A STUDY OF MUSCLE IN CONTRACTURE: THE PERMANENT SHORTENING OF MUSCLES CAUSED BY TENOTOMY AND TETANUS TOXIN.

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INTRODUCTION AND DEFINITIONS.

The term contracture is used to designate a variety of conditions of more or less permanent muscle shortening. Some of these need only be mentioned. The delayed relaxation seen in a nerve-muscle preparation after a series of rapidly repeated contractions, the delayed relaxation caused by veratrine poisoning, the prolonged shortening caused by nicotine and acetylcholine in frog muscle and in denervated mammalian muscle, and Tigel's contracture are chiefly of interest to the physiologist. With the exception of the first-mentioned, which is generally, though perhaps mistakenly, regarded as a fatigue contracture due to the accumulation of lactic acid in the muscle, not enough is known about the physico-chemical factors involved in these phenomena to make them of any value in the solution of the problems to be considered in this paper. Nor are the structural factors any better understood. The relative parts played by the myofibrils and sarcoplasm are still matters of controversy. It is not known whether or not nicotine and acetylcholine produce contracture by stimulating a special receptive substance in the muscle associated with hypothetical parasympathetic endings, as maintained by Riesser and Neuschloss¹ and Frank, Nothmann and Hirsch-Kauffmann². Certainly this conception has received no support from the recent work of Gasser and Dale³.

As ordinarily used in clinical literature, contracture designates a condition of persistent tonic shortening of muscles which will, however, relax during sleep or under anaesthesia. To this class belong the contractures seen in some patients with lesions of the pyramidal tract. This sort of muscle-shortening is maintained by a continuous stream of nerve impulses reaching the muscles from the central nervous system and affecting one group of muscles predominantly or to the complete exclusion of their antagonists. It is, in fact, nothing but an exaggerated and continuous state of hypertonus and is characterized by the deformity which it produces, by its steady persistence during the waking hours, and by the fact that it disappears during deep sleep or anaesthesia⁴. Hysterical contracture and the reflex contractures, so often seen during the war, which bear some obscure relation to more or less trifling wounds, also have as

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their basis an excessive and continuous tonic innervation of the affected muscles. Muscle-shortenings of this sort, whatever their cause, fall in one great group and may properly be designated as hypertonic contractures.

But apparently even in contractures of pyramidal origin there may develop a certain amount of intrinsic shortening in the muscles, for according to Foerster while these contractures are due in large part to hypertonus, they are also in part due to the fact that each muscle group adapts itself to the approximating of its points of insertion by the gradual development of contraction and permanently retains this condition of shortening. If this actually occurs, the affected muscles should not fully relax under an anaesthetic and the residual shortening that persisted under deep anaesthesia would belong in the class to be described next.

Under the general heading of myostatic contractures we propose to include a group quite different from the preceding. It includes all contractures that are caused by the fixation of the muscle at a given length for a considerable period of time. Familiar examples are the contractures which restrict the movements of joints after immobilization for weeks in plaster casts, the permanent shortening of muscles after division of their tendons, and probably, in their early stages at least, the paretic contractures due to the unequal paralysis of antagonistic muscle groups in anterior poliomyelitis and multiple neuritis. This group, because they all are caused by fixation of the muscle for a long time at one unchanging length, might not improperly be designated as immobilization-contractures, but we feel justified in using the word myostatic as more accurately descriptive of the condition.

By myostatic contracture we mean to designate a condition of permanent shortening in resting muscle which is maintained in the entire absence of nerve impulses, the muscle having acquired, usually as a result of prolonged immobility, a new and shorter resting length. It is the condition which has been designated "Ruheversteifung" by Spiegel and Shiboya. These investigators and others as far back as 1886 (Moll) have clearly demonstrated that if the bony attachments of a skeletal muscle are immobilized for several days so as to prevent the changes in length which normally result from spontaneous and reflex movements, there occurs a fixation of the muscle at the length thus imposed upon it. If taken sufficiently early, this contracture can be overcome by active or passive movements, but if it is left untreated for some time the damage becomes irreparable.

It may be that adhesions and ankylosis play a part in the limitation of motion in a joint that has been enclosed for weeks in a plaster cast, but these factors are certainly secondary to the shortening of the muscles. This is clearly shown by the following experiment (Moll). After the hind leg of a rabbit had been enclosed in a plaster cast for 12 days, division of the muscles completely eliminated all of the resistance to passive movement which had developed. This showed that within this time no adhesions, thickening of the joint capsule or changes in the articular surfaces had developed and that the limitation of movement was entirely due to a shortening and fixation of the muscles.
Fröhlich and Meyer\textsuperscript{*} have shown that this fixation of an immobilized muscle is dependent upon the nerve impulses which reach it from the central nervous system. Immobilization of a denervated muscle does not cause it to become set or alter its extensibility as would be the case if the nerve supply were intact. According to these authors section of the dorsal roots supplying the muscle is sufficient to prevent the contracture, indicating that the integrity of the local reflex arc is essential for its development.

Spiegel and Shiboya\textsuperscript{6} removed the left motor cortex from cats and found that in the right hind leg the extensor muscles became set more quickly than the flexors, while flexor rigidity was more easily induced on the left side. They carried out other experiments, transecting the spinal cord in the lower thoracic region and then putting the limbs up in plaster casts in the flexed position, and found that setting occurred in the fore limb more quickly than in the hind limb. These experiments indicate that the brain centres which control tonic innervation of the muscles play an important part in the genesis of this phenomenon.

Myostatic contracture is then the fixation or setting of an immobilized muscle in a new and shorter than normal resting length. In its genesis it is dependent on the nervous system and it is probably an abnormal manifestation of tonic innervation. It is, however, only in its genesis that myostatic contracture is dependent on the nervous system. After it has once developed, it is independent of the innervation and persists under deep anaesthesia and even after section of the motor nerve. It is a contracture of muscle at rest and is in this respect to be sharply distinguished from the hypertonic contractures previously mentioned.

It is surprising that practically nothing is known concerning the chemical, physiological, or structural alterations in muscle which are responsible for the various types of contracture. Attempts at an explanation have dealt almost exclusively with the hypertonic forms and have been for the most part only reviews of the literature on the controversial questions of muscle tonus. The papers of Buscaino\textsuperscript{8}, Cooper\textsuperscript{10}, Crocq\textsuperscript{11}, Noica\textsuperscript{12}, Piéron\textsuperscript{13} and Ott\textsuperscript{14} contribute nothing of importance to the solution of the problem. Probably the most significant single observation is that attributed by Oppenheim\textsuperscript{4} to Brissaud, namely, that an anaemia of the affected extremity produced by an Esmarch bandage has a relaxing effect on the shortened muscles. It suggests that the shortened state is not maintained by structural alterations in the muscle, but by a chemophysical equilibrium that is upset by the anaemia.

We shall be concerned in this paper with the experimental myostatic contractures which can be produced by tenotomy or by tetanus toxin. After section of its tendon a muscle shortens. It is freed from the frequent stretching which normally results from the contraction of its antagonists. Under these conditions it undergoes the same changes in extensibility which occur in the muscles of a limb rigidly fixed in a plaster cast. It is what Spiegel and Shiboya have called "Ruheversteifung," as already remarked. Dixon and I\textsuperscript{15} found
that 15 days after section of the tendo achillis the gastrocnemius muscle of the white rat had not only undergone shortening but it had become set at this new length and was less extensible than normal. This was due to a change in the muscle itself, because it persisted after section of the sciatic nerve. But in its genesis this contracture was dependent on nerve impulses, because it did not occur if the sciatic nerve was cut at the same time as the tendon. These experiments may be briefly summarized as follows.

In the white rat the tendo achillis was cut in both legs. The right sciatic nerve was also cut at the same time. The left sciatic nerve was cut at the time of the experiment, which was performed 15 days after the original operation. By that time the left gastrocnemius muscle, whose nerve had been intact up to the time of the experiment, was shortened, plump and firm. On the right side where the nerve had been cut at the same time as the tendon the muscle was of normal length, but was soft and flabby. While the circulation in these muscles was still intact they were stretched in an identical manner and for the same periods of time with loads increasing by ten gram intervals up to 100 grams. This method of stretching caused a permanent elongation of 2.82 mm. in the right gastrocnemius whose nerve had been cut at the same time as the tendon. This figure represents an average of 6 experiments. But in the left gastrocnemius whose nerve was intact until just before the stretching began the same weights acting for the same periods of time caused in the 10 experiments an average permanent elongation of 1.34 mm. Ten normal gastrocnemii from other rats gave, when stretched in the same way, a permanent elongation of 3.08 mm. This permanent elongation must not be confused with elastic stretch which disappears immediately after the weight is removed. We found that the elastic stretch was not decreased in the muscles after tenotomy; but the permanent elongation or ductility, which corresponds to what Blix called "Nachdehnung," and Langelaan called "plasticity," was reduced by half.

Taking into consideration the fact that all muscles are under continuous tonic innervation it is not hard to see why a muscle should shorten after section of its tendon. But it is not at all easy to understand why it becomes set in this shortened length and has its extensibility reduced; nor do we understand why this reduction of extensibility affects only the permanent elongation and not the elasticity of the muscle. It is clear, however, that the cause is to be found in some change in the muscle fibres, because it persists after section of the motor nerve. It is equally clear that in its genesis this change in the muscle fibres is dependent on the nerve supply, because it does not occur if the nerve is divided at the same time as the tendon. Whatever may be the factors involved they are the same as those that produce the muscular rigidity which limits the use of joints after immobilization.

A very similar, if not identical, condition develops in the late stages of experimental local tetanus. When an appropriate dose of tetanus toxin is injected into one limb of a highly resistant animal like the cat or rat, the spasms
develop only in the injected leg. When this local tetanus is fully developed all the extensor muscles are in continuous tonic contraction. This is not interrupted by fatigue and persists for days without relaxation. The limb is held continuously in rigid extension; it is in extensor contracture. In the early stages of local tetanus the limb relaxes when the animal is anaesthetized, indicating that the condition is one of hypertonic contracture and is maintained by the continuous discharge of nerve impulses into the affected muscles. But in from 5 to 7 days after the injection, if the dose has been an adequate one, the muscles become set in this posture of rigid extension and fail to relax under deep anaesthesia or even after section of the motor nerve or the death of the animal (Ranson and Morris). There are then two stages of tetanus rigidity which are to be sharply distinguished: hypertonic contracture, which disappears after nerve section; and myostatic contracture which, after it has once developed, is independent of the nervous system. Although these facts have been known since 1903 when Meyer and Ransom published their studies on tetanus, no serious attempt has been made to analyse these phenomena or to ascertain their significance for neurology.

Dixon and I found that the ductility of the gastrocnemii in the myostatic contracture of local tetanus was reduced to an average value of 1-28 mm., which is a reduction of 60 per cent. from the average normal value of 3-08 mm. The tests were made in the same manner as in the experiments with tenotomy. As yet we have not been able to find a satisfactory explanation of this decrease in ductility; but experiments looking toward an elucidation of this problem are now under way.

One thing is certain. Microscopical sections of muscle in contracture following tenotomy or the injection of tetanus toxin reveal no structural change which could account for the muscle having taken on a new and shorter resting length. There is no increase in connective tissue and very little change in the muscle fibres except that they have shortened. This change in the length of the fibres will be discussed in detail in a subsequent paragraph. The slight changes in microscopical appearance of the muscle fibres, namely a decrease in the clearness of the cross striations and, in some instances, especially late in tetanus contracture, an increase in the number of nuclei, do not account for the fixation of the muscle in a shortened length.

OBJECT AND METHODS OF STUDY.

The primary object of this investigation was to determine what change, if any, occurs in the function of a muscle in myostatic contracture. Is its capacity for shortening during contraction and its speed of relaxation altered from normal? We have also studied the changes in weight and length of the muscle and in the length of the muscle fibres in tetanus contracture and in that following tenotomy. Cats and albino rats were used for the experiments.

A series of seven young adult rats were injected subcutaneously in the upper part of the right thigh with tetanus toxin. The first one died from an
overdose; but kymograph records of the contractions of the gastrocnemius muscles were obtained from the other six. The rats varied somewhat in their susceptibility to the toxin, and, since it was desired to produce in each as nearly as possible the same stage of local tetanus, the dose and time necessarily varied. Rat T-2 received -01 cc. of the toxin 5 days before the tracings were made; T-3, -01 cc. 7 days before; T-4, -01 cc. 4 days and -005 cc. 3 days before; T-5, -015 4 days before; T-6, -015 cc. 6 days before; and T-7, -01 cc. 8 days, -005 7 days and -005 2 days before the tracings were made. When a high grade local tetanus had developed in the right hind leg, the rat was anaesthetized with ether. If the affected leg relaxed the animal was allowed to recover from the anaesthetic and in some instances was given another dose of toxin as mentioned above. It was tested with ether again a day or two later. If there was very little relaxation of the injected leg under deep ether anaesthesia this was considered as evidence that the desired stage of contracture had developed and the experiment could proceed.

Before discontinuing the ether anaesthesia, 1 cc. of 5 per cent. chloral hydrate was injected intraperitoneally. The sciatic nerve was then exposed throughout its length in the thigh and was cut as close as possible to its point of exit from the pelvis. The tendo achillis was then cut close to the posterior tip of the calcaneus, after a thread had been securely fastened around the tendon. The gastrocnemius was freed of all fascial attachments, up to its two heads. The soleus was separated from the gastrocnemius and divided above its insertion into the common tendon. Damage to the vessels of the popliteal space and the branches supplying the gastrocnemius was avoided. The posterior surface of the middle third of the femur was exposed and a special clamp rigidly secured to the bone. During the dissection, the nerve and muscle were frequently moistened with Ringer's isotonic solution, at 37° C.

The animal was then placed on a support with the body resting on a sloping platform, and the prepared leg partially immersed in Ringer's solution, kept at 37° C. The femur was then rigidly held by the clamp, so that after adjustment was completed no movement was possible.

The heavy linen thread attached to the tendon was passed over a pulley and fastened to an approximately isotonic marking lever, having an initial tension of 28 grams in the first experiment and 50 grams in the other experiments. This tension was exerted by a long band of rubber and necessarily increased somewhat when the lever was raised by the contracting muscle.

This method of loading offered no serious obstacle to the shortening of the muscle and yet left the lever practically free from inertia. By means of an adjusting screw the initial tension could be removed between periods of stimulation and the muscle permitted to sink into the Ringer's solution, which was kept at 37° C., the level of the solution being so adjusted that the muscle dropped into the fluid when the tension was removed.

The stimuli were delivered through an inductorium with interruptions in the primary circuit at the rate of 50 per second. By means of a special
device run by clock-work and employing a platinum-mercury contact it was possible to close the primary circuit for a constant period of time. Nine or sometimes ten successive make and break shocks at a constant rate of 50 per second were obtained in this way from the secondary of the induction coil. A marker in the primary circuit indicated the number of stimuli and the length of stimulation on the kymograph.

The sciatic nerve was stimulated by means of platinum electrodes. For direct stimulation of the muscle one lead from the secondary was placed in the Ringer’s solution and a second electrode of very fine wire was attached to the tendon. In this way the current had to pass down the entire length of the muscle. It was necessary to increase the intensity of the current because of the increased resistance of the solution and the fine wire, but no change was made in time or rate of stimulation. For indirect stimulation the secondary coil was usually set at 9 but for direct stimulation it was moved up to 5 or 6. The stimulus used in each case gave a just maximal stimulation, producing a typical tetanic contraction of the muscle. In some instances, where faulty contact between the nerve and electrodes occurred, an incomplete fusion was present. These curves were not included in the data presented. A time interval of two minutes was given between successive periods of stimulation to obviate any variable factor caused by fatigue. A tuning fork of 100 vibrations per second furnished the time record.

The right leg, which had received the injection or on which the tenotomy had been performed, was always tested first, in order that any ill effects due to poor circulation, cooling, prolonged anaesthesia, shock, etc., which might cause a decrease in the height of contraction or a slowing of relaxation, would show chiefly in the normal or control leg. Such deviations from the normal as were detected in the right leg must therefore be attributed to tetanus or tenotomy and not to any of the factors listed above.

Two cats with local tetanus were studied. One and one-half cc. of tetanus toxin was injected into the left quadriceps of one normal cat. After seven days the animal was prepared in the same manner as the rats with the exception that instead of fixing the femur by means of a clamp, a drill was passed through the femur, just above the origins of the gastrocnemius, and by means of a frame and cross-bars the femur and tibia were held rigidly in position. The leg was not immersed in Ringer’s as in the rat series, but the muscle was prevented from drying by covering it with cotton saturated with Ringer’s solution at 37° C. between periods of stimulation. A second cat was injected with one cc. of tetanus toxin in the left quadriceps. The next day the dorsal roots from the third lumbar to the third sacral were cut on the left side. After a period of eight days following the injection, the animal was prepared and tested in a manner identical with that of the first cat.

After the animals were tested they were killed, the gastrocnemii were excised along with the lower end of the femur to which they were left attached,
and fixed in 10 per cent. formalin. They were suspended with the femur as the upper support, and were kept under tension by a weight of 25 grams attached to the tendon until fixation of the muscle was complete.

In another series of experiments the tendo achillis on the right side was severed with aseptic precautions in six rats and three cats. After the lapse of from fifteen to twenty days following the tenotomy the animals were prepared and kymograph records of the contractions of the gastrocnemii obtained. The procedure used in the experiments was identical with that used in the tetanus series. The records were analysed in the same manner.

In order to determine the height of contraction and the rate of relaxation the kymograph records were analysed as follows.

The base line was prolonged below the curve and a perpendicular AC was dropped to the base line from A, the point of termination of the contraction plateau and the beginning of relaxation (Fig. 1). The measured length of AC indicates the height of contraction as recorded on the tracing. A line BE, parallel to the base line, was drawn through the perpendicular at a point equal to three-fourths the distance AC, measured from A, cutting the relaxation curve at D. A line DI, parallel to AC, was then drawn through D. AC was prolonged to H. The time in hundredths of a second between H and I represents the period required for three-fourths of the relaxation. It would be very difficult to measure the time required for full relaxation because of the uncertainty as to the exact point at which the curve of relaxation first touches the base line.

![Figure 1](http://jnnp.bmj.com/)

Fig. 1 is a tracing of the kymograph record of a typical contraction of the normal rat's gastrocnemius (left gastrocnemius of Tendon Rat 4). It illustrates the procedure followed in analysing the records.

The relaxation time was corrected for error due to the arc of the lever, as follows. The line AF, parallel to the base line, was drawn through A to the arc FE. A perpendicular to the base line was dropped from F. Where it
intersected the line BE, a point G was obtained. The distance GE represented the distortion due to the arc, and the time required for the kymograph to travel this distance measured at H, is the error due to the arc of the lever. The true time for three-fourths relaxation was then obtained by adding or subtracting the correction from the value previously found. Where, as was usually the case, this correction was less than one two-hundredth of a second it was disregarded in the calculations.

The relaxation rate was determined by dividing the distance AB in mm. by 5, the magnification of the lever, and by the corrected time HI in hundredths of a second. In order to determine the amount of shortening in the muscle during contraction the height of contraction AC was divided by the magnification of the lever.

RESULTS.

The muscle contractions were recorded with an isotonic rather than an isometric lever. It was thought that only by allowing the muscle full opportunity to shorten would the muscle, already shortened as a result of tenotomy or the action of tetanus toxin, betray its inability to undergo the normal amount of shortening when stimulated. The strength of the muscle, that is, the tension it could develop against an isometric lever, would be quite another matter. And, since, as Fulton30 has pointed out, the phase of relaxation is a purely passive one, dependent on the viscosity of the muscle, the isotonic record, which allowed full shortening of the muscle and hence a large amount of elongation during relaxation, would be more likely to reveal by a slowing of the relaxation rate any change in viscosity of the muscle which might underlie the contracture.

![Graph](image.png)

Fig. 2 is taken from the kymograph record of the experiment on a rat with tetanus contracture in the right hind leg (Rat T-5). The secondary coil was set at 9 and the time was marked in hundredths of a second. A is the tracing made by the normal left gastrocnemius, and B that made by the right gastrocnemius which was in myostatic contracture. The height of contraction and the rate of relaxation are less on the side of the contracture.
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As is shown by Fig. 2, the height of contraction and the speed of relaxation are reduced in the muscle in tetanus contracture as compared with the uninjected muscle of the opposite side. The results of this series of experiments are summarized in Table 1. Several contractions were recorded from each muscle, analysed, and the data averaged to give the figures recorded in this Table. Analyses of individual contractions from one experiment are recorded in Table 2. The average amount of shortening of the gastrocnemius in tetanus contracture was 4.66 mm. as compared with 7.38 mm. in the control. In a series of six normal muscles the average shortening amounted to 7.52 mm. as shown in Table 3. It is not surprising that muscle in tetanus contracture failed to shorten during contraction as much as normal muscle since, as shown by Table 4, the fibres of the gastrocnemius on the side of the injection had become fixed in a shortened state and were only about one-half their normal length.

TABLE I.
Summary of data on the contractions of the gastrocnemius muscles in response to faradic stimulation of the sciatic nerve in animals with local tetanus.

<table>
<thead>
<tr>
<th>Animal Number</th>
<th>Muscle shortening in mm.</th>
<th>Time for ( \frac{1}{4} ) relaxation in 1/100 sec.</th>
<th>Rate of relaxation in mm. per 1/100 sec.</th>
<th>Muscle shortening in mm.</th>
<th>Time for ( \frac{1}{4} ) relaxation in 1/100 sec.</th>
<th>Rate of relaxation in mm. per 1/100 sec.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rat T2</td>
<td>4.73</td>
<td>11.60</td>
<td>305</td>
<td>6.50</td>
<td>12.00</td>
<td>406</td>
</tr>
<tr>
<td>Rat T3</td>
<td>2.66</td>
<td>5.00</td>
<td>399</td>
<td>7.90</td>
<td>11.00</td>
<td>538</td>
</tr>
<tr>
<td>Rat T4</td>
<td>5.45</td>
<td>10.75</td>
<td>380</td>
<td>7.40</td>
<td>11.75</td>
<td>472</td>
</tr>
<tr>
<td>Rat T5</td>
<td>4.88</td>
<td>9.00</td>
<td>407</td>
<td>7.50</td>
<td>8.66</td>
<td>649</td>
</tr>
<tr>
<td>Rat T6</td>
<td>4.42</td>
<td>14.00</td>
<td>236</td>
<td>7.53</td>
<td>8.40</td>
<td>705</td>
</tr>
<tr>
<td>Rat T7</td>
<td>5.87</td>
<td>10.14</td>
<td>433</td>
<td>7.46</td>
<td>9.33</td>
<td>599</td>
</tr>
<tr>
<td>Average</td>
<td>4.66</td>
<td>10.08</td>
<td>300</td>
<td>7.38</td>
<td>10.12</td>
<td>561</td>
</tr>
<tr>
<td>Cat T1</td>
<td>3.03</td>
<td>18.0</td>
<td>126</td>
<td>4.85</td>
<td>12.85</td>
<td>282</td>
</tr>
<tr>
<td>Cat T2</td>
<td>1.52</td>
<td>12.2</td>
<td>0.93</td>
<td>3.52</td>
<td>16.37</td>
<td>161</td>
</tr>
<tr>
<td>Average</td>
<td>2.27</td>
<td>15.1</td>
<td>109</td>
<td>4.18</td>
<td>14.61</td>
<td>221</td>
</tr>
</tbody>
</table>

There was not much change in the time required for three-fourths relaxation. This, when expressed in one-hundredths of a second, was 10.08 for the muscle in contracture, 10.12 for the control and 9.45 for the normal muscle (Tables 1 and 3). But due to the differences in height of contraction or, to put it otherwise, in the amount that the muscle had to elongate during relaxation, there was a marked difference in the rate of relaxation in spite of the
similarity of the times involved. The rate of relaxation in the injected leg was \(0.360\) mm. per one-hundredth of a second, in the control leg \(0.561\) and in the normal leg \(0.603\). The decrease in the height of contraction and in the rate of relaxation is more marked when the comparison is made with the normal than when it is made with the control muscle of the opposite side because the tetanus toxin had some effect on the control muscle.

**TABLE II.**

Analysis of kymograph records of the contractions of the gastrocnemius muscle of the right (injected) hind leg of Rat T-4 in response to indirect and direct faradic stimulation.

<table>
<thead>
<tr>
<th>Position of Secondary Coil</th>
<th>Indirect Stimulation</th>
<th>Direct Stimulation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Muscle shortening in mm.</td>
<td>Time for (\frac{1}{2}) relaxation in 1/100 sec.</td>
</tr>
<tr>
<td>9</td>
<td>5.6</td>
<td>12</td>
</tr>
<tr>
<td>9</td>
<td>5.6</td>
<td>12</td>
</tr>
<tr>
<td>9</td>
<td>5.4</td>
<td>10</td>
</tr>
<tr>
<td>9</td>
<td>5.2</td>
<td>9</td>
</tr>
</tbody>
</table>

The two cats were in a more advanced stage of tetanus. Cat T-1 had received 1\(\frac{1}{2}\) cc. of tetanus toxin into one hind leg 7 days before the experiment and Cat T-2, 1 cc. 8 days before. The average muscle shortening during contraction on the side of the injection was \(2.27\) mm. as compared with \(4.18\) mm. for the control muscles, and \(6.10\) mm. for a series of 3 normal muscles; and the average rate of relaxation was \(0.109\) mm. per one-hundredth of a second as compared with \(0.221\) for the control and \(0.254\) for the normal (Tables 1 and 3). It will be noticed that throughout both tables the rate of relaxation was considerably greater for the rat than the cat. Although the circulation in the muscle was undisturbed in every case the cat muscle seemed to suffer more from exposure than the rat muscle did.

It is clear from these experiments that a muscle which is already shortened in tetanus contracture cannot undergo as much shortening in a nerve-muscle tetanus as can a normal muscle, and that its rate of relaxation is reduced. This slowing of the rate of relaxation would, according to Fulton’s interpretation of the curve of relaxation as an expression of the viscosity of the muscle, indicate that in tetanus contracture the viscosity of the muscle is increased.

When a large dose of tetanus toxin is injected directly into the quadriceps extensor muscle of the cat there often occurs within 24 hours a setting of the muscle in a condition of only partial contraction. The knee is partly flexed, cannot be moved voluntarily and offers considerable resistance against passive flexion to a more acute angle. This and other interesting manifestations of
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TABLE III.
Analysis of kymograph records of the contractions of the gastrocnemii muscles after section of the right tendo achillis and the development of contracture.

<table>
<thead>
<tr>
<th>Animal Number</th>
<th>Muscle shortening in mm.</th>
<th>Time for $\frac{1}{2}$ relaxation in $\frac{1}{100}$ sec.</th>
<th>Rate of relaxation in mm. per $\frac{1}{100}$ sec.</th>
<th>Muscle shortening in mm.</th>
<th>Time for $\frac{1}{2}$ relaxation in $\frac{1}{100}$ sec.</th>
<th>Rate of relaxation in mm. per $\frac{1}{100}$ sec.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rat Tend. 1</td>
<td>5.56</td>
<td>7.20</td>
<td>-579</td>
<td>7.76</td>
<td>9.20</td>
<td>-632</td>
</tr>
<tr>
<td>Rat Tend. 2</td>
<td>5.20</td>
<td>6.50</td>
<td>-400</td>
<td>7.33</td>
<td>8.16</td>
<td>-673</td>
</tr>
<tr>
<td>Rat Tend. 3</td>
<td>4.70</td>
<td>6.25</td>
<td>-564</td>
<td>7.43</td>
<td>10.50</td>
<td>-530</td>
</tr>
<tr>
<td>Rat Tend. 4</td>
<td>4.47</td>
<td>5.75</td>
<td>-582</td>
<td>7.05</td>
<td>8.25</td>
<td>-640</td>
</tr>
<tr>
<td>Rat Tend. 5</td>
<td>3.27</td>
<td>5.00</td>
<td>-490</td>
<td>7.00</td>
<td>11.00</td>
<td>-476</td>
</tr>
<tr>
<td>Rat Tend. 6</td>
<td>4.40</td>
<td>6.00</td>
<td>-550</td>
<td>8.60</td>
<td>9.62</td>
<td>-670</td>
</tr>
<tr>
<td>Average ...</td>
<td>4.60</td>
<td>6.11</td>
<td>-560</td>
<td>7.52</td>
<td>9.45</td>
<td>-603</td>
</tr>
<tr>
<td>Cat Tend. 1</td>
<td>3.80</td>
<td>11.50</td>
<td>-248</td>
<td>4.95</td>
<td>16.75</td>
<td>-221</td>
</tr>
<tr>
<td>Cat Tend. 2</td>
<td>5.10</td>
<td>21.00</td>
<td>-182</td>
<td>6.57</td>
<td>28.37</td>
<td>-173</td>
</tr>
<tr>
<td>Average ...</td>
<td>5.11</td>
<td>14.45</td>
<td>-291</td>
<td>6.10</td>
<td>19.65</td>
<td>-254</td>
</tr>
</tbody>
</table>

Local tetanus will be discussed in another paper. Here we only wish to call attention to the fact that as the animal walks about there is a stiffness and apparent paresis of the affected limb. It must have been some such observations as these that led Fröhlich and Meyer* to state that in tetanus contracture central motor innervation is weakened or paralysed. The failure of the gastrocnemius affected with tetanus to shorten as much as the normal controls might be interpreted as evidence of such a paresis if it were not known that the muscle fibres were in a shortened state to begin with and could not therefore be expected to contract as much as they otherwise would.

In order to rule out the possibility that the defective contraction was due to an impairment of the nerve or nerve-endings, a comparison was made of the effect of direct and indirect stimulation in four experiments. In no case was there any evidence of involvement of the nerve or nerve-endings. The results obtained with Rat T-4 are given in Table 2. With the secondary coil at 9 the strength of the induction shocks was sufficient to produce a maximum contraction through the nerve. But on direct stimulation, because of the greater resistance in the circuit, the secondary coil had to be moved up to 5 in order to secure a maximum response. When a full response was obtained, the results of direct and indirect stimulation were similar both as
to the amount of shortening and the rate of relaxation. This demonstrates that the innervation was intact and shows that the alterations in function already noted were due to changes in the muscle fibres.

Within fifteen days after section of the tendo achillis the rat gastrocnemius goes into a contracture very similar to that caused by tetanus toxin. As shown in Table 3, the affected muscles shortened on the average 4-6 mm. during contraction as compared with 7-52 mm. for the normal leg. The time required for three-fourths relaxation was distinctly less on the side of the tenotomy, but because of the greater height of contraction of the normal side the rate of relaxation figures out to be about the same on the two sides. The difference between -560 and -603 in favour of the normal muscle is probably not significant. In the contracture following tenotomy, while the ability to undergo further shortening during contraction is reduced to the same extent that it is in tetanus contracture, the rate of relaxation is practically normal. There is therefore no evidence that in this sort of contracture there is any increase in the viscosity of the muscle. So far as one can judge from the character of the recorded contractions the muscle in contracture following tenotomy has merely taken on a new and shorter resting length.

In Table IV there are given the data relative to the length and weight of the rat’s gastrocnemius and the length of a selected, easily identifiable group of fibres in that muscle. In the first series the right gastrocnemius was in contracture due to tendon section. In the second series the right gastrocnemius was in contracture due to tetanus. The left side served as control in both series; but, because the action of the tetanus toxin is not strictly confined to the side of the injection the figures for the control leg in the tendon series are to be regarded as representing the normal; and there was some shortening of the fibres in the control gastrocnemius of the tetanus series.

The measurements were made after the muscles had been fixed for 24 hours or longer in 10 per cent. formalin under a tension of 25 grams. The length of the muscle was measured from the attachment of the medial head on the femur to the last insertion of muscle fibres into the medial side of the tendon. The tendon was cut away at this level and the heads of origin separated from the femur. The weight of the muscles when thus isolated is recorded in the Tables. The length of the fibres was determined as follows. On the anterior surface of the outer head two longitudinal septa are visible separating three groups of muscle fibres. The middle group of fibres which runs obliquely between the two septa was put on a stretch by spreading the muscle out and pinning it down on a cork plate. The length of the fibres was then determined with dividers and a millimetre scale. The measurements were in reality not of individual muscle fibres but of small bundles of fibres. But histological examination showed that in these bundles the fibres ran without interruption through the entire length of the bundle.
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Showing the effect of tenotomy and of tetanus contracture on the weight and length of the rat's gastrocnemius and on the length of the muscle fibres. Tend. designates the rats in which tenotomy was performed and T. those with tetanus. The left leg severed as control.

<table>
<thead>
<tr>
<th>Rat</th>
<th>Right Gastrocnemius, Contracture.</th>
<th>Left Gastrocnemius, Control.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Length in mm.</td>
<td>Weight in gms.</td>
</tr>
<tr>
<td>Tend. 1</td>
<td>27.5</td>
<td>1.480</td>
</tr>
<tr>
<td>Tend. 3</td>
<td>24.1</td>
<td>0.820</td>
</tr>
<tr>
<td>Tend. 4</td>
<td>25.5</td>
<td>0.831</td>
</tr>
<tr>
<td>Tend. 5</td>
<td>26.9</td>
<td>1.180</td>
</tr>
<tr>
<td>Tend. 6</td>
<td>26.0</td>
<td>1.535</td>
</tr>
<tr>
<td>Tend. 7</td>
<td>22.0</td>
<td>0.820</td>
</tr>
<tr>
<td>Average...</td>
<td>25.3</td>
<td>1.111</td>
</tr>
</tbody>
</table>

| T-2         | 24.7          | 1.075          | 4.5                  | 30.1          | 1.364          | 8.1                 |
| T-3         | 28.2          | 1.494          | 4.8                  | 31.0          | 1.494          | 9.0                 |
| T-4         | 24.2          | 1.479          | 4.2                  | 26.8          | 1.405          | 7.8                 |
| T-5         | 25.0          | 1.210          | 6.8                  | 28.5          | 1.242          | 10.1                |
| T-6         | 24.7          | 1.304          | 6.0                  | 28.6          | 1.390          | 10.0                |
| T-7         | 28.7          | 1.470          | 6.6                  | 29.5          | 1.569          | 7.8                 |
| Average...  | 25.9          | 1.338          | 5.48                 | 29.08         | 1.410          | 8.8                 |

It will be seen from Table 4 that after tendon section or the administration of tetanus toxin the length of the muscle in contracture is somewhat less than that of the control, but the difference amounts to only about 10 or 12 per cent. of the length of the normal muscle. The difference in the length of the fibres is more striking, amounting to 45 or 50 per cent. of the length of the same fibres in normal muscle. The muscle in the control leg in the tenotomy series is taken as the normal standard, since the tetanus toxin circulating in the blood stream caused a slight shortening of the fibres in the control leg of the tetanus series. The obliquity of the fibres in the gastrocnemius accounts for the much greater percentage of shortening in the fibres than in the muscle as a whole.

The muscles in tetanus contracture have a normal weight. In the six experiments the average weight of the gastrocnemius on the side of the injection amounted to 1.338 grams as against 1.410 grams on the control side and 1.364 on the normal side of the tenotomy series. These differences are much less than the differences in the individual muscles of a series and are therefore of
no significance. In tetanus contracture, therefore, the weight of the muscle is not affected either by atrophy or swelling; on the contrary, the weight remains normal.

After tendon section, however, an appreciable amount of atrophy occurs. As a result of tenotomy the weight of the right gastrocnemius was reduced to an average value of 1·111 grams as compared with the average of 1·364 grams for the corresponding muscles of the opposite side in the same animals.

Lipschütz and Audova\(^3\) studied the atrophy resulting from tenotomy in rabbits, found that it was nearly as great as that caused by nerve section, and considered that the atrophy was due to inactivity. After tenotomy as after nerve section they found the relative amount of water in the atrophied muscle distinctly greater than normal.

It is, however, questionable if inactivity forms an adequate explanation of the atrophy following tenotomy. Meyer\(^2\) has shown that if a flexed joint is put up in a plaster cast the flexor muscles atrophy but the extensor muscles hypertrophy. Froboese\(^2\), who studied these hypertrophied muscles for Meyer, found that except for the hypertrophy of the individual muscle fibres they presented a normal appearance under the microscope. These results seem to show that moderate tension causes hypertrophy and relaxation causes atrophy of muscles in an immobilized limb.

The atrophy of a muscle following its release from tension after tenotomy and the setting of the same muscle in a condition of permanent shortening are phenomena which are closely related. But neither the atrophy nor the setting has as yet been satisfactorily accounted for. Myostatic contractures form a field in which further investigative work is needed both because of the practical importance of the subject and because a solution of the problems there presented would throw light on the function of normal muscle.

**SUMMARY.**

Contractures may be separated into two groups depending on whether or not they are maintained by a continuous discharge of nerve impulses into the muscle. Hypertonic contractures, including those developing in the course of spastic paralyses and those of reflex origin, are dependent on a continuous tonic innervation of the affected muscles. On the other hand, myostatic contractures, including those which follow tenotomy or the immobilization of a limb in a plaster cast, are due to a shortened condition of the resting muscle and are entirely independent of the nervous system. Very little is known about the nature and cause of the changes in muscle which are responsible for its becoming set in a shorter than normal resting length. The work of Meyer, Fröhlich and Meyer, and Spiegel and Shiboya, serves to define the problem, but contributes little to its solution.

A muscle in myostatic contracture due to tenotomy or to the action of tetanus toxin maintains its shortened state after all nerve impulses have been cut off by the section of the motor nerve or by the death of the animal. It
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has acquired a new and shorter than normal resting length. Moreover, its extensibility has been decreased and this affects only its ductility or "Nachdehnung" while its elasticity remains normal.

Microscopic sections of such a shortened muscle reveal no structural changes which could be responsible for keeping it shortened. There is no increase in connective tissue and little or no change in the appearance of the muscle fibres. After tetanus contracture has persisted for several weeks, secondary changes, such as an increase in the number of nuclei, occur, but these appear late and are in no way responsible for the shortening.

In tetanus contracture and in that caused by tenotomy a selected group of fibres in the rat's gastrocnemius shortened 45 or 50 per cent. of their normal length; but owing to the obliquity of the fibres the muscle as a whole underwent a shortening of only 10 or 12 per cent. The weight of the muscle remained normal in tetanus contracture, but after tenotomy the average weight was reduced to 1-111 grams from an average normal of 1-364 grams.

Kymograph records show that a muscle in myostatic contracture cannot shorten as much as a normal muscle during nerve-muscle tetanus. In these experiments the average normal shortening of the rat's gastrocnemius during contraction was found to be 7-5 mm. and this was reduced to an average of 4-6 mm. for the muscles in contracture. While the capacity for shortening during nerve-muscle tetanus was decreased to the same extent in both types of experimental myostatic contracture, the rate of relaxation was unaffected by tenotomy but was considerably reduced by tetanus toxin.

The height of contraction and the rate of relaxation of a muscle in tetanus contracture are the same whether the muscle is stimulated directly or through its nerve. This shows that the defective contraction cannot be explained on the basis of an involvement of the nerve or nerve-endings.

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A Study of Muscle in Contracture: The Permanent Shortening of Muscles caused by Tenotomy and Tetanus Toxin

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