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ASTASIA-ABASIA.

WITH THE REPORT OF A CASE OF PAROXYSMAL TREPIDANT ABASIA ASSOCIATED WITH PARALYSIS AGITANS.[1]

By PHILIP COOMBS KNAPP, A.M., M.D.,

Clinical Instructor in Diseases of the Nervous System, Harvard Medical School.

THE earliest account of this curious and rare motor disturbance was given by Jaccoud in 1864, under the name of "Ataxia by defect of automatic coördination." Weir Mitchell, in 1881, reported a case under the heading of "Hysterical motor ataxia." Two years later Charcot and Richer described several cases under the title of "A special form of motor impotence of the lower limbs by defect of coördination relative to standing and walking." Other cases were reported by various observers, but it was not until 1888 that Blocq, collecting all the cases reported and adding several new ones, gave to the disturbance observed in these cases, eleven in number, the name "Astasia-abasia"—a name which has since been generally adopted. "We thus designate," says Blocq, "a morbid

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state in which the impossibility of standing erect and walking normally is in contrast with the integrity of sensibility, of the muscular strength, and of the coördination of the other movements of the lower extremities." Subsequent observations have not served to alter materially Blocq's definition. The cases are still few; I have succeeded in collecting only forty-nine, and of these, thirty are reported by French observers. Only three cases have thus far been reported in this country, which may justify me in calling attention again to the subject, and in reporting a fourth case, which presents some of the symptoms of abasia, but which is not wholly typical.

Let me say first that astasia-abasia cannot be regarded as a morbid entity, like typhoid or tabes; it is rather a special symptom-complex, like athetosis, chorea, or eclampsia, occurring sometimes apparently independently, at other times being associated with other affections, such as hysteria, exophthalmic goitre, or the intention psychoses. As a symptom-complex and not as a morbid entity it must therefore be studied.

OBSERVATION I.—On the 25th of February, 1891, Charles Murphy presented himself at the out-patient department of the Boston City Hospital. He was born in Ireland, he had been a marble-worker, and he was fifty-eight years of age and married. His mother, one brother and one sister died of phthisis; his father died at seventy-nine of old age. Two brothers and two sisters are alive and well. No history of neuropathic taint could be elicited. His own health has always been fairly good; he had a mild attack of small-pox some forty years ago, and he had malaria and dysentery during the war; his habits are temperate, he has never used alcohol and he denies any venereal disease. In 1889 he had some catarrh, after which he began to have trouble in walking and what he calls "trembling" in the legs. At the same time he had some pain in the shoulders, chiefly over the upper part of the left trapezius and some difficulty in turning the head to the right. He has also had some cramps in the legs.

The difficulty in walking is as follows: while walking, when starting to walk, and especially when turning, he is attacked with spasm in the legs. He is very slightly bow-legged. The steps become shorter and shorter, and the

cadence is more rapid; there are rapid, almost rhythmical flexions and extensions of the legs on the thighs, and the thighs on the pelvis; the feet seem to cling to the floor; he rises slightly on the toes, carrying the trunk and head a little forward; the steps grow shorter, the cadence more rapid, suggesting the action of a locomotive on a slippery track. Finally the spasm becomes almost tonic, and the feet for a moment almost cease to move. At this point he exhibits a tendency to fall forwards, but he often recovers himself and he can go on with a natural gait. Excitement increases the trouble; it is very noticeable when

he enters a room, and it was once noticed in a very marked degree when he found himself in front of a team. He has a feeling of weakness, but there is no emotional disturbance, or any distress or anxiety during the attack. At times he falls in these attacks, most frequently falling backwards. Of late he has complained of a similar feeling of trembling in the arms, so that he cannot use an axe to chop wood.

He was somewhat depressed, from his inability to work, but no other psychical disturbance could be detected. His vision had failed a little, he was somewhat costive and the

sexual power was a trifle diminished. He had no abnormal sensations, but his limbs felt weaker. For five years he thinks his memory has failed.

The man was fairly developed and nourished. The head was carried bent a little forward, and the face was rather immobile, suggesting the facies of paralysis agitans. The pupils were very small, and on one occasion unequal, but they reacted normally. *Vod* $= {}^{17}/_{15}$, *vos* $= \frac{16}{20}$ —, *vos c* + 0.75 $= \frac{17}{15}$ —. The visual field was normal for form and color; the color sense was good. Further examination of the

eyes and ears was negative, as was examination of the chest, abdomen and urine. The reflexes were not remarkable. Sensibility in all its forms was everywhere good. All the ordinary movements could be performed with fair strength, and good coördination. There was no tremor, ataxia, or Romberg's symptom. The electrical reactions were normal.

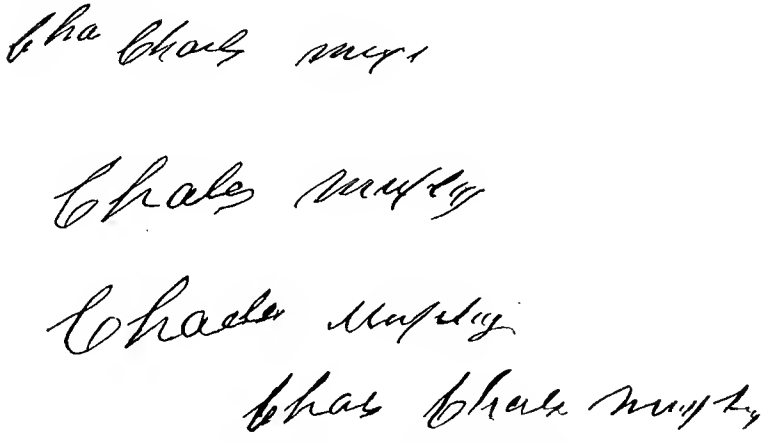
On attempting to hurry him, to take him into another room for consultation, or to bring him before a class the trouble became more marked. It seemed to me that the spasm was largely dependent upon idea, and so, in order to break up the morbid train of association, I directed him, when

the spasm began, to practice the "balance step" of military drill. This arrested the spasm so effectually that it was hard to demonstrate it to the class two days later, or to demonstrate it before a medical society a week later. Unfortunately he heard me explain my theory of the action of the remedy and its efficacy declined. He was treated also with tonics, including strychnine and faradism, but there was not much more improvement and he grew discouraged.

At that time the trouble seemed to be very distinctly abasic, and to be confined chiefly to the legs. At my request he came to see me again on the 11th of September, 1891. His condition had grown worse since I last saw him in April. He had repeated spasms on walking from my waiting-room into my office, taking not more than four or five steps without them. The character of the spasm is unchanged, but he complains now much more than formerly of falling backwards, and a moderate pull or push will cause him to take several short quick steps backwards and to fall—a distinct retropulsion such as is sometimes seen in paralysis agitans. The forward movement has also more of the character of propulsion. He claims that this retropulsion has existed for eight months, but in February he had little to say about it. He complains also of a general trembling, which is worse in bed. He always feels warm, but not uncomfortably so, and his legs sweat freely. For six months he has had vertigo, objects apparently moving from side to side. He has much more general disturbance with the muscles; he thinks he cannot talk as well; the trouble in chopping wood is worse, he cannot write as he used, he even has trouble in holding a newspaper. He says that were it not for the "balance step" he could not get about at all. Aside from the muscular trouble he feels perfectly well.

The pupils are small, less than two millimetres in diameter, and react slightly to light, but more to convergence. There is no stiffness of the muscles of the neck, but he cannot move the head quite so far to the right, and there is a spot which is somewhat tender to pressure over the upper part of the left trapezius. There is no spinal tenderness. He can move the muscles of the face very well, but on making repeated movements of showing the teeth, after a few times the movements are made less regularly and there is a slight tendency to tonic spasm. The voice is feeble, rather monotonous and high-pitched. He can drum with the fingers quite well, but on writing, as will be seen, the spasm soon appears, and after the first few letters, the writing tends toward a series of perpendicular lines, made

with considerable difficulty. He used, he says, to write well. On attempting to hop on one foot or with both feet together he has much difficulty, and the spasm develops. He can go about on his knees or on all fours, apparently not very well, but he says without any of the "trembling" feeling. The muscular strength in the legs is fairly good. The grasp shows right = 13 kg., left = 15 kg. There is no tremor or incoördination of arms or legs, even with the eyes closed. Sensibility remains unimpaired. There is no cardiac or other distress with the attacks of spasm, but the pulse is a little rapid, 106. After a time the attacks were less frequent and he walked about my office for a number of steps quite well, the spasm appearing as he turned and as he prepared to sit down. The balance step can still be



Handwriting of Charles Murphy, September 11, 1891.

performed during the spasm, and it seems to relieve it. There is no muscular rigidity or contracture and no tremor can be felt in the muscles. There is no albuminuria or glycosuria.

On the 15th the retropulsion was still more marked and quite characteristic of paralysis agitans. There was also much trouble on trying to turn. No tremor of the hands or legs could be detected, but there was occasionally a fine tremor of the head.

The diagnosis in this case is not entirely clear. There are certain affections from which abasia must be distinguished, but we need only to name them to render it evident that we have to do with none of them. Among them are tabes, disseminated sclerosis, Friedreich's ataxia,

alcoholic neuritis (with high-stepping) cerebellar vertigo, tic convulsif, paramyoclonus, Thomsen's disease, professional neuroses and the intermittent claudication of diabetes. It is more probable that the disturbance is due to minor "functional" or "dynamic" changes in the nerve-cells than to permanent gross lesions, otherwise the trouble would be more constant. Hence a spastic or ataxic paraplegia from organic changes in the cord is not probable, although the gait somewhat resembles the spastic gait. The absence of exaggerated reflexes, contractures and rigidity moreover renders spastic paraplegia very improbable.

Hysterical ataxia or hysterical paraplegia may be excluded by the absence of ataxia or paraplegia. Whether the present affection be hysterical must be discussed later, but there are certainly none of the stigmata of grand hysteria.

Rhythmical chorea may readily be excluded by the absence of any unusual movement when the patient is at rest.

Saltatory reflex spasm has been confused with abasia. and Thyssen and Cahen have considered one case (Obs. XL.) as abasic, which Brissaud, in whose charge it was, distinctly says was not in his opinion abasia, but saltatory reflex spasm. In this affection there are repeated contractions of the muscles of the lower extremity as soon as the feet touch the floor, which result in throwing the body into the air in a succession of leaps. It is plain that we have here no such condition. The spasm may also appear when pressure is made upon the sole when the patient is sitting or lying down.

To one who reads the report of the present case the resemblance to paralysis agitans would probably be less striking than if he were to see the patient. To-day, more than in the spring, there is a strong suggestion of paralysis agitans about him, in his facial expression, his attitude and the propulsion and retropulsion. I believe that in time he will develop into a typical case of paralysis agitans, but, if he does have the affection, it is paralysis agitans *sine agitatione*. If this be paralysis agitans it is associated with attacks of inability to walk, and with the preservation of muscular force and coördination, with ability, during the attacks, to make various coördinated movements akin to walking. Such a condition corresponds to the definition of abasia, yet I know of no recorded case where abasia occurred with paralysis agitans.

On the other hand, if the case be simply one of abasia,

it differs materially from other cases. In a few cases there has been some implication of the arms, but here there is a morbid state which seems to involve almost all the muscles. After a muscle has made a few contractions, a condition of spasm supervenes which prevents the normal, harmonious action. This is seen in the muscles of the face and in writing. It differs from the spasm in Thomsen's disease in that that appears at the beginning of muscular effort and diminishes. This, although in the legs it may appear in the beginning, in the other muscles comes on after several contractions and increases to a tonic spasm. It resembles the spasm in writer's cramp and seems like a writer's cramp affecting all the muscles of the body, but chiefly the legs.

My original diagnosis last February was abasia with a suspicion of beginning paralysis agitans. To-day my suspicion has grown stronger, and I think that time may show that the abasia was really a symptom of paralysis agitans *sine agitatione*.

Before considering the nature of this curious symptom-complex it will be well to analyze, as briefly as possible, the recorded cases, to furnish us with data for our judgments.

Abstract of Reported Cases of Astasia-Abasia.

Observation II.—(Blocq,[4] from Charcot.) Girl. Father's family gouty; father died of myelitis. Mother's family rheumatic, gouty, alcoholic; grandmother neuropathic; mother rheumatic, nervous, hysterical. Child had convulsions while teething; neuralgia at the age of five, typhoid with convulsions at two and a half. Child fell while at play, striking her back; the next day she complained of pain in the back and difficulty in standing. Dorsal decubitus avoided; marked hyperæsthesia of back. Crises of pain at night. Walking gradually became impossible. Marked hyperæsthesia of the back and some of the thighs. The legs can be readily moved in all directions while in bed, but passive movements can not be easily performed on account of the hyperæsthesia. No ankle clonus or contracture. She can stand with support. She cannot walk, but she can jump a short distance with the feet together. Contraction of the visual fields. Recovery six months and a half after onset and eight days after treatment.

Obs. III.—(Blocq,[4] from Charcot.) Boy, 14. Mother had migraine. He had an address to make, was very nervous

in consequence and had headache and weakness of the legs. The next day he could not get up, stand, or walk, but he could move his legs in bed. Other functions were performed regularly. Muscular strength and coördination in the legs was perfect. No anæsthesia or exaggeration of the reflexes. The boy could only drag himself along. Isolation. Two months after onset the boy could get about by jumping first on one leg then on the other. Two weeks later he walked normally. Four months later, without apparent cause he had another attack, lasting a month,

OBS. IV.—(Blocq.[4]) Boy, 15½. Mother and maternal grandmother rheumatic. In March, 1887, typhoid fever. On recovery he could not stand. When supported under the arms, the legs bent under him and could not support him. When sitting nothing abnormal could be detected in the legs. He could go on all fours, or hitch himself along in a chair, but he could not jump or hop on one foot. He could shin up a tree, but rather poorly. Since this illness he has not dreamed of walking. Nothing abnormal in the legs; sensibility in all its forms intact; knee-jerks lively; no ankle clonus; no hysterical stigmata; slight limitation of the visual field. Cure by transfer, June 27th, four days after entering the hospital.

OBS. V.—(Blocq,[4] from Romei.[32]) Boy, 11. Congenital strabismus. No heredity. Sudden severe fright. After this he kept in his room for two days saying that he felt weak. Then he had a severe headache which kept him in bed four days. On recovery he found that he could not walk or stand. Sensibility and muscular strength normal. Slight pressure on the spine was painful. Moved feet perfectly in bed, but he could not stand; legs seemed dislocated when he tried to walk with support. He walked like a child just learning. He recovered from this and a year later had rheumatism in the left foot. A year after this he walked bent over, had trouble in ascending stairs, his legs failed and he walked as if drunk. The dorsal vertebræ were tender. Four baths relieved the pain.

OBS. VI.—(Blocq,[4] from Weir Mitchell.[27]) Woman, 20. Asthmatic. Sudden loss of speech with unconsciousness and convulsions. Five weeks later a peculiar paraplegia. She could move the legs fairly in bed, but on standing or walking she swayed to either side, and her efforts to maintain herself were in excess and threw her to the opposite side.

OBS. VII.—(Blocq,[4] from Charcot.) Boy, 15. No heredity. Well and strong. In 1882 hystero-epileptic attacks

(arc du cercle, passionate attitudes). Recovery in 1883. Soon after anorexia and emaciation, followed by present trouble. Cannot stand or walk. Hands kept closed. Legs extended, stiffen at attempts to flex them. Voluntary flexion possible. Knee-jerks exaggerated. He can jump but cannot stand. On supporting him limbs stiffen at right angles to trunk. Voluntary coördination, except for jumping and voluntary flexion, is impossible. Considerable anæsthesia; achromotopsia.

OBS. VIII.—(Blocq,[4] from Charcot.) Girl, 14½. Father sexual criminal, sister hysterical. Menstruated at 13½. Six months ago pain in left flank, later in back, followed by trouble in legs. Corset applied, patient was in bed for some months. One day ran, under stress of emotion, then became paralytic again. Spinal hyperæsthesia. Knee-jerks normal. No anæsthesia or loss of muscular sense. In bed legs are flexed and cannot be extended voluntarily. She can raise them from the bed. Legs can be extended passively, and unconsciously by the patient. On standing the legs remain half-flexed, without incoördination. The movements of walking cannot be performed.

OBS. IX.—(Blocq,[4] from Erlenmeyer.[15]) Man, 28. No heredity. Previous history good. Overwork for ten months, followed by localized and general muscular twitchings, insomnia, and excess in the use of alcohol. Fell in the street and lost consciousness. Weak and sleepless for a fortnight after it. Two months later the left leg began to give out when walking. He recovered but the right leg was later affected. This was followed by a convulsive jump, the body being thrown into the air by the feet, as the knees gave way, followed by a certain number of regular jumps, the feet not quitting the earth, the body being thrown up when it was advanced. The legs were finally so flexed that the back of the thighs touched the calves. Knee-jerks exaggerated. No disturbance of strength or coördination. When the sole is put squarely on the ground the knee is flexed and the body makes a compensatory movement forwards. The jump is made only after the primary flexion of the knee. It is not seen when the patient is supported, nor is any movement caused by pressing on the sole while the patient is in bed. Sensation normal. Percussion of the ligamentum patellæ, while the patient is standing, will produce a similar flexion at the knee.

OBS. X.—(Blocq,[4] from Babinski.) Woman, 27. Father neuralgic. Mother impressionable, had fits of rage in which she lost consciousness. Gastro-intestinal troubles. For

nine years headaches, gloomy ideas, nightmare, desire to weep, fear of solitude. In 1883, after some disturbance, could not stand on rising from knees. She made a partial recovery with two or three relapses. In December, 1883, had an attack after which she grew worse. In February, 1886, nothing abnormal noted while sitting. All movements could be performed, but she had a sensation of shock in the knees. On standing she had jerking movements, a flexion of the legs on the thighs, and of the thighs on the pelvis. In walking these movements increase, and become more rapid. She stops and tries to recover equilibrium. The trunk is flexed on the pelvis, the head is flexed and rotated, and the forearms are flexed on the arms. She is obliged to sit down, looks fatigued and sweats. There is no incoördination. She jumps and walks on all fours. The movements cannot be produced artificially when she is sitting or lying down. Slight left hemianæsthesia. Knee-jerks almost absent. Visual field contracted on left. Hearing and taste diminished on left. Hysterogenous zones, pressure on which cause attacks of muscular twitching. Walking easier after attacks.

OBS. XI.—(Blocq,[3] from Babinski.) Woman, 22. Cousin insane. Irritable. Infelicity since marriage. Difficult labor two and a half months before. Some days later nervous crises, throbbing of the temples, constriction in the throat, loss of consciousness, rigidity of the limbs, followed by a feeling of fatigue. These crises resumed several times a day. Poor appetite. On recovery from confinement she could not walk. After taking five or six steps twitchings begin in legs, which rapidly increase, so that she cannot stand. The thigh flexes on the leg and the pelvis on the thigh; the heel is raised, striking the ground two or three times. The patient tries to regain equilibrium and the trouble comes on in the other leg. The trouble increases, she walks, carries the foot back, but cannot advance, the legs flex and extend, the trunk is carried forwards and backwards, the feet striking the ground in a sort of marking time and she falls. Sitting, the muscular strength is good. Slight left hemianæsthesia and left ovarian tenderness. The trouble disappeared at once by hypnotic suggestion.

OBS. XII.—(Blocq.[4]) Woman, 52. No heredity. Previous history good. Fell on her back, losing consciousness; in bed for three months. Six months after fall, without apparent cause, severe lumbar pain. On recovery, three days later, she found she could not stand. For two months she

could not use her arms at all, then they recovered completely, but the legs still troubled her. Anuria for three days. Pain and formication in legs. Lumbar pains persist. Anæsthesia of legs up to the groins. Reflexes normal. Muscular strength diminished but coördination is good. She can stand with great difficulty. When standing she has oscillations and flexion of the pelvis on the thighs and the thighs on the legs. On walking the knees are flexed, and the trunk is projected forwards and backwards. The feet do not drag. Standing and walking are impossible with the eyes closed. Trouble worse on the left. Right visual field contracted. Taste defective. Hyperæsthetic points.

OBS. XIII.—(Brunon.[1]) Boy, 8. No heredity. Previous history good. After a slight febrile disturbance with eruption, it was found he could not stand or walk. His legs bent under him. In bed he could raise his legs with good strength. General condition good. No ataxia or sensory disturbance. Reflexes normal. Ten days later had gained strength. Staggered as if drunk. Two days after that took some steps with hesitation; could jump and make coördinated movements. Moved legs very slowly and walked like a young child. Rapid recovery.

OBS. XIV.—(Berthet.[2]) Woman, 25. Father alcoholic. Anæmic for eight years. Menses irregular, leucorrhœa. At seventeen began to be nervous. Impressionable; had palpitations and constriction of the larynx. At twenty-three had crises preceded by palpitations; in these she loses consciousness and cries and is weak after them. Fourteen months before had severe crises, after which she could not walk and vomited everything but oranges. Standing is difficult and walking impossible. If she stands she trembles, the body making great oscillations and the head and arms making rhythmical movements. After rest in bed she can use her hands in sewing, etc. Slight hyperæsthesia on left to pain, touch and temperature. Sight and hearing better on right. No motor trouble in bed and no ataxia. Tender points, pressure on which gives rise to dyspnœa. She got nearly well then had acute rheumatism, after which trouble returned. When she stands, walks or sits the oscillations return—undulations like the progress of a reptile. She falls, sweats profusely, there is marked dyspnœa and the limbs become inert. With a corset she can do better. She walks on all fours and hitches along on a chair. Intense hyperæsthesia, no pharyngeal reflex, convergence and light painful. Mild faradic current caused intense lum-

bar pain. No trouble when suspended. Trouble reappeared in bed when told to resist lifting the feet. In bed or sitting no trouble in legs, but some trouble in arms. Relief from cold douche.

OBS. XV.—(Grasset[18]–Mathieu.[26]) Man, 29. Nervous crises and convulsions in childhood. A year ago had loss of consciousness without diminution of intellectual force, with trembling of the legs. Similar crises since, with true hystero-epilepsy. Pain and paræsthesia in legs. He has a dancing gait, has difficulty in detaching the right toe from the ground, leaps on it and advances in a painful manner, the body and head oscillating. Walking becomes impossible, the rhythmical oscillations increase, the heel repeatedly strikes the ground and he finally falls powerless. In bed he has no trouble. He can walk cross-legged or by raising the legs high or on all fours. Anæsthesia, loss of pharyngeal reflex, concentric limitation of visual field. After some attacks of abasia he has a marked procursive impulse without loss of consciousness.

OBS. XVI.—(Charcot.[11]) Girl. Probable epilepsy for three years. Since that time she has been unable to walk without support. Cannot go up or down stairs. Fair strength in limbs. Reflexes normal.

OBS. XVII.—(Charcot.[12])* Boy. Cannot walk, but can go on all fours and shin up a tree. Cure in three days. Hysterical cough and feeling of suffocation.

OBS. XVIII.—(Charcot.[13]) Man, 41. Neuropathic heredity. Night-terrors, migraine; timid, impressionable. Profound nervous shock three years before followed by crises of marked malaise and weeping. Poisoning by carbonic oxide; unconscious for three days. Leg burned by sinapism; insistent idea that he might be unable to walk. Legs grew weak and stiff and finally, twenty-five days later, he suddenly became unable to walk. Muscular strength in bed good. No rigidity, contracture or sensory disturbance. Knee-jerks normal. No trouble while sitting and he can stand perfectly well. On trying to walk inclines forward, the legs together; rises on toes, which glide along the floor in a jerky tremulous fashion as if he were impelled forward. At the moment the knee is flexed to carry the foot forward, a contrary movement of extension occurs. He can jump with the feet together or on one foot, go on all fours or walk with a tragic stride.

OBS. XIX.—(Charcot.[14]) Man, 44. Grandmother nervous, mother alcoholic. Always nervous and emotional.

* Possibly the same as Obs. III.

Ten years ago severe nervous crisis after emotional shock. Crises at times ever since. Two years ago had a severe attack, lasting two days; on getting up found he could not stand. He could move his legs in bed. The trouble lasted four months. Seven months ago another crisis of trembling followed by the present trouble. He can move his legs perfectly in bed, he can go on his knees, on all fours, or hitch on a chair. There is no pain or rigidity. The reflexes are normal. He cannot stand at all, his legs giving way under him.

OBS. XX.—(Charcot.[14]) Man, 49. Mother had spinal disease; cousin alcoholic. Night terrors. Eight years ago left hemiplegia, lasting a short time. Since then demented. Abasic for two or three years. He can walk at times and move his legs perfectly in bed. On starting to sit down or on rising he has a peculiar trembling, similar to that shown by patient of Obs. XVII. He can go on all fours or hop with the feet together. Contraction of visual field; no dyschromatopsia. Left hyperæsthesia. No pharyngeal anæsthesia. Taste, smell and hearing normal.

OBS. XXI.—(Charcot.[14]) Man, 75. No heredity. Previous history good. Six years before, without known cause, he had a feeling of weight in the left hip, and he had to carry his left leg forward first in walking. Eight months ago feeling of weight in the nucha and occiput and on the shoulders, and the trouble in walking extended to the right leg. In bed there is no trouble with the legs; no ataxia or anæsthesia. Knee-jerks normal. Stands well. If slightly pushed, sudden movements of flexion and extension in legs. The trunk is carried forward and the feet strike the floor as in Obs. XVII. He can stand on one foot, hop with the feet together or on one foot, go on all fours, perform the movements of swimming, and march in military fashion, marking time. Ordinarily now he walks with great strides with arms extended, striking the ground with a cane at every third step. Visual field normal. Later on he had nervous crises, globus, throbbing of the temples and loss of consciousness followed by weeping.

OBS. XXII.—(Féré,[17]) Man, 39. Sister hysterical. Had convulsions in infancy. Walked at the age of three. Night terrors, enuresis. Always weak in the legs, a poor runner. At the age of twenty-four had painful cramps in the right leg, caused by emotion. At twenty-six had general convulsions with clouding of consciousness and later complete unconsciousness. Vertigo. Paræsthesia right leg. Two years later, cramps and weakness of the legs. Right leg

smaller, left hand stronger. Sensibility slightly diminished in right thigh. He can make all movements with good strength when in bed, even with his eyes shut. On standing the legs flex beneath him. He can walk on his knees, get up and sit down and make the motions of walking if supported. Condition has lasted twelve years.

OBS. XXIII.—(Helfer.[20]) A girl had erysipelas and parotitis after influenza, with hallucinations of smell, long continued vomiting and weakness. She could move her limbs in bed, but she could not walk or stand.

OBS. XXIV.—(Pitres.[30]) Boy, 10½. Profound anger. Some days later convulsive attacks with contractures, photophobia, delirium. Incoherent, talked in negro dialect and used foul language. He was isolated and recovered. A month later he became unable to walk. He could move his legs in bed with much strength and the muscular sense was normal. If he stood up the legs flexed under him at once. He could stand on his knees or go on all fours. If he was suspended he could use his legs perfectly. If his feet rested on the floor he had pain in the knees and an approximation of the articular surface of the knees caused acute pain. There were islets of anæsthesia in the arms and shoulders. No sign of joint disease. Field of vision normal, sensation, except as stated above, normal. No hysterogenous zones. Cure by hydrotherapy.

OBS. XXV.—(Pitres.[30]) Boy, 8½. Always well. Had a slight stomatitis, after which he said he could not stand, but he could move the legs with good strength. He recovered in two months. Two months later the trouble reappeared after a reprimand. He could stand a moment then the legs gave out. He took ten or twelve steps and then fell or came near falling. Contraction of the visual field. No other symptoms.

OBS. XXVI.—(Pitres.[30]) Boy, 11. Rheumatism in mother's family. Father epileptic. In March, 1888, he had pain in the right thigh with swelling of the gland. The swelling disappeared, but the pain persisted. He could not bear the right foot on the ground. Then the pain passed to the left leg, which became powerless and he could not bear that foot on the ground. The trouble persisted till July. All movements were possible and painless when lying down. He was sent to the hospital but got well on the journey. In October he began to have pain in the knees, back and hips, and the abasia returned. All movements were again possible and painless in bed. He could walk on his knees or on all fours, he could stand and mark time,

but on trying to walk he would hold himself up on his toes, making short steps, three or four inches long, jumping like a magpie. If suspended he could move the legs freely. No anæsthesia.

OBS. XXVII.—(Salemi Pace.[33]) Woman, 27. Father rheumatic; mother convulsions in childhood. Rheumatic. In 1885 had rheumatism, paresis of the legs, inability to walk for seven months. Sudden cure. In 1888 after a walk she felt unusual weight in the legs, walked with fatigue, and the knees gave way. Went to bed and the next day felt well, but on trying to rise the legs flexed under her. In bed she could move her legs perfectly. Some lumbar pain. Hyperæsthetic spot in spine. Hyperæsthesia in left leg. Sensation normal. Knee-jerks normal. Left leg somewhat weaker. Sensation of cold in legs somewhat exaggerated; temperature in legs slightly reduced. Hyperæsthesia and weakness five days later transferred to right leg, then three days later transferred again to left, and other transfers followed. Recovery in a month.

OBS. XXVIII.—(Cahen,[8] from El Siglo Medico.) Boy, 7. Slight febrile disturbance. A week later, after exposure to cold intense pain in back of neck. No signs of meningitis. He convalesced, but refused to get up, and it was found he could not stand. He moved the legs perfectly in bed, the sensation, reflexes and nutrition were normal. There were no pains. Six months later the condition was unchanged.

OBS. XXIX.—(Cahen,[8] from Gillet.) Boy, 6. Father epileptic, father's aunt nervous. Slight rachitis, intellect slightly impaired. Slight febrile attack. Sudden fright after which he could not walk. He could stand without support, but it was hard to get his feet from the ground; he dragged them, spread them and oscillated from side to side. No contractures; muscular strength, coördination and sensibility normal. Pharyngeal reflex normal. Electrical sensibility diminished. Knee-jerk diminished on right. Treated by faradism. Ten days later the current caused great pain; the right knee-jerk became normal. He gained rapidly, but a month later after an attack of pulmonary congestion he relapsed.

OBS. XXX.—(Cohen,[8] from Vallet.) Woman, 27. Neurotic. In 1883 miscarriage, which was followed by malaise and attacks of abasia, yawning and a feeling of exhaustion. Three weeks later unable to walk and did not recover power for three months. In November, 1884, relapse after sore throat. In June, 1885, relapse after migraine. In April and November, 1885, February, April, October, 1886,

repeated relapses, one being associated with pregnancy. In April, 1887, new attack, cured by Bernheim by hypnotic suggestion In December another attack, cured less readily by suggestion. In June, 1888, another attack where suggestion acted less efficiently and some weakness persisted. In July another attack lasting eight days. In November, 1888, had new attack with malaise, fatigue, pain in the left side of head and left blepharospasm. Suggestion cured this and made her walk a little. The attacks come on suddenly or gradually and most commonly after an attack of migraine; the onset is attended with malaise, yawning, faintness, dimness of vision and giddiness and the attack ends with weeping. At times she has cramps in the abdomen. Hypnotism is now far less effective. She can stand for a few minutes, then she trembles. The tremble is worse when the eyes are shut and there is then incoördination. She cannot move one foot before the other or stand on one leg. She can walk on her knees, on all fours or hitch along sitting in a chair. She has trembling of the legs in bed and of the arms when grasping. Knee-jerks exaggerated, sensation normal. Ovarian tenderness, left. Twitchings on going to sleep. Blurred vision. Recovered in January, 1889. In February, a new attack with ptosis of left eye, recovered in spring. Light attack at end of year and in October, 1890.

OBS. XXXI.—(Cahen,[34] from Vallet.) Woman, 48. Gout, migraine and deafness in mother's family. Attacks of torticollis, two fractures of fibula. In 1887 gave up active life, had vertigo, malaise and diarrhœa. About this time third fracture of fibula followed by lassitude and increased fatigue on walking. In May, 1888, after emotional excitement, transitory recurrent incoördination in walking, which in August became permanent abasia. She starts off well, then the feet are thrown out, come down hard like a tabetic; she cannot advance, the movements are exaggerated, the body is thrown backwards, but she never falls. She can jump and raise the legs in bed. No tremor, knee-jerks normal, œdema of legs, vague pain in calves, no anæsthesia. Hyperæsthetic joints, normal visual field. Red sweat in axillæ. Poor sleep. Bruised feeling (courobature).

OBS. XXXII.—(Seglas and Sollier.[35]) Woman, 43. Insane after childbirth, Can neither stand nor walk, but throws the legs about as a tabetic. In bed movements normal. Muscular sense absent, tactile sense diminished. Delusions of persecution, hallucinations, confusion. Husband spiritualist, induced his delusions on her.

OBS. XXXIII.—(Möbius.[29]) Young woman, could not walk or stand.

OBS. XXXIV.—(Möbius.[29]) Girl, 10. After influenza could not stand. Brought in on her mother's back, clinging to her mother with her legs. In bed motion, sensation and reflexes normal. On feet legs gave way. Recovered in four weeks.

OBS. XXXV.—(Eulenburg.[16]) Woman, 18. Neurotic heredity. Anæmic at thirteen. Mental overwork. Two years later exophthalmic goitre developed, with exophthalmos, goitre, palpitation, vaso-motor disturbances and amenorrhœa. Weakness, lack of energy, sleeplessness, depression. Great loss of flesh. Right ventricle dilated; chlorosis. Diminished resistance to galvanism. Some improvement under treatment. Later trouble in legs. Legs flexed under her when she tried to stand or walk and she felt pain in them. All movements could be performed in bed. Tibial nerves slightly sensitive. Sensation, motion, reflexes, normal. Before this attack she had had a sort of agoraphobia, fear of crossing bridges. Recovery after two painful treatments with the faradic brush.

OBS. XXXVI.—(Kusneszow.[21]) Woman, 26. Headache, irritability, anxious for years. Uncertain gait for eight years. For six years unable to walk or stand. Increased irritability. False hearing. All movements possible in bed, but she sunk at once to the floor on trying to walk. Knee-jerks and sensibility normal. Hysterical; headache, globus, neuralgias, hyperæsthesia. Gradual recovery in a year and a half. Attempt at suicide, after which there was again some trouble in walking.

OBS. XXXVII.—(Thyssen.[25]) Girl, 11. For three years had epilepsy and difficulty in walking. A year later, after typhoid, walking became impossible. She walked with pronounced titubation, but without vertigo. She could hop or go on all fours perfectly. No motor trouble, no incoördination and no anæsthesia in the legs while sitting. After a fit limitation of the visual field and improvement in walking.

OBS. XXXVIII.—(Thyssen.[25]) Girl, 12½. Neuropathic family. Vertigo, vomiting, headache, spinal hyperæsthesia in 1887. In 1888 disordered movements of legs. Convulsive crisis with cephalic aura and tendency to contracture. In October loss of consciousness as soon as her back is removed from the back of a chair or she is put on her legs. As soon as her back is supported she returns to consciousness. She can move her legs perfectly with the eyes open,

but not with them shut. She can swim and get about in a wheeled chair with the aid of her legs. Painful hysterogenous points. Gradual recovery by the aid of douches and isolation.

OBS. XXXIX.—(Thyssen.[37]) Women, 19. Neuropathic family. Attacks of weakness. Legs weak for six months, and now she cannot stand alone. Choreic movements for a week. Flexion and extension of legs well resisted. Left side a little weaker. If leaning back in a chair cannot rise or sit up straight without using her arms. Cannot stand without support, oscillating from side to side and from front to back. She rises on her toes and makes various contradictory movements. Drags the right foot in walking.

OBS. XL.—(Thyssen,[37] Brissaud.[6]) Man, 26. Four years at Tonquin. In October, 1889, had lumbar cramps. Pain in the lumbar region, paresis and dysæsthesia in legs, cramps in legs, exaggerated knee-jerks, epileptoid trepidation, incontinence. Walking became difficult and he jumped in trying to walk. In February, 1890, walking became impossible and he rebounded on his toes. Anæsthetic to the knees. In March he did not walk at all; in May he was much better. In January, 1890, he had spasmodic trembling in bed, produced by extension of the legs or even by uncovering them. Later all movements became possible in bed, but the tremor comes on when trying to walk. (Thyssen and Cahen consider this a case of abasia, but Brissaud thinks it is different from true abasia).

OBS. XLI.—(Henoch.[21]) Boy, 7. Masturbated for two years. Enuresis. Sleepless. For a fortnight unable to walk, cannot sit, stand or walk without support. Staggers, complains of vertigo and has marked ataxia. The symptoms increase on closing the eyes. In bed all movements are possible, but less strong. Sensibility intact, but plantar reflex diminished. Anæmic, emaciated. Hard to control evacuations. In a month cured by tepid baths with cold douches and prevention of masturbation. [Henoch speaks of having seen several cases of hysterical paralysis in children where they could not walk, but could move the limbs in bed, and quotes two cases where the legs were paretic, with inability to walk, but he does not state whether in these two cases, the legs could be moved when lying down].

OBS. XLII.—(Hammond.[19]) Woman. Old Pott's disease. Later she became neurasthenic and had aphonia. While in bed with neurasthenia she could move her legs perfectly. Now in walking she advances the left leg and

draws the right one after her. If she tries to advance the right leg or stand on it she revolves to the right and falls. If this be prevented she makes a profound salaam or flexes the thigh on the trunk. No paralysis. Knee-jerks normal. Slight loss of muscular sense in right leg. Sensibility otherwise normal; visual field normal. She has similar trouble if she tries to go on all fours. It requires a greater mental effort to move the right leg in bed.

OBS. XLIII.—(Hughes.[22]) Woman, 32. Always delicate. Rheumatic, neurasthenic. Spinal and peripheral neuralgic pains. Cardialgia. Transient delusions. While in bed can move limbs perfectly, but she cannot stand without support, or put feet forward while standing. She can put feet forward while sitting. No anæsthesia. No signs of hysteria.

OBS. XLIV.—(Souza-Leite.[36]) Negress, 38. Convulsions in childhood. Rheumatism. In June, 1887, saw a woman in convulsions which caused her to have malaise. In July she had abdominal pain in a car, and in getting out to walk her legs felt wooden and trembled. Paræsthesia in feet. Relief on sitting. That night she had hallucinations. Three days later sense of position diminished, ovarian tenderness, right plantar reflex increased. Dyschromatopsia. Sensibility slightly diminished in legs. On rising legs tremble as in spinal epilepsy, but less if the foot is flat on the floor. Knee-jerk increased, greater on left. In walking she puts a cane in front of her, rises and falls, flexing and extending legs at the knee and rising on heels in rhythmical oscillations. Well in a week.

OBS. XLV.—(Souza-Leite.[36]) Girl, 12. Neuropathic family. Poor sleeper. After a slight febrile attack (measles) she grew forgetful and committed unusual faults. The legs felt heavy and crawly. The arm twitched and she twisted herself about. She cannot walk alone and would fall without support. Back hyperæsthetic; left ovarian tenderness; diminished pharyngeal reflex; hallucinations of sight; convulsions. On trying to walk she flexes and extends the legs. Both these cases occurred in an epidemic of chorea.

OBS. XLVI.—(Ladame.[25]) Man, 54. Much exposure, malaria and yellow fever. Twenty-five years ago after a venereal sore, sudden seizure, vertigo and distress, pallor, feeling of impending death. Some days later a similar attack in which he could not walk or cry out. Gradual recovery with much distress on first trying to walk, but he soon was able to keep on with a long march. Four years

later a new attack with pallor. Some days later attack with inability to walk and severe neuralgia in the heel. For years could walk only a short distance without an attack. In them he feels a shock. Feels perfectly well when sitting or lying down. Loses consciousness in them if on horseback. Pains in the head and back. His hand grows tired if he writes. Vision good. Sensibility normal. Knee-jerks normal. Romberg's symptom absent. No weakness or ataxia. No hyperæsthesia. Sudden attack after walking in which he turns pale, raises his arms and sits down. If he tries to walk further his feet tremble and cannot be detached from the floor.

OBS. XLVII.—(Binswanger.[3]) Man, 55. Neurotic taint. Syphilis and overwork. In 1883 after a hearty meal had a feeling of weakness, vertigo and distress, and could not stand erect for a few minutes. Six months later he had an attack with vertigo and a feeling of inability to stand or walk, and increased sensitiveness to sounds. In 1885 he had other attacks with distress, tremor, tinnitus. The feet clung to the ground and he had much cardiac distress. A crowd or an extended space caused much distress. He was neurasthenic and syphilophobic. He has repeated attacks with frightful unrest and cardiac distress. Arteries rigid. Knee-jerks exaggerated. No ataxia, strength good. Sensation normal, visual field slightly contracted. If he is put in the middle of a room his head trembles, he looks restless and anxious, puts his hand to his occiput and rushes for a support. He has similar disturbance in sitting. Weight in head, numb feeling in feet. No vertigo. On walking slowly he has pressure in the occiput, cardiac distress and an idea he can go no further. He stands still or turns and hurries home with long quick strides, a stiff swaying gait, with his head sunk downwards.

OBS. XLVIII.—(Binswanger.[3]) Man, 58. Brother nervous. Always nervous. Overwork. One morning, having been well the day before, he woke suddenly with intense anxiety and a feeling of heaviness in the head. He jumped out of bed and felt blinded; he staggered as if drunk, rushed to the window and had a feeling of complete loss of power and approaching death. He was perfectly conscious. The next day he was well, but afterwards he had spells of feeling uncertain in walking and standing. He staggered and heard people call him drunk. He thought of death and became irritable. He could not walk from fear. New impressions increased the trouble. He felt best on his back. Exertion, conversation, etc., were painful. Knee-jerks ex-

aggerated. Sensation, visual field, ocular movements normal. No ataxia or Romberg's symptom. Sleep poor; digestion slow. Partial recovery in six weeks.

OBS. XLIX.—(Binswanger.[3]) Man, 35. Mother neuropathic. Dreamy, effeminate. Overwork. Power for intellectual work grew weak and he became excitable and had a sense of weight in the head. During a short walk he fell, complained of headache, had a rapid pulse and dim vision, could not speak or get up. He had a feeling of annihilation and impending death. This lasted fifteen minutes. Afterwards he had headache and gloomy ideas. When he tries to walk he has a sudden trembling and a feeling of distress and he would fall without support. He has titubation and intellectual weakness. On his back he feels well and makes all movements without tremor or ataxia. On sitting or standing he has great distress. He walks more easily in the dark. He recovered after a year's treatment.

OBS. L.—(Seglas.[34]) Woman, 40. Overwork. Sudden attack two years before of pain and weakness in the leg with a feeling as of falling if she tried to walk. Later headache, loss of appetite, indigestion. Trouble in walking increased. Vertigo. Trouble in walking especially in the morning and when fasting. Sense of weakness in the legs with oscillation, a feeling as if the ground were rising; objects swam before the eyes and the head felt light. Later on intellectual disturbance, slowness of association, poor memory, failure of attention, irritability. At present she cannot stand or walk and if she is raised she has extreme anguish and epigastric oppression, pallor, sweats, palpitation and a sensation of impending death. This ceases when she is put back in bed. She has this anxiety in sitting if the back be not supported, but she can ride in a carriage easily. Sensibility and muscular sense normal. Reflexes slightly exaggerated. Internal organs normal. Special senses normal. No paralysis or incoördination.

Astasia-abasia has been observed at all ages from six to seventy-five; nineteen cases were under twenty, twenty-five over; twenty-five cases were in men, twenty-five in women. In eighteen cases there was a distinct neuropathic heredity, in nine cases no heredity taint could be elicited. In twenty-one cases it was associated with hysteria, in three with chorea, in two with epilepsy, in four with intention psychoses and in one each with dementia, confusional insan-

ity and exophthalmic goitre. Many of the cases were also neurasthenic.

It must, as I have said, be regarded not as a disease, but as a symptom. As a symptom what are its relations, and what is its pathogenesis?

Binswanger regards it, apparently, as allied to the intention psychoses, agoraphobia, claustrophobia, etc. His own cases, three in number, are certainly not typical cases of astasia-abasia as described by Blocq and others. In his cases there has been the intense anxiety, cardiac distress and inability to stand or walk consequent upon that distress. The patient is profoundly conscious of his trouble and his impotence is due to fear and secondary to it. Ladame's and Seglas's cases resemble those of Binswanger. Eulenburg's case shows a certain transition, in that the patient had previously had agoraphobia, although the abasic trouble was not attended with any anxiety. In forty-five cases there was no anxiety or distress and the patient was not dominated by any morbid terror. It is therefore manifestly incorrect to base a hypothesis upon a symptom exhibited only by a small percentage of the cases, and it is by no means clear whether we are justified in classifying Binswanger's, Ladame's and Seglas's cases as genuine astasia-abasia.

Various French writers, noting its frequent occurrence in hysterical subjects, are disposed to regard it as *prima facie* a symptom of hysteria. This seems to me unwarranted. In twenty-one cases the patients had also well-marked hysteria, but in seventeen others evidence of hysteria was absent, and it would be an utter begging of the question to say that those cases were hysterical because they had astasia-abasia.

Walking and standing erect are acquired faculties, acquired slowly after long practice. The first attempts are made as distinct conscious voluntary processes, every movement being a separate act of volition, begun by a discharge from the highest cortical cells in the motor tract. Gradually the element of conscious volition grows weaker, the movements become almost automatic, and the individ-

ual can talk, read or turn his attention to diverse objects while standing or walking. He may even perform these acts with complete unconsciousness during sleep. It is assumed that these functions have, in the process of education, been assigned to groups of cells in a lower level, below the level of consciousness. The process of walking, moreover, is differentiated from other movements of the legs in somewhat the same fashion that, in highly educated persons, writing is differentiated from other movements of the hand, or, in the sensory sphere, the understanding of speech is differentiated from the understanding or perception of sounds.

In the pure cases of astasia-abasia, and in my own case, the motor apparatus is, at least to a degree, in working order. A definite motor impulse set free as an act of conscious volition in the highest cortical cells can be and is conducted perfectly well to the muscles. The movement is made with strength and accuracy. Hammond suggests that there is in these cases a loss of power in adjusting muscular contractions, and other writers have attributed abasia to a disturbance of muscular sense. This is hardly tenable. In the majority of cases the muscular sense, as tested, has been unaffected, and highly volitional movements are perfectly well performed. My patient, in the height of his spasm, could make the balance step with comparative ease. It is only, or chiefly, when the action of the highest centres is taken off, and the subconscious centres are called into play that abasia becomes manifest. Charcot regards these centres as spinal and hints that abasia may accompany organic spinal disease, but no case as yet has been found to substantiate this hypothesis. Salemi-Pace and Hughes go still further and attributed the trouble to a failure of memory in the spinal cells, a partial spinal amnesia.

Although birds and frogs can stand and walk when their heads are cut off, that is, under the influence of the spinal cord alone, I doubt whether the spinal mechanism in man is sufficient to regulate completely the movements of locomotion. If so, why might we not expect something

more than the simplest reflex movements in cases of transverse lesions high up in the cord? I confess a difficulty in imagining in the comparatively simple nervous mechanism of the cord a morbid process which can affect certain forms of movement and leave the rest free. It seems more plausible that the mechanism affected is higher up.

We can more easily imagine in the brain, below the highest layer of motor cells, cells that act only by conscious volition, a subordinate group of cells in the layer of the subconscious—a part of James's hidden self, perhaps, to be revealed in rare instances, as Janet has done, by the hypnotic trance—which has been educated to preside over the movements of walking. The cells of this group are knit together by many processes of association, and, as a rule, act harmoniously in producing regular coördinated semi-conscious movements.

Now let some process break up the associations between the cells in this subconscious group and the result is obvious. Their harmonious interaction and the production of subconscious movements is lost, but the motor tract is still open for the conduction of voluntary conscious impulses. We have then for example a retention of voluntary movements in the legs,* but a loss of the semi-automatic power of walking.

Such a morbid process would probably be what is termed "dynamic." A gross lesion, such as hæmorrhage, thrombosis or neoplasm, would, in all probability, do more than break up associations. Association tracts, cells and the motor tract itself would probably be destroyed together.

Some support to this hypothesis has been brought out through hypnotism, which puts in evidence many of these subconscious processes and which probably affects the cerebrum rather than the cord. Blocq hypnotized a patient and told her that she no longer *knew how* to walk,

* I say of the legs, but it seems a little doubtful whether in some cases the trouble should not be referred to the trunk muscles, producing inability to maintain the erect posture, and, *ipso facto*, inability to walk. In my own case I was unable to find any disturbance of the trunk muscles, but their condition should be studied with care in subsequent cases.

and ataxic abasia was at once produced. When he said that she *could* not walk, the idea of complete impotence became paramount and paralytic abasia was produced. In astasia-abasia the onset is usually sudden and the trouble has often developed after some emotional disturbance, fright or trauma, or after an illness which has kept the patient in bed. In this way, perhaps, in the field of the unconscious, ideas of inability to walk have developed, which, by a process of suggestion, have acted on the lower cerebral centres for automatic walking and have inhibited them, or broken up the normal association process. Thus astasia-abasia comes under the heading of the association neuroses, to which Prince has lately called attention.

This process is entirely distinct from that of agoraphobia. Here some external factor presents itself to consciousness, the attention to such a phenomenon is morbidly increased and the idea thus aroused dominates the field of consciousness and inhibits voluntary effort. The associations in the subconscious centres are not affected, the action of the highest motor centres is inhibited; the patient has not forgotten how to walk, but an external factor has excited his morbid terrors so that he is afraid to walk. In true insanity of doubt—which differs distinctly from agoraphobia and the allied intention psychoses, the imperative representation is ever-present in consciousness and dominates the entire life. It is independent of the patient's surroundings. The victim of agoraphobia suffers only when in an open space, the victim of the insanity of doubt suffers everywhere and all the time.

As the association neuroses are seen most commonly in neurotic subjects, especially in the hysterical, it is not remarkable that in the majority of cases we find astasia-abasia associated with hysteria or some pronounced neuropathic taint. That does not mean, however, that it is necessarily a symptom of hysteria.

Some psychical shock, as I have said is frequently the exciting cause, affording the occasion for the unconscious auto-suggestion. In my own case, however, I am disposed to attribute the suggestion to more distinctly physical

causes. It seems not improbable that the beginning disability of the legs, due to the organic disease, acted as the suggestion and superinduced upon the organic disability the functional disability of abasia.

I have spoken of the ætiology and psycho-pathology of the trouble, and I would add a few words as to the symptomatology. The manifestations of the disturbances of standing and walking vary materially both in kind and degree, as the cases collected will show. Blocq was the first to attempt to classify these variations, basing his classification on the degree of disturbance, as follows:

Gait { Abolished. / Diminished. / Disturbed.

Grasset adopted a classification based on the kind of disturbance:

Gait { Weakness. / Incoördination. / Cadenced Movements.

Charcot suggested one more satisfactory:

Astasia-Abasia { Paralytic. / Ataxic. { Choreiform. / Trepidant.

Thyssen and Cahen, finally, have suggested the most elaborate classification of all:

Astasia-Abasia { Paroxysmal (Par accès) (Ladame). / Continuous. { Paralytic. / Ataxic. { Choreiform. / Trepidant (Charcot, Grasset). / Saltatory (Brissaud).

This is hardly satisfactory, for it includes too much, and yet does not include enough.

In the first place we must distinguish the cases where abasia is not an association-neurosis, but an intention-psychosis (Binswanger, Ladame). I have already spoken of them and touched upon their pathology. They differ dis-

tinctly from the other cases, and if included at all under the heading of astasia-abasia—which I am not disposed to do—they must be set in a class apart.

This throws out almost the only case of the paroxysmal, intermittent type (abasia par accès, Obs. XLVI.). If this type exists, as my case would indicate, it probably will be found to present the same features as the continuous forms. The *cases*, therefore, may be divided in two ways:

$$\text{Astasia-abasia} \begin{cases} \text{Paroxysmal.} \\ \text{Continuous.} \end{cases}$$

$$\text{Astasia-abasia} \begin{cases} \text{With distress (intention-psychosis).} \\ \text{Without distress (association-neurosis).} \end{cases}$$

Either one of these classifications may probably be applied to the other, and the third classification, that of *forms* of astasia-abasia, may be applied to both.

The commonest form of astasia-abasia, present in twenty-six cases, is the paralytic. Here the legs simply give out as the patient attempts to walk, and bend under him as if made of cotton. There is no rigidity, no spasm, no incoördination. In bed, sitting, or even while suspended, the muscular strength is found to be good.

In other cases the motor disturbance is manifested by some form of spasm or ataxia. This was present in some form in twenty-four cases. The commonest variety seen twelve cases is the trepidant, where walking is hindered by contradictory movements which stiffen the legs and consist of a sort of trepidation recalling that of spastic paraplegia.

In ten cases there were sudden flexions of the legs, such as is seen after a sharp blow on the ham-string muscles; the body is nearly thrown down, and the exaggerated and sudden flexions are seen also in the arms. These movements recall those of chorea, and hence Charcot has given to this variety the title of choreiform. In some cases, as in Souza-Leite's, the trouble seems to be associated with a form of epidemic chorea, and Rodrigues describes a choreiform abasia, more closely allied to epidemic rhythmical chorea than to true astasia-abasia, for the ataxic and choreiform movements are present in repose, as not in-

frequent especially in the spring in Bahia and other parts of Northern Brazil.

Whether we must admit into this classification Thyssen's saltatory variety is still doubtful. It rests only on the cases of Erlenmeyer and Brissaud, (Obs. IX, XL.). Of the latter I have already spoken, as more probably saltatory reflex spasm than astasia-abasia. The same may also be said of the former, but in this case the spasm could not be produced by pressure on the soles and it did not come on until after the feet had been on the floor for some seconds; hence its position is doubtful.

The prognosis of astasia-abasia, taken by itself, is usually good. Children almost invariably recover completely. In adults, however, relapses are commoner, and in a few cases, especially in those of advanced years, it seems to be permanent. It never, however, threatens life.

The treatment employed has been various. The best results seem to have been obtained by adopting the usual treatment for hysteria, isolation, feeding, rest and moral control. Hydrotherapy has proved of distinct advantage, and electricity, usually as the static spark or as the induced current, seems to aid the cure. The essential factors seem to be to treat the underlying neuropathic condition in the most approved way and then to break up the morbid association by moral appeals. These must, of course, vary with the individual. In my own case it seemed to me that the substitution of a distinctly volitional for an automatic act in the midst of the paroxysm might so far break the morbid association as to render the normal automatic act again possible. Hence I suggested the balance step. Its success at first was striking and it still proves of some help, but the progress of the underlying disease naturally renders the hope of any lasting relief vain.

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CURRENTS OF HIGH INTENSITY IN CANCER OF THE UTERUS.

Dr. Wernitz, of Odessa, relates four cases of cancer of the uterus treated by means of galvanism, with favorable results.

The results obtained were only palliative, but very satisfactory. Currents of from 100 to 200 milliampères were employed, according to the tolerance of the patient. The large indifferent electrode was placed upon the abdomen, while the active electrode, in the form of a ball, was placed directly in contact with the tumor. Treatment of eight or ten minutes were given every day or every two days.

He also uses a platinum needle, which he introduces into the neoplasm and connects with the negative pole of the battery. As to results, the secretions and the tendency to hæmorrhage have diminished.

The pains have been so far relieved, that patients who could never get along without opiates have been able to discard them entirely.

The results are such as to stimulate the employment of this agent in uterine cancer. W. F. R.