

one great disadvantage, and that is the long period of four weeks which must elapse between each introduction. I had some time ago a patient under my care who had a perineal abscess and fistula caused by its application in America.

There is, seventhly, the method of over-distending the stricture by introducing an instrument with two blades, which can be expanded. It is very painful on application, and the results are of a most ephemeral nature. Fortunately, it has only a few advocates.

So much, gentlemen, for what I may call the manipulative treatment of stricture. At the next lecture I shall commence with the operative treatment.

ON TUMOURS OF THE CEREBELLUM.¹

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CHARLES M.—, aged thirty-five, was admitted into the London Hospital under the care of Dr. Sutton, Aug. 30th, 1879. Dr. Sutton made the diagnosis of disease of the cerebellum. The patient was later on transferred to my care. (The notes of the case were taken by Mr. Lawton and Mr. Lloyd Jones; Dr. Sutton's house-physician and medical assistant; and also by my house-physician, Dr. Jones, and by Mr. Richards, medical assistant.)

The patient was a healthy-looking man, and had had, except for rheumatic fever, a healthy life until the fatal illness. The duration of the illness altogether was from about January, 1879, but at first there was only a complaint of stiffness of the neck. The marked symptoms were of about three months' duration. He was at his work as a law writer until his admission, Aug. 30th, 1879.

The first thing he noticed beyond mere slight stiffness was that when writing his neck would become suddenly stiff and painful at the nape, preventing his bending his head over to the desk. The attacks came on irregularly, and at first he ascribed them to excitement, caused by having to control what he called the "drunken set" he had to overlook. The attacks of neck pains and stiffness generally began with a throbbing pain in front of his throat, and this pain, as he expressed it, "flew backwards, drawing his head back." Sometimes for a day his head would be drawn a little back, and go further back in paroxysms. He slept well, having no paroxysms at night. About a month before his admission he felt his feet tottering when he got up to walk, but he walked well when fairly started. About fourteen days before admission his eyesight became dim. On admission, Aug. 30th, patient had pain in the back of the head. He had double optic neuritis with impaired vision. His other special senses were normal. He had occasional vomiting. His head was constantly drawn back, and occasionally he had paroxysms of greater drawing back, each lasting two or three minutes. On one occasion, to relieve himself, he lay on his belly, with his chin resting on the pillow. Sometimes the occiput would touch the spine, and in the earlier notes of his attacks his body is said to be drawn back, and that he is in a state of opisthotonos. On Sept. 25th he had an attack of greater range, in which there was twitching of the limbs with greater retraction of the head and opisthotonos; there was also some nystagmus, the exact nature of which is not mentioned.

He was transferred later on to my care. On Oct. 6th I saw him. His head was then drawn back, but at my examination he was not in a paroxysm. When got out of bed he could walk, but reeled very slightly. When sitting, his legs acted strongly whilst endeavouring to overcome our resistance to flexion, extension, and to raising his knees. His arms were not affected. Patellar tendon-reflex was normal.

He continued subject to the paroxysms. On Oct. 11th he had paroxysms nearly all day long. A diagrammatic sketch was taken of the patient in one of them by Dr. Stephen

Mackenzie whilst the patient was standing. It was of necessity taken rapidly. I noticed spasm of no other parts than of the muscles of the spine, and in particular that the masseters were not engaged. Possibly part of the incurvation of the back was compensatory. To my astonishment the patient died the same day.

There was found, post mortem, a tumour about the size of a walnut in the inner part of the left lobe of the cerebellum reaching up to the middle lobe. It was a sarcoma, cystic in its interior. The symptoms may be put in two classes.

1. *Those which pointed simply to the existence of local gross organic disease of some kind.*

2. *Symptoms which localise—(a) retraction of the head; (b) paroxysms of increased retraction; (c) an unsteadiness of gait.*

Before considering either set of symptoms, it may be remarked that tumour or abscess of part of the cerebellum may exist without any marked symptoms. This may be affirmed of the cerebrum also. A patient may for a long time have symptoms so slight that he takes no notice of them, when suddenly he has a convulsive seizure, or becomes apoplectic, or dies suddenly; after death we find abscess or tumour of the cerebrum or cerebellum. In the case of tumour we find occasionally hæmorrhage from that tumour. Or a patient is under our care for some non-cerebral ailment, and we find tumour or abscess post mortem, although the existence of any kind of intracranial disease was never suspected during the patient's life.

1. *Symptoms pointing only to gross organic disease of some kind within the cranium.*—(a) Double optic neuritis was found on the patient's admission. His sight began to fail about fourteen days before admission, but no doubt his discs were abnormal before that time. It is well known that very strikingly abnormal ophthalmoscopic appearances may exist before the sight becomes defective, and that neuritis may pass away, probably always leaving some relics, without leaving defect of sight. Optic neuritis is of no value in localising disease in any part of the cerebrum or cerebellum. It has been said that tumour of the cerebellum, of the middle lobe especially, produces blindness by pressure on the corpora quadrigemina. Admitting that this may be so, it remains that in all cases I have yet seen, or heard of, of defect of sight with cerebellar tumour, there has been optic neuritis, and thus the defect of sight may be accounted for in another way than by the pressure hypothesis. In one case of tumour of the middle lobe there was optic neuritis when the patient's sight (a child) was such that he could see a hair on his pillow. As a matter of fact, a defect of sight from intracranial tumour is not caused in the majority of cases because the tumour involves any known part of the optic nervous system (corpora quadrigemina, optic tracts, or optic nerves); it is, therefore, in most cases not caused by destruction of any part of the cerebrum or cerebellum. (It may be that symmetrical disease of Ferrier's centres of vision produces defect of sight). Optic neuritis results in an indirect way. By some unknown process the tumour (or other adventitious product) leads to changes in the optic nerves, on which blindness may or may not follow. Again, in some cases of even large intracranial tumour, there is no optic neuritis. It may come on years after the first symptom of intracranial tumour. (b) Headache. Probably the pain at the back of the head was partly soreness, the result of dragging by the retracting muscles. There appears to have been no excessive pain. Pain at the back of the head is not to be trusted for the diagnosis of cerebellar tumour. (c) Vomiting is not a symptom peculiar to cerebellar tumour. Probably it depends rather on acuteness of process than on the position of the tumour.

To resume. The three symptoms mentioned are of no value in localisation. They are not decisive evidence of local gross organic disease; such disease may exist without them. They are, nevertheless, the best evidence of gross organic disease (renal disease being excluded).

2. *Localising symptoms.*—As aforesaid, there may be no marked symptoms in cases of cerebellar tumour or cerebellar abscess. I have seen a tumour in each lateral lobe of the cerebellum in a man who, fourteen days before death, had no motor symptom except paralysis of one arm, which was accounted for by tumour of the hinder part of the uppermost frontal convolution. Beyond question, whether we have motor symptoms or not depends on the size of the tumour, on the rapidity of its growth, on the softening it produces, and, of course, on the exact position of the tumour.

¹ Read before the Medical Society of London, Jan. 12th, 1880.

Then we have to bear in mind that, during the last part of the illness, in most cases, the patient is obliged, by pain and misery, to keep his bed, and testing his locomotion is not possible. (Tumour under the tentorium probably produces more disturbance by pressure than cerebral tumours do.) I think Nothnagel is right in his opinion that disease of the middle lobe is more likely to be attended by reeling than disease of the lateral lobe. Reeling is the earliest motor symptom from cerebellar disease.

It may be well here to speak of the several motor symptoms found in some cases of cerebellar tumour.

There are three motor symptoms which may occur with tumour of the cerebellum; they have their analogues in cerebral disease. (a) Reeling. (b) A certain persisting rigidity. (c) Tetanus-like paroxysms. Reeling, I think, is, or at least depends on, a cerebellar paralysis, the analogue of the hemiplegia a cerebral paralysis; persistent rigidity is the analogue of the well-known rigidity in hemiplegia and the tetanus-like seizures of unilaterally beginning convulsive seizures from cerebral cortical disease. (After some unilaterally beginning convulsion there is temporary paralysis of the parts first and most convulsed. I have yet no facts as to anything analogous in cerebellar disease.)

(a) Reeling is the earliest motor symptom. It is not the gait of locomotor ataxy, nor that of paraplegia. It is like the walk of a drunken man. It occurs in some cases of ear disease—often acutely in paroxysms (Menière's disease), or sometimes chronically. It is essentially a swaying of the trunk from back to front and from side to side. It is masked in some slight cases by the patient's walking with his legs apart; by this means small inclinations of the trunk are neutralised. The gait is worse when he walks with closed eyes. In early stages, in slight reels, I believe the real fault is paresis of movements of the spine answering to the swayings of the trunk. The legs act erratically, but in an early stage they are blameless; they act erratically because they have to "run after" the trunk, to prop it up in its various over-inclinings. The legs, often blamed, are really doing more than their duty. In cases of paralysis of the extensors of the feet the patient lifts his legs highly. To a layman his defect would appear to be that of lifting the legs highly. But this movement is "necessitated" by the paralysis of the extensors. To blame the legs in an early stage of cerebellar reel would be like blaming the leg-lifting movement in paralysis of the extensors of the feet. If the true fault in cerebellar reeling be paresis of the movements for bracing up the spine, we see that the reel compares with hemiplegia; the essential fault in the reel is a partial paralysis.

I suppose no one nowadays would confess to a belief in a *faculty* of co-ordination, nor to a belief in a co-ordinating centre, but that all consider every nervous centre, whether spinal, cerebellar, cerebral, or sympathetic, to be a centre of co-ordination, and that only in the sense of its being a part where these or those impressions and movements are *represented* in this or that degree and order. So those who hold that the cerebellum is the centre for the most special and complex co-ordinations of locomotor movements mean that it is a centre where the most special and complex of these movements are represented. This is only another way of saying that the cerebellum represents especially those movements which are bilateral or alternate. If we take count of all kinds of locomotion (and, indeed, of equilibration, whether statical, as in standing, or continually changing, as in progression) we see that the usual statement as to the chief functions of the cerebellum is equivalent to saying that it represents movements of all parts of the body. We must not use the term locomotion for walking only. In swift running the body is bent forwards, the jaws and face are in action, the arms swing, and professional runners carry corks in their hands to prevent the nails hurting the palms. We have to study all degrees of locomotion, from getting up to swift running, with the aim of ascertaining the order in which movements of different parts of the body are represented in the cerebellum. We see, I think, that muscles first in action are the spinal; they get into very strong action in swift running; the body would fall forwards if not held up. The reel is, I think, owing to paresis of movements of the spinal column. If so, the true morbid condition in the reel compares with hemiplegia and not with chorea, the movements of which, even on a very superficial glance, differ *toto caelo*. If we look at the choreal limbs and at the legs in the reeling man we may properly say that they are both disorders of co-ordination, but in the former case the movements occur at any time (from increased cerebral discharges)

in the latter only when the patient is trying to move, and then they are "necessitated" over-movement to compensate for the loss of other (spinal) movements. A truer analogue of the cerebellar reel is the tremor of disseminated sclerosis in which there is also paralysis. (Judging by the analogy of other cases, the condition will be more complex than this. From enervising of the diseased centre there will probably be underdoing of some and overdoing of other spinal movements.)

It is quite true that in cases of complete paralysis, with atrophy, of some of the spinal muscles, say the erectores spinæ, we have no such reel as in cerebellar disease. But the cases are different. A lesion of the anterior horns of the spinal cord destroys the action of some muscles altogether; this is a very different thing from loss of some complex movements of the spinal column. Again, in the paralysis, with atrophy, of some muscles, the patient walks by "dodging" the paralysis (just as we see other patients "dodge" the effects of paralysis of an external rectus); even then he walks badly, has a veritable inco-ordination of locomotion, although not one so complete as a reel.

(b) *Cerebellar rigidity*.—Just as rigidity ensues in some cases of cerebral paralysis (hemiplegia), so it does in some cases of cerebellar paralysis. In the latter the spinal muscles first become rigid, drawing back the head and curving the spine; ultimately the legs and arms are rigid. This universal rigidity, as a condition arising from cerebellar tumour, I have seen only when the tumour has been of the middle lobe. I may here say that I am indebted to Dr. Stephen Mackenzie for the drawings I send round, and also for most valuable help in the investigation of cases of cerebellar disease. In cases of hemiplegia the rigidity is contemporaneous with the ensuing of Wallerian wasting in the spinal cord. Whether there is a "descending" wasting in cerebellar rigidity I do not know. Cerebellar rigidity differs from the rigidity of paralysis agitans; in the latter the body is bent forwards, in the former backwards.

(c) *Tetanus-like seizures*.—Besides rigidity we find, in some cases of tumour of the middle lobe, attacks very like, if not quite like, those of ordinary surgical tetanus. I pass round drawings by Dr. Stephen Mackenzie, to whom again I gratefully acknowledge my indebtedness for help in the investigation of cerebellar disease.

I have only once seen seizures like tetanus in a case of local cerebral disease, and in that case they occurred during the last hours of life, during dying. Dr. Gowers discovered no cerebellar lesion; there was large cerebral tumour corresponding to hemiplegia. In the case of extensive rigidity and tetanus-like seizures, the tumour has been of the middle lobe, and has been large. I suppose the cerebellar rigidity to be owing to unantagonised cerebral influx (rigidity in hemiplegia being owing to unantagonised cerebellar influx), and the tetanus-like seizures to be owing to cerebellar discharges analogous to those cerebral discharges which produce unilaterally beginning (epileptiform) convulsions. But there is the obvious objection that the two symptoms, with the cerebellar disease, may be owing to interference with the corpora quadrigemina (electrical excitation of which Ferrier finds produces tetanus-like states), or to interference with the medulla oblongata.

Hitzig and Ferrier have convinced most people that some parts of the cerebral cortex represent movements, and Charcot, by a very different kind of evidence, clinical and pathological, has confirmed their opinions. I have long wished to ascertain if the cerebellum cannot be broken up into regions, each representing especially some movements of the body. In this case, the subject of this paper, there was a very local movement, a local tetanus. That the drawing back of the head in paroxysm was owing to discharge of some part where the movements for drawing back or supporting the head are represented is a truism; but I do not draw the inference that it was the part in which the tumour lay, for, besides objections already stated, I have but one case. If the discharge were of the cerebellum, it may have been of some part of the middle lobe. Of course the discharge would be of *nerve-cells* rendered highly unstable by action of the tumour, and it does not follow that the *nearest* cells would soonest become unstable; probably small cells become unstable before large ones. Another important thing is that one would suppose the movements of drawing back or supporting the head to have a wide representation, and we could, I think, only expect to find some part of the cerebellum, on each side doubtless, where movements of the head are *chiefly* represented. There are clinical difficulties,

drawing back of the head occurs in cases of meningitis; but plainly, the best cases for investigation of regional localisation are cases of small tumour producing a chronic illness.

Occasionally we meet with other local tonic spasms, sometimes of the jaws. Of the pathology of these cases I know nothing at all. I would suggest that the cerebellum should be carefully searched as well as other parts in all cases of local tonic spasm, including ordinary torticollis.

I think there is evidence that in some parts of the cerebellum there is a unilateral representation in the order from trunk movements to movements of the hands and feet. I have no doubt that Brown-Séquard is right in saying that tumour pressing on the crus cerebelli produces paralysis of the same side, and I believe this is mainly of movements of the trunk and those passing to the upper parts of the limbs. The erratic movements of the arm in some of these cases partly depend, I think, on paresis of muscles attaching the limb to the trunk.

There are indirect symptoms from tumour of the middle lobe and large tumours under the tentorium. There is vast increase of fluid in the lateral ventricles of the cerebrum in some cases. In children the head enlarges; there is great hebetude of mind, a slow failure of mental faculties. This I used to suppose was caused by pressure, as the veins of Galen. Stephen Mackenzie, in an admirable paper on Cerebellar Disease, recently read before the Hunterian Society, suggests another explanation.

A MODIFIED CLOVER'S BOTTLE FOR THE RAPID EXTRACTION OF DÉBRIS AFTER LITHOTRITY.

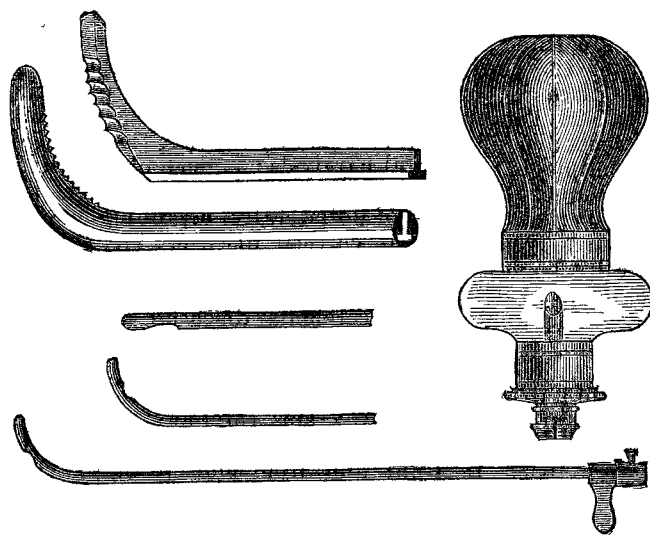
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DR. BIGELOW'S bottle and tubes for exhausting débris after lithotritry are manifestly a great improvement on the apparatus previously employed for that purpose. The improvement consists, first, in providing a wider passage for the current to and fro between the bladder and the exhausting bottle; and, secondly, in furnishing a more forcible stream than was possible with the small bottle of Clover. But as Bigelow's bottle has considerable disadvantages in being costly, cumbersome, and especially in being much longer than is necessary, it occurred to me that Clover's bottle might, by some small modifications, be rendered a more rapid exhauster of débris than that of Bigelow. For some time before Dr. Bigelow's visit to this country I had been employing urethral tubes for washing out débris larger than those commonly used for this purpose, habitually employing Nos. 24 and 26 French, instead of No. 22 or No. 23 (Nos. 12 and 13 English), the customary sizes. Consequently, when Dr. Bigelow brought before the profession his rapid method of clearing the bladder of débris, I was prepared to appreciate the improvement he has effected in this department of surgery. By the apparatus which I have now used on several occasions—the outcome of repeated alterations—I find I can exhaust a given quantity of débris more rapidly than I can by Bigelow's bottle. This advantage I attribute mainly to the shorter distance the fragments have to travel from the neck of the bladder before they are securely lodged in the trap below the current. The length of the tube from the beak to the interior of the receiver is only eleven inches, or twenty inches less than that in Bigelow's apparatus.

The woodcut shows the form of the apparatus very well. A stout india-rubber bottle, holding ten ounces, with a neck one inch across internally, is fitted to a glass receiver, or trap, blown into a discoid shape resembling that of a flat turnip or onion, having a transverse diameter of $3\frac{1}{4}$ in. The mouth of this receiver is closed by a cap, to which the washing out catheters can be fitted in a joint fastened by a bayonet-catch. The catheter is prolonged into the centre of the receiver by a short tube, $\frac{1}{4}$ in. (about No. 40 French) in diameter, to prevent the reflux of the fragments that have already been sucked out of the bladder. As the fragments issue from the tube they at first swirl about in the receiver,

then fall rapidly into the lower part, where they lie snug and still while the water is being injected again into the bladder to bring forth another quantity of débris. Probably the main advantage of the apparatus is its shortness. A moment's reflection will show that when the outflow ceases part of the débris sucked from the bladder lies in the tube, to be driven into the bladder again by the next inflow of water; consequently the nearer the trap or receiver can be



placed to the neck of the bladder, the more rapid will be the removal of the fragments. In Bigelow's apparatus, the receiver is placed at the farthest extremity of the bottle. This large bottle is placed for convenience of working at the end of 14 in. of flexible tube. Thus the fragments have to travel $11\frac{1}{2}$ in. along the silver tube, then along $14\frac{1}{2}$ in. of flexible tube, making a total distance of 26 in., before they reach the bottle itself, down which they gravitate during the pauses between each act of injecting and exhausting to the glass receiver placed at the further end of the bottle. Thus the fragments do not reach the trap or resting-place, until they have traversed 31 in. of distance. The tubes I use have an internal capacity equal to sounds Nos. 24, 26, and 30 French (equal to Nos. 14, 16, and 18 English). They are provided with large eyes, carefully bevelled off to prevent their gaping edges from lacerating the mucous membrane. Some have the eye placed at the convexity of the beak, some have it at the concavity, and one tube (the largest) is straight throughout. This I have seldom used, as it is rare for the urethra to admit its introduction without a preliminary free incision of the meatus urinarius, which it is not always expedient to do. Except in being shorter, and in the lesser sizes smaller, my tubes do not greatly differ from Bigelow's, and their mode of attachment to the washing-bottle is copied from Bigelow's pattern.

The woodcut also shows the beaks of a powerful lithotrite made for me by Mr. Coxeter, for breaking large hard stone that cannot be safely managed by ordinary lithotrites. It was constructed for me in imitation of Sir Henry Thompson's new lithotrite, and in some respect, like other imitations, advances beyond the first embodiment of the new idea. His recent alterations on former patterns Sir Henry Thompson has already brought before the profession in this country, and on the continent. They are chiefly two. First, the male blade is fitted to slide along the female blade until its heel comes flush with the outer surface of the beak of the female blade, which is cut away for that purpose. By this arrangement fragments cannot lodge nor lock the blades, but are driven through the female blade before the advancing male blade. The second novel point is, the knife edge form given to the beak of the male blade. This knife edge enables the male blade to penetrate a hard, bulky stone with facility, and split it into fragments, that can afterwards be readily comminuted by the usual lithotrites, which have more pulverising action. The beaks of the lithotrite depicted in the drawing differ from those of Sir Henry Thompson's instruments in having a somewhat sharper knife-edge and a wider notching of the edge; there is also a slight jaggling of the opposing edges of the female blade to afford a better hold of the stone before it is broken. Whether this increased sharpness of the knife-edge is an additional advantage can only be determined by comparative trial of the two instruments. Mine was simply an endeavour