DETERMINATION OF EXCITABILITY OF HUMAN MUSCLES

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It has been shown by D. N. Nasonov and D. L. Rozental (3) that chronaxie "is not a measure of the speed of a tissue reaction, or of the excitability of the given tissue." For the characterization of excitability, the use of the constants b, the intensity excitability threshold (rheobase), and a, the duration threshold of excitability, was proposed. These constants may be derived from the formula

$$v = \frac{a}{t^n} + b,$$

where v is the threshold potential, in volts, and t is the time of action of the current, in milliseconds (msec).

If t^n is large enough, v = b. If b is small enough, then $v = a/t^n$, when a will be "the threshold potential which causes stimulation within a given time of action, chosen by us as a unit, for sufficiently short intervals." Taking v in the formula $v = a/t^n$ (for n = 1) as unity, a will define the duration of the stimulation. Chronaxie will in no way define this time, since it follows from the formula $v = a/t^n + b$, when v = 2 b, that

$$2b = \frac{a}{Chr} + b$$

and hence that

$$b = \frac{a}{Chr}, \quad \text{or} \quad Chr = \frac{a}{b},$$

i.e., chronaxie will depend on the ratio of a to b, and these may vary relatively independently of each other. On the strength-duration curve chronaxie defines the point of inflection, where one expression, dependent on time, is replaced by another, time-independent one.

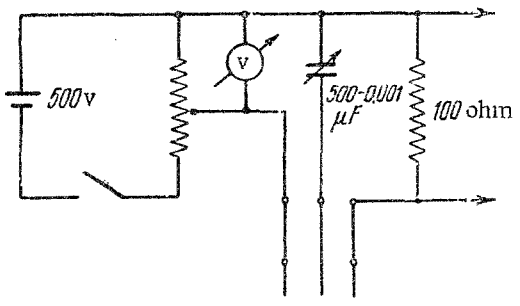

Fig. 1. Circuit diagram for determining excitability.

Inasmuch as chronaxie expresses some property of tissues, the possibility remains to be investigated that, although it is not a measure of excitability of tissues, it might still serve some clinical purpose, possibly a diagnostic one. The chronaximetric method is widely applied clinically, since chronaxie is regarded as a parameter characteristic of the speed of development of excitation. No independent significance is attributed to the rheobase, which serves only for measurement of chronaxie. In their reviews of chronaximetry, D. A. Markov [1] and Yu. M. Uflyand [6] frequently present data for chronaxie, without giving the corresponding rheobase values.

We have examined the dynamics of short- and long-term excitability of normal and pathological human muscles, as also their chronaxie. In our investigation we used a modified chronaximetric method, in which the Lapicque shunt was replaced by a 100 ohm shunt [3]. The circuit is represented in Figure 1.

We did not, as was recommended by D. N. Nasonov [2], include a resistance in series of 50-100 ohms, since this is in effect replaced by the resistance of the tissues through which the current passes to the nerve. The

thresholds of excitability of the nerves and muscles of the intact organism are higher than those of the isolated tissues, but because of the 100 ohm shunt we were able, using a current of 400-500 v, to obtain the data for a full strength-duration curve for the intact organism.

Most modern condenser chronaximeters used for determination of rheobase make use of d.c. impulses. The subject of such measurements usually experiences painful sensations (6). In our experiments, determination of the rheobase was painless, using condenser discharges of considerable duration.* It is the usual practice in chronaximetry to apply the electrode to the motor point for each measurement of excitability. We think that this procedure suffers from a number of disadvantages. A displacement of a few millimeters of the electrode from the motor point, or its firmer application, lead to quite different results of excitability measurements. Tying the electrodes down, as is sometimes practiced, is not to be recommended, since the electrode readily undergoes displacement under such circumstances [1]. The degree of moistening of the electrode is also of importance. It is not surprising that only experienced operators are able to achieve reproducible results.

For these reasons we modified the usual method, for the study of the dynamics of excitability over short periods, not exceeding a few days.

As a measuring electrode we used a chlorinated silver cup, area 1 sq. cm, and depth 4 mm. The cup was filled with cotton-wool impregnated with 2% agar made up with physiological saline. Having found the motor point, we attached the electrode by means of sticking plaster. This modification assured a constant moisture level, constant pressure of the electrode against the skin, and firm fixation to the motor point. Errors are thus largely eliminated, although displacement of the motor point may still take place if the position of the arm is changed.

We determined the strength-duration curves in each experiment, which could very conveniently be achieved with the attached electrode. If we had applied the electrode separately for each reading we should either have had to find the point of minimum excitability anew for each capacity, or to apply the electrode each time to a previously marked motor point. In the former case, we would have had to devote very much time to the preparation of the curves, while in the latter case, in addition to all the usual shortcomings inherent in this procedure, we would still be left with the possibility of errors due to shifting of the motor point.

Having once constructed the whole of the strength-duration curve, we could thereafter confine ourselves to the measurement of the constants $\underline{a}$ and $\underline{b}$. The value of the constant $\underline{a}$ was obtained in the following way: we selected the threshold potential for a sufficently short time interval (the maximum potential and the smallest capacity achievable with our equipment). Since it follows from Horweg's formula that when $n = 1$, $a = (v - b)t$, then from the threshold potential found by us we could derive the value of the rheobase and of the difference $(v - b)$, expressed in mv, and this multiplied by time, in msec., gave the value of $\underline{a}$, expressed in millivolt-milliseconds. Determination of the rheobase and of the constant $\underline{a}$ takes no more time than is needed for measurement of chronaxie. **

It is possible to derive the value of $\underline{a}$ from the formula $(v - b)t$, or from vt, only in the case when $n = 1$; in other cases, t^n has to be taken. We found that $n = 1$, or is very close to 1, for the strength-duration curves of human arm muscles. A logarithmic curve of this sort is shown in Figure 2. This also gives the values of the constants $\underline{a}$ and $\underline{b}$, and of chronaxie, and $\underline{n}$ is calculated as $\tan\alpha$. In order to derive chronaxie from the curve, a distance of log 2 is marked off on the ordinate axis, above the point corresponding with unit $\underline{b}$, and a horizontal line is drawn parallel to the abscissa axis, to the point of intersection with the curve. A perpendicular dropped from this point to the abscissa axis gives the value of log Chr.

* According to L. K. Rubin [5] it is inadmissible to determine chronaxie and rheobase using impulses of current of different forms, as it would not be possible to know whether chronaxie was determined for the same fibers as those for which rheobase was determined, or for different ones. This is a further argument supporting the view that rheobase should be determined using condenser discharges of prolonged duration.

** The value of the rheobase is not taken into account in measuring the value of $\underline{a}$ for frog nerve muscle preparations, since it is much smaller than the threshold potential for currents of short duration. In this case $\underline{a}$ is given by the product of $\underline{v}$ and $\underline{t}$ [4].

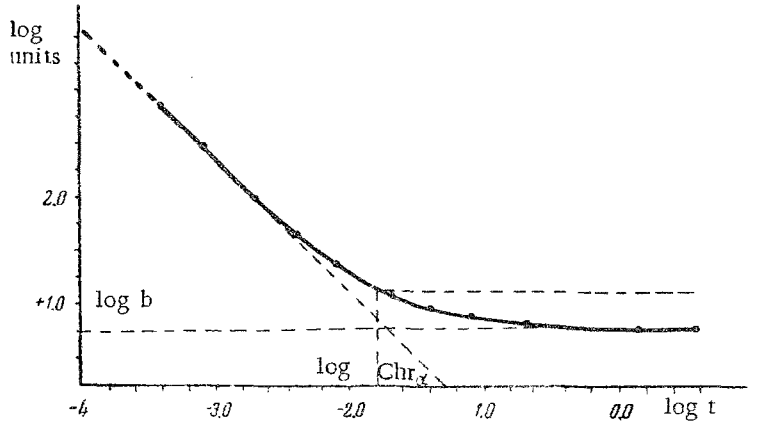

Fig. 2. Logarithmic strength-duration curve for the patient F.
n = 1; a = 118 mV in 1 millisecond; b = 6.6 v ; Chr = 0.0159 msec.

The majority of the experimental strength-duration curves found by us for human arm muscles are in fairly close agreement with the Horweg-Weiss formula. This is clearly evident in Table 1, which gives the deviations of the experimental from the calculated results. Since n = 1 for all the curves of Table 1, we could apply the formulae $v = a/t + b$ or $a = (v-b)t$. The numerical values of threshold potential for the biceps muscle of the patient F are given in Table 1 (calculated values v_1, and experimental values v_2).* For the other patients we give only the differences between the calculated and experimental values (expressed as percentages of the calculated values). In all cases, the deviations are so small that we are justified in assuming that the experimental curves for human muscles correspond with those calculated from the Horweg formula, i.e., with the theoretical curves. It should be noted that the deviations of experimental curves for frog nerve-muscle preparations from the theoretical ones are considerably greater, especially around the inflection point of the curve.

TABLE 1

Threshold Potentials for the Biceps Muscle, Found Experimentally and Calculated from the Formula $v = a/t + b$.

	Patient F., 10/10;			Patient A	Patient R	Patient V	Patient A	
							10/27	11/10
	v_1 calculated from the formula	v_2 experimental*	$\frac{v_2 - v_1}{v_1}$	$\frac{v_2 - v_1}{v_1}$	$\frac{v_2 - v_1}{v_1}$	$\frac{v_2 - v_1}{v_1}$	$\frac{v_2 - v_1}{v_1}$	$\frac{v_2 - v_1}{v_1}$
			in %					
90	—	6.6	—	—	—	—	—	—
30	—	6.6	—	—	—	—	—	—
5	7.17	7.2	+0.4	+ 6.3	— 0.3	+4.4	+ 4.3	— 3.86
2	8.0	8.2	+2.5	+ 2.28	— 1.58	+1.95	+ 2.6	+ 1.54
1	9.47	9.4	—0.074	+ 2.2	+ 1.77	+3.6	+ 6.1	+ 2.6
0.5	12.35	11.2	—0.93	+ 7.8	—13.3	+0.62	+ 7.5	— 9.7
0.2	21.0	20	—5	+ 8.1	— 6.25	0	— 8.0	— 3.1
0.1	35.3	34	—3.7	— 0.26	— 3.62	—9.8	— 2.0	— 2.7
0.05	64	62	—3.12	— 2.16	— 7.25	—2.6	— 2.2	— 8.4
0.02	150	150	0	+ 2.16	+ 7.2	0	+ 2.9	— 9.2
0.01	293.6	310	+5.6	— 0.87	— 8.2	—8.6	0	+ 9.4
0.005	—	—	—	+12.1	—	—	—12.9	—16.8

* The numerical values contained in this column were taken for the construction of the strength-duration curve of Figure 2.

In our studies of pathologically altered muscles we examined leg muscles chiefly; their excitability thresholds were higher than those of the upper extremities, probably owing to the greater resistance of the intervening skin layer. For this reason, we could not get enough points on the strength-duration curves for the accurate determination of n. We believe, however, that the slope of the strength-duration curves for leg muscles is the same as for arm muscles, i.e., that $n \simeq 1$.

A comparison of the excitability of normal and pathologically changed muscles (this is possible only on the condition that there is no great difference between the resistance of the two extremities) shows that the strength-duration curves for diseased muscles lie above those for normal ones (Figure 3). This means that both the intensity and the duration thresholds have been raised. The chronaxie changes are in most cases in the same direction

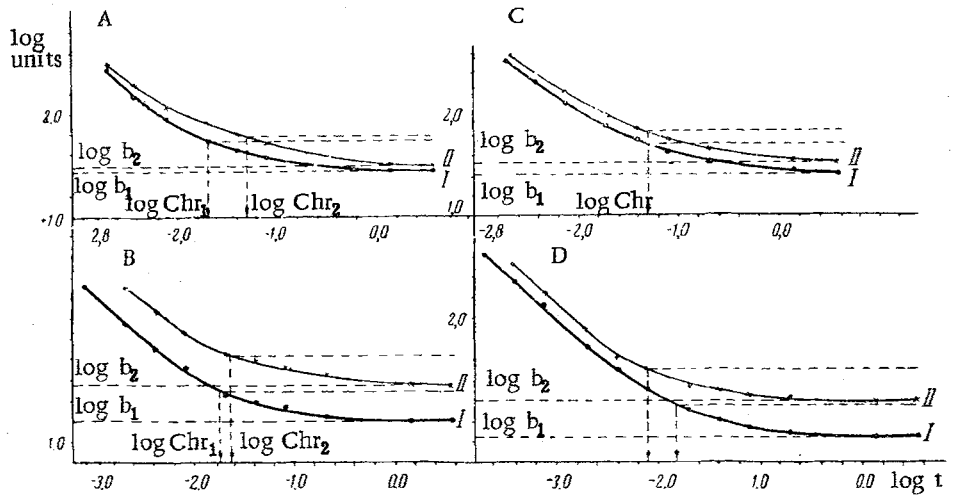

Fig. 3. Logarithmic strength-duration curves for normal and pathological muscles.
A) Patient S. Ankylosis of the right knee-joint; Rectus femoris muscle: .–.–.–healthy leg: a=503 mv in 1 msec.; b = 28v; Chr = 0.0209 msec.; x–x– diseased leg: a= 580 mv in 1 msec.; b = 30v; Chr = 0.0502 msec. B) Patient A. Sequelae of osteomyelitis: Rectus femoris muscle: .–.–.–healthy leg: a= 248 mv in 1 msec.; b = 16 v; Chr = 0.0174 msec.; x–x–diseased leg: a = 532 mv in 1 msec.; b = 36v; Chr = 0.0219 msec. C) Patient S. Ankylosis of the right knee-joint; Muscles of the semi-group: .–.–.–healthy leg: a = 592 mv in 1 sec.; b = 24 v; Chr = 0.68 msec.; x–x–diseased leg: a = 696 mv in 1 sec.; b = 24v; Chr = 0.61 msec. D) Patient G. Residual symptoms of spastic paralysis of the left arm: .–.–.–healthy arm: a = 80mv in 1 sec.; b = 7v; Chr = 0.016 msec.; x–x–diseased arm: a = 127 mv in 1 sec.; b = 16 v; Chr = 0.008 msec.

as are the changes in the strength-duration curves, i.e., the lowering of excitability of the pathologically changed muscles is associated with prolongation of chronaxie. This correlation is, however, basically a qualitative one. We very frequently encountered a large increase in chronaxie together with a small rise in excitability threshold (in Figure 3A, b is increased by 7%, and a by 14%, while chronaxie is increased by 150%).* Figure 3B illustrates another not infrequently encountered case, in which there is a considerable, though uniform rise in excitability threshold (b by 111%, and a by 112%), with only a small increase in chronaxie (by 20%). There are cases in which chronaxie gives a distorted picture of the functional state of the muscles. Two such cases are represented in Figures 3C and 3D. Figure 3C shows the strength-duration curves for the semitendinosus and semimembranosus group of muscles of the diseased leg (ankylosis of the knee joint) of the patient S.

* Here the increase in chronaxie is due to the deviation from the calculated values curve in its middle part.

There is a marked fall in excitability (with a corresponding prolongation of reaction time), but in all the points of the strength–duration curve for the diseased leg the chronaxie of the muscles is the same as for the healthy leg. From chronaxie data alone we should have come to the conclusion that the functional states of the healthy and the diseased muscles are identical.

An even more instructive example is given by Figure 3D. The upper curve, for patholgically changed muscle is considerably displaced from that for normal muscle. The duration excitability threshold is raised from 130 v for the healthy leg to 170 v for the diseased one, and the rheobase from 7 to 16 v, with considerable shortening of chronaxie, from 0.016 to 0.008 msec. The reason for this was that the rheobase had risen more than the duration excitability threshold; an inexperienced observer might have drawn the erroneous and somewhat paradoxical conclusion that the diseased muscles were in a good functional condition.

Although such cases may be seldom encountered, their possibility is a sufficient objection to using chronaxie measurements for diagnostic purposes. We have not encountered any such discrepancy between the clinical picture and the values of the constants $\underline{a}$ and $\underline{b}$. In all the cases presented by us, the curves for pathologically changed muscles showed higher duration and intensity excitability thresholds. We found no exceptions to this among the 14 patients whom we examined.

We were able, by following changes in the values of the constants $\underline{a}$, $\underline{b}$, and chronaxie, to detect postoperational changes in excitability. We shall here give one such example. Transplantation of the femoral extensor muscles (biceps and semitendinosus) was performed in the patient G. to the lower part of the femur (for treatment of postpoliomyelitis paralysis). Table 2 presents the tabulated data for duration (a) and intensity (b) excitability thresholds, and for chronaxie, for the semitendinosus muscle before operation, and 2 months after operation, when the plaster cast was removed.

TABLE 2

Values of the Constants $\underline{a}$ and $\underline{b}$, and of Chronaxie, for the Semitendinosus Muscle of the Healthy and Diseased Legs of the Patient G., Before and After Operation

Constants	Healthy leg			Diseased leg		
	before operation	after operation	percentage change, compared with the calculated pre-operational value	before operation	after operation	percentage change, compared with the calculated pre-operational value
b (v)	28	15.6	−44.2	30	17.1	−43
a (mv in 1 msec.)	541	320	−40.7	470	504	+ 7
Chronaxie (msec.)	0.033	0.32	−3	0.024	0.05	+108

We observed considerable lowering of the intensity threshold (by 43%) for the transplanted muscle, while the duration threshold remained practically unchanged (a rise of 7% is too small to be considered as significant). At the same time, chronaxie was doubled. We know from the case history that the transplanted muscles functioned well after two months, and that the patient walked better after the operation than before. The increase in chronaxie seen in this case did not reflect change in the functional condition, but was due to diminution in the term $\underline{b}$ of the fraction a/b, while $\underline{a}$ remained without change. Excitability changes were also evident in the healthy leg, in the form of a considerable fall in intensity (by 44.2%) and duration (by 40.8%) thresholds of excitability. Since the values of the constants $\underline{a}$ and $\underline{b}$ fell parallel, chronaxie was unchanged in this case. Had we confined our examination only to chronaxie measurements we would not have perceived any changes in the functional state of the muscles.

The data presented in this paper, on changes in excitability of normal and diseased muscles, can only be regarded as being of the nature of a preliminary communication. It is necessary to accumulate more factual material, and to relate it more closely to the clinical data. We can, however, on the strength of the examples quoted, state that chronaxie can give a misleading impression of the functional state of muscles, and that a much more trustworthy picture of the functional state is afforded by intensity and duration excitability threshold data.

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EXCITABILITY OF HUMAN MUSCLES DURING SLEEP AND WAKEFULNESS

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L. Lapicque [5] has developed the concept of the subordinative effect of centers, based on the observed increase in excitability and prolongation of chronaxie following transection of a nerve. It has, however, been shown by Nasonov and Rozental' [8] that these changes in excitability are due to impulses proceeding from the point of transection of the nerve. The apparent paradoxical increase in chronaxie, with heightening of excitability over the whole of the strength–duration curve, was explained as resulting from the unequal diminutions in the duration (constant b) and intensity (constant a) thresholds of excitability.

The changes in peripheral excitability observed by a number of workers to take place during sleep have been ascribed to disturbances in the subordinative influences of the centers on the periphery. G. Bourguignon and J. B. Haldane [14] have reported prolongation of chronaxie when the subjects fall asleep. This observation was confirmed by P. A. Kiselev and F. P. Maiorov [2, 3]. They examined a number of narcoleptic patients and of healthy individuals during sleep, and found that chronaxie is prolonged during sleep.

In their further researches, F. P. Maiorov and his co-workers studied chronaxie changes during sleep due to various causes, viz., during alcoholic intoxication [1], in aged individuals [11], in juveniles [4], hypnotic sleep [6, 12], and in narcoleptic states [9, 10].

Researchers have shown that increase in chronaxie takes place during sleep (to an extent depending on the depth of sleep), together with regular changes in the ratio of flexor and extensor chronaxies. These findings, which are of great importance for the study of sleep and of subordination, were based exclusively on the use of chronaximetric methods.

Our investigations of the effect of severing a nerve on its excitability [8] have led us to take up a critical attitude towards current concepts of subordination, although we do not reject the existence of central influences at the periphery. We therefore thought it worth-while to reexamine the above-cited data on the subordinative effects of the centers on the periphery during sleep, applying the new methods put forward by D. N. Nasonov and D. L. Rozental [7] for the evaluation of excitability.

EXPERIMENTAL METHODS

We examined peripheral excitability during wakefulness, during sleep during the day, both natural and reinforced by hypnotics, and during nocturnal sleep. We recorded strength–duration curves for the biceps brachii and flexor digitorum sublimis muscles, using a slightly modified condenser chronaximeter, in which the Lapicque shunt had been replaced by a 100 ohm shunt [7]. This permitted the recording of strength–duration curves of

*In Russian.