

THE TENDON-JERK AND MUSCLE-JERK IN DISEASE, AND ESPECIALLY IN POSTERIOR SCLEROSIS.¹

BY S. WEIR MITCHELL, M.D.,
MEMBER OF THE NATIONAL ACADEMY OF SCIENCES,

AND

MORRIS J. LEWIS, M.D.,
OF PHILADELPHIA.

OUR recent researches² have, as we believe, given to symptomatology an enlargement as to certain nervous maladies. The present brief paper is an effort to apply what we have learned to a single disease—posterior sclerosis of the cord. This disease was chosen because we were able to gather readily for examination a representative group of cases, and also because its natural history descriptions, having come from masters of their art, are both definite and reasonably complete.

The knowledge on which our reëxamination of muscular conditions in ataxia is founded is very recent. However unfamiliar it may now be, like much other medical knowledge it will soon become common property, if once it be recognized as useful.

It will be necessary to state sharply the facts and inferences on which our research rests, in order that a clear comprehension of the analysis of the muscular failures in ataxia may be obtained.

Nothing can be more valuable to the physician than to know clearly the relative readiness with which a centre acts, a nerve carries its mandates, or a muscle responds to various forms of excitation.

Whether a muscle acts rapidly and completely when the nerve-force, volitional or reflex, calls upon it, or is inert and slow and incomplete in moving, would probably be one of our best tests of local or general health. The analysis of the quality of muscular reaction escapes us, in a measure. It may not always do so.

It is possible, however, to test muscle and nerve states by the study of coarser agents, and of these there are but two—electricity and mechanical

¹ Read before the Association of American Physicians, June 17, 1886.

² Physiological Studies of the Knee-jerk and of the Reactions of Muscles under Mechanical and other Excitants, Philadelphia Medical News, February 13 and 20, 1886.

excitation. The first gives us information of value, but, strangely enough, is to be regarded as a ruder agent than the other, and in some ways inapplicable beyond a certain point.

The mechanical excitation of muscle began to have value when, in 1875, Westphal described the diagnostic use of what we now call knee-jerk, and what is usually known as tendon reflex.

That the knee phenomenon is a direct muscle response, and not a true reflex, is now little questioned.

In 1883 Jendrassik¹ showed that violent exertion, as lifting weights, clenching the fists, increased the response to a blow on the patellar tendon. No addition was made to this inviting discovery until we added a collection of facts, which are certainly of interest, and which seem likely to prove also valuable. We showed that the knee-jerk and like acts, from striking a stretched tendon, are the most refined means we possess of deciding as to the tone of a muscle; that wherever we can get such movements, as at the knee, ankle, elbow, and jaw, every distinct muscular exertion, even if as slight as winking, increases the jerk, if it be timed correctly.

Also every strong sensation on the skin, whether it be heat or cold, or hurt of any kind, at once gives increased responsive power to the muscle the tendon of which has been struck.

If we combine sudden exertion with violent sensations, like that caused by a rhigolene jet, or the faradic wire brush, the coincident knee-jerk equals that seen in spastic disorders.

What is familiarly true of the knee is also true of the elbow, where the biceps responds beautifully to a blow on its insertion tendon, and gives us an excellent test for the presence of ataxia in the upper limbs.

And now, as concerns the muscle itself, if we strike sharply with a percussion-hammer the belly of a muscle, the fibres shorten in response to the blow.

Coincident distant motions or sharp sensations at once reinforce the muscle act, and exaggerate the effects of the blow. This muscle act obeys the rules which apply to tendon-jerks.

Therefore, all forms of mechanical irritation of a muscle admit of these reinforcements. Such additions to muscular excitability disappear when the muscle is cut off from connection with the spinal centres.

Section of the nerves, or disease of portions of the cord, as in posterior sclerosis, ends all response to tendon jerks; whilst a blow on the muscle still evokes local motion in its tissue.

The explanation is not far to seek. A muscle moves when struck, because of its innate capacity to twitch when irritated, but it does not move when excited by a blow on its tendon, unless it has besides its own

¹ Beiträge zur Lehre von den Sehnenreflexen, von Dr. Ernst Jendrassik, Deutsches Archiv für klinische Medicin, vol. xxxiii. p. 175, 1883.

excitability a constant influx of tone-waves from spinal centres. These aids to motility are born of two sets of causes: 1st, the afferent impressions due to muscular metabolic changes, a constant source; and, 2d, the intermittent influx of like waves due to overflow on to its own spinal ganglia of the excess of nerve force arising from remote muscle acts or sensory impressions. Without the constant tone flood no tendon-jerks can occur, and hence this delicate response fails when the nervous arc is broken in its sensory or motor branch, or in their connecting centre, but increases with every coincident increase of tone.

The jerk from a blow on the muscle continues even after such ruptures, but both forms of response cease then to be reinforced by distant sensations or volitions.

A blow causes a muscle-jerk extending in both directions up and down, but not transversely beyond the area struck. In some cases of disease this reaction lessens or is lost, and this irregularly, not all over a muscle but in limited areas; as regards the stimulation of a blow, certain muscular regions respond better than others in health, the extensors better than the flexors, and naturally those parts near to the motor points of nerves. At the point struck, the muscle fibres may, in addition to their sudden contraction, rise somewhat slowly into a little hump or eminence which, unlike the instantaneous twitch, subsides slowly. This hump is best seen in thin and in feeble people, and in the muscles late in ataxia is sometimes unusually large and lasts longer than is common. As seen when the percussion-hammer strikes the thin and feeble chest muscles of the tubercular patient, it is familiar to most of us, but it is observable in some healthy muscles at all times. Dr. Buzzard long ago remarked, with his usual sagacity, on some of the direct muscular reactions of nervous disorders, but although Erb and others have also given the matter attention, there are still wanting full and systematic studies of these reactions in connection with disease.

We have spoken so often of tone reinforcements, that it seems well to give some explanatory statement of what we mean by tone.

The very existence of this addition to the excitability of muscles has been doubted, but our own experiments leave us, we think, little possibility of indecision on the subject.

All over the body we find muscles which possess a variable amount of intrinsic capacity to shorten under the influence of nerve force, electricity, and mechanical excitation, as abrupt stretching or a blow.

From the spinal centres there is a more or less steady flow of nerve influence which is the final preparation for prompt muscle response to excitations.

One source is probably in the muscular chemistries. Some afferent force thus created reaches the cord by the sensory nerves of each muscle, and the current returns through the motor track and keeps the muscle

ready to react. If we were to conceive of this as a sort of tuning of the muscle, a final preparation for ready reply to the will, it would perhaps be not remote from the truth.

There is another source of tone which needs no further demonstration. Remote muscle acts, or sensory stimulations also remote, at once increase the readiness of the muscle to react and numerically add to its power of motor response.

Clenching of the fist or chilling of the arm abruptly increases the knee-jerk.

This may be explained in various ways. We now hold tentatively that will-force does not confine itself to one channel in this case. Besides the needed stimulation of the hand centres, there is an overflow of gentler excitation which floods the whole spinal group of ganglia, and for the moment adds to the flow of toning influences which constantly prepare the muscle to move and increase its excitability. This is an abrupt addition to muscle tone, and hypothetically accounts for what we call reinforcements of knee- and muscle-jerks.

In childhood this overflow is competent to cause extensive movements when only one is willed, until, as we grow older, the constancy of habitual effort creates a growing tendency, on the part of nerve force, to take and keep certain very definite paths, and only a slight overflow reaches centres other than those involved in the volition.

Remnants of this difficulty are seen in our individual incapacity to move voluntarily certain muscles without unwilled action on the part of others.

Illustrations abound in disease, as Hughlings-Jackson and Buzzard have shown when certain centres become over-excitabile, and their related muscles move in irregular response to so much nerve force as overflows upon them from acts of will or from involuntary acts, meant to evoke only specific results. Here that which usually only throws on the centres mere tone waves, becomes, through their acquired excitability, competent to cause unwilled and irregular motion.

We are now in a position to understand that incessantly there are flowing variable amounts of nerve force upon and through the centres to the muscles. A blow on one of them reveals the amount of this addition to their power to respond by motion. It is well seen in the fact that a partly exhausted hand can act better on the dynamometer when its fellow is willed to consentaneous action.

A single violent act tunes, so to speak, every other muscle in the body through the overflow it sets in movement. The first blow a man strikes thus makes him more competent to strike the next, and a hurt of any kind adds directly to this readiness for action.

A beautiful illustration of overflow lies in the ease with which a torpid bladder empties itself during or just after the chilling shock of a cold

bath, and helps also to explain many results of cold affusion commonly accepted as reflex.

It is probable that in certain states of enfeeblement the tone waves grow less and that in this way we may account for the difficulty of active motion which these conditions exhibit. We have ourselves observed that on dull, damp, warm days the response to knee-taps was in some men distinctly lessened, and also that reinforcements did not give us the usual ample gain in motion nor give rise to exceptionally violent responses, common at other times.

We have then tendon-jerk, its increase, its loss, and its reinforcements by distant muscle and sensation acts. We have also muscle-jerk, its increase, its loss, and reinforcements, and other peculiarities. Having acquired new knowledge as to these latter facts, we wish to apply them to disease. To do this, so as best to see relations at a glance, we have made the first extensive effort known to us to represent symptoms and their force by signs. We thus express in one table with the eloquence of condensation what we venture to call the equation of each case, and also make easy the recognition of any one symptom over a set of cases. Table I. shows the varying symptoms in twenty-three cases of locomotor ataxia, expressed in signs which may easily be interpreted by referring to the explanation at the foot of Table II.; many of these signs, however, need no elucidation. Where two signs occur in the same space, and where R (right) and L (left) are not mentioned, the first refers to the right and the second to the left side; or the first refers to the hands and the second to the feet, as may be seen by the context. Table II. is taken from Table I., and is a distinct effort to class the cases by some decisive symptom. For this we select station, the relative power to stand steady, with eyes open or shut. This symptom can be made numerically accurate by standing the patient in front of a bar marked in inches, and placed on a level with the ears. The extent of lateral sway of the head may thus be easily observed; a like observation records the anterior tendency. The first is rarely over half an inch in health; the second does not usually exceed an inch, even with closed eyes. Any large increase is suspicious. The observer should be seated, in order to eliminate his own sway, and should be at a set distance from the patient. Dividing our ataxics by this test into classes we have, as a starting-point, Class 1, normal station; then Class 2, slight impairment; Class 3, great impairment; and Class 4, the paralytic stage. The division as between Classes 2 and 3 is difficult, but the check of the average duration of disease shows our classifications to have been reasonable. Now let us group the muscle phenomena in relation with the integrity and the lack of station.

We have long been looking for the tests of a preataxic stage in posterior sclerosis, which stage might be amenable to treatment; but usually the nearest approach we can make is to find pains, or stra-

TABLE II.—A PARTIAL STUDY OF TWENTY-THREE CASES OF LOCOMOTOR ATAXIA.

No. of case	CLASS I. Station nor- mal.		CLASS II. Station slightly impaired.						CLASS III. Station greatly impaired.						CLASS IV. Paralytic stage.							
	2	11	3	6	10	12	15	16	18	1	5	7	8	9	13	14	17	19	20	21	22	23
Ankle- and knee-jerks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Reinforcement	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Elbow-jerks	R-LO	N	0	0	0	0	R+L+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Reinforcement	R-LO	N	0	0	0	0	R-L+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Muscle-jerks in legs	0	N	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Reinforcement	0	N	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Muscle-jerks in arms	N	N	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Reinforcement	N	N	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Chin-jerk	P	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Associated movements	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Mouthing	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Proniption of eyes	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Hearing	N	N	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Vision (not carefully tested)	H	N	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Age of patient	42	37	57	47	46	44	60	52	38	49	44	56	52	52	44	40	47	51	51	58	29	52
Age of disease	3	4	7	6	7	9	2½	8	4	4	7	4	?	18	14	5	9	10	6	6	22	10
Average age of disease	3.5 years.	6.2 years.	7.6 years.	10.8 years.																		

EXPLANATION OF SIGNS USED IN THE TABLES.

- M = male.
- F = female.
- N = normal.
- 0 = absent.
- = slight, or diminished power or response.
- + = very slight, or greatly diminished power or response.
- ++ = marked, or increased power or response.
- ± = irregular, or much increased power or response.
- ∅ = natural order of affairs reversed.
- I = incontinence.
- R = right.
- L = left.
- A = ataxic.
- S = sluggish.
- P = present.
- H = hypermetropic.
- △ = small pupils.
- = large pupils.

bismus, and with these, loss of knee-jerk. A glance at the double row of zeros, in the lines ankle- and knee-jerks, and reinforcement, shows that all of our cases, at least, have reached this stage, and give no information of use so far as the legs are concerned. If we turn to the arm symptoms, we are in a more fertile field. We should have reflected long ago that as ataxy of the arms comes on even years after the legs have gone through their stages of failure, we might expect, with watchfulness, to trace in the arms of recent cases preataxic alterations and the fatal progress of the arm muscles in the direction of impaired tone. But even the newest of our own cases have gone too far to yield all we wish to know or hope yet to see. We note that the arms seem to advance through the same changes as the legs. The biceps-jerk (elbow-jerk) and its reinforcement are found only in Classes 1 and 2; with a much impaired station or station loss (Classes 3 and 4) they are gone. In one case in Class 2, the elbow-jerk, though absent, is seen on reinforcement. Had we a much larger number of cases we should probably observe more instances of lost elbow-jerk with reinforcement yet possible, in various degrees.

In one instance the elbow-jerk and reinforcement are in excess, and this, with some like facts, makes us query as to the possibility of their being a representation of a preataxic stage of excitability. Several cases recently studied by us present some of the earlier symptoms of posterior sclerosis, and yet show an increase of the knee-jerk with an increased reinforcement. May not this represent an irritative condition of the cord which will subsequently fade and give place to more decisive and distinctive local trouble in the posterior columns?

Bramwell¹ believes that an exaggeration of the reflexes may result from increased excitability of the gray matter. In such cases it is but too likely that a diagnosis of disease of the lateral columns would be accepted as the sole explanation.

The very interesting investigations of Norris² tend to corroborate this view of the case. He discovered an increase of the knee-jerk in eighteen out of thirty-seven cases, which also exhibited varying degrees of degeneration of the optic nerves and were, presumably, early ataxics. Later examination showed, in some of these, a diminution or absence of the knee-jerk.

In Classes 3 and 4 the elbow-jerk is lost, just as the knee-jerk was long before. The forearm muscle-jerks and their reinforcement are interesting—both normal in Class 1. As the cases advance, the muscle-jerks show irregularities, and are not lost utterly in a single case, up to the paralytic stage, while reinforcement of them is not possible in some of Class 2, and in Classes 3 and 4 is absent.

¹ Diseases of the Spinal Cord, by Byrom Bramwell, M.D., F.R.C.P. Edinburgh, 1882, p. 112.

² On "The Association of Gray Degeneration of the Optic Nerves with Abnormal Patellar-tendon Reflexes," by Wm. F. Norris, M.D., 1885. Transactions of the American Ophthalmological Society.

TABLE III.—A STUDY OF THE MECHANICAL REACTIONS OF TENDON- AND MUSCLE-JERKS IN LOCOMOTOR ATAXIA.

	1st stage.	2d stage.	3d stage.	4th stage.	5th stage.	6th stage.
Tendon-jerk	Diminished or absent.	Absent	Absent	Absent	Absent	Absent
Reinforcement	Fair	Absent	Absent	Absent	Absent	Absent
Muscle-jerk	Normal	Normal	Normal	Increased, regularly or irregularly distributed.	Diminished and irregularly distributed.	Absent
Reinforcement	Normal	Normal	Absent	Absent	Absent	Absent

NOTE.—A pronounced tendon-jerk capable of excessive reinforcement is sometimes seen in the earliest stages of locomotor ataxia. Moulding of the muscle at the point struck sometimes occurs in stages 4 and 5, and occasionally even in 3.

From these data Table III. is made up. It represents the mechanical reaction changes in the muscle- and tendon-jerks of the arms, and theoretically in those of the legs. They may be seen to alter in this order. The tendon-jerk is lessened or lost, but can still be reinforced in the first stage. In all successive stages both are absent. Meanwhile the muscle-jerk, say of the extensors of the hand, is healthy in stages 1 and 2, and reinforcible in both. In stage 3 the muscle-jerk is normal, but there is no reinforcement. In stage 4, with station much impaired, the muscle-jerk becomes increased. It is quicker and larger, as Buzzard observed in 1878, and Erb also, but now it is irregularly distributed, and may be less in places, and cannot be reinforced. In stage 5 the muscle-jerk is lessened irregularly, and no reinforcement is possible. In stage 6, that of complete paralysis, rarely attained because death is apt to intervene, all the reactions, tendinous and muscular, and all reinforcements vanish. Of the chin-jerk, little can be said. It is sometimes absent in health, or is hard to get, and seems more common in stages 1 and 2 than in 3 and 4.

The various changes here mentioned probably represent incompletely, the totality of alterations of muscles in posterior sclerosis. The gradual cutting off of spinal tone waves is shown in the successive loss of tendon-jerk and its reinforcement, and of muscle reinforcement. The increase of direct muscle-jerk which ensues may be due to some irritative changes in the muscle, not as yet to be fully understood. It is followed by lessening of muscle-jerk, and at last by paralytic loss of all muscle reactions. Some microscopic studies of muscle tissues are still needed to explain these late phenomena.

While studying the effects of voluntary motion on the tendon- and muscle-jerks it was noticed that associated movements occurred in many of the cases. Thus, on clenching the fist, movements occurred in the other hand, or even in one or other of the legs. This we discovered too

late to note it in all our cases. It apparently belongs to an advanced condition of the disease. To observe it, the hand should rest in passive supination on the thigh, while the opposite fist is being clenched; the patient's attention should not be called to the possible result of the experiment.

In Class 4 it was seen in six out of seven cases, and not looked for in one. In Class 3 it was present in three out of the six cases, absent in one, and not looked for in two.

In Classes 1 and 2 it was seen twice, absent five times, not sought for in two patients—a percentage of 64.75, exclusive of five cases where the symptom was not looked for. Muscle-moulding was found irregularly, without positive relation to the state of the disease.

A new symptom, distinct prominence of the eyes, with a full appearance of the surrounding tissues not due to œdema, was found six times, chiefly in late cases. Hearing was lessened in 44.4 per cent. of the cases in Classes 1 and 2; in 66.6 per cent. in Classes 2 and 3; and was normal in but one of Class 4. Vision was not tested with care.