the slower curve is determined in the usual way, i.e., it is the duration corresponding to a strength twenty times that at infinite duration. To determine the chronaxie of the more rapid part of the curve the strength required when the discontinuity
appears is supposed to be the strength at infinite duration. This amounts to the assumption that the rapid curve if it could be investigated at long durations would not fall appreciably below the value corresponding to the horizontal part of the curve. On this assumption the chronaxie is the least current duration required when the strength is twice that corresponding to the horizontal part of the curve. In some cases this must be found by extrapolation as the actual strengths of current were not great enough.

The table brings out very clearly the agreement between the rapid part of the complex curve and the curve for muscle with intact nerve supply. The slower part of the curve agrees more or less with that for completely denervated muscle, but the close agreement of the average values is to some extent fallacious, since the extremes show a much wider variation in the case of incompletely recovered muscle than they do in denervated muscle.

**INTERPRETATION OF RESULTS.**

We have seen that in muscle with intact nerve supply and in denervated muscle the strength-duration curve is of a constant form, but that the time constant is much greater in the case of denervated muscle than it is in intact muscle. Further, the results obtained on muscles which are recovering from incomplete nerve lesions make it clear that the slow curve characteristic of denervated muscle does not return gradually and without any discontinuity until it approximates to that for intact muscle. Instead of this the curve for recovering muscle is complex and is always made up of two distinct curves, of which the slower corresponds more or less with that for denervated muscle and the more rapid with that for intact muscle. In the earlier stages of recovery the more rapid curve does not appear until the current strength is several times the minimal value. As recovery progresses the rapid curve becomes evident with weaker and weaker current strengths until eventually it would seem to oust the slower curve altogether.

This result is of very great importance, for it shows that during recovery the current takes effect upon two distinct excitable mechanisms with very different time constants. At first sight it might seem possible to explain this on the assumption that the current affected two groups of muscle fibres in different states of recovery. In poliomyelitis the different fibres of a paralysed muscle do not always recover at the same rate, and a part of the muscle may have voluntary power and response to faradism while
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the rest is completely paralysed and responds to galvanism only. These fibres might be so intimately mixed that it would be impossible to distinguish one set from the other by mere palpation. Consequently the strength duration curve might be composite because it was made up of curves for several sets of muscle fibres in different stages of recovery. However, if this were the case, we should expect to find not one discontinuity in the curve but several. In fact, there would be as many discontinuities as there were different stages of recovery in the fibres under investigation. Moreover, there would certainly be curves in which the time constant had a value intermediate between that for denervated muscle and that for muscle with intact nerve supply. Neither of these possibilities is realized in figs. 6 to 11. The only other possible explanation is that the mechanism upon which the current takes effect in muscle with intact nerve supply is quite distinct from that in denervated muscle, and that the great difference in the time factor in the two cases is not due simply to an alteration in the conditions of one and the same mechanism. On this hypothesis the recovery of the muscle is shown by the appearance of the mechanism characteristic of normal muscle in addition to the mechanism characteristic of denervated muscle. In the earlier stages of recovery the former mechanism needs a much stronger current to excite it than does the latter, but as recovery progresses the rapid mechanism becomes more and more excitable until eventually it replaces the slower mechanism altogether.

It is not difficult to find a satisfactory explanation of the nature of these two mechanisms. In the frog, Lucas and Lapicque have shown that the nerve fibres in the trunk of the sciatic and the nerve fibres in the substance of the sartorius and gastrocnemius react to much more rapid currents than do the muscle fibres excited directly. Now the great difference between normal muscle and denervated muscle lies in the fact that the former possesses healthy nerve-fibres running in the substance of the muscle, whereas the latter does not. Evidently in normal muscles there are two distinct excitable mechanisms, the nerve-fibre and the muscle-fibre, whereas in denervated muscle there is one mechanism alone. From the results obtained on the frog we should expect that the nerve-fibres would have a much shorter chronaxie than the muscle-fibres, and hence it is easy to see why the intact muscle gives a much shorter chronaxie than the denervated muscle.

During recovery from injury to the nerve, traumatic or toxic, the intra-muscular nerve-fibres become active again, but at first
we may suppose they are less excitable than usual. Under these circumstances we should expect to find a double curve, weak currents of long duration affecting the muscle-fibres directly and strong currents of short duration affecting the intra-muscular nerve-fibres. A process of this kind has been demonstrated experimentally in the frog’s sartorius by Keith Lucas. The strength duration curve of this muscle may be either a rapid curve characteristic of the nerve-fibres or a slower curve characteristic of muscle, or else a double curve showing both components. The type of curve in any particular case depends on the relative excitability of the nerve and muscle-fibres and on the distribution of the muscle-fibres in the neighbourhood of the electrodes. When the muscle is treated with curare the nerve-fibres gradually cease to transmit impulses to the muscle and consequently the more rapid component of the curve disappears, to return again when the effect of the curare works off. Fig. 13 shows a double curve obtained by Lucas,¹ from the sartorius of the frog and the resemblance between this and the curves for human muscle in figs. 6 to 11 is self-evident.

So far, then, the explanation is simple enough. However, there are one or two points which call for further discussion. In the

¹ *Journ. of Physiol.*** xxxv, p. 328, 1907.
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first place the mechanism which has been excited by currents of short duration has been identified with the intramuscular nerve fibre. If this is so, we should expect to obtain a curve with the same constants when the current is applied to the nerve trunk instead of to the muscle itself. The average value of the chronaxie when the cathode is applied to the external popliteal worked out at 0·00025 second for four different cases. This is slightly longer than the value for the tibialis anticus. The difference may, of course, be due to experimental error. However, there is another possible explanation which deserves mention. In the frog's muscle, in addition to the muscle and nerve fibres, Lucas has shown that there is a third excitable substance which appears to correspond in distribution with the nerve endings in the muscle. This substance has a shorter time constant than the muscle fibre, the chronaxie being 0·00017 second as against 0·0005 second, for the nerve. In the case of human muscles it is conceivable that a current applied to the muscle may excite a mechanism in the nerve endings rather than in the intramuscular nerve, and this may explain the lower value of the chronaxie for the nerve trunk. However, in the absence of more reliable figures, this cannot be insisted upon.

There is another possibility which must be discussed shortly. It is sometimes held that voluntary muscle is made up of two components, a fibrillar substance which reacts rapidly and a sarcoplasmic substance which reacts slowly. These two substances might perhaps be identified with the two excitable substances in human muscle, the fibrillar substance disappearing when the nerve degenerates. However, the idea has little or nothing in its favour. The separate existence of the fibrillar and the sarcoplasmic substances has never been proved, and there is nothing to show that they have the distinct properties with which they are credited. Furthermore, the electric reactions of the cold-blooded tissues are certainly due to the effect of the current on muscle fibres and nerve fibres, and it is difficult to believe that the reactions in human muscle are due to an entirely different mechanism. Much the same objection may be brought against the view that the two excitable mechanisms are to be identified with the white and red fibres of the muscle. It is true that the existence of these two sets of fibres is not in doubt, and it is true also that the white fibres degenerate more rapidly than the red. Again the white fibres give a brisker contraction when they are stimulated, and therefore they might well react to currents of shorter duration. In human muscle the two types of fibre are intimately mixed, and so
we might expect to find evidence of two different constants in the strength-duration curve. However, we have seen that the chronaxie of the rapid component of the double curve is equal to or slightly shorter than that of the nerve trunk itself. This in itself is very much against the suggestion that the rapid curve is due to the white muscle fibres rather than the nerve fibres or nerve endings, for, setting aside the comparatively close agreement between the chronaxie of the rapid curve and that of the nerve, no other case is known in which a muscle fibre has a time constant equal to or shorter than that of the nerve fibre which supplies it. Moreover, if the short chronaxie were due to the white muscle fibres, we should not expect it to disappear until the muscle showed definite signs of wasting, or to reappear until the wasting began to improve. Consequently we must revert to the idea that the two mechanisms in question are the muscle fibre and the nerve fibre.

A further point of interest concerns the value of the chronaxie for denervated muscle. After complete section of the sciatic, the chronaxie always lay between 0.013 and 0.009 second, and it has been suggested that this value is characteristic of muscle fibre excited directly. It is naturally impossible to verify this by direct experiment on a human subject, as the effect of the nerve fibres would have to be eliminated by curare or some such means. In the frog the chronaxie for muscle fibre is about 0.007 second, at 15°C., although we might naturally expect it to be longer than that for the muscles of a warm-blooded animal. This suggests the possibility that the chronaxie for human muscle fibre would be really shorter than 0.01 second if it could be measured when the fibre was in a healthy condition, and that after the nerve is destroyed the chronaxie becomes longer on account of the abnormal state of the muscle. This view is upheld by the fact that in Case 9, where the nerve injury was incomplete, the chronaxie of the slower component of the curve was 0.004 second, a value which is less than half the average for denervated muscle. In spite of this, it seems on the whole more probable that the figure of 0.01 second is the average value for muscle fibres, and that the short value in Case 9 is due to an individual peculiarity, and not to the improved condition of the muscle. If this were not the case, we should expect the chronaxie to become longer and longer with the lapse of time following section of the nerve. The figures in Table II show that this lengthening does not occur. Again, in Case 7 (poliomyelitis) the chronaxie of the lower part of the curve is 0.02 second, although there was some return of voluntary power
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in the muscle, and although the chronaxie for the other tibialis anticus, which did not show any sign of recovery, was only 0.016 second. Consequently the restitution of functionally active nerve fibres is certainly not followed by an immediate shortening of the chronaxie for muscle fibres. This does not imply that the chronaxie for muscle does not become longer as the muscle wastes, and becomes converted into fibrous tissue. The muscles examined in Table II had all been treated by massage and electricity, and they showed very little wasting; on the other hand, in a few cases of ulnar and median injuries, with marked wasting of the small muscles of the hand, the chronaxie appeared to be considerably longer, 0.025 or 0.03 second at least. These results are only rough approximations, as the method is very uncertain when applied to small denervated muscles in close contact with small healthy muscles. Still, no doubt, the chronaxie of human muscle fibre may vary from 0.004 second to 0.03 second or longer, according to the state of the tissue. The important fact remains that functional connexion with the central nervous system may be restored, although the chronaxie of the muscle fibres is twice as long as the average value for denervated muscle. The precise bearing of this result will be seen in the following section.

The conclusions arrived at in the present section may be stated briefly as follows: In muscle with intact nerve supply there are two distinct mechanisms upon which an electric current may take effect. These mechanisms are: (a) the intramuscular nerve fibres, and (b) the muscle fibres themselves. The nerve fibre responds to currents of much shorter duration than the muscle fibre, and in healthy muscle the strength-duration curve is always continuous and characteristic of nerve fibre alone. In muscle, which is in process of recovery after a nerve lesion, the curve is discontinuous, and is made up of two simple curves, one with the short-time constant characteristic of nerve, and the other with the long-time constant characteristic of muscle. After complete degeneration of the nerve, the curve is continuous, and has the long-time constant characteristic of muscle. The constant may vary with the condition of the muscle, but its precise value does not affect the possibility of reunion with the nerve.

The Bearing of these Results on Diagnosis and Prognosis.

If we accept the foregoing interpretation of the changes which follow injuries to the nerve, we can draw certain conclusions regarding the possibility of electrical testing in general without
reference to any particular method. For instance, it is clear that after a lesion which has produced complete degeneration of the nerve fibres, the electric reactions of the muscle itself can give no information at all as to the state of the nerve at the site of the injury. All we can hope to decide is whether the muscle is or is not in a fit state to recover its voluntary power when the nerve fibres have regenerated. We cannot tell whether the lesion is one which makes regeneration impossible and therefore demands operation, or whether regenerated fibres are already growing down towards the muscle. The present observations show that the condition of the muscle itself, as indicated by the time constant, is of no great importance. It may remain for long periods unchanged after the complete degeneration of the nerve, and voluntary power may return although the constant is twice as long as that six months after complete section (Case 7). The all-important factor is the condition of the nerve at the site of injury and this we cannot hope to determine by electrical methods.

The position is altered when there are some nerve fibres peripheral to the lesion which retain or have regained their excitability to electric currents. The presence of these fibres may be detected from the complex nature of the strength-duration curve and the production of a response in the muscle to currents of very short duration. If these fibres become more and more easily excitable it is safe to assume that the condition of the nerve is improving. In such cases the determination of the electric constants would show that the prognosis was favourable. However, these determinations would be of value only in those cases in which the possibility of recovery could not be deduced by simpler methods. For instance, if there is any return of voluntary power in the muscle there is clearly no need of an electrical examination to tell us that some of the nerve fibres have regained their function. In three out of four of the cases of commencing recovery which were examined by the present method, the return of a slight degree of voluntary power appeared to coincide with the reappearance of the nerve fibres which would respond to stimulation by electricity. In the fourth case (gunshot wound of the right thigh, Case 9) the strength-duration curve showed that some of the nerve fibres could be excited electrically although there was no return of voluntary power in the tibialis anticus. Even so the electric constants were not needed to show that the nerve was recovering as there was already a considerable return of power in
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the calf muscles. Nevertheless in so far as its history is concerned the case is typical of a large number of incomplete nerve injuries caused by gunshot wounds, in which the paralysis clears up without operation and much more rapidly than it should do if the nerve fibres below the site of injury had degenerated completely. In these cases it is evident that the nerve fibres below the injury must be still in some kind of trophic connexion with the motor cells in the cord though they are unable to transmit impulses from the central nervous system to the muscle. Unfortunately Case 9 was the only one of this class of injury which could be investigated by the present method, but it is not unreasonable to suppose that most if not all of them would agree with it in showing evidence of nerve fibres peripheral to the lesion and yet capable of responding to electric stimuli. If so the determination of the strength-duration curve would give valuable evidence of the likelihood of recovery. When the curve does not show the characteristic discontinuity, we can only say that the nerve fibres peripheral to the injury are inexcitable and that the possibility of recovery must remain in doubt. In such a case we may say with certainty that there is no method of electrical testing which will help to solve the question, since the current takes effect on the muscle fibres alone and the state of the muscle fibres gives no indication of the state of the nerve.

In conclusion we may say that a certain prognosis can be given only in those cases in which the nerve fibres peripheral to the injury retain some measure of excitability. The presence of such fibres is shown by the complex type of strength-duration curve and the efficacy of currents of very short duration. How far their presence may be detected by the simpler methods of testing in vogue at present is a question which will be discussed in the following section.

The value of these results in diagnosis is another matter. To a certain extent any method of diagnosis which gives additional information as to the state of a diseased tissue must be counted of value even though the information has no direct bearing on the treatment and prognosis of the case. The strength-duration curve gives definite information as to the state of the excitable mechanisms in nerve and muscle fibre and this information may be expressed in a quantitative form. Furthermore Lapicque, Kejth Lucas and A. V. Hill, have put forward theories as to the nature of electric excitation in which the constants of the strength-duration curve are given definite physical interpretations. These theories...
are based on Nernst's original hypothesis that, in order to excite, the current must bring about a certain concentration of ions at the surface of a semi-permeable membrane in the substance of the fibre. Thus in Hill's theory the constants of the curve enable us to tell the charge on the ions, their rate of diffusion, the distance separating the membranes on which they take effect and the degree of concentration which must be attained in a given time for excitation to take place. These may be calculated as readily in the case of human excitable tissues as in those of the frog, and in this way it should be possible to relate a change in the condition of the muscle to a change in the diffusibility of the effective ions or the degree of concentration which must be attained at the membranes, etc. No attempt has been made to treat the present results in this way, because they are too few for such an elaboration and because the present theories of excitation are admittedly incomplete and in need of alteration. For the present it must suffice to point out the possibilities of exact diagnosis which might be made on these lines.

Present Methods of Electrical Testing.

We have seen that the most important function of electrical testing is to determine the presence or absence of excitable nerve fibres peripheral to the damaged area. If these fibres are present some weeks after the injury the prognosis is good and recovery may take place without operation. If they are absent the prognosis is uncertain, but there is evidently no need to delay operation. It follows that the different methods of electrical testing must be valued according to their ability to detect these fibres. When the complete strength-duration curve is determined the presence of active fibres is shown by the complex type of curve and the response to currents of very short duration. A current of 0.0004 second will excite nerve fibres if it is strong enough, but it will not excite muscle fibres however strong it may be. So if we could always use a current of this duration and of variable strength, there would be no need to determine the full curve. It remains to be seen how far this may be done with the methods in use at present.

(1) The Faradic and Galvanic Current.—At first sight it might appear that the ordinary method of testing with the induction coil would be accurate enough, since the faradic current is of such

\[ Arch. f. d. ges. Physiol., cxxii, p. 275, 1908. \]
a duration that it will excite nerve but not muscle fibre. However, it is evidently not at all suited to detect the presence of nerve fibres if these are relatively inexcitable. It is well known that a muscle may regain some degree of voluntary power long before it shows any response to faradism, and in such a muscle there must be some functionally active nerve fibres. Such a condition was found in all the cases of incomplete recovery described in this paper. The explanation of this state of affairs may be seen from fig. 7. In this case both legs had some return of voluntary power and the left tibialis anticus reacted slightly to faradism whereas the right did not react at all. The strength-duration curves show that active nerve fibres are present in both muscles, but in the right leg the current must have a strength of 40 if it is to affect the nerve, whereas in the left it need not be stronger than 29. With a short duration it is quite possible to employ a constant current of strength 40 or more without causing much pain because the whole of the current is effective in stimulating the tissue. However, a faradic current of equal stimulating effect would be far too painful because the useful part of the current is only that fraction of the discharge when the electromotive force is at or near its maximum, and the remainder adds to the pain without taking any effect on the excitable tissue. Consequently the right tibialis anticus was put down as inactive to faradism because it was inactive to faradic currents of a strength which could be borne by the patient without an anaesthetic. Presumably the same holds good for every case in which there is voluntary power but no response to faradism. The nerve fibres need a strong current to excite them and a faradic current of this strength cannot be tolerated because of the pain it would cause.

(2) The Condenser Method.—This is clearly superior to the induction coil method because it is possible to select the duration of the discharge as well as the strength. Indeed, with some elaboration, it would be possible to map out the full strength-duration curve with a series of condensers of different capacities. The discharge is more painful than that of a constant current because the electromotive force falls off rapidly and the latter half of the discharge is useless. The duration of the discharge depends on the total resistance in the circuit as well as in the capacity of the condenser, but for practical purposes the difference in the resistance of two similar limbs is not great enough to matter. Consequently it should be quite possible to select a capacity which would affect nerve fibres without affecting muscle and to use this
for detecting the presence of active nerve fibres in incomplete lesions. It would be necessary to have some means of varying the strength of the discharge, or else to use a discharge so strong that there would be no chance of missing relatively inexcitable nerve fibres.

The ordinary method of using condensers gives information of very doubtful value. If the muscle will respond to discharges of very short duration, 0.05 mf. or less, we may safely infer that the nerve contains active fibres, but if a longer discharge is necessary, its precise value tells us nothing unless we know also the strength of the discharge relative to the strength required when the duration is infinite. If we do not know this we cannot fix even one point in the strength duration curve, and a change in the least capacity required to excite might be due to nothing more important than a change in the general excitability of the tissue brought about by increased skin resistance or excess of fluid in the subcutaneous tissues.

Consequently, there is nothing to be gained by stating the fact that a muscle responds to the discharge of a condenser of, say, 1 mf. capacity unless we can add that the strength required must be a definite multiple of that required at infinite duration. Without this information it is clearly quite impossible to determine the constants of the tissue.

(3) Otto May’s Method.—This method has not come into general use, but it is interesting from a theoretical point of view. May uses Leduc’s commutator for delivering a series of constant currents of short duration at definite intervals and determines the duration at which the contraction of the muscle is at its maximum. This duration becomes longer when the nerve is destroyed. Unfortunately, the height of the contraction depends on very many factors, all of which might be influenced in one way or another by alterations in the nerve supply. For instance, the height of the contraction will depend to some extent on the number of stimuli which take effect within a given time, on the rate at which the contraction develops and subsides, on the refractory period of the tissue, etc. The optimal duration of a series of discharges is certainly related to the state of innervation of the muscle, but owing to the many factors involved it becomes well-nigh impossible to deduce the precise condition of the tissue from the data given

1 Brain, 1913.
by May's method. In addition to this there is a certain amount of practical difficulty in measuring the strength of the contraction, though no doubt this could be overcome if the method were likely to afford information of much value.

(4) Laugier's Method.—This amounts to an attempt to determine the constants of the strength-duration curve by means of two stimuli of different duration. These are furnished by the make and break shocks of an induction coil, since the durations of these shocks are fixed quantities depending on the dimensions of the coil, and the duration at make is several times as long as that at break. A comparison of the strengths of current required to excite at make and at break gives a quantity which Laugier calls the indice de vitesse of the tissue and which is proportional to the slope of the strength-duration curve between the points corresponding to the durations of the make and break shocks. The index is not affected by simple changes in the excitability of the tissue, and a change in the index always denotes a change in the slope of the curve. The method is ingenious and simple to apply, but it is based on a misconception of the changes which take place when the nerve is damaged. If the rapid curve characteristic of normal muscle were transformed gradually and without discontinuity into the slow curve of denervated muscle and vice versa the slope of the curve between two fixed points should give all the information necessary to determine the condition of the tissue. However, we have seen that the transition is abrupt and that the two curves are quite distinct, the rapid curve becoming more and more prominent as the process of recovery advances. When the curve is discontinuous the slope between two fixed points will depend on the position of these points in relation to the discontinuity, and although a change in the slope will imply a change in the complex curve, the notion of the indice de vitesse loses its simplicity and is no longer a quantity definitely related to the constants of the tissue under examination.

Practically the method has the disadvantage that it cannot be applied to a muscle which has lost its excitability to faradic currents, since the stimuli are necessarily of very short duration. In any case it cannot be said to take the place of the complete determination of the strength-duration curve, since the double curve cannot be defined by two points only, whatever may be the durations corresponding to these points.

1 *Brain*, 1913.
The curve which expresses the relation between the least strength and the least duration of the current required to excite has the same form for human tissues as it has for the tissues of cold-blooded animals. The time constant of the curve is determined by the duration at which the current strength must be twice the minimal value. This duration is characteristic of the tissue examined and it has been named the "chronaxie." In healthy muscle with intact nerve supply the chronaxie is very short, 0.00016 second on the average, and it is slightly longer when the electrode is applied over the nerve trunk instead of to the muscle directly. When the nerve has degenerated the chronaxie is very much longer. Its average value is 0.011 second, and, in the case of the tibialis anticus at any rate, it shows surprisingly little variation with increased lapse of time following the injury. When the muscle is in process of recovery the slow curve of denervated muscle does not pass gradually into the rapid curve of healthy muscle; instead of this the first sign of recovery is marked by the appearance of a discontinuity in the strength-duration curve. With very strong currents the curve has the short time constant characteristic of healthy muscle, but with weaker strengths a slower curve appears and this has the long time constant of denervated muscle. The production of these double curves shows that there are two distinct mechanisms upon which the current may take effect; only the slower mechanism is present in denervated muscle, and as recovery takes place the more rapid mechanism comes into play and eventually predominates to the exclusion of the slower mechanism. Reasons are given for the belief that these two mechanisms are to be identified with the muscle fibre and the nerve fibre or nerve ending. Thus the appearance of the discontinuity in the strength-duration curve and the response to currents of very short duration shows that there are some excitable nerve fibres peripheral to the injury. This is to be expected in those cases of incomplete injury where the trophic influence of the nerve cells is not abolished although impulses cannot pass from the central nervous system to the muscle. When the curve is continuous and has the slow chronaxie only, all that can be said is that there are no excitable nerve fibres peripheral to the injury. The possibility of regeneration depends on the state of the nerve at the site of injury and this cannot be determined electrically. The condition of the muscle is not of very great importance, for voluntary power may begin to return although the chronaxie of the muscle fibres is twice
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as long as it is nine months after complete degeneration of the nerve. It follows that the most important function of electrical testing is to decide whether there are any excitable nerve fibres peripheral to the injury. If there are, and if they are becoming more excitable, then there is no need to operate, if not we cannot tell whether the nerve will regenerate or not and there is no reason to delay operation.

The different methods of testing in vogue at present give some information as to the presence or absence of these fibres. The method of condenser discharges is the most reliable, but the usual method needs several modifications; in particular the strength of current must be a definite multiple of that required at infinite duration, otherwise a change in the general resistance of the limb, etc., may be mistaken for a change in the time constant of the muscle.

The present investigations were carried out on the military cases at the National Hospital for the Paralysed and Epileptic, Queen Square, and I am deeply indebted to the medical and surgical staff of the hospital for allowing me to make use of these results.