Tremor

Robert H. Townley

University of Nebraska Medical Center

Follow this and additional works at: https://digitalcommons.unmc.edu/mdtheses

Part of the Medical Education Commons

Recommended Citation

Townley, Robert H., "Tremor" (1940). MD Theses. 834.
https://digitalcommons.unmc.edu/mdtheses/834
TREMOR

by

Robert H. Townley

Senior Thesis
Presented to the College of Medicine
University of Nebraska
Omaha, Nebraska
1940
Introduction

The phenomenon known as tremor has been recognized since the time of Galen. It has been associated with various recognized pathological conditions, and regarded as an integral unit of many clinical pictures. That so common a physiological manifestation should have stimulated so little research is indeed lamentable.

This paper which I present in an attempt to bring together the widely scattered "intellectual gems", as related to the subject TREMOR, offers few opportunities to form definite conclusions, but it does center ones attention on several observations, which might well serve as a nucleus from which the tendrils of understanding might spring.
Tremor may be regarded as an oscillating tonus, created by groups of impulses sent from the nerve centers which serve as a background, broken by intervals of a lesser or a greater duration, and by groups of impulses which crowd in and produce short tetanic convulsions.

The course of a tremor curve may be estimated by its basic components:
1. The rhythm and amplitude of the alpha waves.
2. The rhythm and amplitude of the beta waves, plus various superimpositions.
3. Correlation between the rhythm and degree of regularity of oscillations.
4. Character of sustaining a general curve level, and falls there from. (1)

These components are apparently, the final mechanical expression of the central rhythm, constituting both tonic and tetanic innervation and altering in accordance with the current state, condition, and activity of the motor apparatus. (1)

Dr. Leon Binet has advanced the following Laws of Tremor:
1. Tremor calls forth tremor, i.e. record normal tremor; have the patient tremor voluntarily; command him to stop, and you will observe that the
tremor which persists is similar to the voluntary
tremor, and not at all like the original normal trem-
or.
2. Tremor increases as the result of muscular con-
traction, and becomes exaggerated under the influence
of work. This occurs first in that part of the body
that has been worked (law of unilaterality), then
in the opposite member (law of symmetry), and finally
becomes general (law of generalization).
3. Tremor increases in consequence of emotion.
4. Tremor may be modified by respiration. i.e. is
increased with inspiration.
5. Tremor is exaggerated under the influence of
cold.
6. Tremor is modified by peripheral impressions,
pain etc. (2)

Types of tremor:
Simple tremor, If produced by a single muscle group
and its opponents.
Compound tremor, If several muscle groups enter into
the production.
Intentional tremor, If appears only during voluntary
movements.
Static tremor, If present only while limb is at rest.
Spastic tremor, If tonus of muscle involved is pathologically increased.

Fibrillary tremor, That which is due to the alternate contractions of the different fibrils of muscle.

Physiologic tremor, Those seen in normal individuals, and manifest under conditions of excitement, during chills, following violent physical exertion and sexual overstimulation, and during convalescence from severe diseases.

Habitual tremors, Those without known cause and which last for years (also idiopathic t.).

Toxic tremor, Arising during acute infectious disease and following the use of various drugs.

Coarse tremor, One in which the excursion is large jerky and comparatively slow.

Fine tremor, Is the reverse of the above.

A cortical tremor has the characteristics of an action tremor.

A subcortical tremor has the characteristics of a rest tremor.

A spinal tremor is clonic due to its lack of central inhibition. (8)
Most of the information which we now possess is the result of simple observation of the extremities. As the study of tremor became more scientific, and more detailed and permanent records became desirable, devices such as, the kymograph, levers, tambours, and finally the string galvanometer were utilized. Although these methods were productive of records which were exceedingly helpful in the study of tremor, it was felt that the inertia encountered rendered the results inconclusive. The utilization of a beam of light offered the only practical means of circumventing this mechanical obstacle. An apparatus consisting of: a source of light, a lens, a timer, and a camera equipped with a motor driven continuous strip of sensitive paper was set up in such a manner that the oscillatory shadows of the patient's finger could be recorded. In order that the record be more sharp and distinct; the hand is supported either at the wrist, or at the metacarpo-phalangeal joint, and an ordinary sewing thimble perforated by a fine needle is slipped over the patient's finger. The shadow of the needle then makes the record.

The results obtained with this apparatus have
shown that there exists a normal tendency to tremor. (4)

This tendency is manifested by at least two tremor frequencies, i.e. the alpha, and the beta. The former have a frequency of nine to eleven per second, and a potential of the order of fifty to 150 microvolts. The latter are characterized by a frequency which is most often reported to be from twenty to forty, and a potential of 25 to fifty microvolts. Travis with his supersensitive Westinghouse oscillograph as recorded these secondary or beta waves at a frequency as great as 600 per second. He has also shown that each large tremor (alpha) endures approximately .1 sec. and is composed of groups of several oscillations occurring at a rate of about 200 per second. (5) (6)

Sollenberger disputes the above mentioned high beta wave frequency. (7)

On the basis of the above one would be reasonably justified in regarding the alpha waves as a manifestation of the phenomenon known as "summation". The beta waves then, might well be the contractions of the individual fibers, or bundles which are maintaining tone. (9)

In as much as tremor investigation has recent-
ly led to a study of brain potentials a brief discussion of the more important electrical phenomenon, as observed in the nervous system will be presented.

About 1514 Defolius said "meanwhile, we will not too anxiously discuss whether spirits are carried along certain hollow channels of nerves as the vital spirits are carried by arteries, or whether they pass through the solid material as light passes through the air".

It is of some academic interest to note that the outstanding contributions to this subject came at about 100 year intervals from the above mentioned date. Thus we see that about 1600, a muscle and its nerve supply were removed from the body, and that the muscle could be made to contract by stimulating the nerve. Proving that spirits did not pass through the nerve and cause the muscle to swell; about 100 years later Galvani stimulated a nerve electrically; and nearly 100 years later Helmholtz measured the speed of nerve impulses. He found that it varied from two to 180 miles per hour, depending on the kind of nerve stimulated. Keith Lucas and Adrian found that a change in potential accompanied nerve impulses, and that as the stimulus was increased
their height stayed uniform, but their frequency increased.

There are in the brain about 9,000,000,000 nerve cells. There are then under one sq. mm. of cortex about 20,000 neuron units. This multiplicity of functional units as would be expected makes the exact interpretation of brain potentials very difficult. The pattern of potentials in the various parts of the brain has been found to vary in resting individuals. It seems that each part of the brain has a fundamental rhythm which is subject to variation under stimulation, depression and disease. We do not know just how, where, or why, these discharges arise, or what becomes of the processes that generate them.

Mercer has advanced the following working theory which is based upon consideration of those processes which generate, store, and liberate force in nature. While it is not supported by inductive evidence in sufficient quantities to amount to a demonstration, it is of value, because it does not conflict with our theories of nerve and muscle physiology. Furthermore it harmonizes with known and related facts in the organic and inorganic world.

One sees that nerve tissue is undergoing a
constant anabolic process, and that only occasionally does it liberate energy. Here then, we study a mode of action of force which is constantly accumulating and only occasionally discharging. One can but wonder; what causes the discharge? What causes it to stop after it has started? We know that force like matter is persistent; that is, it continues in the same way with the same intensity, until altered by some other force; so that the discharge of a nerve cell, once begun unless influenced from without, can never stop until the energy of the cell has entirely exhausted itself. As a matter of fact healthy cells always stop short of this, and as a rule immediately after stimulatory cessation.

The doctrine of persistence of force, completely proven to hold throughout both the organic and inorganic worlds absolutely requires an active force as the cause of this cessation. Mercer has postulated a force which he has termed inhibition; and it is his theory that "this force is constantly exerted by each nerve center on the center just before it in importance". This coincides very nicely with our present idea concerning the corpus striatum, for it is believed that the putamen exerts an inhibitory action on the pallidus.

All motion and all progress, moral, mental and
physical is not uniform, but rhythmic, consisting of oscillations on either side of a mean point. This of course also holds true for nerve tissue, for here we see two forces, the inhibitory one being a constant and the cell force being one of a gradually increasing intensity. (10)

Based on the above, is the following explanation for the phenomenon which we recognize as tremor.

The condition of equilibrium within the cell becomes more and more unstable, and finally the inhibiting force is overcome. A discharge results. It is rhythmical, and consists of a succession of puffs, or outbursts, which in the case of a motor ganglion cell, gives rise to successive contractions of the corresponding muscle. (This is the case in health) If for any reason storage occurs more slowly, or inhibition is stronger we see at once from analogies in nature, that the separate discharges will be more powerful and will take place at longer intervals.

The epileptic convulsion is the result of discharges so wide spread and intense, that it furnishes possibly the best instance in which to study the opposing forces which are readjusted in every muscular act. The liberation at the beginning of
every convolution is enormous, and as the inhibitory force is at this time little, or nothing, the result is a series of discharges which follow each other so rapidly as to be practically merged, and the result is a tetanic contraction of all muscles. As inhibition increases the discharges follow each other more and more slowly, until they are no longer completely merged, and a slight tremor begins to manifest. This tremor becomes coarser as inhibition begins to preponderate, until, finally after a few tremendous contractions of all of the muscles together, inhibition predominates and the attack ceases. This, I believe illustrates Mercer's theory, and brings the phenomena of convulsions into harmony with that which has by physiological methods, been proven in regard to the contraction of muscle in health. This view of inhibition also explains the tremor of fatigue, alcoholism, poisoning by tea and tobacco, in fact of all conditions in which the storage of force is diminished. (10)

The American J. of Med. Sci. 1887 contains the first article ever to be presented on hereditary tremor. I will briefly review that article. (11) The affection consists of a fine tremor constant-
ly present in typical cases during waking hours, voluntarily controlled for a brief time, and affecting nearly all of the voluntary muscles. It is chronic beginning in early life, not progressive, not shortening life, and not accompanied with paralysis or any other disturbance of nervous function. It resembles to some extent the tremor of paralysis agitans, still more a simple neurasthenic tremor. A most striking clinical feature is its marked hereditary family type, and its transmission along with other nervous diseases.

The following is the summary of "a most complete history":

A. Grandfather very intemperate, but never insane.
B. Father insane, but not intemperate. He had nine children. They all had the tremor.
C. One son insane from early life.
D. One a lover of strong drink, but not a confirmed inebriate.
E. Two were peculiar and would be called now-a-days "cranky".
F. Two died without marrying.

All grew to adult life and most of them lived to an advanced old age.

Several showed intelligence and mental activity
much above the average.

Seven of these nine produced thirty four children, and all of them that lived showed the family tremor.

In none of these cases did the tremor increase with age. It is a curious coincidence that the family early embraced spiritulism, and that that passed through the different generations along with the tremor.

Pintus G. Sulla has studied the transmission of essential tremor in 200 members of the same family. In some of the members he found the tremor to be coarse, and in others to be fine. He feels that trembling is transmitted, as are diseases of predominantly monogenic type, by crossing between heterozygous and normal individuals, and that neither homology nor homocrony is respected. He states that dominance repeats itself after Mendelian principals. (12)

We see a form of tremor occurring during chill and fever, which is due to an upsetting of the splanchnic peripheral balance. This tremor overshadows all associated phenomena. It cannot be inhibited by will, is independent of central inn-
ervation, and even persists after the motor nerves have been severed. (13)

The tremor is characterized clinically by the fact that it is practically inexhaustible in duration and degree. Even the cachectic and exhausted patient will react in his twentieth chill with the same intensity and vigor, and will have a severe tremor hour after hour as at the onset of the infection. True, the chill and rigor are associated with a feeling of fatigue which, however, begins at the onset of the tremor. This occurs despite the actual feeling of lassitude which persists after the chill has passed.

The tremor affected by intense cold on the untrained skin, is similar to the above in that it too is involuntary, and is not associated with effects of objective fatigue. It differs, however in that it is rapidly overcome when voluntary motor impulses supervene. This would indicate that these two forms of tremor are similar in character, but differ in intensity.

If during a chill a patient is asked to execute some voluntary movement, he will usually respond that it is impossible for him to do so; if he does carry out the muscular act, one can observe that in
spite of an intact voluntary nervous system, the vegetative responses have been totally suppressed. This is shown by the following facts:

1. There is a total absence of increased volume.
2. There is no increase in temperature.
3. There is no increased circulatory response.
4. There is no secretion of sweat.

These normal vegetative reactions which occur simultaneously with the onset of voluntary nervous stimuli are probably truly autonomous, originating locally as reflexes. They are not wholly dependent on the connections with the higher vegetative centers, for even after the sympathetic connections have been severed, they remain intact.

If one observes a previously healthy adult who is suddenly seized with a chill, one is struck by the prompt loss of muscular strength. This is the crucial difference between the feeling of loss of power which is immediately incident to the onset of a chill and which persists during its duration, and the fatigue which becomes apparent after long continued normal muscular effort. The muscle in rigor becomes functionally disabled along with the feeling of fatigue. This state is immediately initiated with the tremor. Despite the feeling of
fatigue and actual diminuation in power, the tremor persists for hours without diminuation, as against the normally working muscle, which is seen to fatigue to a degree commensurate with the degree and duration of the work preformed.

The conclusion is reached that the decrease of muscle power during chill does not depend on fatigue as such, but on an inhibition of the normal vegetative correlation; that is, on the ability of the vascular bed to dilate. This inhibition is due to impulses which persist during the period of the chill.

The tremor is then, presumably a clonic effect in muscle with increased tonus, and may be associated with local effects of metabolic products accumulated when vascular constriction takes place, rather than a phenomenon due to the existing anoxemia. The tremor is not associated with increased temperature and is to be regarded as a phenomena analogous to that studied in smooth muscle during so called "muscle blockade".

That tremor can be entirely on a functional basis is shown by the following case which was presented by James J. Walsh M. D. (14)
A cook age 58 had felt for years that a tremor was developing in her arms. This was noticed at first only when she sat down to a meal. Its intensity gradually increased, until she found that she could not serve liquids to her mistress. (It is interesting to note that the Pt. lived in constant dread of being fired for spilling something on her lady, and that she always slopped things in her lady's direction — never away from her.)

Following many medical consultations, and much useless therapy the patient visited Dr. Walsh. He was able to completely eradicate tremor. His treatment consisted of Psychotherapy, and various exercises, i.e. He had the Pt. go through the motions of serving, eating etc. with objects such as paper cups (empty), pencils and metal pitchers.

He reports a very similar case observed in a banker. This patient could not sign his name in the presence of others. This disadvantage was circumvented by the usual encouraging advice, and by changing the patient's manner of holding his pen.

Realizing that inexperience immediately removes all possibility of theorization on my part, and being cognizant of the utter futility of any such
attempt. I feel that the following observations and Neophytic reactions to these observations will not be regarded as unwarranted presumptuousness.

As have most people who see at least a part of what they look at, I have noted a tremor, functional in nature, wide spread in occurrence, and one which is practically ignored by the literature. Surely its frequent occurrence and psychological sequelae, merit more than a passing remark, or the disdainful admission that it does exist.

We note the tremor that mars the otherwise unruffled calm of the occasional toastmaster; the trembling hand of the student who is undergoing the tortures of the examining chamber; the vibrating knees of his more unfortunate brother who has been asked to come before the class to recite, but what of the mechanism involved? Must we accept the simple and meaningless explanation that they are brought about by an anxiety state, or can we arrive at a more scientific answer?

It seems logical to me that the explanation of this perplexing problem might be along one of the following lines:

We know that metabolite accumulation will cause a muscle to tremble. We also know that an unexplained
localized vaso constriction which manifests itself as "spot-blanching" can occur. Might it not be possible that the cortical activity which we recognize as apprehension could bring about a similar vascular change within some muscle or muscle group, and thus result in tremor?

It has been shown by several of those individuals who have the happy faculty of being keen observers, that they can by some slight volitional variation in muscular tension initiate a tremor which may be either localized or involve an entire appendage. This would lead one to believe that tremor might be due to some reflex activity.

That the endocrines may be related to tremor is suggested by the action of thyroxin and adrenaline. Perhaps further study along these lines will show that functional tremors are due to some transitory endocrine imbalance, or more probably to some yet undiscovered hormone.

Simultaneous records of finger tremor, muscle action potentials, and cortical potentials have shown that the frequency of normal tremor movements correspond closely with the frequency of potential rhythms previously discussed. During sleep the
rhythm frequency decreases to around five per second with bursts of 13 to 14 per second.

Sensory stimulation may decrease the tremor as it does the cortical potential, or it may cause a disassociation between cortical potential and tremor rhythms, and be manifested by an increase in tremor amplitude and decreased cortical potential.

It would seem that there is an interrelationship existing between the cortical and subcortical centers which allows a grouping of impulses reaching the final motor pathway, and that sensory stimulation may disrupt this synergy. Also that subcortical centers may dominate the tremor movements, i.e. disassociated rhythm.

Likewise, under periods of general excitement one may see the cortex dominating the picture.

Experimentation on advanced bilateral paralysis agitans patients has shown the normal functional integration between cortical and subcortical motor centers to be interrupted with only a mild, or unilateral involvement of the stria pallidial motor system. More extensive bilateral involvement may produce subcortical activity which may take over control of cortical rhythms. (15)

Stanley Cobb refutes the above. (16)
Lucy and Case report the case of a man age 33 years, who after severe cerebral injury had a mild right hemoparesis, a slight expressive aphasia with considerable slurring of speech, and a coarse unilateral tremor present both at rest and in association with voluntary muscular activity. This latter was marked in the right upper extremity and slight in the right lower extremity.

Scopolamine hydrobromide, pheno-barbital and bulbo capnine failed to influence the tremor. Soporific doses of other barbiturates abolished the tremor only during sleep. The patient was operated, and the arm area of the precentral region (areas 4 and 6 of Brodmann) was decorticated. After the operation the following observations were made:

1. There developed a complete right hemiplegia. The paralysis of the face and lower extremity soon largely disappeared. The paralysis of the upper extremity improved, but the movements remained slow and awkward and discrete fine movements of the hand and fingers did not return.

2. A complete motor aphasia appeared and began to subside on the eleventh day after operation, and speech soon returned to its preoperative level.
3. The tremor both at rest and on voluntary movements completely disappeared. (This has been adequately documented by electromyographic studies, by studies of tremor with a photoelectric cell, and by motion pictures.)

4. The surface temperature of the right arm was lower than its preoperative level by as much as from .4 to 2.7 °C for 24 hours following the operation.

From a study of these observations it is concluded:

1. An essential part of the mechanism of tremor both at rest and on voluntary movement, lies in the precentral region, i.e. areas 4 and 6 of Brodmann. Whether tremor is mediated by the parapyramidal fiber systems alone or by the parapyramidal and pyramidal systems together cannot be stated. Certainly, experimental data has demonstrated that the pyramidal system alone is not responsible.

2. The remaining precentral region, i.e. the leg and face areas of the same hemisphere is capable of integrating considerable crude and rather awkward movement in the upper extremity after the precentral arm area has been removed. Fine well coordinated movements however, are abolished.

3. The precentral region exercises a certain degree of control over the vasomotor mechanism,
since removal of the arm area of areas 4 and 6
result in a temporary lowering of the surface temp-
erature of the contralateral upper extremity. (17)

The cerebellum is the chief coordinating cen-
ter for muscular movements, and it receives its
afferent impulses from the muscles and peripheral
structures via the posterior collums, and poster-
ior nuclei, and partly via the direct cerebellar
and ascending antero-lateral tracts. (18)

The direct cerebellar reaches the cerebellum
by the restiform body, and the smaller tract through
the superior peduncle. The cerebellum also has
crossed connections through the middle peduncles
with the opposite frontal and motor cortex, while
its efferent paths reach to the spinal cord through
the opposite red nucleus, and through Deiters nucleus
on the same side. The cerebellum has therefore,
indirect control on the musculature by its crossed
connections to the opposite frontal and motor cortex,
and the rubro spinal tract, and also direct efferent
paths to the anterior horn cells via Deiters nucleus
and the vestibulo-spinal tract of the same side.

J. F. Fulton in attempting to work out the re-
lation of the cerebrum to the cerebellum, has summar-
ized his experiments on cats as follows:

1. In cat, following complete decerebellation, tremor does not appear until the third or fourth day (muscles of the neck), and is not fully developed until eight or ten days after operation.

2. Involuntary opisthotonic spasms are unassociated with tremor.

3. No tremor is seen until the animal attempts voluntary movement.

4. When the cerebral hemisphere is removed, the extremities on the opposite side pass for several days into a state akin to decerebrate rigidity. Well marked associated movements ultimately appear in the hemiplegic extremities, but no tremor is seen.

5. When both cerebral hemispheres are removed thus producing a thalamic preparation with out a cerebellum, vigorous locomotor movements occur quite unassociated with tremor.

Conclusions:

The cerebellum exerts a controlling influence over the reflexes in chronic decorticate cats.

The cerebral hemisphere is an integral part of the nervous mechanism responsible for genesis of cerebellar tremor. (19)
There exists a chronic progressive form of cerebellar tremor, the most striking characteristic of which is a generalized volitional tremor. It begins locally and gradually progresses.

In its advanced stage the disorder of motility is comparable in severity and violence with that of Huntington's Chorea, or the generalized athetosis. There is however, this difference, that in a position of rest and muscular relaxation the tremor movement ceases.

An analysis of the motor disorder shows a marked disturbance of the ability to properly control and regulate coordinated movements. This is shown by the presence of hypermetria, dysmetria, adiadokokinesis, hypotonia and intermittent asthnia.

All of these symptoms including the volitional tremor, coincide with the classical symptomatology which results from a loss of the cerebellar control over voluntary movements. This disorder is therefore regarded as of cerebellar origin.

The local onset, gradual progression, and chronic course indicate a progressive degeneration of certain special structures of cerebellar mechanism presiding over the control and regulation of muscle movements.
For this chronic progressive disorder of cerebellar mechanism the name dyssynergia cerebellaris progressiva has been suggested.

The accompanying pathological lesions were an atrophy of the motor cells of the corpus dentatum and superior cerebellar peduncles. There was no atrophy of other cerebellar systems and none of the nucleus ruber. The lesions of the cerebellar mechanism were, therefore confined to the short and important inter nuncial common pathway which conveys efferent impulses from the cerebellum to the spinal cord. (20)

The author in further observations on this subject came to recognize a combined form of tremor. This tremor combines the one just previously described with a new set of characteristics, i.e., there is a spontaneous rhythmical tremor characteristic of striatal disorder, and in association with the intention tremor of cerebellar disease. This clinical differentiation was confirmed by autopsy findings. (21)

The destruction of mid line structures of the cerebellum, or those belonging to the archi and palio cerebellum have been found to produce no tremor. In these instances however, the dentate
and pontine nuclei, and the greater part of the neo-
cerebellum must be left intact. (22)

It has been pointed out by Holmes that with
gunshot wounds of the hemispheres tremor as such is
less conspicuous than in the cerebellar atrophies
in which case the deep nuclei are more extensively
involved. This would indicate that the synergizing
activities or centers then must be in the deeper
cerebellar structures.

The first of the basal ganglion syndromes to
have clinical recognition was the shaking palsy originally
described by James Parkinson in 1817. It may
occur in association with vascular changes of old
age, or in younger individuals it may follow enceph-
alitis. The tremor, unlike that of cerebellar disease,
occurs during rest and is usually inhibited by vol-
tional movement; it also ceases during sleep. The
characteristic tremor in the fingers suggests the
movements used in rolling a pill. It is usually
controlled by effort, and is made worse by emotion.

Recent study of the Pathology of Paralysis
agitans indicate that it is due to changes in the
corpus striatum. The essential changes envolve the
large cells which are particularly common to the
globulus pallidus of the lenticular nucleus. (23)

In 1918 and 1919 Woolsey noted a postmeningetic tremor which he has since been able to define.

By the experimental production of various lesions in, and by stimulating various parts of the vestibular apparatus he has made observations which enable him to set forth the following conclusions:

Destruction of the ampullae of both horizontal canals, or of the left anterior vertical and right posterior vertical canal, or the right anterior vertical and left posterior vertical canal, or of all the vertical canals produces first an eye nystagmus, second a head nystagmus, and third a fine head tremor (which he has called a vestibular tremor). (24)
BIBLIOGRAPHY


(2) Laws of tremor (Dr. Leon Binet) Lancet 198:265; 1920.

(3) The American Medical Dictionary (Dorland).


(5) Proceedings of the Staff Meetings of the Mayo Clinic 12: 837; 1939.


(8) Characteristics of certain forms of tremor; (J. Belloni) J. Exper. Psychol. 16:644-656; Oct. 1933

(9) Proceedings of the Staff Meetings of the Mayo Clinic 12:837.


(13) Splanchno peritperal balance during chill and fever (W.F. Peterson and E.F. Muller); Arch. Int. Med.; 40:575-593; Nov. 1927.

(14) Functional tremor (J.J. Talsh); Internat. Clin. 1:124; Mch. 1933.

Physiology: 1:87-100; Mch. 1938.

(16) Brain potentials (Stanley Cobb) J. of Neuro Physiol. 2:241; Jan. 1939.


(18) Tremor ataxy and spasm (W. Harris) Lancet 2:1145-1149; Nov. 24, 1934.


(21) A combined form of tremor (J.R. Hunt) Arch. of Neu. and Psy. Vol. 8: 1922.

(22) Cerebellar tremor (J.F. Fulton) Physiology of the Nervous System, pages 517,531,533,536.

(23) Resting Tremor (J.F. Fulton) " " .