to all the irregularities left by the fracture, is much lighter in colour than the substance of the calculi. (See Fig. 1.)

Fig. 1.

The calculi are composed of uric acid and urate of ammonia intimately mixed. The outer light portion, which has the look of a phosphatic deposit, is, in fact, urate of ammonia. When this is dissolved in liquor potassæ, it leaves a gelatinous matrix and a great abundance of spores and mycelium, strongly resembling those of microsporum furfur. The central portion of the original calculus is wanting in all the fragments. Under the microscope, the mass of the calculus is found to consist of compact crystalline matter with a radial fibrillation. No separate crystals are to be obtained.

The cause of the original fracture of the calculi appears to me to have been an expansion of the central portion, or some expansion at the centre, acting as the exploding powder in a shell. There is evidence that, for some time preceding the appearance of the fragments, there was enough disturbance of the general health to render it probable that the chemical constitution of the urine was altered. There is also evidence in the nature of the secondary deposit that the state of the urine was different after the fracture from the state accompanying the formation of the calculi. I infer that such change was accompanied with a tendency to alkalinity, and that as a result a chemical influence was exerted upon the calculi, tending to cause swelling of any mucoid matters which might enter into their composition; that the centre which has disappeared was different in some respects from the circumference which remains; and that this difference determined an unequal swelling of the centre, resulting in disruption of the spheres.

That this was the case rather than a disintegration or falling to pieces through general failure of cohesion, is an idea supported by the perfection of the individual segments and the compactness of their structure. The regular form of the fragments opposes the idea of fracture having arisen through collisions among calculi. Such conditions would produce irregular chipping rather than radial fissure and segmentation. An inspection of the fragments resulting from lithotripsy will show the essential nature of the difference. It is not uncommon, indeed, to find calculi that have been cut in two and preserved, as commonly found in museums, fissured radially round the centre. This is, to my mind, usually an acquired, and not an originally present, condition, not existing when the calculi were in the bladder, due to the unequal drying of the centre and circumference. The condition, however, indicates that under exposure to certain circumstances of altered moisture, radial splitting, beginning from the centre, may occur in calculi; and what I suggest is, that a similar sort of process led to the segmentation in this case under circumstances which determined swelling instead of shrinking of the nucleus.

The spontaneous fracture of vesical calculi is apparently not a common event. The late Mr. Southam, however, published in the BRITISH MEDICAL JOURNAL two cases observed by himself and one of Mr. Luke, quoting also a case of Mr. Liston and giving a drawing of a calculus from the Dupuytren Museum of Paris. The paper was illustrated by excellent drawings, which have been reproduced in Dr. Beale's work on the *Urine*. The calculi in Mr. Southam's two cases were larger than those now exhibited, and the hypothesis of rupture by internally developed gas was advanced. The fragments were only two in each case, and were less regular segments of spheres. One was composed of lithic acid with a coating of hard oxalate of lime, and the fragments, after disruption, had become coated with fresh phosphate. It is, therefore, quite possible that changes in the composition of the urine played a part in producing disruption. The composition of Mr. Southam's second calculus is not stated. In Mr. Luke's case, the calculus was composed of triple phosphate, and there was no history of violence. In Mr. Liston's case, the calculus is supposed to have broken up suddenly under the influence of a violent bodily shock. The fragments were numerous, and the result was fatal. No analysis is given. In the last case only the drawing is given, showing a fragment separated by fissures, but not detached. The fissures do not reach the nucleus, and the fragment is held in place by fresh enveloping deposit.

Mr. Coulson has also recorded a case in which a sort of natural lithotripsy occurred, two or more calculi being crushed together and broken by one another as nuts may be broken in the hand. In such cases, the calculi are mostly of loose consistence, and the fragments are irregular and not segmented. (*Pathological Transactions*, vol. xv, p. 143.)

I have been fortunate enough to find in the Museum of the Royal College of Surgeons a preparation (Calculi, A 177) very closely resembling my own. It is figured at the end of the catalogue in Plate 1, figs. 6, 7, and 8. There is no history, but the preparation is described as a pisiform calculus (Prout's term) "separated into triangular portions, possibly the result of spontaneous disintegration". The segments are pointed, instead of being, like these, truncate; that is to say, the centre remains. They are composed, according to Mr. Taylor's analysis, of uric acid, and appear to have been coated by fresh material after fracture.

In relation to the analysis of these calculi, I may add a few remarks illustrating the use of the microscope in these processes. After the general facts of organic composition, of the presence of uric acid and ammonia had been determined, some of the central part was chipped and examined under the microscope. The characters of the fragments have been already noted, and it may be observed that the whole of the original portion of the calculus was built up of fine concentric laminæ, perfectly compact, perfectly continuous with one another, and showing a radial crystalline structure. The superficial portion, on the other hand, consisted of spherules imbedded in a clear matrix, the spherules being dense at their centre, less dense and lighter in colour, with marked radial crystalline structure, at their circumference. This is the form taken by urate of ammonia in the presence of colloids. The spheres were slowly disintegrated by hydrochloric acid, and rapidly dissolved by caustic potash, leaving the matrix and the contained fungoid structures.

The central mass, on being powdered and boiled in distilled water, dissolves sparingly, giving a strongly acid solution, from which large very thin flat lancet-ended crystals are thrown down. These are exactly like the crystals which I have shown elsewhere to be produced when uric acid is added in large excess to hot solutions of urate of ammonia. As I believe them to be crystals of a highly acid urate, they will indicate that only a comparatively small relative quantity of ammonia is present. After three or four exhaustions of the powder with boiling distilled water, rectangular plates of pure uric acid are deposited. There is clearly, then, also a large proportion of uncombined uric acid, representing, in fact, more than half the bulk of the calculus. The solution yielded by the surface is neutral. The quantity of material being small, and the material itself comparatively soluble, crystallisation was not effected. No oxalate crystals were obtained in any process.

[To be concluded.]

AN ADDRESS

ON

OPHTHALMOLOGY IN ITS RELATION TO GENERAL MEDICINE.

The Annual Oration delivered before the Medical Society of London.

By J. HUGHLINGS JACKSON, M.D., F.R.C.P.,

Physician to the London Hospital, and to the Hospital for the Epileptic and Paralysed; etc.

III.

THERE are still other valuable deductions to be made from the facts of cases of ocular palsy. In a case of paresis of the right external rectus, when the patient tries hard to turn the lamed eye out, the other eye turns in too much; there is secondary deviation. From the attempt to move the paralysed or weak part, there is a double effect; there is, firstly, failure to move it, and, secondly, overmovement of associated muscles.

Later on, we shall see that there is evidence, both by cases of disease of the brain and by Ferrier's experiments, that there are centres for conjoint movements of both eyes. This principle bears in a most important way on the interpretation of locomotor ataxy and of disorders of co-ordination in general. It has already been applied by Wundt to explain the disorder of co-ordination produced by experiments on animals; but, so far as I know, it has not been yet applied by physicians to explain cases of disorder of co-ordination by disease. I do not, however, maintain that in locomotor ataxy there is solely a motor difficulty; there is sensory defect, and the joints and bones even become diseased. Let us, however, apply the principle so far as it will

go. Generally speaking, the principle is that, when a centre discharges and when one route for the current is stopped, the current, so to speak, overflows in other channels, in those for associated movements.

There are other examples supplied by cases of paralysis illustrating the principle, although the ocular palsy is the clearest. When the flexors are atrophied, an effort to shut the hand is considerable; and then, to use Duchenne's language, a very intense nervous current reaches the extensors, giving rise to exaggerated extension—not to the desired closure, which is, of course, impossible. The flexion here is the analogue of the secondary deviation of the healthy eye in paralysis of one external rectus.

The principle of double effects in paralysis must be considered in all cases of disorder of co-ordination. I think it applies evidently to locomotor ataxy. To call this or any other symptom a disorder of co-ordination is to speak with conventional correctness, but the word co-ordination has such misleading implication that it is scarcely safe to use it. It is frequently used metaphysically, it being implied that there is such a thing as a faculty of co-ordination residing in this or that centre, and that the centre has nothing to do with movements except to regulate them autocratically.

I believe the essential thing in locomotor ataxy is paralysis. This will, no doubt, seem a strange remark. It will, in the first place, be asserted that there is no loss of power. It is quite true that the patient has great power in his legs, can stamp vigorously, and can resist flexion and extension. I only suppose there to be some loss of power to execute, or to execute effectively, the more special movements of walking.

In locomotor ataxy, there is central disease—viz., of the posterior column; hence there is not likely to be paralysis of some muscles, but paresis of complex movements. It is just what happens in cases of disease of the higher centres: we never have from disease of any part of the brain simple paralysis of any one or more ocular muscles, but loss of movement—loss of power to look to one side, for example. Moreover, the pathological change in locomotor ataxy is wasting of nerve-fibres, and this could cause nothing active. A wasting of nerve-fibres—in effect, a loss of them—could not make a man reel; it could only directly cause paralysis of motion or sensation, or of both. It would, I submit, lead indirectly to disorder of co-ordination, as palsy of an ocular motor nerve demonstrably does.

In detail, I think the state of things is this: the first failure in locomotor ataxy is of that movement which is most important in locomotion, of that in which the peroneus longus takes the lead—the movement by which the body is swung over on to the ball of the big toe. In early stages of locomotor ataxy, we see that the patient has a difficulty in, so to speak, biting the ground with the front part of his foot; and we see, also, that there is strong contraction of the tibialis anticus, the antagonist of this muscle—in ophthalmological metaphor, there is secondary deviation of it. The explanation I give (limiting the illustration to these muscles) is, that there is a two-fold effect. Suppose some higher centre, perhaps the cerebellum, energises. It is absolutely certain that there is activity of some centre, and that cannot be made up of wasted nerve-fibres, or the patient could not walk at all. The centre may be the cerebellum, although it matters not, for the principle of the explanation, which centre it is. The current developed cannot, on account of the wasting of fibres in the posterior column, reach the muscle for the most special movement, or cannot reach it in full degree, just as in the ocular palsy it cannot reach the external rectus. But as in the case of the ocular palsy the estimate is by the central discharge, so in locomotor ataxy a strong movement of the peroneus is estimated, although not performed; then, as the analogue of the secondary deviation of the healthy eye, there is over-action of the associated movement, that in which the tibialis anticus takes the lead. In short, there is over-estimation of the intended but underdone peroneal movement, and actual overdoing of the tibialis anticus movement. If this be so, we see that we have no need to invoke a faculty of co-ordination, nor a special co-ordinating centre. We see, too, that locomotor ataxy compares not with such diseases as chorea, but with such as hemiplegia. There is paralysis leading to secondary effects.

Duchenne believes that some cases of flat foot are owing to what he calls functional impotence of the peroneus longus. It seems to me that he gives an essentially similar explanation of the symptoms of this affection as I have given of those of locomotor ataxy. He speaks not only of the impotence of the peroneus longus, but of the reflex contraction of the extensors of the foot—so to speak, secondary deviation of the foot.

So far, we have spoken only of paralysis of ocular muscles from disease of nerve-trunks. The physician often sees cases of palsies of ocular muscles, or rather paralysis of certain ocular movements from

disease of nerve-centres. Let us suppose that the right corpus striatum is extensively damaged, there will be left hemiplegia, and for some hours or days the two eyes turn to the right.* The head often turns in the same direction. The eyes and head turn to the right side for the same reason that the face does. The turning in all three is from the side paralysed, and therefore towards the side of the brain injured. There is no strabismus, for the eyes, although turned to one side, remain parallel. The implied healthy nervous arrangements for this lateral movement of the two eyes have been likened by M. Foville, junior, to the arrangement of reins for driving two horses. What occurs in the lateral deviation is analogous to dropping one rein (the right one, we will suppose); then the heads of the horses (supposing the left rein to be, so to speak, in tone) turn to the left. Here the secondary deviation in paralysis of the external rectus may be illustrated. Using a clumsy mixture of illustrative terms, we may say that when the centre for one rein energises strongly, the current developed cannot reach the head of the horse on the right, as the slip of rein to that side is paralysed; but in the effort to make it move, a strong current passes by the other slip to the head of the horse on the left—that horse's head undergoes strong "secondary" deviation. The lateral deviation shows that after the nerve-fibres of the ocular nerve-trunks have entered the central nervous system (if we may, for convenience, trace them backwards way), they are—partly probably by the intermediation of several centres—redistributed. It shows that far above their points of entrance, or rather exit, the nerve-fibres of the ocular muscles are rearranged in each cerebral hemisphere in complex ways for particular movements of both eyeballs. There is no such thing as paralysis of the muscles supplied by the third nerve, or sixth nerve, from disease above the crus cerebri. But there is clear evidence that the movement for turning the two eyes is represented still higher than the corpus striatum. I have long believed that ocular, and indeed all other movements, are represented in the cerebral convolutions: if they be not, the occurrence of convulsion from disease of them is unintelligible; without admitting such a constitution of ideational centres, and certainly unless ocular movements are therein represented, what is called the physiology of mind is, I think, an impossible science. Hitzig and Ferrier's experiments show, it seems to me, that movements are represented in at least some of the convolutions. For many years I have believed that every part of the nervous system is made up of nothing else than of nervous arrangements representing impressions and movements.

In Hitzig's and Ferrier's experiments the centres are stimulated; in the experiments by disease, of which I have been speaking, there is destruction. Ferrier finds that stimulation in several parts of the cortex cerebri produces movements of the eyes. I have already mentioned that he finds lateral movement of the two eyes from stimulation of what he calls the auditory centre: he finds, too, that irritation of the visual centre—the angular gyrus—produces lateral movements of the two eyes in an upward or downward direction, according to the particular part of the gyrus stimulated. He concludes, and I think fairly, that in both these cases the primary irritation is of sensory fibres representing the auditory and retinal expansions, and that the associated ocular movements result from a propagated irritation to what he calls the special motor centre for ocular movements. This special centre is situated at the hinder part of the superior and middle frontal convolutions. Stimulation on the right side causes the two eyes to turn to the left, and at the same time there is dilatation of the pupils. I have seen him produce this movement, well defined, exactly according to his prediction made, before applying the electrodes. This, to renew an old illustration, is like pulling the rein which goes to the right sides of the two horses' heads. It is just what one so frequently finds in cases of epileptiform convulsions, due to, at any rate occurring with, lesions of the cortex cerebri by disease. Ferrier is able to produce a convulsion by experimentally irritating the surface of the cerebrium, closely like that resulting from the discharge of that part in disease. It is to lesion in some part of what Ferrier calls the special centre for the movements of the eyes that I should attribute the loss of lateral movements in some cases recorded by Priestley Smith. He has recorded cases in which loss of the lateral movement of the two eyes was the sole paralytic symptom. Such a symptom of necessity points away from lesion of nerve-trunks, and to a lesion of nerve-centres: that centre might be the pons Varolii or cerebellum; but the most likely seat of lesion is, I think, the convolitional centre spoken of.† At one time I did not believe that destruction of parts of the cortex produced local paralysis, but Ferrier's experiments on monkeys go contrary to this opinion. It has been recently found by Charcot and

* This was first pointed out by Vulpian and Prevost, Humphry, Hutchinson, Lockhart Clarke, Russell Reynolds, and Broadbent.

† Priestley Smith has just published a paper on Double Vision in the *Birmingham Medical Review*, highly deserving the attention of physicians.

others, exactly in confirmation of Ferrier's experiments on monkeys, that from destructive lesions of parts of the cortex there occur limited palsies, as of the face, of the arm; and as Charcot finds *post mortem* wasting of fibres "descending" from the parts of the cortex destroyed, the cases must, I think, be admitted as conclusive. The most likely explanation of some of Priestley Smith's cases is, that there is a monoplegia, to use the French term, comparing with other monoplegias, such as paralysis of an arm only, or of the face only, as in cases of cortical lesions described by Charcot.

Further, there is clear evidence from Ferrier's experiments, and from other experiments, that the movements of the eyes are represented not only in the cerebrum, but in the cerebellum. From experimental destruction of the right crus cerebelli in some quadrupeds, the right eye is turned upwards and outwards, and the left downwards and inwards. This which I call a skew deviation, it will be observed, is just one-half of the movement for the estimation of differences of distance. In the adjustments for distances there is more than convergence and divergence; in convergence the eyes are slightly directed downwards, and in divergence slightly upwards. Ferrier finds that several different associated movements of the eyes are developed by irritation of several particular parts of the cerebellum; but he only develops the skew deviation in animals whose eyes are placed laterally. He did not develop it in monkeys; yet such a deviation occurs in man. Lesion of the crus cerebelli was diagnosed correctly by M. Nonat from observation of skew deviation.

We see from the experiments of disease, and very evidently from Ferrier's experiments, that the ocular muscles are represented far apart in each of the two great divisions of the nervous system—in the corpus striatum and in the hinder parts of the superior and middle frontal convolutions, and also in the crus cerebelli and cerebellum. There are cerebral and cerebellar movements of the eyes.

In an earlier part of this address, it was suggested that the cerebral ocular movements are for the estimation of the superficial extension of bodies, their superficial size and shape. I would suggest that the ocular movements for the estimation of distance are chiefly represented in the cerebellum. It is of course understood that these estimations by the eyes are symbolic of tactual and locomotor movements. I believe the movements of the eyes which are represented in the cerebellum are concerned in the symbolic estimation of depth of bodies and of depth of space, and also of resistance, which is, physiologically speaking, arrested locomotion. I founded the latter part of this doctrine chiefly on the experiments of Adamük, who asserted that different kinds of movements, either parallel or convergent and divergent, of the eyes are producible by irritating different parts of the corpora quadrigemina. However, Ferrier, although he finds that irritation of the corpora quadrigemina produces movements of the eyes, has not been able to corroborate all Adamük's statements.

It interests me greatly that Priestley Smith has recorded a case in which, with loss of power to look to one side, convergence was intact. For this shows, by another kind of evidence, that parallel movements and convergent movements are separately represented. It does not, of course, follow from this that the convergent movements are represented in the cerebellum; however, Ferrier has been unable to produce convergence by stimulation of any part of the cerebral hemisphere; but he suggests that convergence might result were we to be able to stimulate simultaneously the special motor centre for the eyes in each cerebral hemisphere.

For the rest, however, I shall speak only of affections of ocular movements from cerebral disease. The lateral deviation of the eyes and head is valuable in many ways.

1. By it we estimate the gravity of a lesion; the lateral deviation is the sign of a grave lesion.

2. It will be helpful in diagnosis betwixt drunkenness and apoplexy from local brain-disease, as Prevost has insisted.

3. The lateral deviation is an exceedingly valuable symptom, helping us to a methodical interpretation of unilaterally beginning convulsive seizures.

4. It is valuable physiologically; it illustrates the order in which symptoms appear in disease of nervous centres. The deviation appears both in hemiplegia and in the hemispasm, which is one of its mobile counterparts at a certain stage of gravity of these affections; it illustrates what I call the law of dissolution, using this term as the opposite of evolution.

5. It is valuable in that part of physiology which corresponds to psychology. Its occurrence in hemiplegia and in hemispasm shows us a definite relation betwixt hand and eye movements, and thus inferentially betwixt what are psychologically tactual and visual ideas.

[To be continued.]

RECOVERY AFTER TAKING EIGHTY GRAINS OF EMETIC TARTAR.*

By F. MASON, L.R.C.P.Ed., Surgeon to the Eye Infirmary, Bath.

TARTARATED antimony has been so seldom taken in large quantities accidentally, or used either for the purpose of suicide or murder, that the number of cases recorded of poisoning by it are very few. Casper, in his work on *Forensic Medicine*, only mentions antimony in the list of irritant poisons, without giving any cases. Taylor, in the third volume of the third series of *Guy's Hospital Reports* (1857), has collected thirty-seven cases, of which sixteen proved fatal; seven of these deaths were in young children; the smallest fatal doses were in two infants, three-quarters of a grain having been prescribed for one for some disorder of the stomach following measles, and a like quantity for another recovering from measles, and, in the adult (a medical student aged 28), two grains taken for gastric disturbance; "but, in this instance, there were circumstances which favoured the fatal operation of the poison". The largest fatal dose was one hundred and eighty grains. In the other cases, the quantity ranged between ten grains and sixty grains. The largest quantities swallowed, and from which recovery has taken place, are a girl 360 grains, a man 240 grains, and a man a tablespoonful, or 478 grains.

In 1855, Palmer was suspected, and afterwards found guilty, of having murdered John Parsons Cook by administering tartar emetic in repeated doses, and finally a dose of strychnine. This case gave rise to suspicion, and the discovery that he had in the previous year poisoned his wife also by repeated doses of tartar emetic. Imitators quickly followed, and, before the summer of 1857, two persons were tried for murder and one for the attempt at murder by administering this mineral in frequently repeated doses.

As Taylor, in the ninth edition of *Manual of Medical Jurisprudence*, published in 1874, only refers to these cases reported in 1857 and to no others, it may be assumed that no case has been recorded in the interval of seventeen years.

Some time ago, the attention of the profession and the public was forcibly directed to one of the most celebrated inquiries in the annals of medical jurisprudence, in which a verdict of wilfully poisoning by tartar emetic was returned. I consider the following case, in which eighty grains of this substance were carelessly given by a prescribing druggist, of sufficient interest to bring before your notice this evening, especially as, from the lapse of time, no harm can now follow the publication.

At half-past 9 A.M., May 12th, 1848, I received a message to go immediately to a druggist's shop in the neighbourhood of my residence. On arrival, the druggist called me to the back of the shop and told me he had by mistake given a man a large quantity of tartar emetic. The man was sitting in the front part of the shop. The mistake had arisen thus. The man had gone to the shop and asked for medicine to relieve some affection of the bowels. The druggist intended to give him a dose of quinine, but was at the time engaged in preparing a quart of antimony wine, and had the eighty grains of antimony in a mortar dissolved in a portion of the wine. He took a mortar precisely similar in size, etc., and, after preparing a draught in it, turned from the counter to get a glass for the man to drink from, when, on coming back, took the wrong mortar, gave the contents a stir, poured them into the glass and handed them to the man to drink. The man left the shop and returned to his work as a stableman before the mistake was discovered. He was then sent for to come back.

I ordered tannin, in some form or other, to be given, and a drachm of tincture of galls in water was at once administered. I also requested some strong coffee or tea to be made directly, for him to drink of it as much as he possibly could.

I went home for the stomach-pump. When I returned, the man had again gone back to his work, and I sent for him. He only complained of pain in the stomach, but not more than he had had for several days, together with diarrhoea, for which he had been under treatment. After he had taken the first cup of coffee, there was slight vomiting; this must have been nearly an hour after the antimony was swallowed. I now passed the tube of the stomach-pump, and pumped in about half a pint of milk, when the efforts to vomit became so violent, that I desisted from pumping, and, before I could reverse the action, the milk was ejected through the tube.

Before using the pump, I ordered decoction of cinchona to be made and now to be given in doses of a wineglassful every twenty minutes or half an hour, and, if he threw up one dose, to give him another imme-

* Read before the Bath and Bristol Branch.