monstrate the existence of methyl-hydantoin in the urine, and thus sums up the result of his investigations.

"Our knowledge, therefore, of the destination of sarcosine in the organism may be formulated as follows. By far the greatest part is excreted unchanged; a smaller portion, one-fifth to one-sixth, is transformed into the uramido-acid we have been discussing, or rather into its anhydride (methyl-hydantoin), and a smaller portion is oxidised into methyl urea."

Sarcosine, then, undergoes little change in passing through the system, but a portion is converted into methyl-hydantoin. If, therefore, when glycocine is absorbed from the alimentary canal, it does not undergo the normal change, we may reasonably suppose that, when brought in contact with ammonium cyanate, it will be acted upon in the same way as sarcosine, and a portion transformed into hydantoin, and thus we have the first step towards the formation of uric acid. Circulating in the system, the hydantoin may, under certain conditions, so affect the central nervous system, and thereby the innervation of the kidneys, that, when it arrives at these organs, condensation of two molecules of ammonium cyanate may take place with elimination of ammonia, forming biuret, and thus be conjugated with hydantoin; or this condensation may take place independently of the nervous system. Condensation of the cyanates readily takes place, as is shown by the action of acetic acid on potassium cyanate, forming potassium cyanurate and potassium acetate.

Such a view, I think, meets all the difficulties which have been raised with regard to previous theories as to the formation of uric acid—difficulties which have been very clearly set forth by Dr. Garrod, in his Lumsian Lectures. Nor is it difficult to understand that if, as I have endeavoured to prove, the final conjugation of two soluble bodies takes place in the kidney forming the very slightly soluble ammonium urate (1—2400), a portion may not be excreted, but remain in the blood—overflow, as it were, and so pass on into the circulation; a result which certainly happens when the ureters are ligatured. The ammonium salt would then, meeting with the soda in the blood, be converted into sodium urate, the form in which it is deposited about gouty joints.

The appearance, then, of uric acid in the secretion is the result primarily of the non-transformation or metabolism of glycocine into uræa—whether that glycocine be derived from the bile poured out into the duodenum, or formed elsewhere in the body.

I now come to the question of the abnormal formation of uric acid in the human system. Just as in diabetes, the essential fault lies in the inability of the system, either in the liver, or it may be elsewhere, to effect the metabolism of glucose, which then passes into the circulation and is discharged by the kidneys, so, in gout or gravel, the imperfect metabolism of glycocine is the primary and essential defect. Unchanged, it passes from the alimentary canal, or elsewhere, into the liver; there, under the action of the gland, it is conjugated with uræa resulting from the metabolism of the other amido-bodies, leucine, etc., and is converted into hydantoin; it then passes on to the kidneys to be combined with other molecules of uræa or biuret, forming ammonium urate, a portion of which overflows into the circulation, and is converted into sodium urate. It is not difficult to understand that, in persons who are addicted to the pleasures of the table, who are fond of port, and who take little exercise, the liver should become "sluggish;" that the gland-cells should, from overwork, become inactive or destroyed; or the terminations of the nerves should, from excessive stimulus, become somewhat paralysed, and the gland in some measure like the submaxillary after the injection of atropine. The result would be the imperfect performance of its function, and the non-metabolism of glycocine. But it is not every toper and gourmand who develops gout, nor is every gouty man necessarily a toper or a gourmand. Further, it is certain that uric acid is also present in excess in the blood under other pathological conditions which have no connection with arthritic mischief. How are these facts to be explained?

MEDICAL MAGISTRATES.—The Lord Chancellor for Ireland, on the recommendation of Viscount Monek, has appointed Dr. Lambert Hepenstal Ormsby, to the Commission of the Peace for the county Dublin.—Dr. Griffin, of Kilkee, county Clare, has been appointed a Justice of the Peace for that county.—Drs. Anderson and Weir have been appointed Justices of the Peace for the county Down.—Dr. Frederick H. Daly, of Amhurst Road, Hackney Downs, has been appointed a Justice of the Peace for the county of Middlesex, by the Lord Chancellor, on the recommendation of Lord Enfield, Lord Lieutenant of the county.

THE GULSTONIAN LECTURES, ON SPASM IN CHRONIC NERVE-DISEASE.

*Delivered at the Royal College of Physicians of London,
March, 1886.*

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LECTURE III.

(Concluded from page 638.)

2. *Spasm produced in a Reflex Manner by Disease of Afferent Nerves.*—There is scarcely a more difficult subject in the whole range of nerve-pathology than that of reflex spasm. Cases of general convulsions, as well as of local spasm, are so frequently explained in this way, and are in so many instances quite insufficiently supported by scientific evidence, that one cannot help coming to the conclusion that many cases are accepted as of reflex origin, which, at any rate, carry very little conviction with them. Some reasons can be adduced for the difficulties which beset the diagnosis. Many of these cases occur where some sensory nerve is the seat of severe pain; or where a part is diseased which is painful to move. A good example of the former class is spasm of the muscles of the face, accompanying facial neuralgia; and, of the latter, rigidity in joint-disease. But the most ordinary method of expressing pain is, by some overaction of the facial muscles; while the ordinary way of preventing pain in joint-disease is by keeping the joint still, and opposing attempts at movement, by contraction of muscles which produce an opposite effect. This is so natural a contrivance, not only in man but in lower animals, that it must be looked upon as almost an involuntary act. Reflex muscular acts are in very different degrees capable of being controlled or modified by the will; and even if it be allowed that the latter plays a part in the limitation of the movements of a diseased joint, this does not prove that the act is not partly involuntary and reflex. In many cases, it is very difficult to form a definite conclusion with regard to this question.

No one who observes the great variety in the degree of reflex muscular contraction, produced by similar stimuli applied to different individuals, can be surprised at finding evidence that persistent spasm occasionally occurs as the product of a reflex act; or that a stimulus, which produces no motor result in one person, gives rise to definite muscular contractions in another, not equally healthy. Thus, in hemiplegia, accompanied by descending sclerosis in the lateral columns, deep reflexes are much more brisk than they were before the hemiplegia occurred; and contractures, Brissaud says, may be suddenly increased by comparatively slight injuries. If this increased reflex excitability be due to the hyperphysiological activity of the spinal centres, which have been loosed from cerebral control, similar disorders of nerve-centres rather than of nerve-fibres are probably the most fertile causes of reflex spasm. Hence, it is scarcely too much to say that the injury, or disease, which supplies the stimulus to the sensory nerve in such cases, though apparently the principal agent, is really so in many instances only from a particular point of view. That is to say, a mine exists in the patient which has been accidentally exploded by a stimulus applied to a nerve in connection with it; but, had the same nerve been connected with a healthy and stable centre, no spasm would have ensued.

In speaking, therefore, of the present division of the subject, namely, spasm produced in a reflex manner by disease of afferent nerves, we can scarcely deal with it apart from the next division, namely, spasm produced by disease of ganglionic cells. For it is questionable how far stimuli applied to afferent nerves in chronic disease would produce muscular spasm if the centres were healthy. I am not aware that there is evidence to show that gross or demonstrable disease of ganglionic cells produces spasm. But impalpable disorders do; such, for instance, as those resulting from their separation from the cerebral centres. Non-demonstrable disorders, however, are what we presume to be the basis of so-called functional spasms; and to these we shall revert presently.

Reflex spasm no doubt occurs, but how frequently it does so, or how far the afferent or efferent nerves, or the nerve-centres, take the leading part in its production, are points which can scarcely be estimated. As good an instance as I know of reflex spasm is recorded by Mr. Clutton

in the *St. Thomas's Hospital Reports*, vol. x, p. 64. A boy, aged 14, had been bitten in the face by a dog eighteen months previously. The spot had been painless until a month before his appearance at the hospital; but, since that time, he had suffered from constant shooting pains in the neck, which always started from the scar. At the same time that the pain was felt, the angle of the mouth was drawn outwards, and the skin of the neck was wrinkled by the action of the platysma; the whole side of the face and neck blushed, and then became bathed in perspiration. This succession of symptoms recurred every time the scar was pinched. Croton-chloral-hydrate in five-grain doses twice a day soon cured the affection.

Weir Mitchell, in his work on *Injuries of Nerves*, gives instances of reflex muscular spasm; and this condition is often referred to intestinal, uterine, or other irritation, but often without sufficient reason.

Charcot has written a good deal in support of the view that various affections of joints may give rise to rigidity of muscles in a reflex way; and it certainly is very difficult to explain them otherwise. The following case appears to me to be one of the kind referred to.

Case of Persistent Muscular Spasm due to Joint-Disease.—F. W., aged 16, was an out-patient at St. Thomas's Hospital, under the care of Mr. Clutton, in February, 1884. I saw the case with him, and the following is the clinical record which he took while she was under his treatment, and which he has kindly put at my disposal. The patient was sent to him by Mr. Mercers, of the Bath Mineral Water Hospital, with the following history. During May, 1883, she had had rheumatic fever, and the joints chiefly affected were the wrists and ankles. For three months the wrists were placed in splints, and in the autumn of the same year she was sent to Bath with perfectly stiff fingers and wrists. Passive movement was employed, which made the fingers pliable, but which left the wrists as stiff as ever. The accompanying illustrations (Figs. 44 and 45) show the condition which existed when the case came under Mr. Clutton's notice. The photograph was taken by Mr. Charles West. Both hands were in the same condition. On attempting to move the wrist-joint, the flexors and extensors alike resisted, and could not be overcome. Taking them by surprise produced no better results. When the patient was placed under ether, the muscles yielded to attempts at flexion and extension, but the rigidity did not entirely disappear. There were but few adhesions in the joint, and these readily gave way under the anæsthetic, but left the state of affairs practically the same as before. That is to say, the wrist could be flexed or extended, but, on removing the force, the hand again assumed the position shown in the illustration. When the wrist was extended, the fingers always became flexed, and when it was flexed, they became extended. The patient could voluntarily extend or flex the phalanges, and could separate the fingers. The thenar muscles also acted normally. The wrist was rigid, and could not be moved in either direction, and there was slight superextension at the metacarpophalangeal joints, which was easily overcome voluntarily when the patient flexed the fingers. Dr. Kilner tested the electric condition of the nerves and muscles, and found it normal.

Let me here briefly refer to a class of spasmodic affections which appear to have the same explanation as tendon-reflexes. Whether explicable as a reflex or as a purely local phenomenon, contraction of muscles occurs when they are put on the stretch by their antagonists; and this has the effect of controlling and steadying movements which might otherwise be jerky and uncertain. When the influence of the lateral columns of the cord is removed, the spinal centres, in which reflex acts are produced in relation with parts below the level of the lesion, are brought into a state of excessive activity, and stretching of a tendon produces an abnormally sudden and extensive contraction. This is very probably the cause of the tremors in the direction of movement seen in cases of disseminated sclerosis. But it is probable that some cases may have a peripheral origin. A medical friend of mine informed me that he once took a vehicle, in order to drive to a house where he was going to stay for a while. The driver put him down two and a half miles from his destination, and drove off. The medical man had rather a heavy bag, and, being unable to get another conveyance, he carried it himself. For four days after this walk he suffered from pretty constant contractions of the triceps muscle of his right arm, whenever he flexed the latter. In this instance, the muscle had been what we call strained, the tendon had been unduly pulled upon, and, when the arm was flexed, the slight increase of tension made the triceps contract. Such a case represents a very small departure from the normal, but suggests an explanation of more troublesome affections; such an one, for instance, as the following.

Case of Persistent Rhythmical Contraction of the Palmaris Longus, due to Injury.—On July 7th, 1878, I saw a girl, aged 19, who had had

her wrist bent backwards and sprained, five years previously. The parts injured had swelled, and she had been obliged to carry her arm in a sling for some weeks. Ever since that time she had suffered without intermission from twitchings of the arm and palm of the hand. On examination, I found that the general power of the arm was unimpaired, but that there were spasmodic contractions of the palmaris longus muscle, occurring with perfect regularity ninety times in the minute. The contractions were energetic, and showed the muscle and its attachments very beautifully. In this case, probably the palmaris longus had been stretched, and the peripheral nerves ending in it had been rendered more irritable; the result being that the slight tension of it, which occurs ordinarily in the act of extending the wrist, caused the muscle to contract. This occurring frequently, gave rise to a neuro-muscular habit.

The following is a somewhat similar instance. Mr. Clutton once showed me a girl, aged 10, who had fallen down a week previously and slightly sprained her right wrist, but had done herself no other damage. I found her suffering from an affection which had come on immediately after the fall. Her right arm was in a position midway between pronation and supination, and was the seat of perfectly regular tremors of very small excursion, such as one sees in paralysis agitans, occurring in a direction transverse to the longitudinal axis of the limb.

Other pathological conditions, as well as injuries, may produce these rhythmical spasms. In January, 1886, a girl, aged 14, was in St. Thomas's Hospital, under the care of Dr. Bristowe, for rhythmical contraction of the occipito-frontalis muscle on both sides, which occurred from fifty to sixty times in the minute, and had been going on for some months. The history she gave was that she had had a very bad attack of "erysipelas of the face," commencing on the forehead and spreading downwards. The onset was sudden, and the description she gave of the affection corresponded with that of erysipelas. Roughness and pigmentation of the skin of the forehead could still be seen. As the disease got well, the contraction of the occipito-frontalis muscle supervened, and continued for months without intermission, except during sleep. The original cause of this condition was probably an irritable state of the muscle and tendon owing to inflammation, and an involuntary neuro-muscular habit was soon developed. Although the girl showed no evident hysterical peculiarities, I found that she had marked localised tenderness over those regions which Charcot has found to be unduly sensitive in hysterical subjects. Moreover, information which I subsequently obtained proved that at least one other member of her family was hysterical. For, while the patient was still in the hospital, her sister, aged 11, was admitted under Dr. Bristowe's care, suffering from a similar affection. The latter had been under me as an out-patient for fits, which were evidently hysterical; and, at the early age of eight years, she had had an attack of rhythmical spasm of the occipito-frontalis muscle, which lasted three months, and then got well. When I saw her in the hospital during the second attack, I found that the muscle was contracting pretty regularly 120 times per minute, and this condition was accompanied with sighing, yawning, slight movements of the tongue, partial loss of sensation on the left side, pain in the left hip and knee, and drawing up of the left leg so as to produce distinct shortening.

Other cases of spasm from peripheral injury are more complicated, but appear to be reflex in their origin, whether the pathological stimulus acts upon the nerves which exist in muscles and tendons or upon other afferent paths. The following is an example.

Case of Spasmodic Movements of the Jaw, the Floor of the Mouth, etc., due to Injury.—On November 20th, 1877, a boy, aged 10, came to St. Thomas's Hospital, on account of a peculiar affection which he had had for five days. There was nothing noteworthy in his family or personal history, except that he had suffered from incontinence of urine. Five days before coming to St. Thomas's, he had been in a playground where his schoolfellows were having a game of football. He happened to be in their way, and one of them took him by the back of the neck, and threw him to one side. His neck pained him at the time, but by the following day the pain was gone. In its place, however, was a peculiar affection, consisting of constant movements of the lower jaw and of the floor of the mouth, and they had increased since then. As he stood in front of me, I saw the lower jaw and the floor of the mouth descend in regular and frequently recurring jerks, and when the child's mouth was open it was seen that the tongue was likewise jerked spasmodically downwards and backwards. In addition to this, the larynx descended slightly, and the depressor muscles in the neck could be seen contracting. Each downward movement was accompanied by a sound like the croaking of a frog.

Neither the faradic nor the galvanic current applied from the nape of the neck to the parts below the jaw had the slightest effect on this curious condition. I gave the child some bromide of potassium, and sent him away. Nine months after that I saw him, and he was nearly, but not quite well.

As I have already said, it is a matter for speculation how far such causes would give rise to spasm, if the nerve-centres were in a normal condition. What is really developed in these cases is an involuntary neuro-muscular habit. We are all familiar with the ankle-clonus which is set up sometimes in healthy people when sitting with the toes on the ground, and the heels a little raised. If we start the clonus voluntarily, or if it have occurred several times in succession involuntarily, it may be difficult to stop it by an effort of the will, unless the position of the legs be altered. This is probably a physiological representative of certain cases of muscular spasm which assume pathological dimensions.

I have now come to the end of the remarks I have to make upon the first two divisions of spasmodic affections in connection with chronic nerve-disease, those, namely, which result from pathological changes in the cerebral motor mechanisms, on the one hand, and the spinal motor mechanisms, on the other. The third division, and that the most difficult of all, remains still to be dealt with, and is co-extensive with what are called functional spasms. As I have already pointed out, the first two divisions should, and we may hope some day will, cover the whole field of spasmodic affections; and, by placing functional diseases under a separate heading, we publicly confess our ignorance, and proclaim that there is a very long list of spasmodic affections which have been carefully observed without success, so far as their pathology is concerned. In lectures professing to deal, however perfunctorily, with spasm, it would be impossible to pass by in silence the class referred to. It remains, therefore, for me to consider how far these functional diseases are capable of explanation or reasonable classification.

III.—FUNCTIONAL SPASMS.

The result of our consideration of those diseases which produce many varieties of spasm, and which are represented by gross lesions of the nervous system, is, that a very large proportion must be put down to the credit of the cerebral system of fibres, or pyramidal tract. The spinal mechanisms are less prolific causes of these affections; but those spasmodic conditions which do occur in connection with them have been considered under the heads: (1) peripheral motor nerves; (2) afferent nerves; (3) centres. It is only reasonable to suppose that, if functional spasms resemble those which result from gross lesions, they may admit a similar explanation. In nerves and muscles which during life have been the seat of motor disorders, we may find no naked-eye or microscopic pathological changes after death. But clinical observation shows that, so far as function is concerned, marked changes have occurred. Now we know that healthy function must depend upon fine molecular rearrangements, which are at present a closed book to us; and we conclude that, if molecular changes form the basis of healthy function, they form that of pathological conditions also. If this be so, it is legitimate to argue that the molecular changes in question might affect the same parts which are the usual seats of the grosser lesions, and consequently give rise to similar abnormalities of function; and then the anatomical classification which has already been attempted in the case of tangible disease, might serve also for those cases which are the result of impalpable alterations in nerve and muscle. I propose, therefore, to consider shortly certain forms of functional spasm, and I shall use the same divisions for classification which I have already used for grosser diseases.

1. Do functional disorders of the cerebral motor mechanism, or pyramidal tract, produce spasmodic conditions at all resembling those resulting from gross disease? They do. In hysteria, not only does hemiplegia occur, but hemispasm, both fixed and mobile, is not uncommon. There is this difference between the hysterical conditions and those which are seen in gross disease; the leg is affected most, the arm less, and the face not at all. But the facts observed in gross disease, where the leg is usually less affected than the arm, are probably to be explained mainly by the fact that the vessels which bleed most frequently, and interrupt the functions of the internal capsule, are those which are situated towards the anterior part of its motor division, that part, in fact, which contains the fibres controlling the face and arm. But apart from these points, the spasmodic contractions of the leg and arm are similar in the two conditions. In functional diseases, there is simply removal of the voluntary impulses which, in health, travel down the pyramidal tract. The muscles and nerves of the limbs remain practically healthy; and if rigidity be

not too great, the tendon reflexes are increased. Fevers and other exhausting conditions, such, for instance, as that of the patient with anemia to whom I have already referred, may give rise to increased reflexes, and even to clonus and rigidity. Moreover, absence of portions of the motor area of the cortex, such as is occasionally seen congenitally, is accompanied by non-development of the corresponding portion of the pyramidal tract, and gives rise to spastic conditions like those found in hemiplegia. Absence of the voluntary impulses, then, is enough to give rise to the phenomena in question; and certain cases of hysteria have died after long continued contraction of limbs, and the most careful examination with the microscope has failed to disclose any palpable pathological changes. Such a case is recorded by Dr. Bristowe in *Brain* for October, 1885. This case I often saw myself, and, as Dr. Bristowe remarks, "at the end of her two years, she seemed as well in general health as when she first came to the hospital; but she was suffering from headache, sickness, ophthalmoplegia externa, complete anæsthesia of the right side, with rigid paralysis of the arm and leg, and repeated hæmorrhages of both ears." Dr. Hadden made a most complete microscopical examination of the central nervous system in this case, but found no evidences of disease. As we know, however, that mere absence of the voluntary motor impulses along the pyramidal tract is sufficient to give rise to this condition, it is legitimate to conclude that suppression of the functions of this tract is the cause of functional hemispasm. We are still further supported in this view by cases like that just quoted, where there is at the same time clear evidence of suppression of the functions of the sensory area as well.

Clinical and pathological observation has shown that monospasms having similar characters may be produced by certain limited lesions of the motor convolutions; and, after what has already been said, it is not too much to assert that, when spasm limited to a leg or arm occurs in hysteria, it is due to suppression of the functions of the pyramidal tract, which in health controls the action of the affected member. In discussing the question of athetosis and other allied mobile spasms, we arrived at the conclusion that they were due to affections of the same tract of fibres, which were not severe enough to stop the passage of voluntary impulses. Now, similar conditions occur in hysteria, and may probably be explained in the same way. Weir Mitchell, in his work on *Nervous Diseases*, records cases of "hysterical motor ataxy," and of "hysterical athetosis." It appears probable that changes, ending in greater or less functional abeyance of the pyramidal tract, lie at the root of unilateral, and sometimes of bilateral, spasm, both in functional and in gross disease. The only difference is that, in the latter case, we can appreciate, by our present methods of investigation, the alterations which have been wrought, while, in the former, they are hidden from our view.

There is another class of functional spasmodic affections which are hardly represented among the forms of spasm due to gross lesions, and which, I believe, have their origin in suppression of the functions of certain portions of the pyramidal tract. I refer to those conditions which are sometimes termed "professional hyperkineses." They include histrionic spasm, writers' cramp, pianists' cramp, telegraphists' cramp, etc. The striking peculiarity of these affections is that, when a voluntary effort is made to perform the accustomed muscular acts, spasm of the muscles involved occurs, and prevents further efforts. Before this stage of the affection is reached, great fatigue often accompanies endeavours to work. In a considerable number of cases, at any rate, the muscles can be employed in other ways without difficulty. What has occurred is probably that, after long repeated acts of the same kind, that part of the pyramidal tract which is used becomes fatigued, and its functions are partly suppressed, so that a condition of "latent contracture" of the muscles, over the voluntary actions of which it presides, is developed; the lower centres are "let go," as Hughlings Jackson says, and are in a state of "hyperphysiological activity," just as the spinal centres of one side are in hemiplegia accompanied by slight rigidity.

In describing "latent contracture" in connection with hemiplegia from gross disease, Ross says: "The patient may perform all the simple movements of the limb, and probably with undiminished power; but when his attention is specially directed to the movements, as when he wishes to perform any manual operation requiring a little dexterity, the muscles instantly become rigid, the fingers are flexed on the palm, and the deformity, which was present during the period of fixed contracture, reappears." Surely this description suggests the true explanation of professional spasmodic affections, which present symptoms resembling very strikingly those which Ross portrays.

Erb divides the professional hyperkineses into spastic, tremulous, and paralytic. The tremulous cases are perhaps due to similar but slighter paralytic affections of the pyramidal tract, and the condition probably

resembles in kind, though not in degree, paralysis agitans. The pathology of the latter disease is not known, but those cases which one meets with where rigidity of limbs and exaggerated reflexes occur, suggest forcibly degeneration in the pyramidal tract as the physical basis of the affection. The paralytic variety of the professional hyperkineses is probably the result of extension of disease to the motor cells in the anterior cornua of the spinal cord, at any rate in those cases where marked atrophy occurs (see *Brain*, vol. vi, 233, a Case of Sawyers' Cramp, by G. V. Poore, M.D.).

I should remind my hearers, in passing, that the term pyramidal tract, as used here, includes not only fibres, but the motor cells of the cortex in which they originate.

2. We have seen that spasms of muscles resulting from gross disease of the pyramidal tract has its representative among functional diseases; let us now consider that which owes its origin to affections of peripheral motor nerves. Is it likewise represented among functional disorders? Actual gross pathological changes in motor nerves give rise to muscular spasm, as has already been pointed out, in two ways—the one direct, and the other indirect. In the latter case, certain muscles atrophy and disappear, and undergo cicatricial shortening, producing distortion; or else their opponents being left to act in their absence, give rise to abnormal positions of the limbs. Now, one of the striking peculiarities in functional nerve-disease is the absence of such degeneration and destruction of muscles. Hence we should not expect to find any functional spasms which were due to such indirect causes; nor do we, so far as I know. We also came to the conclusion that direct irritation of motor nerves is a very rare originator of spasm; and there is no reason to suppose that it is less rare a cause of functional affections. Nerve-centres seem much more prone to functional disorder than nerve-fibres. I am not sure that the latter condition, originating in centres, may not so affect even peripheral nerves which are connected with them, as to give rise to symptoms which are found in gross disease only when peripheral nerves are involved. Dr. Norris, of Windsor, sent to me, for treatment in the hospital, a girl, aged 16, who had lost the use of her left hand for eighteen months. She was a strong, healthy girl, and free from evident hysterical tendencies. The affection commenced with swelling and blueness of the fingers, such as is seen in chilblains; and the hand was cold and numb. When she came to me, the hand was completely paralysed, and had a swollen puffy look; the skin was paler and smoother than that of the right hand, and the backs of the fingers far less wrinkled. The left hand was much more influenced by external conditions than the right, and rapidly became warm when covered, and cold when exposed. She suddenly recovered after the application of a blister to the wrist, but then lost power in the left leg. The latter also suddenly recovered power. But, although slight alterations, such as those which were observed in this patient, may occasionally occur in peripheral motor nerves in cases of functional disease, I am not aware that they give rise to muscular contraction.

3. Nor is there any proof that chronic spasm is produced by molecular changes of afferent nerves alone. Even in gross disease of an afferent nerve giving rise to reflex spasm, the lesion is often so trivial as to make it very probable that functional disorder of the centres is present as well, and is the main factor in the production of spasm.

When speaking of reflex spasm from gross disease, I pointed out certain cases of what appeared to be rhythmical spasm due to affections of tendons; and I cannot help thinking that this class has frequent representatives in functional diseases. But they are due more to the abnormal condition of the centres than to that of the peripheral nerves. There can be no question that one of the peculiarities of nerve-centres in hysteria is their abnormal irritability; so that slight afferent impressions give rise to muscular acts, which pass with great ease into neuro-muscular habits. Such a condition is well exemplified in cases due to imitation. The patient sees a certain form of muscular spasm, and the idea produces the same in her. For instance, a healthy girl, aged 17, came to my out-patient room complaining of involuntary movements in her right hand and tongue. On examination, it was seen that the thumb was rhythmically adducted, and there was simultaneous flexion of the fingers, most markedly of the index, which moved in such a way that she appeared to be rolling something between it and the thumb. At the same time, she was troubled by being suddenly unable to finish a sentence, owing to the tongue refusing to act properly. The girl worked in a confectioner's shop, where one of her comrades became affected with uncontrollable movements in the left hand and catching in her speech. After a short time, my patient became similarly affected.

The following is another case of suddenly developed neuro-muscular habits, though it originated in a somewhat different way. A healthy

dark-haired girl aged 16, who had never had any illness, and had no evident neurotic peculiarities, went out for a row on the Thames with some friends in the summer of 1885. She rowed without much intermission for five hours, which was to her quite an unaccustomed length of time for such recreation. She used to row every morning at Wandsworth, where she was at school, but only for an hour at a time. About an hour after returning from this long row, her arms began to twitch, and movements continued uninterruptedly, and remained of a precisely similar kind up to December 29th, 1885, when Dr. Bristowe, under whose treatment she had been, very kindly asked me if I should like to see her.

Both arms were the seat of similar and synchronous movements, occurring regularly about one hundred and sixty times a minute. They consisted of rapid elevation and retraction of the arm at the shoulder and of the scapula, partial flexion at the elbow, and slight extension of the wrist; in fact, they bore a very marked resemblance to the movements of the arms and hands in rowing, the extreme rapidity of the "stroke" making up for the limited extent of the movements.

In these rhythmical contractions, it is not at all improbable that the slight stretching of the tendons of the affected muscles which naturally results from the action of their antagonists gives rise to another contraction, and so on, for an indefinite number of times. Such a condition, though much less marked, is sometimes seen when the spinal centres are cut off from connection with the cerebral cortex by gross disease.

A case of caries of the cervical vertebræ was under my care in St. Thomas's Hospital last summer, in which paraplegia, with rigidity, had been present for five years. The legs were always flexed at the knee and hip, but could be extended by continuous gentle traction. As this was being performed, movements of flexion and extension, which were involuntary, occurred. When the tendons of the flexor muscles were stretched, the latter contracted, and, in so doing, they put the extensor tendons on the stretch, and so caused their contraction.

Weir Mitchell has noticed these "alternate spasms" in hysteria, in which, as he remarks, the action of the flexors calls the extensors instantly into play, and this, in turn, summons the flexors into activity. "These spasmodic motions," says he, "were the more curious in the last case I saw, because of the general and profound paresis, which made every volitional effort excessively difficult." Such an absence of voluntary power, in the case of which Weir Mitchell speaks, shows that the functions of the cortical motor centres were in abeyance. Instances of alternating spasms are far from rare in hysteria, and probably owe their presence to excessive irritability of the spinal centres, and the development of a habit through the inter-mediation of tendon-reflexes. "Saltatory spasm" is a good instance of this class of cases.

4. The fourth and final heading, under which we have to compare spasmodic conditions arising in connection with gross nerve-lesions, with functional disorders, is disease of the spinal motor centres. We have already seen that tangible pathological alterations in these cells give rise to paralysis and subsequent atrophy of muscles, but we could quote no facts to show that muscular spasm ever originates from such disease. Those impalpable molecular changes, which result from the anatomical separation of the cerebral motor centres from the spinal centres produce a condition of hyperphysiological activity of the latter, to which many spastic conditions are due. Does abnormal functional activity ever exist primarily in the spinal centres and result in spasm? This is a question which is very difficult to answer. In the case of gross lesions which interrupt the continuity of the fibres of the pyramidal tract, even where no actual spasm has resulted, there exists a condition of excessive excitability of the spinal centres, which gives rise, on the slightest provocation, to rigidity of muscles. An injury, even of a trivial nature, may, in such patients, produce contracture. Exactly the same state of things is found in hysterical subjects; and Charcot, in his lectures on nerve-disease, gives instances of the most trifling injuries suddenly producing spasmodic conditions of limbs in such patients. But we must remember that persons may have those peculiarities of their nervous system which are usually embraced under the term hysteria, without ever presenting striking emotional or other tendencies which are wont to call attention to the existence of the disorder. The first evidence of the latter may be the sudden supervention of spasm from a very slight external injury. If this condition of the spinal centres depends mainly upon the amount of cerebral control exerted over them by the functional activity of the pyramidal tract, it is evident that there must be infinite gradations between the normal and abnormal. Indeed, it is impossible to give any strict definition of health and disease. All we can say is, that

the more cerebral control retires into the background, the more likely are spasmodic conditions to come to the fore.

It is quite impossible to answer the question, whether hyperexcitability of the spinal centres occurs in the absence of diminution of cerebral influence to account for it; but it probably does. We know that strychnine, in certain doses, produces an excitable condition of the spinal centres, which Charcot has likened to that which is seen in "latent contracture" due to gross lesions of the pyramidal tract, and to hysteria. In comparing strychninism with the latter, Weir Mitchell says (*Lectures on Diseases of the Nervous System, especially in Women*, page 100), "Perhaps I shall, in a measure, clear your minds as to the nature of what I mean by functional spasms if I recall to you the influence of strychnine in large doses, such as you have seen given here many times. You will remember that, in certain spinal maladies, such as those of syphilitic birth, it is my habit first to give iodides in heavy doses, then to suspend them for a time, and to give strychnine up to the limit of physiological endurance, that is to say, until I cause an approach to spasm. When, for example, you give hypodermically the one-fifth to the one-eighth of a grain daily, the patient will have little or no annoyance, if you are careful to insist that he remain at absolute rest in bed for two hours after each injection. If there be any tendency to spastic twitchings of the muscles, the will is competent to control them, unless—and this is the point I would make—the patient attempts to exercise. Should he do this, the effort results at once in irregular movements of an inco-ordinate character, and in slight or more grave spasms of the muscles employed. While at rest, there is no obvious trouble, but voluntary movement occasions spasms, which are the offspring of the poison. They are, in a word, functional spasms, and would not be seen at all, with limited use of strychnine, were it not for the efforts of voluntary action." If, then, a substance circulating in the blood can directly produce this condition of the spinal centres, in which they may almost be said to be watching their opportunity to produce muscular contraction, it is not improbable that molecular alterations may arise from other causes, and produce a similar excitable condition of these centres. We know, however, little, if anything, about the changes which act thus. What are called phantom tumours of the abdomen, and sometimes of other parts, may have some such origin; Weir Mitchell records one, in which all the abdominal muscles had remained violently contracted for a year. But whether this be the explanation of such cases or no, the hyperphysiological activity, due to lack of cerebral control, which exists in the spinal centres of some subjects, is probably the principal factor in a very large number of tonic and clonic functional spasms.

In the comparison which has been attempted between the results of gross disease on the one hand, and molecular or so-called functional alterations on the other, the pyramidal tract stands prominently forth as the great offender in the production of muscular spasm. Its action is indirect, it is true, as it only looses the reins of the spinal centres, which it should keep well in hand. Still, it rules the situation. Spasm rarely ensues directly from injury or disease of peripheral motor nerves; and, although it frequently results from reflex causes, it is very likely that the spinal centres are, in a considerable proportion of cases, more at fault than the afferent nerves. It may be thought somewhat curious that, in speaking of diminution of cerebral control, the voluntary motor tract should alone have been referred to as liable to suffer from depression of its functions. But the only reason why it has been thus signalled out is, that these lectures have dealt with motor phenomena. The sensory functions of the brain, suffer, too, in a very striking manner, so that complete hemianæsthesia is far from a rare occurrence in hysteria, the loss of function being evident in the realm of the special senses, as well as in that which has to do with common sensation. And although at first sight these functional defects appear to be confined to one side, careful investigations show that they are not. For, in cases of hemianæsthesia, both fields of vision are contracted, although that on the anæsthetic side is the more affected. Again, in cases where the loss of function appears to be confined to the sensory area, a careful comparison of the muscular power on the two sides will show that this is not so. In a case of the kind which I lately saw, I found that on the hemianæsthetic side the patient could only reach 45 on the dynamometer scale, although this was the right side, while with the left she reached 50. In such hysterical patients, it is probable that nerve-power is deficient in all parts, and on both sides of the brain, although this deficiency is more marked on one side than the other, and in some portions of that side than in others. Considering how late the pyramidal tract develops in man, and what a high pitch of evolution it represents, it is not to be wondered at if it is one of the first parts to suffer in the process of dissolution.

If we are right in considering deficient nerve-power, especially in the brain, as the condition which is at the root of functional nerve disease, the term *neurasthenia*, a product of modern times, would be a more suitable general title for such disorders, than the older term, *hysteria*. When we review the history of nerve-disease in families, we cannot but conclude that people are born with very different physical bases, both in their nervous and in their other systems. And no line can be drawn between those who are normally stable and enduring under unfavourable circumstances, and those who are liable to lose equilibrium. Some patients would remain perfectly well had they not accidentally met with some catastrophe; and then they fall into the category of hysterical patients. And yet there are many of their friends and acquaintances who are as liable to functional disorder as they are, and who yet may never have their weakness brought to light, owing to their not having been exposed to a sufficiently severe trial. Even small portions of nerve-centres may become hysterical, if I may be allowed to say so, as, for instance, in writers' cramp, pianists' cramp, and other professional hyperkineses. All that we know of this matter is, that functional nerve-disease, even in the case of apparent exceptions, such as spasm, is due to deficiency of nerve-power. How the condition is actually brought about we cannot tell. Some think the centres are starved by contraction of their nutrient vessels. It seems to me more likely that the nerve-centres are exhausted by their own action. Thus, sudden emotional excitement may bring on hysterical phenomena, indicating nerve-exhaustion, such as hemi-anæsthesia, paralysis, or spasm. It is important to remember, too, that exhaustion of one area seems to affect the whole system. This may indicate that the various differentiated centres draw nerve-force from some common supply. Considering the enormous amount of cells which are found in the cerebral grey matter, many without evident connections, is it going too far to suppose that a large number of these are factories for the production of nervous energy, which is necessary for the continuous and regular action of the highest centres, and which flows in increasing quantities towards those centres which are from time to time brought into action? Constant supply would mean constant power in all centres; and as the latter are all bound together so as to produce a physiological equilibrium, constant power would involve constant control of one by the other. Where the store of constantly produced force (represented, of course, by structural peculiarities) is by inheritance small, or where it has been exhausted by unnatural calls upon it, there would be insufficient to supply all the centres; and those to which the nerve-paths are most freely open by habit would command it, while others would be starved, and loss of equilibrium, or the natural control of one centre by the healthy action of others, would be the inevitable result. If we do not suppose any such reservoir of nerve-energy, how can we explain the exhaustion of all, by the excessive action of some centres? Why is a person who has had excessive mental work less able for the time to do active bodily work? and why, when bodily fatigued, are we less ready to do intellectual work?

But I have gone, I fear, too far into a region of speculation, which it is hardly legitimate for me to have even entered with so small an equipment of facts. I shall, therefore, say no more, but bring these lectures to a conclusion by offering an apology for having chosen a subject which is so extensive in range, and at the same time so obscure and little understood, that my treatment of it must fall far short of that which is expected on occasions like the present.

ROYAL FREE HOSPITAL, GRAY'S-INN ROAD.—The Report read at the Forty-eighth Annual Meeting of the Governors of the Royal Free Hospital, Gray's Inn Road, stated that the receipts of the Institution from all sources during the year had amounted to £7,556. A handsome brass cot, with a suitable inscription, had been placed in the Milne Ward, as also a brass tablet had been erected in the large room used for meetings, in commemoration of Dr. Samuel Rabbeth, the senior resident medical officer of the institution during the previous year, who sacrificed his life in endeavouring to save that of a child upon whom he was performing the operation of tracheotomy, by sucking the tube in order to clear the child's throat. The expense of the cot was defrayed out of a fund raised by public subscription, and the tablet by subscription at the hospital. The total expenditure for the year had been £11,085, and to meet this the committee had been compelled to sell out £5,000 of consols. The number of in-patients admitted during the year had been 1,728, and the daily average in the wards 135. The number of out-patients treated was 21,853. Several changes had taken place in the medical staff, and some slight structural alterations had also been effected.