cavity a dense layer of lung tissue with shreds of disintegrated lung were found still adherent to the chest wall. After thorough evacuation this cavity was washed out with an antiseptic solution, and gauze was introduced as a drain.

This operation afforded great relief, except that the patient would not dare to turn from one side to the other, and preferred to lie on the sound side. He said that when he tried to turn, something gave him a violent wrench, as if "his heart were being pulled out." This was attended by a distressing angina-like spasm. The reason for this sensation, which the patient experienced, was not understood at the time, though it was afterwards fully explained. After four weeks it was evident that the great space on the left side of the chest was not closing, and besides there was a persistent and free purulent discharge from this large cavity, which, if it were allowed to continue, would soon have exhausted the small amount of strength which the patient still possessed.

Under these circumstances it was deemed advisable to perform thoracoplasty, both with the view of thoroughly exploring the cavity and removing the shreds of lung tissue still remaining, and also with the hope of reducing the dimensions of the cavity by permitting the external wall to be involuted.

Excision of portions of the fourth, fifth, sixth, and seventh ribs was performed, each portion extending from the angle of the rib to the costal cartilage. It had been intended to remove three other ribs, but the patient's condition was such that their removal had to be deferred. The view obtained of the interior of the left side of the thorax by this free opening was such as to enable one to see that there was no vestige of the left lung remaining, save shreds adhering to the walls of the chest, a portion at the apex and a stump representing the larger bronchi and the corresponding blood vessels, somewhat fused together, and covered by granulation tissue. Behind this stump the descending aorta was seen and felt, and in front of the stump the pericardium lay freely exposed and non-adherent by pathological adhesions. The absence of the pericardial pathological adhesions was so marked and the normal attachments so relaxed from over-stretching of the diaphragm downwards and pressure of the heart toward the right side, that when the patient lay on his sound side the heart rested against the anterior mediastinum, and when he turned on to his back the heart flopped backwards over the stump of the bronchi, and the lower portion of it within the pericardium swung free in the thoracic cavity. The pericardium was somewhat thickened, but not so much as one would have expected, owing to the great destruction of the lung tissue occurring in its immediate vicinity.

The free movement and flopping of the heart inside its pericardium explained the strong objections which the patient expressed, previous to this second operation, to being turned from one side to the other, as he said each time he was turned it gave his heart a wrench. This had not been experienced by him before the evacuation of the fluid from the chest, but had been markedly felt in the interval between the first and second operation. There were several shreds of disintegrated lung tissue still adherent to the diaphragm, to the outer walls of the chest and to the apical part of the thorax. The former were removed, the latter cut short, their proximity to the large subclavian vessels rendering it undesirable to attempt complete removal. In order to prevent displacement of the heart, and to hasten adhesions between the pericardium, the mediastinum, and the diaphragm, the large cavity was packed with gauze, which was changed at frequent intervals. Subsequently adhesions quickly formed between the pericardium and the diaphragm, one of these first appearing as a comparatively thin band which stretched somewhat during the dressing. It gradually became thicker until it attained the diameter of a finger. These adhesions were then able to hold the over-stretched pericardium in its place, and thus to prevent the swinging of that organ, which it was previously subject to when the patient was turned from one side to the other. Soon after these adhesions had formed, the pericardium became attached to some of the structures in the anterior mediastinum and later to the anterior wall of the chest and to the stump of the bronchi behind. Gradually all the structures presenting on the left side of the mediastinum became covered with granulation tissue and fused

together so that their individual outlines were lost, a continuous wall having been formed over the left side of the mediastinum from the anterior to the posterior portion of the thorax. The right lung likewise expanded to the right side. Portions of the third, eighth, and ninth ribs were removed at a later date to further facilitate contraction. The osteoplastic resection thus extended from the third to the ninth rib inclusive. After the interior had become aseptic and a layer of healthy granulation tissue had filled the various recesses, the skin was laid over the now thickened pericardium and diaphragm and stump of lung, and soon became adherent and filled up the cavity, a small sinus at the upper end persisted, coming from a point where shreds of the apical portion of lung remained. On several occasions, while the cavity of the thorax was still open, these were attempted to be detached, but on each the adhesions were found too firm to permit of easy separation. The adhesion of the apical portions to the subclavian vein precluded further detachment. The loose portions were trimmed. His general health meanwhile improved, the râles disappeared from his right lung and he gained strength and weight, and was able to walk about and enjoy life.

It is now eleven years since this man was operated on, and since his convalescence he has been able to engage in light work, supporting his wife and family thereby. He can walk about easily while taking time, and generally enjoys good health.

Examination of his lung at present time:

On June 3rd, 1906, this patient was examined by three physicians, who reported that there were no breath sounds on left side of the chest, except the transmission of bronchial sounds heard under the clavicle on the left side. The right lung appeared normal. The heart sounds were good. The patient states that he has been regularly at work as a light porter; he can go upstairs without trouble. He mounted the eighty odd steps of the infirmary stairs before he was examined, and did so without pause and with very little inconvenience. He states that since his operation he has walked ten miles without fatigue, but he does so at what he calls an "ordinary easy rate." Since the operation he has never been ill and has regularly been at work.

Gentlemen, allow me to thank you for the honour you have done me by inviting me to deliver the Cavendish Lecture for 1906, and also for your patient and courteous attention during its delivery.

Clinical Lectures

ON THE

BORDERLAND OF EPILEPSY:

VERTIGO.

DELIVERED AT THE NATIONAL HOSPITAL FOR THE PARALYSED AND EPILEPTIC.

BY SIR WILLIAM R. GOWERS, M.D., F.R.S.,

PHYSICIAN TO THE HOSPITAL; CONSULTING PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL.

LECTURE I.

GENTLEMEN,—Epilepsy is a many-sided malady. The prominent symptoms that attend its various forms differ widely in character, and some of them are common to it and diseases that are of different nature. Some of these maladies resemble epilepsy closely, and their distinction from it may be difficult. It seems sometimes that there is a gradation from the one to the other, and occasionally that an actual transition may occur. It is not surprising that this should be so with such diseases, considering how complex are the functions of the brain, and how close are their interrelations. Functional diseases are often less defined than our definitions. Thus, epilepsy has a borderland of disorders which resemble it in some of its special features, and their distinction from it may be a matter of considerable difficulty. The difficulty may be real, or may disappear with knowledge and consideration. It is important, therefore, to know the features of these borderland cases, the symptoms they present in common with epilepsy, and their distinctive characteristics. They fall into groups according to their nature and dominant

feature, and I propose to consider to-day the affection which constitutes by far the largest province of the borderland—vertigo.

As you know, vertigo is the most conspicuous subjective feature in a large number of cases of true epilepsy; it is also common from other causes. But, as a fact of observation, the only form of vertigo which bears a close resemblance to epilepsy is that which is produced by labyrinthine disease. The resemblance is, of course, to minor epilepsy alone. When there is either tonic rigidity or clonic convulsion, the nature of the initial vertigo is clear. In the minor attacks it is often the only symptom of which the sufferer is conscious, and it is these that the labyrinthine form may closely resemble. "Aural vertigo" and "labyrinthine vertigo" are practically synonymous terms; whatever part of the ear may be primarily affected, it is through the semicircular canals of the labyrinth that the symptom is produced. As you know, it is sometimes called "Menière's disease," but the cases described by him were of intense severity, and, at least in one case, due to haemorrhage into the canals.

I need hardly remind you that the function of these peculiar structures is believed to be concerned solely with the perception of the position and movements of the head. Nerve impulses are produced by the pressure of the endolymph on the hairs in the ampullae. These impulses seem to give rise to no conscious sensation. It is thought that they pass to an equilibrical centre—perhaps in the middle lobe of the cerebellum—which receives other impressions—some from the limbs, and others determined by the state of the muscles of the head and eyes, which afford guidance as to the relation to the body of objects that are seen. All should harmonize, and their effect guides the cerebral perceptions and the motor activity. If they are disordered, vertigo is the result.

In essential character the vertigo of epilepsy bears a close resemblance to that of labyrinthine disease. The latter must be perceived through the cerebral hemispheres, and we can thus understand at least the fact that they may be similar. Consider the vertigo of epilepsy. There may be a sense of turning to one side (subjective vertigo), or of a movement of objects to one side (objective vertigo), or the patient may actually turn (motor or actual vertigo). As a rule, objective, subjective, and actual vertigo are in the same direction. The facts can be understood on the explanation, suggested by Dr. Hughlings Jackson, that vertigo is a sensation of motor origin. In epilepsy we may ascribe it to an inequality in the motor discharge in the two hemispheres, often manifested in the attack by the deviation of the head and eyes towards the side most convulsed. A commencing unequal discharge, too slight to cause movement, may produce a subjective sense of movement. During this, objects in front of the eyes seem to be moving in that direction. If a person is actually turning, and an object remains opposite him, he necessarily infers that it is moving also, and in the direction in which he is turning. Thus, even the objective vertigo, the sense of movement in what is seen, is intelligible as the result of a motor process.

Is the same explanation applicable to the vertigo of labyrinthine origin? On first consideration this would seem to be different in nature. It has rather the character of a pure sensation of the disturbance of equilibrium. But its motor origin is possible. The nearest physiological analogy to labyrinthine vertigo is that produced when rotation of the body is suddenly stopped. The tendency to turn continues in the same direction, and may cause a fall. Objects before the eyes seem moving, also in the same direction as the preceding rotation. Yet during the turning, the apparent movement of objects was in the opposite direction. The rotation thus leaves a motor tendency in the same direction as the previous motion, which involves seen objects, as already explained. Apparently the excitation of the semicircular canals by the motion induces a motor process of the same character in the cortex of the brain. It seems to produce that which would cause it, by a sort of backward association.

It does not excite an opposing activity, as might be imagined, but an activity of corresponding character, as if the rotation were the result of volition. The stimulation of the canals and its central effect seem to be long in subsiding. Hence it is possible that even the conscious vertigo of labyrinthine disease may be produced

through the agency of the motor cortex, under the influence of the derangement of the impulses from the equilibrical centre. But the precise features of such vertigo vary much, and are sometimes so peculiar as to suggest a direct sensory effect—not, indeed, from the canal nerves themselves, but by an indirect mechanism through the centre for equilibration. Some of these special features will come under our notice presently. One of them is not easy to explain; it is the apparently opposite direction of subjective and objective vertigo. The sufferer falls one way and objects seem to move the opposite way. It may be due, in some way, to a process of secondary origin, opposed to the primary. But it is rare, and some doubt attaches to sensations in a moment of supreme confusion. In connexion with it we may note that the objective vertigo sometimes predominates over the subjective sensation; indeed, the patient may only be conscious of the sense of movement in objects. These difficulties we cannot yet explain; but before I end you will cease to be surprised, so various and peculiar are the forms met with.

Very little is known of the features that are common to aural vertigo and epilepsy; I must, therefore, make good my ground by describing the facts as shown by individual cases. I must also make the evidence secure by mentioning the evidence of the labyrinthine origin of the symptoms. However briefly I do this, it must make a large demand on your patience and attention; but if the supply is equal to the demand, you will find that facts presented through cases secure a firmer hold upon the memory than those that are given as general statements, easily received and easily lost. I will describe the symptoms which aural vertigo presents in common with epilepsy, with as much method as the cases permit, but patients seldom present facts in the systematic way that would be convenient to us.

In the cases described, hearing was tested up to 2,000 vibrations by C tuning forks. By "middle C" is meant that between the treble and the bass, 256 vibrations. The precise number varies with the pitch, and it is easier to remember if we call it 250. The C within the treble is C¹, 500 vibrations, and that within the bass, C₁ 125 vibrations. Above C², 2,000 vibrations, hearing is observed by Galton's whistle, and recorded by the number of vibrations, which are indeed convenient for lower notes. Tinnitus is a symptom of great importance, as evidence of labyrinthine irritation; the word "continuous" is used in opposition to "pulsating," and "constant" is employed in the sense of persistent.

SUDDENNESS.

The sudden onset of the attacks of aural vertigo is well known; one moment the sufferer may be in his normal state, and the next unable to keep upright without support, or on the ground and unable to rise from the severe sense of vertigo. We cannot regard the suddenness of the onset as the result of a sudden change in the impressions from the labyrinth; at any rate in most instances there is no other evidence of such a change. In rare cases there is an increase in the tinnitus at the time of the onset, which suggests a sudden increase in the labyrinthine irritation, but this is not common. Too much significance has been ascribed to it. The suddenness of the onset may be more reasonably regarded as of central origin. It suggests that the equilibrical centre has a certain degree of stability, which suddenly gives way under the cumulative effect of a persistent derangement of the impressions that reach it. We must consider that this centre acts upwards on the cerebral cortex, and its sudden deranged action may be compared to the process of "discharge" in epilepsy. The constancy of the features of the attacks in many cases suggests that, as in epilepsy, the repetition of the process facilitates its occurrence in the same form. Indeed, if the middle lobe of the cerebellum is the equilibrical centre, the facts of many cases suggest the conception of a cerebellar epilepsy, but this would carry the analogy farther than is quite justified. The suddenness of the onset is too familiar to need illustration, but it is not so with the feature next to be considered.

BREVITY.

We conceive aural vertigo as of prolonged duration even when sudden in onset, and that of epilepsy as brief,

almost momentary. But the former may also be brief as well as sudden, and this constitutes one important point of resemblance which often gives rise to doubt. When the severe vertigo is brief it may be followed by much slighter giddiness for a time, or by nausea, which suggests its nature; and usually the patient has, or has had, attacks which present no resemblance to epilepsy. But it is not always so, as the following case shows.

A man of 60 had suffered for several years from attacks of momentary giddiness, a sudden impulse to go forwards, instantly over, but followed for a few moments by a sense of nausea equally brief. He never lost consciousness, and had no other form of attack. He had been slightly deaf for six years, hearing on the left side only the highest tuning-fork, 2,000 vibrations, and not much higher notes on the right; while for the same time he had tinnitus referred to both ears, at first louder in the left, lately in the right—a continuous hissing, sometimes increased for a moment to a loud “whizz.” Such evidence of labyrinthine irritation as tinnitus affords is always important. In this case the aural condition had evidently made little progress during the years of its persistence, and this is of interest in relation to the uniformity of the vertigo. The facts suggest that, as I have just said, a tendency to uniformity had been developed by repetition. Such a case, although not epileptic, is distinctly “borderland.”

A similar case, in which the sudden brief vertigo often caused an actual fall, was that of a man of 42. The sudden sensation was that everything before him was coming on top of him. Even when he fell, there was no impairment of consciousness; in a few seconds he was able to get up, but a slight sense of unsteadiness remained for ten minutes. Such a fall in epilepsy would have been attended by momentary unconsciousness. Other attacks, however, showed their nature. Occasionally he had time to take hold of something, and did not fall, but he felt himself and objects turning to the right. Such attacks were formerly followed by considerable nausea, and by diarrhoea, and with great coldness. There was also clear evidence of labyrinthine changes; hearing had been impaired in the left ear for five years. He had constant left tinnitus, which presented the rather unusual feature of being increased by external sounds. It was “buzzing” in character, occasionally louder, and then attended by a sense of pressure in the ear which seemed to prevent hearing. At the onset of the attacks in which objects seemed to be coming on to him, the noise changed for a moment to a shrill whistle. This affords confirmation of their labyrinthine nature. Note also the two after-symptoms, both suggestive. One was coldness of the surface, which must be ascribed to superficial vasomotor spasm. The other is the diarrhoea; it frequently follows the vomiting after vertigo and sometimes even nausea. It may be the result of impairment of digestion, due to the nerve disturbance which causes vomiting, or possibly to dilatation of the intestinal vessels, coincident with the contraction of those of the skin. It is evidence of the readiness with which disturbance of the equilibrium centre deranges the gastro-intestinal tract.

In the last case there was an increase of the tinnitus at the onset of the sudden vertigo, which, as I have said, is rare; in the next it was increased after the attack. A man of 34 had moderate deafness and constant pulsating tinnitus on the right side, with continuous slight giddiness. He had also sudden exacerbations of giddiness in which, as in the last case, the floor seemed to rise and pass over his head, and he fell, but without the least confusion of mind. In a few seconds the attack was over, but the tinnitus was much louder for an hour or more after each attack. Slighter attacks, equally brief, were attended by a sensation that he himself was turning to the left. Such a variation in character is not uncommon. The objective sensation of objects coming to or over the person is usually a feature of the more severe attacks, and often causes a fall.

Many of the cases I shall have to mention in connexion with other symptoms will afford further illustrations of the brevity of attacks.

LOSS OF CONSCIOUSNESS.

If such cases of a sudden fall with almost momentary giddiness resemble epilepsy and are often mistaken for it, much more is this true of those in which the attack is accompanied by definite brief loss of consciousness. It is

not generally known that such loss occurs in purely aural vertigo, and the symptom is often regarded as unequivocal evidence of epilepsy. But the cases I am going to describe afford clear demonstration of its occurrence in vertigo. As I have said, the condition in any attack of intense vertigo is one of imperfect perception of the surroundings, and of their relation to the sufferer, a condition which itself involves imperfect consciousness. We can understand that vertigo coming on with suddenness and momentum may for a moment sweep consciousness completely away before it.

A man of 61 had suffered for ten years from bilateral labyrinthine deafness, greater on the right side, with tinnitus louder on the left, and also from attacks of giddiness. They were severe, with a “whirling” sensation. Eight years before I saw him he had consulted Sir Samuel Wilks, who diagnosed “Menière’s disease.” The attacks had continued, but had become much briefer, lasting only a few seconds. Objects and himself seemed moving to the right, and when these attacks were severe, he fell. Almost immediately afterwards he could recall the giddiness and its character, but not the fall—clear proof of loss of consciousness. On coming to himself he felt dazed for a few moments, and burst into perspiration. The latter is a common feature of pure vertigo; the “dazed” recovery is a feature of epilepsy. But it is so as an effect of the loss of consciousness, and when this occurs in vertigo the slowness of the return to normal consciousness is not rare. The sudden perspiration is a feature of vertigo.

Another instance of the effect on consciousness of pure aural vertigo is of value on account of the high intelligence of the sufferer. He was a clergyman of 37, who had suffered for seven years from left-sided deafness and tinnitus, due to chronic changes in the middle ear, spreading to the labyrinth. His first attack of giddiness, three months before I saw him, was intense, making him fall from a chair on which he was sitting and he afterwards vomited. By a common diagnostic error, the vomiting, which was the pure consequence of the vertigo, was regarded as evidence that the attack was “gastric vertigo.” Afterwards he had two slighter attacks, in which objects seemed to go to the left; mere retching followed these. Then occurred one of startling severity. When dressing in the morning before a looking-glass, this and the table on which it was standing, and the floor below, all seemed to rise up and come down upon him, as he fell. He knew of the fall, but was certain that he was then unconscious for a few seconds. He afterwards had others slighter, with objective giddiness to the left, and after each attack the tinnitus was louder, and became pulsating instead of continuous. (This change in character often attends an increase in degree: a uniform hissing, which becomes louder, may beat like machinery.) This patient was a careful observer, and he noticed that an increase of his tinnitus, with this change in character, often followed, for a short time, the act of rising from the sitting position. This fact is curious. A change of position often induces giddiness in labyrinthine vertigo, presumably because it causes a sudden change of pressure of the endolymph on the hypersensitive structures in the ampullae of the canals. The pressure of the endolymph must be similarly varied in the part of the labyrinth concerned in hearing, and if the changes are so situated that the influence exerted by rising acts upon them, we can understand the increase in the tinnitus that may be thus induced, although we may be unable to trace the process. Indeed, the pulsatile character of tinnitus must be due to the sensitiveness of the structures to the changes of pressure caused by the arterial pulsations. In this patient, the left ear gradually became quite deaf, and then all giddiness ceased.

Again, a man of 41 had suffered for some years from hissing tinnitus on the left side, on which he could hear no note of Galton’s whistle, although it was well heard on the right. Recently he had had attacks in which a momentary “wave” seemed to pass over his head, with a movement of objects to the left; he fell to the left, and was unconscious for a second or two. Sometimes the apparent movement was to the right, and then he fell in that direction. The resemblance of such attacks to minor epilepsy is so close that doubt may be felt whether the association with labyrinthine changes is sufficient ground for regarding the case as essentially vertigo. But, before the attacks became so brief, he had slighter more pro-

longed attacks of giddiness, in which the ground seemed to recede, and if walking he would fall, distinctly from the unsteadiness. These attacks were followed by nausea, and they make the nature of the case certain.

I may mention, lastly, the case of a gouty woman of 67, who had complete left-sided deafness through air and bone, with left tinnitus "beating like an engine." Attacks of giddiness had occurred during three years, with a subjective sensation of being turned round and round. Lately they had begun very suddenly; they were so intense that for an hour she could not open her eyes, and were followed by prostration and nausea. In one she suddenly fell backwards, and afterwards remembered nothing of the fall; other attacks followed, also with brief unconsciousness, but followed by giddiness during many hours, slowly lessening.

Loss of Sight.

The facts I have described show clearly that intense and sudden vertigo may affect the cerebral functions with such momentum as to cause brief unconsciousness. As I have said, all intense giddiness involves some failure in perfect consciousness, so far as clear perception of the environment is concerned. The sensation of a swift movement of seen objects is distressing in vertigo; the fact that often the eyes cannot be opened testifies to the effect of visual sensations. It is not, therefore, surprising that when consciousness is not entirely lost, sight may be disturbed. In cases of partial syncope, sight may be alone affected, and sight may be lost before consciousness in epilepsy—and, indeed, may alone be lost. So in some of these epileptoid forms of pure vertigo.

A lady of 42, with gouty ancestry, had for years deafness of the left ear, distinctly labyrinthine, and also tinnitus—a noise like machinery, which seemed to be behind her. During eighteen months she had suffered sudden brief attacks of giddiness, lasting only a few seconds, in which she felt as if "going heels over head" backwards, and, at the moment, she could see nothing. There was no unconsciousness. An attack would sometimes occur in bed, and she would then beg to be held down, so intense was the subjective sense of movement. Although the feeling usually ceased suddenly, a very severe attack was followed by slighter giddiness for some hours, at first sufficient to prevent her raising her head from the pillow. During vertigo, when the sufferer is lying, the act of raising the head usually intensifies the distress. It causes a fresh impression on the nerve endings in the semicircular canals, which, slight though it must be, is sufficient to intensify the existing disturbance. The fact is easily verified. If a person turns round a few times with the head bent forward he feels sufficiently giddy to have to steady himself by taking hold of some support, but if he raises his head as soon as he stops, the giddiness is so much more intense that he may have great difficulty in preventing a fall.

Sight was lost, for a few seconds, in sudden falls to which a man, aged 61, was liable, in which he seemed felled to the ground. They had succeeded slighter attacks of giddiness from which he had suffered for two years. He had middle-ear disease and deafness on the right side, with destruction of the drum, and on the left side he was unable to hear high notes, and he had continuous tinnitus.

The fact that the loss of sight depends on a special disturbance of the visual centres was shown in a peculiar way by a lady of 50, who had suffered for three years from left labyrinthine deafness. Only the middle C tuning-fork (256) could be heard; no higher or lower octave. There was buzzing, pulsating, tinnitus, changing sometimes for a few minutes to a sound of bells. Attacks of vertigo had been severe and distressing, compelling perfect rest for the time. There was a sense of movement to the right in herself and external objects; when lying with her eyes closed the bed seemed turning over to the right. Later the attacks became brief, lasting only a few minutes, beginning with a rush to the head and a sense of movement within the head so bewildering that she "hardly knew where she was." In one of these everything was dark in the upper half of the fields of vision for about a minute. Her account was very precise. Such transverse hemianopia is met with rarely in functional disturbance of the brain. These brief attacks were seldom followed by vomiting, but generally by great coldness and shivering, no doubt from vasomotor spasm of the skin,

another illustration of the relation of the vasomotor centre to that for equilibrium. Her longer attacks were frequently followed by recurring cramp in the legs. This effect may have been the result of their position, although we may remember that the equilibrical centre must readily influence the muscles of the legs for the maintenance of the erect posture, by whatever mechanism the influence is exerted.

We pass next to a remarkable symptom of sudden attacks which is probably characteristic of vertigo, although it has a striking resemblance to an epileptic aura.

SENSE OF IMPULSION.

Intense aural vertigo of sudden onset, causing the person to fall, may be attended by a sensation that the fall is actually compelled. The feeling of giddiness is so intense as to involve a sense of compulsion, in which sometimes the vertiginous element is hardly realized. In epilepsy there may be a feeling of involuntary motion but not of being "felled or hurled to the ground," as the patient described it whose case I mentioned just now, and as many do who suffer from aural vertigo. I need not give you other special illustrations of this. The fact is carried still further in the remarkable symptom to which I pass.

Not only is there a feeling of being a passive object on which an impelling force is acting, but this impression may be so intense as to have, as it were, a backward influence. It may thus cause a definite sensation of an external physical agency which seems the cause of the sudden disturbance of equilibrium. The sensation may be gentle or violent. In the former case it is merely a sense of an impulsive force. For instance, a lady of 50, with slight nerve-deafness on the left side, had also loud pulsating tinnitus, referred to the head, compared to machinery in motion, sometimes changing to a sound of drums beating. For six months she had suffered from attacks which began with a sudden distressing sense of pressure at the vertex, passing to the front of the head, with a feeling that she was going down, down, through the floor. With this there was a slight tendency to fall to the right, and objects seemed to oscillate from side to side. Thus the chief subjective feeling of sinking down was accompanied by the perception of a physical force, pushing her down.

Much more violent in character was the sensation experienced by a woman of 34, with progressive nerve-deafness and tinnitus on the right side. For five years she had had sudden attacks, at first simple giddiness, and while it lasted she was unable to hear distinctly. Then they began with a sudden sensation of being struck on the head, just behind the vertex. Sometimes she fell and it seemed to her that the fall was the consequence of the blow. She never lost consciousness, nor had she any definite perception of movement at the onset; the sense of the blow, with the fall, seemed to prevent any vertiginous feeling, but, after the fall, she was distinctly giddy for an hour, usually with nausea and sometimes with vomiting. The fall naturally caused a feeling of alarm and sometimes of dyspnoea. She also had slighter attacks, with the sense of a much more gentle blow and a tendency to fall forward, which also seemed to her the direct effect of the blow. After such an attack there was a slower movement of objects to the left and back again, and she sometimes fell to the left. Even when sitting still she occasionally had the feeling of the sudden blow, which seemed to impel her forwards. Occasionally she almost lost consciousness, and it was actually lost in one attack. Afterwards the attacks occurred also during sleep (a point to be considered presently), and she was once waked up from sleep by a sensation of being struck on the right side of the head, and she seemed to be knocked over to the left by it. She afterwards remembered putting out her hands to save herself, but nothing more until she found herself on the ground. Her right-sided deafness steadily increased, hearing being lost from above down until she could only perceive the bass C ($C_1 = 125$).

That the vertigo in this case was purely aural we cannot doubt. Some special character in the process may have determined the perception of a seeming cause, but the constancy with which the feeling of a blow accompanied the attacks, whether they were slight or severe, is remarkable.

So definite a sensation of a blow is very rare, but other

cephalic sensations at the onset of the attack of giddiness seem to be of the same general nature. It was described as a "clutch" in the case of a single woman of 29, in whom the aural nature of the vertigo was less certain than in the cases I have previously described, since there was no deafness. But the patient had persistent tinnitus in the left ear, and such tinnitus means irritation of some of the labyrinthine structures, which, however trifling, may have a disturbing effect on the centres out of proportion to its intrinsic degree, and may be greater in the part of the labyrinth concerned with equilibrium. The patient had brief attacks of giddiness, in which the ground seemed heaving up and down, and each began with a sudden momentary sensation of a "clutch" in the head. Later, this clutching sensation occurred when going off to sleep, and was followed by palpitation of the heart, the tinnitus became much louder, pulsating like a steam engine at work, and she wondered that other people did not hear it. Sometimes it changed for a short time to a loud continuous hiss, compared to an engine blowing off steam. Strange to say, her hearing continued perfect, but there must have been definite labyrinthine irritation, sufficient to make it probable that the other symptoms had the same origin. This would not have been suggested by her nocturnal sensation, which, considered alone, bore a striking resemblance to an attack of minor epilepsy.

NEURASTHENIA: ITS NATURE AND TREATMENT.

By DAVID DRUMMOND, M.A., M.D., D.C.L.,

SENIOR PHYSICIAN, ROYAL INFIRMARY, NEWCASTLE-ON-TYNE; JOINT LECTURER ON PRINCIPLES AND PRACTICE OF MEDICINE, UNIVERSITY OF DURHAM COLLEGE OF MEDICINE.

THE NEURASTHENIC CONDITION.

THE importance of this subject at the present time arises on the one hand from the fact that the condition of things known by the rather unsatisfactory name of "neurasthenia" is very greatly on the increase through all ranks of society, and on the other because instruction respecting it forms no part of the curriculum of the medical student, nor of the training of the nurse. Year by year cases of neurotic illness assume even larger proportions in the work of the general practitioner, and his inability to deal with them successfully is both a disadvantage to himself and the occasion of much avoidable pain, discomfort, and protracted invalidism in his patients.

By a curious and unfortunate misconception "neurasthenia," and in a tenfold greater degree "hysteria," have come to be regarded as terms of opprobrium, as though their victims were, as such, a kind of moral delinquents wilfully hugging maladies of a more or less imaginary character, which a mere fiat of their own might abolish if it so pleased them. It is to be hoped that in the light of fuller knowledge and more discriminating judgement of conditions this hurtful misconception will before long take its way into the limbo of other time-honoured but exploded prejudices.

Neurasthenia, as it comes under the doctor's notice, shows itself physically in a wide variety of ways, of which one of the commonest is general malnutrition, often leading to great emaciation. There may be indications of numerous forms of illness for which on examination no physical basis can be discovered—dyspepsia and vomiting without disease of the stomach; cardiac symptoms, even anginal in character, with a perfectly sound heart; paralysis or spinal pain, with reflexes intact; altered secretions from organs in a normal state. Or, again, there may be no local disturbances, but only general debility and depression—a state in which the patient can do nothing so well as formerly, and finds every little exertion a trouble; and often enough this is accompanied by a feeling of insecurity and self-distrust—a dread of facing life and its demands.

This condition of things may have been brought on by a sudden shock—some fright or distress—though in such a case it usually does not appear at once but after an interval of incubation. But very much oftener it is the upshot of a long, gradual deterioration in health due to some steady cause of wear and tear. The important point to grasp and keep in mind firmly is that the physical state is *secondary*. The originating cause is always to be

sought for in the patient's mental life and history. So markedly is neurasthenia a disorder or disease of the mind that the gravest cases—such, for instance, as those in which the patient is subject to one fixed idea, often most difficult to dislodge—reach a border-line where they cannot with certainty be distinguished from the still more unfortunate persons who must be definitely handed over to the alienist. These, however, are extreme cases, and are, happily, not numerous; and, in the great majority of instances, it is comparatively easy to decide whether a given patient is or is not likely to respond to the treatment appropriate to neurasthenia and to be successfully treated.

But at present the neurasthenic patient complaining of some definite local pain or disturbance runs two opposite risks at the hands of an inexperienced practitioner: Either the pain and other symptoms may be taken to indicate the existence of some recognized physical disease for which the patient is in consequence treated by the ordinary recognized methods; or the doctor, finding no physical grounds for the symptoms, concludes, and perhaps gives his patient to understand, that there is nothing really the matter and nothing to be done. On the contrary, there is something really wrong, only it is not what the patient thinks, and has to be combated by means quite other than those directed to the cure of organic diseases.

This point has to be specially emphasized, that neurasthenia is essentially an affection of the *mind*; and that in consequence of its prevalence we medical men are in fact called upon to treat—perhaps more frequently than any other class of ailments—mental disease, and that often at a stage when as mere functional disturbance it is fully amenable to treatment. This fact becomes all the more striking and worthy of attention when we think that not one out of a hundred medical men knows anything of even the elements of psychology, and most doctors are consequently unable to explain to patient and friends intelligibly, and in a fashion suited to their requirements, the nature of the disturbed or perverted function. Such an attempt at explanation is often made, but only too often the result is disastrous alike to the patient's feelings and the doctor's reputation. Frequently it ends in the patient's going off to consult another doctor, either because the language employed is capable of bearing another interpretation than the one intended, or because it cannot carry conviction when the necessary tact and confidence begotten of knowledge are lacking. How often have I been consulted by a patient glibly dubbed a "neurotic," who informed me that the doctor said there was nothing the matter, and on my rejoinder that I was sure Dr. So-and-So could not have meant that, has replied, "Well, if he did not say so he implied it." Neurasthenia must be regarded as a real disease, something to be diagnosed and treated. The diagnosis may not be easy; but it can be made, after an honest and searching inquiry the aim of which is to exclude organic disease, and then when made it must be adhered to with courage, and the proper line of treatment adopted.

Where, then, are we to look for some central explanation of the protean variety of symptoms exhibited by different neurasthenic patients—some master-key to fit locks of apparently such varied patterns?

Let us consider very shortly our ideal of a normal, healthy human being, of sound mind in sound body, and possibly it will help us to find out where and how the neurotic patient breaks away from the normal. We recognize that health is wholeness, and that the whole being is one whose personality functions—that is, feels, thinks, acts—through and by the aid of an enormously complex organism in which, as in a well-governed State, each minutest part has its assigned work to perform in harmony with all the rest. Little by little, through the continuous efforts of myriad generations, has this organism been evolved to its present stage in which, through tiny acts countless times repeated, much that must at first have been achieved with difficulty is now, as it were, so registered in the structure as to go forward automatically—that is, without conscious attention or effort on the part of the present head of the State. Intelligence marks every step, and is still in control, but now it is intelligence so drilled and educated as to carry on its business without continual supervision from the conscious will; it works, as we say, subconsciously. So the reflex movements follow their