I hereby certify that the accompanying thesis is entirely my own work.

C. S. Huxley

April 24, 1882.
Thesis

on

Cerebral Motor Discharge and Myotatic Irritability.
Cerebral Motor Discharge + Neurasthenic Irritability

The first public observations on the effect of convulsions on motor reflexes are contained in a paper by Hughlings Jackson in the Medical Journal & Gazette for February 12th, 1861. In this paper the author details the case of a patient who, suffering from syphilitic disease of the cerebral cortex, had his fourth digit under observation. The lesion began in the left foot, was limited to the left leg, arm, was accompanied by unconsciousness of the left affected limb, particularly the hand. The paralysis lasted for some hours during that time the left knee jerk was slow, there was left ankle clonus. Hughlings Jackson suggested exhaustion of the lateral columns of the spinal cord as an explanation of the change in myasthenic irritability. Stokes (Selden, p. 105) gives the following generalization as the result of a systematic investigation of this symptom after epileptic seizures: after slight attacks there may be no change in the myasthenic contraction; after fits of greater severity there may be foot clonus, a mere weak knee jerk; while after very severe fits there may be a very short time loss of knee jerk followed by a stage of recovery. The transient loss of knee-jerk is due to temporary exhaustion of the grey matter of the spinal cord.

The following are, with one exception, probably all cases of cerebral illness with convulsions: at any rate, they all agree in this, that the convulsions were followed by temporary hemiplegia. In the few cases of epilepsy proper I have had the opportunity of examining after a fit the knees I have been normal there has been no ankle clonus.
Kate King, Oct. 29, married, was admitted February 19th, 1881.

Patient has been epileptic since childhood; the fits, which have been chiefly left-sided, occurring on an average once a year. They have never been followed by paralysis. On Feb. 18th (the day before admission) she had a fit in which for the first time she did not lose consciousness; otherwise it was identical with the others. She has suffered much from headache chiefly about the right temple during the last three or four weeks. The patient has a neural type which was followed by distinct secondary symptoms.

The patient was a dark well-nourished woman without any mark of syphilis. Physical examination of the various systems gave normal results.

The fits began at 10:00 p.m. on the 18th and at 4 a.m. on the 19th, when they ceased for a time. She was 75. They were always separated by intervals of at least several minutes, were preceded for a minute or two by a somewhat worrisome uneasy feeling. The first objective warning was a fairy twitch of the left foot, repeated in about half a minute; after a like interval the toes of both feet were flexed, the foot pointed, the left leg adducted. The left arm was raised by the side; raised; the right arm was sometimes flexed and held over the head. The head and eyes were turned to the left. The respiration was embarrassed. In a short time clonic spasm began—equal in the legs; the right arm sank on the chest; the left, slightly, the left, strongly. There was much twitching of the left side of the face and of the right side of the face. The whole fit lasted about a couple of minutes. It was clear that she did not lose consciousness for she was able to describe what took place about her during the fit. On the (9th) fit she spoke in
Enter this on the Dean's list as a Date.

Jesus.

It is not blame

Faith.

M. Rutherford

20 June
Answer to a question throne of course not infrequently. In the intervals.

she was flushed restless irritable. Her temperature never rose

above 102°. Immediately after the first fit there was slight

tremor in the left foot on dorsiflexion & this was again noticed at

(26th) 1.30 a.m. on the 26th after the 7th fit. At that time she complained

of her left arm feeling weak but she could move her fingers

raise her arm from the bed. She could also walk & her knee

jerk was equal.

At 11 a.m. after 28 fits she was still able to walk helping herself

with her right hand & swingy her left leg forward from the

pelvis. She did not use her left arm & when asked to move it

only moved it feebly from the shoulder. There was no trace

of ankle clonus: the left knee jerk was slightly less than

the right.

At 7 p.m. shortly after 47 fits a little left facial paralysis was

noticed, also some indistinctness of speech. Paralysis of left arm

leg was complete, both limbs being extended equally free from

rigidity.

At 4 p.m. before 57 fits marked facial paralysis, no ankle clonus.

At 8.30 p.m. after 67 fits the patient had slept a little. Facial

paralysis was gone & speech improved. Left arm & leg still complete

paralyzed. No clonus.

At 8 a.m. on the 27th the patient had slept twice in 24 hours. She

had her last (75th) fit. Speech normal. No facial paralysis.

She could raise her arm from the bed that a good grade

could move her leg feebly. No ankle clonus. The right knee jerk

was normal, the leg rising fully at each tap, while the left was

either feebly to absent but never equal to right.

At 9 a.m. she could walk with difficulty. No ankle clonus.
Knee jerks unchanged.
22° 23° 24° 1°-25° (morning). Patient continued to gain power. Could at last move her arm freely, though the movements were still weak. Could walk fairly well. During this time the left knee jerk improved, the characters on the last date being—sometimes normal, in extent, readiness, usually positive, sometimes absent. The last-taken mass in the above group of days was on the morning of the 15th. At 9 p.m. on that day the patient complained that for the last hour or half she had had twitching of her left arm, very similar to what she sometimes had before a fit. It was observed that at somewhat irregular intervals, usually of about 15 minutes, there was a slow twitch of the left forearm and left foot. The knee jerks had changed much: the left leg more than the right, and on the goniometer the movement being quite equal to a normal jerk but not equal to the right which had also increased its range. There was no ankle clonus.

On the morning of the 20th the twitchings were still present but weaker. The left knee jerk had fallen off a little from the description of the 15th (morning). At 8.30 p.m. the patient had a fit in which for the first time since admission she lost consciousness. She was seen at 6.30 p.m. when there was distinctly less power in her left arm. No ankle clonus. The right knee jerk had diminished since morning not rising so easily or so far to an ordinary tap. The left was absent.

On the 27th (morning) the right jerk was normal, the left rarely equal to the right. Usually right or absent. The paralytic was lean; the twitching still continued.

March 1st. Patient had 3 fits the morning but her jerks were not taken until the 2nd when they were found as described on Feb. 27th. On the 9th she had a fit but was not seen till Shrove Tuesday, when
her knee jerk were found unchanged. Her arm was again weaker.
16th no more fits. She had still slight twitching.
23rd discharged. Left knee jerk was occasionally absent or 
abnormal. Left grasp slightly weak. Walk normal.
She was afterwards seen on March 28th, April 6th, 12th. She was 
working at hat-binding. She noticed no defect in her left hand better 
in its power or delicacy of movement. The grasp was no longer 
perceptibly weak. Her gait was natural. The knee jerk on the 
left side still remained a little impaired. She had had no fits 
since her discharge.

while she was in the Infirmary, her eyes were repeatedly examined
the right eye was a little tender than the left; but no Maritio 
paralysis was seen at any time. When her knee jerks were 
tested her knees were laced when leg allowed to hang free.

The peculiarity of the case lies in the behaviour of the tendon 
jerks with regard to motor discharge. After the 7th fit the knee jerks 
were equal while after the 28th the left had diminished. 
I regret that they were not tested while the hemiplegia was complete 
but on the 21st the hour after the last fit, when the paralysis 
was so far disappeared as to permit full movement of the 
left limbs, the left knee jerk was faint or absent but never 
equal to the right. After the 25th improvement of jerk accompanied 
to a certain extent the improving power of the limbs.

The next motor discharge began on the evening of the 25th; for the 
first 20 hours showed itself as a slight flexor movement of 
the left forearm and foot. Then the patient had a fit in which she 
lost consciousness which was therefore more severe than her 
former ones. The change which took place in her knee jerks during 
the motor aura, as it may be called, after the fit was most
Remarkable. The slight motor discharge in this instance increased both knees jerks but especially the left; while the exhaustion either of cerebral nerve cells or of fibers connected with them abolished the jerk in the opposite limb I diminished it in that of the same side. The extinction of jerk was not due to exhaustion of gray matter of the spinal cord because it was observed 2 hours after the fit. It is to be noted, however, that the twitching reappeared it had apparently no influence on the jerk.

Elisabeth Parkinson, aged 6 admitted at 8 p.m. July 24, 1881.

The patient had been ailing all day. Was sick about 6 p.m. had a fit while being brought to the infirmary.

On admission she was unconscious, comatose. The convolution consisted throughout of clonic spasm of the right side of the face, right arm and leg, also of the left side. Left leg had in much lesser degree. Without exception each fit began in the face, then spread to the arm and lastly to the leg. As a rule they were distinct, but occasionally overlapped—the next fit appearing in the face before the former one had ceased in the leg. They progressed deliberately, e.g. twitching began in the face at 9:46 p.m., in the hand at 9:47 in the leg at 9:47, ceased in the leg at 9:50. Spasm ceased in the one of it invasion—face, arm, leg. Between the fits the patient was as a single unconscious where right limbs were always placid motionless while the left elicited movement. Sometimes moved. Left to 11:15 p.m.

The fits occurred at intervals of 2 minutes each. At 11:21 p.m. there had been no fit since 11:15. At this time she sat up shifted her position, opening her eyes, turning her head, moving her left
arm stiff but not her right. No facial paralysis.
At 11:28 p.m. she became conscious apparently but did not speak when questioned but put out her tongue when asked to.
At 11:37 p.m. the last fit ended.
At 11:45 p.m. she swallowed a little milk but did not speak or move her right limbs.
She was left at 12:10 a.m. when she was sleeping. The nurse, meantime to observe the following points, reported next morning that she had no more fits, that the first moved her right arm they at 2 A.M. that she woke at 2:15 A.M. but could not be induced to speak till 5:30.

With regard to myotatic irritability the following facts were repeatedly observed. On the left side a well defined elbow jerk could always be obtained by tapping the radius where there was always an ordinary left knee jerk except once immediately after a fit. On the right side while the limbs were placed there was neither elbow nor knee jerk; but soon after the twitching appeared at the hand an elbow jerk was obtainable. A knee jerk soon after twitching began in the foot. These tendon jerks were quite distinct from the movement of spasm. Spasm of rest of the face had no influence on the jerk of either limb; neither had spasm nor paralysis of one limb any influence on the jerk of the other.

Myotatic irritability in a limb appeared invariably with spasm and disappeared invariably with paralysis. It was absent at midnight 23 minutes after the end of the last fit.

On the following morning she had no paralysis when speech was natural. Her elbow and knee jerks were present on both sides and equal. Acro-athetoid sign examined on the 18th were normal.

I had once before noticed the presence of the jerk with spasm in its absence with paralysis in a boy of five who also had myotatic irritability.
Conclusions.

According to Flourens' theory Parkinson should have had exaggerated dystonic contractions in her paralysed limbs for, to judge from her paralysis, no exhaustion of the motor tracts of the spinal cord could have been more complete. But when sheam ceased dystonic irritability ceased also. In seeking an explanation of this, it is to be noted (for contrast with the following case) that the paralysis was not only complete as far as voluntary motion was concerned but also that there was never any rigidity of either limb. Directly sheam ceased the limb became flaccid at the same time tendon reflex disappeared. The tendon for RA did not cease from exhaustion of the grey matter of the spinal cord for two reasons: first, dystonic irritability co-clearly came at once with the onset finding of motor discharge; second, it was absent for periods of time varying from one to at least twenty minutes. Whether therefore the paralysis, which resulted from the palsy, be due to exhaustion of the cortical brain cells or of the fibres passing down from them, it was the actual loss of function of the cerebral motor area, that produced the loss of dystonic irritability. Directly these motor areas again began to act as shown by paresis the tendon reflex reappeared. How did cessation of motor discharge abolish the palsy? The position of the limb the blow on the tendon were identical during sheam during paralysis, but in the latter apparently the tendon could no longer send the limb did not produce the condition of muscular irritability necessary for tendon reflex. But this condition is brought about by reflex action. (Sowerby 'Practical Med.' 2nd Ed. p. 249). To the question how loss of function of a cerebral motor area prevents this reflex action there seem to be two possible answers: first, that it is controlled directly by the motor area; second, that it is inhibited by a lower centre which is in its turn restrained by the cortical centre.
Of these the second seems the more probable for exact control has the fact against it that the knee jerk persists after division of the spinal cord in its upper part, whereas destruction of the cerebral motor tract by hemorrhage or softening. Again, it is difficult to understand how loss of function of one part of the brain can abolish a spinal reflexion except by allowing another part of the brain to act uncontrolled to inhibit it.

Another interesting observation was made during spasm of the arm. The twitching usually began in the muscles of the back of the thumb, some time before it spread to through the fore arm or upper arm in a distinct elbow jerk or spasm. In King's case, it may be remembered, the twitching of the motor aura affected the foot only i.e. the muscles of the leg but not at all those of the front of the thigh from which latter, however, a greatly increased knee jerk was obtained. It seems, therefore, that not only did motor discharge in these cases produce an increase of spastic irritability but that it did so without causing contraction of the muscles from which the jerk came; or to put the conclusion otherwise— that increase of tendon jerk demonstrates a furler motor current than spasm.

Frederick Ogilvie, aged 4, was admitted at 8 p.m. on the 20th December 1881, unconscious and convulsive. The spasm was clonic right-sided but in addition there was slight twitching of the left eyelid, leg, occipital region of the left arm. Convulsions ceased at 2.45 p.m. but he did not regain consciousness until about an hour later. From 2.45 to 2.57 p.m. his right elbow was bent at a right angle, the arm rigid. It required some force to straighten it. When released it immediately assumes its former position. The right leg was
also rigid though not in the same degree. About 3 p.m. he moved his right limbs for the first time; at 7 p.m. they were still weak. At 10 a.m. he felt trace again at 3 p.m. Exaggerated knee jerk and ankle clonus were obtained on the right side but at 4 p.m. the ankle clonus had disappeared. The right foot was only a little more active than the left. At 8:30 p.m. the jereals were equal.

There are two points of difference between the paralysis in Ogilvie's case and in Parkinson's: first, the difference in motoric irritability; second, Ogilvie's paralysis limbs were rigid while Parkinson's were flaccid. The rigidity was evident as soon as sleep ceased and gradually disappeared in 12 months. How was it caused? Not by any action of the motor discharge on the spinal cord for it was absent in both Parkinson's whom it never was severe as Ogilvie's; the same objection holds against it being a purely muscular phenomenon. Neither could it be due to any uncontrolled action of the cerebellum for Parkinson's paralysis was complete the limbs placed. We are forced to conclude therefore that the rigidity was of cerebral origin due to a continuation of the motor discharge. For as physiological a muscle requires a certain measurable quantity of nerve force to produce its contraction, it is illogical so long as our only source of motor discharge is contraction of muscle, to infer that because the spasm of convolution stops suddenly, the motor discharge must also in all cases cease as suddenly. A priori, it seems quite improbable that the cessation might be gradual. But if this continuous motor discharge exists as Ogilvie case it helps to explain the presence of exaggerated motonic irritability which co-
backed so vividly with its total absence in Parkinson's case; and has been shown that in such cases motor discharge increases without necessarily producing contraction of muscles. Both suggest the condition of increased myoelectric irritability; therefore, seem to be due to the same cause: viz. the persistence of motor discharge in diminishing intensity after the end of the fit; but of these two effects the latter being the better marked in some cases is the better sign of such motor discharge. Indeed as will be seen more fully, may be slight or even absent after some attacks.

Thomas Street, aged 48, admitted November 16, 1881.
In March of that year having been in perfect health previously, he had his first fit. It commenced with tremor of the right arm, was not observed to be unilateral, but was followed by weakness of the right hand for half an hour. Since then he has had about 10 fits. Some after the first have been confined to the right side, followed by temporary right hemiplegia. In all of them he has lost consciousness early in the attack. His first aura occurred before the fit; it described it as occurring before all his subsequent attacks, consisting of a dull pain feeling. This usually starts from a definite spot at the right costal border in the axilla and branches either up to the shoulder down to the arm or down the leg. When it reaches either the hand or the ankle, there begins in the limbs, but not constantly. As he has pure, sensory anesthesia; after the pain has lasted some minutes, he has a fit. Neither, however, in this constant as he often has attacks of combined motor and sensory aura which do not.
Go on to a fit. In fact, between the slightest aurora to a severe fit he has every degree of attack. His right limbs are weak both after a combined aurora or after a pure aurora one but the weakness is less after the latter. A week before admission his speech altered but remained impaired. Since the fits began he has been at times giddy but has neither headache nor sickness. No history direct or remote of syphilis.

The patient was well developed muscular. Pulse 82, after thickened. Respiratory system normal. Heart: after not displaced slight systolic aortic murmur. Blood, acid. Sp. gr. 1053. No album. There was neither tenderness nor tumour over the scalp. No cranial, facial, or ocular paralysis. The pupils were equal firm, small reactive; the optic discs were normal. The grip of the right hand was perceptibly weaker than that of the left but still powerful. No cluminess observed in buttoning. He dragged the right leg a little in walking.

Reflexes: Plantar present but feeble on both sides. No ankle clonus. Kneejerk equal. Cremasteric present but not active. Abdominal cremasteric better marked on the left side.

The patient was stupid but could always name objects correctly and ask for what he wanted though his sentences were always short and ellipt of the shorter words. His articulation was hesitating. He occasionally used 'yes' for 'no' and vice versa. His memory was defective he could give no connected history of his illness. He has never been able to read or write.

After admission he has a few slight attacks but it was not till December 6th that they were specially observed.
At 9:35 a.m. on that day, the patient called the attention of the nurse, who immediately afterward observed his right leg drawn up. There was only one movement of flexion by which as he sat his foot was drawn towards him along the floor. No spasm of face or arm; no change in speech. When he was seen at 9:38 a.m. he said that he still had the feeling (aura) in his ankle but that it was beginning to pass off, that there had been no aura in arm. At that time there was ankle clonus or exaggerated knee jerk on the right side; no ankle clonus or normal jerk on the left. This was repeatedly noted between 9:38 and 9:45. A little before the latter date he walked with help across the ward, dragging his right leg more than usual, and about the same time there was some tremor in right hand.

At 9:45 the aura ceased at foot, ankle clonus was less marked, at 9:47 clonus ceased. Knee jerk still exaggerated. The difference between the knee jerks gradually diminished but was still perceptible at 10:40 a.m., doubtfull at midday and absent at 2:50 p.m. At midday his leg felt a little weak but no increased dragging was detected; at 2:30 p.m. he was as well as usual.

On December 7th the aura was felt on the side at 10:35 a.m., passed down to foot. When he was seen there was distinct right ankle clonus before the aura ceased but the knee jerk was not increased until the aura was passing off. At 10:34 clonus had ceased while the jerk was still exaggerated but at 11:10 the jerks were equal. Although the aura was a fairly dense one it was followed by a feeling of weakness in the arm and leg. The grasp was for a short time weaker than usual. His walk seemed unaltered.
December 27th. Patient was again seen in a sensory aura. As long as the aura was confined to the side there was neither ankle clonus nor increased knee jerk, but when it reached the ankle, clonus appeared and paresis of the knee jerk became exaggerated 4 mm. After the beginning of the attack the aura ceased. Clonus was disappearing at 9 mm. 1 was absent at 12 mm. When the knee jerk was still increased some tremor was felt in right quad. At 23 mm from beginning the jerks were equal although the leg felt weak. No change was noticed in the patient's gait. There had been no aura in arm two affection of speech.

Three notes of 8 other auras in 6 of which there was movement in 2 none. In all there was ankle clonus of various degree but always distinct generally beginning when the aura had reached the foot or was disappearing casting from 1 to 4 mm. in all but once a slight sensory aura which traveled from the side to the knee only — there was exaggerated knee jerk beginning as a rule after the ankle clonus lasting from 10 to 20 minutes.

December 27th. At 10.40 a.m. Clonic spasm of the right arm began 1 after this had lasted for 4 minutes, his head and eyes turned to the right, he lost consciousness, the tonic stage of the fit set in. The right arm was flexed across his chest; his right leg extended rigid while his left arm was twitched to his left upper arms from the floor. The Clonic spasm which soon replaces the tonic affects the right limb only. The whole fit proper was over in about 13 min. On its termination right ankle clonus & exaggerated knee jerk were readily obtained. At 10.51 he tried to speak but was unintelligible. He moved his right foot very stiffly, but when asked to move his right arm he left it with his left hand. At this time
his right thumb & finger were noticed twitching. At 10.56 his right hand closed involuntarily. The eyelid opened without using his left. Right facial paralysis noticed. At 10.58 when he was asked to open his hand the two middle fingers at once closed. Could stand with help. At 11 a.m. he could move his fingers freely. Tongue deviated to right. At 11.33 ankle clonus increased. Knee jerk still present. Draped right leg much in walking.

On the same day he had a second fit of which the following are the outlines:— the arm began to jerk at 1.12 p.m. at 1.20 the fit ended: for 2 minutes after this the right leg was rigid. Sticking nearly straight out when the thigh was lifted; during the time there was ankle clonus & the knee jerk, though limited in extent, by the tension of the leg was produced by the slightest tap on the shin. First movements of arm & leg observed at 1.35; at 2.24 clonus was slight, knee jerk still increased.

On January 14th at 11.34 a.m. he informed me that a fit was coming on. At that time his right elbow was bent at a right angle, his arm rigid, his hand closed. There has been neither a nor movement in the leg but there was ankle clonus & exaggerated knee jerk on the right side. The arm was now forcibly straightened on it becoming rigid. Extension was again flexed & this passive movement was kept up for 2 minutes or two. At 11.58 he said that his arm felt well again except for a little stiffness about the shoulder & it was found that he could not raise it fully above his head. At 11.40 clonus was very slight. Jerk still present. At 11.42 clonus doubtful. At 11.45 clonus again became well marked at the same time twitching of the right fingers was noticed. At 11.46 twitching repeated. At 11.48 clonus marked. At 11.50 clonus full. At 11.51
Clonus increased again. Patient said he felt sick & expected an aura. At 11:52 aura in arm & leg. At 11:53 finger twitches. Clonus marked. At 11:58 twitching occurred again. The Clonus, which had become feeble began to increase about a minute before spasm appears. At no time in this attack was there any movement of the right foot or leg or no leg aura until 11:52.

On January 13th Kneepkoks Equal: No ankle Clonus.

In the attack just described it is impossible to believe that the Clonus & increased Kneepkoks could be caused by exhaustion of the motor tracts of the cord. The movement was much too slight to admit of such an explanation. But the theory of continued motor discharge here advances readily explains their presence after so feeble a spasm.

The pure sensory aurae, while accompanied or followed by Clonus or increased Kneepkoks were also followed by a feeling of weakness in the right limb. In one instance by an objective loss of power in the hand. The neural explanation given of the Conjunction of Sensory aurae with loss of power is that of inhibition of the motor centre by the sensory; but since extinction of the function of a motor-centre in Parkinson's case abolished myotatic irritability in the limb it is to be expected that diminution of function by inhibition would cause weakness is K. In Sweet's case, however, the sensory aura increased myotatic contraction. Against any attempt to explain the weakness by the theory that it resulted from motor discharge there has always hitherto been the sufficient objection that there was no evidence of any discharge. But after what has been said to show that increase of myotatic irritability may in certain circumstances
he taken as such evidence the theory above indicated may not be so absurd as at first appeared. At any rate the probability seems to be in favour of direct sensory aurae, being accompanied by a slight motor discharge to which rather than to inhibition the weakness was due.

Concerning the first fit it is to be noted that at 10.51—11.01 minute after the beginning of the attack while he could not move his right hand his thumb and fingers twitched; that at 10.55 his right hand closed; he had to use his left to open it; that at 10.58 his two middle fingers flexed instead of extending. After the second fit the leg was rigid for 29 minutes. The twitchings show at the possibility of motor discharge occurring during the paralysis after a fit but are of course no evidence of the existence of that slighter continued current advocated in the paper. The rigidity after the second fit resembles that commented upon in Ogilvie case 1 admits of the same arguments in favour of it being cerebral rigidity.

In the attack of January 14th—although perhaps the most remarkable of all his observed seizures there was movement of the arm but none of the leg at any time a no leg aura like about 16 minutes after he was first seen, but even at first foot clonus increased this jerk were readily obtained. Still more noteworthy was the way in which later the clonus increased when the fingers twitched diminished in the intervals. Inhibition as a cause of the clonus is out of the question because there was no leg aura at first so also is exhaustion of the motor tracts of the cord for there was no movement of the leg at any time. But it is quite probable that while the arm centre was discharging strongly the neighbouring leg centre might also be in action with sufficient intensity to elicit a
the myotatic irritability of the limbs. In what other way can
explain the regular increase of the clonus when the fingers
jerk?

From these four cases, too small a number to give certainty of
perfect, stable conclusions; I admit, the following inferences are
drawn. During the temporary paralysis after convulsions
myotatic contraction may be either absent or increased. In the former
case cerebral motor function has probably ceased; the limbs
are flaccid; in the latter, motor discharge does not cease
when the fit ends but gradually diminishes. Etti; as long as
the tendon jerk is excessive while the limbs may be rigid for a
short time after the fit or may show motor discharge by twitching.
Muscles receive a motor current that will not cause their
contraction. The duration of this discharge of not its intensity is
measured by the increase of myotatic irritability. The inhibitory
reflex part of the phenomenon of tendon jerk is inhibited by a lower
cerebral centre which is controlled by the cortical motor areas.
In some cases where a sensory aura is followed by loss of power
it is possible that it may be accompanied by a slight motor
discharge.