there have been several (none under my own care), have almost always been young patients, and generally, I think, for strabismus operations. Then, again, for young infants, in whom chloroform seems to be exceptionally safe, I think I shall still continue to prefer it. For such operations as hare-lip, for instance, chloroform is more convenient.

At Moorfields we have for long been in the habit of using methylene. It is certainly more rapid in its action than chloroform, but I have never been sufficiently impressed, either with its superior convenience or its additional safety, to be induced to employ it either in private practice or here. I have seen several cases in which alarming symptoms occurred during its use, but never an actual death.

Our one reason for abandoning chloroform is its danger. If we could get rid of this, it would certainly stand without rival. My own experience of chloroform has been very considerable, and for a great variety of cases. I have had but a single death in my own practice, but I have been the witness of several in the practice of others. I have also had amongst my own patients several very alarming occurrences, in which it was with the greatest difficulty that death was prevented. In the case of a girl at Reigate, in whom apparent death occurred during excision of the knee, we had to perform artificial respiration for at least ten minutes before any signs of reanimation occurred. In another case at the London Hospital, in an amputation of the thigh, the period during which I thought the patient dead was almost as long. In both these instances, death-like pallor of face and absence of pulse (cardiac syncope) were the alarming symptoms; and in both artificial respiration, irritation of the surface, and the use of brandy enemata, were the means resorted to. In addition to them, I have had many cases in which the pulse failed temporarily, and in which we had to begin artificial respiration in order to ward off the more dangerous condition. During the last four or five years, I think I have had fewer alarming cases of this kind than formerly; and I attribute this in part to the skill and care of those who have given chloroform for me, and in part to the fact that I have been very particular as to the administration of stimulants (brandy) before using the anæsthetic. I have never of late used chloroform without knowing that my patient had fasted for some hours, and had taken a dose of brandy within the last half-hour. These seem to me the two principal points; but, if you insist upon the one (the fasting), it is additionally important that you attend to the other. In a fasting and exhausted state of the system, we have the condition in which cardiac syncope is the most likely to occur.

The case which I have alluded to as the only one in which I have had death actually occur, was that of a man much exhausted by a month's suppuration from a compound fracture with his elbow-joint. I saw that he was very nervous when he was put on the operating-table, and I ought to have given him brandy before proceeding. His heart failed within five minutes of the commencement of the inhalation, and before I had begun the operation.* If your patient is unusually feeble, I do not think it well to do an operation early in the morning—i.e., before the first meal. Indeed, in all cases in which an early appointment is made, it is best to tell the patient to take some fluid food, coffee, beef-tea, or milk, about two hours before the time fixed. Under most other circumstances I should insist upon a four hours' fast, with the double object of preventing the danger which attends vomiting during the anæsthesia, and the inconvenience and suffering which it sometimes produces afterwards. As regards the quantity of brandy to be given, that should be in proportion to the patient's age and habits, bearing in mind that it is better to give too much than too little. It is given in order to act as what is called "a diffusible stimulant;" which, I suppose, means that it is to keep the blood-vessels well open, and thus facilitate and sustain the circulation. Ether is an agent of a similar class, and hence its freedom from risk as compared with chloroform. Opium also acts in the same way, and I have no doubt that the administration of a good dose of opium, or the inhalation of ether for a few minutes before using chloroform would, either of them, make the latter much more safe.

There is just one other little matter to which I wish to advert. You may have observed that in cases in which the anæsthetic is used in order that the actual cautery may be applied to some part of the face, as we not unfrequently do for lupus and rodent carcinoma, I always give chloroform. Sometimes we give ether for a few minutes first, but we always take care that the vapour has had time to pass away before the cautery is brought near to the face. The reason for this is obvious; the vapour of ether is very inflammable, and a little thoughtlessness might easily lead to a terrible accident. It may be well also, for the same reason, to avoid employing ether by candle-light.

* This case occurred about ten years ago, and was published at the time.

CLINICAL LECTURE ON CASES OF PARALYSIS AGITANS.

By C. HANDFIELD JONES, M.B. Cantab., F.R.S.,
Physician to St. Mary's Hospital.

[Concluded from page 223 of last number.]

THE first point for our consideration is, to what recognised place in a nosology the foregoing cases are to be referred. The prominent symptom in all was tremor, persistent and continuous. Paralysis could hardly be said to be present in any, except perhaps the last. The question then arises, Does such tremor as existed in these cases sufficiently characterise them as instances of paralysis agitans? Eulenberg, one of the best recent authorities, would, I suppose, say no; for in some passages he questions the reality of recorded instances where there was no evident paralysis, not even excepting Dr. Reynolds's case of paralytic tremor. In others, however, he speaks of tremor as the initial and essential symptom of the disease, and of paralysis as a secondary one, which ensues after a longer or shorter time. Thus he appears to consider that the diagnosis of paralysis agitans cannot be made in the absence of some amount of paralysis; and that, therefore, in the early period of the disease, we are for the most part unable to recognise it. He regards, if I understand him aright, simple though continuous tremor as a distinct disease. This, I think, is over-refining. Dr. Sanders, in his excellent article in Reynolds's *System of Medicine*, vol. ii, p. 198, takes a sounder position. He distinguishes *simple* or *passive* tremor, occurring only during a voluntary action, from *spasmodic* or *active* tremor, taking place even during rest and when the parts are supported. All tremors he admits to be signs of weakness in the nerve-centres; but the *passive* tremor he regards as evidently due to want of power only, while the *active* implies some irritation in the motor nerve-centres. The latter is characteristic of paralysis agitans, and furnishes the ground of diagnosis. This seems to me, under the circumstance, the best rule to follow. If, indeed, we knew anything positive respecting the pathological change which accompanies the malady, if the results of necropsies had not been so frequently negative, it would be better to be guided by the *post mortem* than by the *ante mortem* phenomena; but, as we are quite in the dark respecting the pathology, we must rely on the symptoms for the present; and, though it may be true that in typical cases there exists evident impairment of motor power, yet the absence of this does not constitute sufficient ground for denying the genuineness of cases and excluding them from this group, which possess its most characteristic features. We might as well refuse to admit a case, otherwise well characterised, to be pneumonia, because of the absence of rusty sputa. The essence of the disorder is clearly not paralysis, but clonic spasm, as we must conclude from this symptom being persistent from the commencement to the close, and giving its special features to the disease. Muscular debility in the later stages undoubtedly becomes, or may become, very great; but even then it is rather the debility of exhaustion and decay, than true paralysis. It is in elderly persons that we find the best marked examples of paralysis agitans; and in them, as Dr. Sanders says, the effects of senile decay (among which muscular weakness figures conspicuously) are necessarily mixed up with the other symptoms. Besides, in bad cases, the continued unrest must tell heavily on the nervous system in the way of exhaustion. It is further to be considered that, when we observe no evident sign of paralysis in a patient, we are scarcely entitled to conclude certainly that nothing of the kind exists. A man may be able to grasp pretty forcibly and walk fairly well; but, unless we had tested by some method the amount and endurance of his muscular power previously to his illness, we cannot positively affirm that they are undiminished. My third case has some bearing on this point. The spasmodic movements in this patient were extremely forcible—more than I could control; yet his force for a continued effort was notably less than mine. His right upper limb was more severely affected than his left. When recovering, he could hold out a weight with the latter steadily for several seconds; but, when he attempted to do the same with the right, tremor came on immediately, and the arm dropped. It was quite clear that tremor coincided with failure of contractile power—that the right arm was, not only less steady, but less strong, than the left. As improvement went on, ability for sustained muscular effort was regained. I think we do not sufficiently estimate the value of endurance—of staying power—as a test of the efficiency of a nervo-muscular apparatus. It is this quality, especially when well developed in the heart, which enables a man to

endure fatigue and withstand and outlast morbid influences; and it is this quality which fails most evidently in a multitude of neurotic disorders.

But, again, there seems to be evidence that the muscular power, at least as regards brief efforts, may be remarkably preserved in paralysis agitans. M. Charcot tells us (*Gazette des Hôpitaux*, April, May, 1869) that, among the inmates of the Salpêtrière suffering from paralysis agitans, though there are difficulty and retardation in the execution of voluntary movements, yet the dynamometer shows that the muscular strength is by no means deficient, being often greater in the limb which trembles most and seems weakest. Trousseau (vol. ii, p. 217) corroborates this statement as regards the early period of the disease. For all these reasons, I accept Dr. Sanders's rule above quoted; and I regard the four histories above given, as well as those contained in my work, as genuine instances of paralysis agitans (*pace* Herr Eulenberg).

Morbid Anatomy.—Not many careful observations have yet been made, but still sufficient to enable us to come to some conclusions. Stoffella, Skoda, and Oppolzer have found well marked sclerosis of the pons Varolii, medulla oblongata, and cord. M. Joffroy (*Gaz. des Hôpitaux*, No. 157, 1871) has published the results of his examination of three aged female patients. He found the central canal of the cord filled up by a cell-growth, probably derived from the lining epithelium. In two of his cases there were nuclear formation in the vicinity of the ependyma, pigmentation of the nerve-cells, especially in Clarke's *columna vesiculosa*, and an abundant accumulation of amyloid corpuscles. In the third case there was some sclerosis in the medulla oblongata near the pons Varolii. In the present case, there was no lesion in the medulla or the pons. Dr. Murchison (*Path. Trans.*, vol. xxii, p. 25) records the case of a male aged 71, who had had paralysis agitans twelve years, and died of typhus. Dr. Cayley found (1) thickening of the cortical layer of connective tissue of the cord, and increase of its nuclei; (2) irregular nucleated tracts and patches of connective tissue in the dorsal and cervical regions of the cord, mostly situated near the posterior roots of the nerves (the reticulum of the cord, especially in the neighbourhood of these patches, was much thickened); (3) the spinal canal was transformed into an oval tract of leucocytes, which occupied also the site of the surrounding "substantia cinerea gelatinosa". There were also some other changes of a recent kind, probably due to the typhus. Eulenberg cites three necropsies made by Ordenstein. In one of these, where the malady had lasted thirty years, there was found only atrophy (rarefaction) of the nerve-tubes; in a second, softening of both cerebral peduncles, and some loss of substance in the pons; in a third, nothing. He states also that Th. Simon examined four cases at Hamburg with a negative result. It is to be wished that we had some examinations of the bodies of younger patients; and it would also be well if we had some of aged persons who had *not* suffered from any symptoms of the malady. It might be the case, that more or less sclerosis occurred without producing any symptoms. Accepting the above statements, the negative as well as the positive, we can but conclude that the symptoms of paralysis agitans may occur with and without discoverable lesion, which, therefore, is not essential. Joffroy's observation, that sclerosis of the pons and medulla is not necessarily present in the most typical paralysis agitans, accords with the experience of Larcher and others that this change induces a set of symptoms differing materially from those of paralysis agitans, especially in the early occurrence of paralysis, and the non-spasmodic character of the tremor.

The *relations* of paralysis agitans, defined as above, are very interesting. Thus it has clearly an intimate affinity with mercurial tremor—so close that we may correctly regard the latter as a *toxic* form of the malady. Most neuroses are capable of being generated in this way, as well as by simple failure or defect of a nervous centre, and by remote irritation; so that we should expect to find the same hold good in the case of this disorder, regarding it as a neurosis. Another near relative of the malady which we are considering is chorea, which might almost be called the paralysis agitans of the young, and is often attended with very great muscular weakness or even actual paralysis. Dr. Sanders describes (*Edin. Med. Journal*, 1869, p. 743) a case of paralysis agitans, with characteristic and well marked symptoms, in a boy aged 16; and remarks that the disorder, being really spasmodic in its chief phenomena, is more nearly allied to chorea than to paralysis. Dr. Mac-lachlan mentions a case of four or five years' standing in a male aged 60, where paralysis agitans was associated with chorea so intimately that it was hard to say which set of symptoms preponderated. With tetanus it agrees in being a disorder of the spinal motor centres, and perhaps of their superior expansions, in being often independent of definite lesion, in being idiopathic, toxic, or reflex, as regards causation, in being non-febrile, and in the exemption of the intellectual centres; but differs in the spasm being clonic and not tonic, in

being rarely excited by a lesion (reflex irritation), and in being non-transitory. Neuralgia resembles paralysis agitans in the absence of definite lesion, in its frequent association with debility, in its being intensified by causes of exhaustion, and especially in its inveteracy in advanced life. Between paralysis agitans and the disorders which depend on sclerosis of the cord, its posterior columns or upper expansion in the medulla oblongata or pons, there seems to be symptomatically no great resemblance; but organically some affinity may be traced, inasmuch as in some instances the same kind of change is found in paralysis agitans. Admitting, as I am inclined to do, that sclerosis may be occasionally the chief cause of paralysis agitans, the variety of symptoms produced by this degeneration in conditions clinically different may depend on differences in its localisation.

Etiology.—What is true of chorea and of many other diseases—viz., that their effective cause varies greatly in different instances—holds true, I believe, in paralysis agitans. The actual condition of the nerve-cells which causes the clonic spasm is no doubt the same in different cases, but it may be generated by different agents; and to bear this in mind may be of material consequence. Debility of nerve-centres, however induced—by mercury, by falls, by violent muscular or excessive mental exertion, by violent emotions, by venereal excesses, by ague—seems to have a potent influence in producing paralysis agitans, just as it has in causing other neuroses. Old age, therefore, when the vital powers are all gradually failing, may well be, as we find it, the period of life especially favourable to its invasion. No special cause may be requisite, more than that certain motor centres should deteriorate in their nutrition after a special fashion. The peculiar molecular condition of the altered nerve-cells may be indiscernible; but its effect on function is, that the centre, instead of becoming simply weak, becomes unduly excitable. Change of this kind is by no means infrequent in the sensory centres from impaired nutrition, and gives rise to the various forms of hyperæsthesia and paræsthesia. This view of course does not account for the peculiarities of paralysis agitans—does not explain to us why it differs from senile tremor or chorea; but it is correct, I believe, as far as it goes. It indicates the kind of treatment requisite, and may be supplemented by the very reasonable hypothesis that change, which is in the main deteriorative, may vary in its *manières d'être*, to borrow Trousseau's phrase; and that such variations may, though slight, materially modify the symptoms. There are, in fact, cases which the best observers regard as forms of paralysis agitans, but which present, nevertheless, notable diversity in the symptoms, such as are related by Trousseau (vol. ii, p. 215).

Now, proceeding on this view—that the motor nerve-cells in paralysis agitans are in an enfeebled condition, probably due to defective nutrition, which, however, is itself the result of failing power—we next observe that the loss of functional capacity shows itself in a special way. In the construction of a motor nerve-centre, it is requisite to create an apparatus which shall be always ready to evolve force, but which shall not do so spontaneously—not without the application of a stimulus of some kind or other, physical or mental. Now, the peculiarity of nerve-cells is, that they possess these two qualities: they prepare material which, by undergoing oxidation or in some other way, generates force; and yet they can prevent this material from so acting, although blood is circulating all round it charged with oxygen. The cells of other organs, as of the kidney, the testes, and the liver, seem to be acting constantly: the only condition requisite for their action is, that they should receive a due supply of blood. But the motor nerve-cells may lie for hours ready to act energetically, and yet scarce act at all. Now, in states of spasm this property of the nerve-cell is lost or much impaired. It still generates force; it does so continuously, but not in obedience to appropriate stimuli. As long as blood is supplied, it works on blindly, draining away the nerve-force from other centres, till at last the heart is exhausted and circulation ceases. If it should be thought that the action of the nerve-cells of the corpora striata and anterior spinal horns is regulated by those of the convulsions, which keep the former quiescent when this is desirable, yet the question recurs, How is the quiescence or regulation of these superior nerve-cells obtained? How is it that, while fully supplied with healthy blood, they can refrain or not from functioning? If we must refer this to a constant operation of the mind, how is it that the muscles are at rest during sleep and insensibility from narcosis or injury? Most of us, however, I suppose, hold that quiescence of the intellectual and volitional centres coincides with the same state of the motor centres, and *vice versa*; so that more need not be said on this point. In states of cerebral excitement, mania, and delirium, this faculty of quiescence is lost in the cells of the convulsions, and in great measure in those of the motor centres too; and here also the pathological change is commonly induced by causes which depress and exhaust nervous energy.

As regards *treatment*, Cases II and III show that, when the disorder

is recent, there is good hope of a successful result; while Case IV corroborates the general experience of the inefficacy of remedies in the aged when the disease has become inveterate. The same influence of age is remarkably apparent in the neuralgia of herpes zoster, which is so slight in the young, and so terrible and persistent in the old. The improvement obtained by succus conii in Case III was very striking, and was also well marked in Case II. Galvanism in Cases I and II appeared useful, but not highly so. Electricity is probably more beneficial in some cases than in others. Dr. Butlin (*Practitioner*, November 1869) records the case of a servant more than eighty years old, who was cured completely by electro-magnetising the affected arm every second day a few times. The disorder had existed a long time before treatment was commenced, and was severe enough to disable her from carrying trays, etc. One case which I have recorded in my work was speedily cured by the same means. The general tenor of experience in this and in kindred disorders is to the effect, (1) that the main indication is to conserve and support the failing power of the nervous centres affected; (2) that this is best accomplished by remedies drawn from the class of sedatives, or by the milder tonics. Henbane, conium, chloral, subcutaneous opiates, bromide of potassium, belladonna, hypophosphites or phosphorus, cod-liver oil, carbonate of iron, and sulphuret of potassium baths, with electricity in one or other of its three forms, appear to me the most hopeful remedies. But steady persistence in appropriate treatment is doubtless essential, and the want of this may account for many failures. Trousseau's adage should be borne in mind, "À longue maladie, longue traitement."

M. Eulenberg uses some very complimentary expressions in accusing me of confounding cases of simple tremor and chorea infantilis with paralysis agitans. I believe I have justified myself sufficiently with regard to the first; and, as to the second, I cannot see any ground for it. There are none of the cases contained in my chapter on Paralysis Agitans which can be regarded as chorea. I think he has quite failed to disprove my point, that there are different affections comprehended under the name of paralysis agitans.

THERAPEUTIC MEMORANDA.

PARACENTESIS ABDOMINIS.

IN the JOURNAL of February 15th (page 185), it is stated that Dr. Lyons described seven cases of paracentesis abdominis, in which he performed the operation with the effect of prolonging life. One woman suffering from cirrhosis had been tapped no fewer than thirty-six times, from fourteen to sixteen quarts being drawn off at each time, and ultimately the ascites remained stationary. Another patient was apparently moribund, when sixteen quarts of fluid were evacuated with immediate relief. All the members who spoke at the meeting when the above remarks were made, were in favour of early tapping.

I have only had very limited experience; but from the result of my cases I am also in favour of early tapping. In my first case, the dropsy had been early detected, and was operated upon before the strength of the patient had been exhausted. I tapped this patient three times during the year 1864, with the effect of greatly relieving the suffering, and apparently prolonging life. My second case was in all respects very similar; it was one of ascites from cirrhosis; early operation gave great relief, and I believe it prolonged life. The only other case on which I have operated was in February last. A lady had been under the care of a practical physician for some months suffering from cirrhosis. I was requested to attend her quite at the end of her illness, and I found her a good deal exhausted from pain caused by a much distended abdomen. The physician being related to the patient I did not insist upon the necessity (indeed, the time to do much good had passed) for operative interference; but, being requested, I tapped on the 13th of the month, and the patient died on the 26th purely from exhaustion. Early tapping, so far as I know, is a course not generally adopted, nor advocated in text-books, if one may judge from the following quotations.

"In this form of dropsy, however, the peritoneum becomes more impaired in its power to absorb the fluid than in most of the others; the fluid is, therefore, seldom reduced, and the patient generally requires the last imperfect resource of art—namely, tapping, or paracentesis."—Dr. Aitken.

"But if there be urgent dyspnoea or other general distress from the dropsy, the fluid ought to be removed by tapping; a proceeding, however, which does not afford satisfactory results."—Dr. Tanner.

MARSHALL MONCKTON, Hurstpierpoint.

ABSTRACT OF A CLINICAL LECTURE

ON

RENAL DISEASE IN CALCULOUS PATIENTS, AND ITS INFLUENCE ON THE CHOICE OF OPERATION.

Delivered at University College Hospital, February 28th, 1873.

By SIR HENRY THOMPSON, M.B.,

Surgeon-Extraordinary to His Majesty the King of the Belgians; Surgeon to University College Hospital; etc.

THE patient, a naval pensioner, aged 60, thin and careworn, had suffered from symptoms of stone for more than three years. In the course of 1872, he had been admitted into a metropolitan hospital, when the stone was crushed several times. After this, the patient continued pretty comfortable for about three months; he then rapidly relapsed, and, when he came under Sir Henry Thompson's care, the old man was in a most miserable condition. He could not hold his urine more than half an hour, even at night, and, as he could only pass it when in the erect position, he was obliged to leave his bed every time, and was greatly reduced by pain and want of sleep. This urine was alkaline, of low sp. gr., contained a large amount of albumen, and an unmistakable granular cast was found under the microscope at the first examination. On sounding him, fragments of phosphatic stone were detected. It was evident that the patient had advanced disease of the kidneys, and that his ultimate fate was settled; still, his principal sufferings were due to the presence of the calculus matter in the bladder, and these could be removed or greatly relieved by lithotripsy.

He was accordingly admitted into the hospital on January 21st; the 24th, Sir Henry removed some *débris*, and repeated the process on the 28th without any unfavourable symptoms: on the contrary, the patient during this time improved in strength, could move about better, and was able to hold his urine for an hour or more at a time. All but a few fragments had in fact been removed when, on February 1st—a cold day—the patient slipped out of the ward and stood for some time in the yard of the hospital smoking. Next day, he had a severe rigor, followed by headache, drowsiness, partial suppression of urine, etc., and, although at first he rallied somewhat under treatment, he never recovered the effects of his unfortunate indiscretion, and died on February 19th of uremia.

Post Mortem Examination.—The external surface of the kidneys was granular; the capsules were opaque and adherent: on section, the cortical layer was thin, mottled with patches of yellow degeneration, and studded here and there with small abscesses; the pyramids were congested. The pelves of the kidneys and the ureters were dilated, and contained puriform matter. The muscular coat of the bladder was hypertrophied; the mucous membrane was much congested, dark, thickened, opaque, and ulcerated in places; the so-called "middle lobe" of the prostate was much enlarged, forming a regular bar across the neck of the bladder; in the deep hollow behind this were a few small and soft fragments of stone, weighing in all twelve grains.

In commenting on this case, Sir Henry Thompson said:—"The question we have to consider to-day, gentlemen, is this—If stone in the bladder be complicated by the presence of chronic renal disease, what should be done? When are we justified in operating? and which operation should we choose? "Chronic renal disease" is a wide term; and, in order to answer the question better, I will consider the chief forms of kidney-disease separately.

We may at once dispose of malignant disease: if this be so advanced that a satisfactory diagnosis is possible, any operation is clearly useless. Chronic Bright's disease, again, is a loose term, and includes several varieties; the two that chiefly concern us now are the large white smooth kidney, and the granular contracted kidney: the so-called amyloid disease is rare. There is no difficulty in the diagnosis of Bright's disease, even when complicated by the presence of stone; the low specific gravity of the urine, the presence of casts and of an amount of albumen out of proportion to the amount of pus present decide the nature of the case at once. Next, there is what may be called the calculous kidney. You will often meet with men who are frequently, or almost continuously, passing crystals of uric acid or small calculi. This never goes on for any length of time without damage to the kidney; on microscopic examination, you will always find blood in the urine. They are often stout red-faced, healthy-looking rustics; but if such a man come to you with a stone