SOCIETIES' PROCEEDINGS

THE SCOTTISH OTOLOGICAL AND LARYNGOLOGICAL SOCIETY

(The Forty-fifth Meeting of the Society was held in the Victoria Infirmary, Glasgow)

May 29th, 1937

President—DR. J. P. STEWART

I. Aural Vertigo

DR. G. B. BRAND

It is with more than a little diffidence, that I endeavour to present to you an opening paper on the subject of " Aural Vertigo ".

I have nothing original to contribute to our knowledge of the subject, and my idea is to bring before you, as clearly as possible, the advances which have been made in our knowledge of this difficult and fascinating branch of our work.

In order to justify, in a way, my position this afternoon, I must give you a little history. I had the good fortune about twenty years ago to be living in the same Mess somewhere in Egypt, as the late Mr. Tweedie. He was such an enthusiast, that even on Active Service he was in possession of a Bárány Chair, and for several months we worked most evenings at the various tests. He would have been a dull dog indeed who would not have been imbued with enthusiasm under such a master.

As we all know, Mr. Tweedie's interest in the subject persisted during his life, and his untimely death was a serious loss to our specialty, as he was still actively engaged in elucidating the mysteries of the various labyrinthine reactions.

To the memory of a great aurist and staunch friend, I pay my tribute.

My enthusiasm has waned with the years, but is now vigorously waxing again, as the result of the study required for this paper.

(1) *What is Aural Vertigo?*

In the first place, let us be clear that it is not a disease. It is only a symptom. W. Russell Brain says, that while there is no

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one feature pathognomonic of aural vertigo, there are features in most cases that make it easy to recognize. I propound the definition that in order to be aural, vertigo must have a sense of rotation, with, or without visceral symptoms, e.g. pallor, nausea, sweating, vomiting, etc., but without loss of consciousness, tinnitus or nystagmus with past-pointing during the attack, and some demonstrable affection of the endorgan of hearing, e.g. diminished hearing, and altered excitability of the vestibular function.

Now a short epitome of the vestibular paths.

Labyrinth—Deiter's Nucleus—and some fibres to the cerebellar cortex. From the vestibular nuclei, fibres enter through :

(A) Post-longitudinal bundle and end in certain cerebral nuclei.
  (*a*) The vagus which accounts for visceral symptoms.
  (*b*) The eye centre which accounts for the nystagmus, and
  (*c*) The nucleus of the accessory which controls head movements.

(B) Certain fibres pass downwards to end around motor cells in the anterior horn of the spinal medulla—to explain alterations in muscle tonus.

The cerebellum is the centre of equilibrium and co-ordination. Fibres pass from the vestibular nuclei to the cerebellar cortex : to the nucleus dentatus then, *viâ* the brachium, to the red nucleus of the opposite side : whence, *viâ* the rubrospinal bundle they cross the middle line to the motor cells in the spinal medulla.

Thus each half of the cerebellum has a connection with the motor cells in the medulla of the same side.

(2) *Physiology of balance and vestibular tests.*

The endorgans of the vestibular apparatus are the saccule, utricle, and the three semicircular canals, and since the days of Flourens, over a hundred years ago, increasing numbers of investigators have been working at this bewildering function of balance and orientation. We are still very much in the dark as to the functions of the various endorgans of the labyrinth. Flourens stated that nystagmus is independent of the brain and cerebellum, and De Kleyn says that it is only necessary to have intact endorgans, vestibular and abducens nuclei, abducens nerve, and external rectus muscle. So that the essential centres must lie in the midbrain.

Have the semicircular canals and the utricle anything to do with skeletal muscle tone, and has the saccule anything to do with the equilibrium ?

We must remember that embryologically the saccule and the cochlea are developed from the same bud of the otic vesicle, whilst

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the utricle and canals are developed from another. Recent work by Tumarkin and McNally, are well worth study, but do they give us much help clinically ?

I quote here from Tumarkin's paper. " In his latest work on the labyrinth, Professor Rigaud makes the following statement. ' Although vestibular examinations have been in widespread use for over twenty years, yet we are still arguing about the nature of the nystagmic tests : about their correct performance and their diagnostic value, etc.,' and finishes by saying that ' the clinician is quite unable to derive any benefit from the results of their obscure researches.' " The main reason for this, is that so far no one has been able to discover a simple method of measuring the labyrinth sensitivity.

Tumarkin's work deals with the fallacies of the rotation test, chief of which is excessive stimulus, and he rotates his patient, suspended in a tank of warm water, blindfolded and with the meatuses stuffed with plasticine, in order to eliminate tactile sensibilities, vision, and hearing.

His work is very ingenious, and manifestly quite unsuited to the consulting room.

In my opinion the recent work of McNally is most important, and must tend to be of great help clinically, in time. McNally favours the tilting test, and has done, and is doing wonderful work with the frog's labyrinth.

He says that the semicircular canals are in close connection with the whole postural body musculature. Nystagmic reactions are only a small part of the semicircular sphere of control. He has been able to isolate the various endorgans in the frog, and has investigated the results with the slow and quick tilt.

He has found that certain stimuli invariably produce characteristic reaction patterns of the musculature. The recognition of the characteristic pattern for each canal will ultimately be of great clinical help.

The utricles and vertical canals have connections with the same muscle groups, and produce the same muscle patterns. With a quick tilt backwards the utricles produce the same pattern as a quick tilt forwards of the vertical canals, and *vice versa*, so that their reactions are compensatory. Ablation experiments on the frog have proved the saccule to have no equilibrial function.

It probably has a cochlear function.

This is in accord with its anatomical development.

The vestibular tests.

I think that most of us nowadays rely on the caloric test for clinical purposes ? The value of the tilt test clinically has to be proved.

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(3) *How much use can we make of the caloric test in suppurative conditions?*

In the chronic cases without exacerbation, there may be little harm in the cold douche, but few of us will have the temerity to apply it in acute cases.

In these latter, Dundas Grant's cold air douche is a safe method of applying the test.

(4) *Differential Diagnosis.*

The routine examination of the nose, throat and ear will reveal the causes of aural vertigo.

[1] Unilateral Eustachian deficiency.
[2] Acute and chronic suppurative otitis media. Some with fistula symptom.
[3] Cases of old suppuration with scarring and adhesions.
[4] Cerebellar abscess.
[5] Otosclerosis.
[6] Labyrinthitis.
[7] Septic foci, tonsils, teeth, sinuses, unerupted teeth, etc.
[8] General toxic conditions of the ear due to syphilis, alcohol, tobacco, etc.
[9] Tumour of the VIIIth nerve,

and a large number which are grouped under the heading Ménière Syndrome, which we may designate " Aural vertigo of unknown origin ".

Vertigo is a common symptom of injuries to the head. The dizziness is of a fleeting type, and is usually accompanied by headache and cerebral irritability. Another common type of vertigo is probably due to vascular disturbances—hypo- and hypertension. It is usually transitory and not rotatory in character, and as a rule can be produced by stooping or sudden rising from a recumbent position. Förster has produced vertigo experimentally in man by stimulation of the cortex anterior to the Rolandic area.

Petit Mal has often an aura of vertigo.

It is usually sudden and momentary in duration. There is, as a rule, a loss of consciousness.

Polycythæmia. Bieling reports a case with over 6,000,000 red cells and high hæmoglobin content.

(5) *Ménière's Syndrome.*

Now we come to a very controversial part of the subject. What is the mechanism of the vertigo, nystagmus, the tinnitus, and the variations in hearing?

Is the condition a local labyrinthine one, or is it due to an affection of the VIIIth nerve, or even more central still?

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Is it unilateral or bilateral, and which is the commoner?

Is a very mobile stapes footplate conducive to labyrinthine disturbance, and can this help to explain the readiness with which some individuals develop travel sickness? Do individuals who are good sailors, etc., ever develop Ménière's syndrome?

Given a physical condition in which vertigo is easily produced. What are the exciting causes?

Why, in longstanding cases of Ménière syndrome, does it so often happen that as the deafness increases, the attacks of vertigo tend to disappear?

Chilow's animal experiments showed that increased pressure in the external meatus raised the fluid in the manometer, in the animal's labyrinth several millimetres, and on releasing the pressure it does not reach its normal level. Is the attack due to increased pressure in the labyrinth? Can it occur owing to disorder of the water metabolism? (Kopetzky—Dederding.) Sodium sensibility? (Furstenberg.)

Is it possible that there is a condition in the labyrinth analogous to choked disc in the eye?

Can protein sensitization produce a sudden labyrinthine pressure?

(6) *Treatment.*

Indications for operation for relief of vertigo. Every means of local and general treatment having been exploited, what can we do for this distressing and crippling syndrome?

In my experience the most successful treatment is ½ gr. luminal twice daily.

Should this fail, and the condition be definitely unilateral, operation must be considered.

Portmann devised an operation on the saccus. Many operators divide the VIIIth nerve, and others attack the labyrinth itself. In the hands of W. M. Mollison, alcohol injection has proved very satisfactory.

Operation cannot be entered upon light-heartedly, but on the other hand, it should not be withheld in suitable cases.

DISCUSSION.

Dr. IVY MACKENZIE said it was only this morning that he was made aware of the test to which he was to be put this afternoon. It did make him feel a little giddy when he heard about it, and he was not sure that he could contribute anything very definite to the points which had been raised. Giddiness, or dizziness was a symptom which was brought to the notice of the physician every day, and for a number of years he had been interested in the anatomy of the brain, and what

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he had to say would have reference to some extent to the structures, the perversion of whose function underlay this very disturbing disorder. Of course there was a great deal of speculation about the cause of dizziness and vertigo. Dr. Mackenzie was reminded of a story he had heard about a man who, finding his way home, arrived at Charing Cross in Glasgow, and having indulged rather freely, was suffering to some extent from toxic vertigo. He noticed a man in the middle of the street turning a water key, and he went up to him and said, " So it is you that's turning the blooming street round about ! " Dr. Mackenzie thought most people were not very much better informed than this speculator as to the actual causes of giddiness. As soon as he got to know anything about anatomy in the practical sense, he had been very interested in the vestibular apparatus, which lay at the very root of the integration of the whole nervous apparatus.

In the fish, the lateral line organs have to do with the orientation of the body in water and the vestibular nuclei are closely related to those of the lateral line organs. The vestibular apparatus has really got to do with the forced movements of the body as a whole in which the nystagmus in the Bárány Chair is the ocular expression of reaction. But, Dr. Mackenzie said, he would just like to point out the way in which he himself approached giddiness as a symptom and give one or two examples of the clinical syndromes related to disorders in which it played a part. Giddiness may be regarded as a disorder of special sense in some cases, as due to affections of the labyrinth with which Dr. Brand has dealt so clearly.

The vestibular apparatus may be compared with the eye. It is obviously from the anatomical point of view quite a sensible proposition that the various parts of the labyrinth are related to certain parts of the muscular conformation, just as the homonymous halves of the retina are related to the contralateral musculature, or one eye in the fish is related to movements on the opposite side of the body.

Labyrinthine disorders apart from local disease are often difficult to recognize because there are other clinical phenomena incidental to the close association between the vestibular, trigeminal and vagal nuclei in the medulla. In the medulla the trigeminal nuclei lie right up against the trigeminal and vagal nuclei. It is not a question of Deiter's nucleus alone ; these three nuclei are all in one bunch and that accounts for the very frequent association of headache, related to the trigeminal nuclei, vomiting to the vagal nuclei and giddiness to the vestibular nuclei. That is to say giddiness, headache and vomiting all go together in far reaching association, and are often provoked by the poisons of fever or migraine. Dr. Mackenzie said he should call these secondary symptoms, related to toxic disorders.

Now there are the phenomena of giddiness that are related to abnormalities quite a distance away from the vestibular apparatus or the medulla. Dr. Brand had referred to the co-operation between the otologist and the physician in the diagnosis of cases. He (Dr. Mackenzie) had very frequent cause for appealing to Dr. Brand's generosity in these matters, and he would like to emphasize one or two points in which the otologist was of very great help to the physician.

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A great number of the cases of giddiness with nystagmus were quite independent of the vestibular apparatus. His attention was first drawn to these a number of years ago, when encephalitis appeared in epidemic form. Acute encephalitis sometimes manifests itself through forced movements which at first sight may suggest cerebellar or vestibular disorder. That is to say, the patients had abnormal ocular movements, rolled about in a peculiar way and showed signs of disturbance which one might quite well relate to cerebellar disease. When, later, these patients went into that stage known as Parkinsonism, they sometimes showed a little nystagmus. Dr. Mackenzie said he was very much interested in that, and for two or three years used the Bárány Chair for examination of these cases of Parkinsonism and a very interesting fact emerged. It was this, that a case of Parkinsonism after rotation on the chair would walk straight to the other end of the room without any feeling of giddiness at all; that is to say, the apparatus which is concerned in the restricted movement of this extra-pyramidal disease lies outside the immediate connections of the labyrinth. It reminds one of one of the effects of complete destruction of the labyrinth. A most interesting phenomenon in connection with ablation of the labyrinth is that deaf mutes, for example, do not become seasick, and when diving into the water are not able to find their way up; that is to say, the labyrinth has something to do with orientation in space, and in a more automatic way than pertains to the organ of vision. At one end of the vestibular apparatus you have the labyrinth itself, whose ablation has the effect of rendering the body insensitive to certain forced movements, while in the basal nuclei (pallidum, striatum and substantia nigra) you have distant relations to the vestibular nuclei whose destruction gives rise to interference with forced movements comparable with that which follows ablation of the labyrinth. We are thus in a very wide field of brain anatomy when considering the problem of giddiness in relation to the vestibular apparatus.

Dr. Mackenzie gave another example which, he said, really accounted for his presence there. He asked Dr. Brand to see a patient who for the last two years had been suffering from very profound giddiness—a woman of 60 years of age who had had progressive giddiness over a period, not associated with headache, or with vomiting, or with nystagmus. She had a certain monotony in her speech, and when she walked she was stiff and stooped. There was a superficial resemblance to Parkinsonism but nothing to suggest cerebellar disease. Her vision was quite good or had been quite good till within the last week. It was difficult to examine her visual fields but they seemed to be normal, and her fundi showed no signs of disease. In addition to her complaint of giddiness, she had a stare in her eyes and on examining their movements it was found that she could not look up above the horizontal line of vision. She could look down a little but that varied from day to day.

Now, it was very important in a case of that kind to exclude a vestibular component in the syndrome and that Dr. Brand was able to do. There was no evidence from experiment that the vestibular apparatus had anything to do with the vertical movement of the eyes, though some of the cases of vestibular nuclear disease from thrombosis

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of the posterior inferior cerebellar artery, for instance, showed a vertical nystagmus. This woman, on examination, showed what one would regard as a perfectly normal reaction to the caloric test. The presumption was that her vertical ophthalmoplegia was due to an interference with supra-nuclear fibres or tectal connections in the mid-brain.

Dr. Mackenzie said he had been very much interested in listening to what Dr. Brand had said because it really opened up an enormous field of clinical interest in which the otologist's knowledge of the labyrinthine reactions was of very great help to the physician in determining the site of disease. One point he (Dr. Mackenzie) discussed with Dr. Brand was the question of nystagmus in cerebellar disease. He was not sure whether cerebellar disease in itself did give rise to nystagmus; he was very doubtful about it. He thought the presumption was that when nystagmus did occur it was usually due to increased tension from an abscess or tumour in the cerebellum pressing on the vestibular nuclei in the medulla. The presence of nystagmus would be in favour of a diagnosis of cerebellar disorder, but one might quite well get a cerebellar abscess or tumour without any nystagmus.

Mr. PATERSON said that when he was invited to attend the Meeting he did not expect to have to say anything, and anything he had to say would be very brief and would simply be in connection with the operative treatment of these cases of Ménière's disease. It was a subject for discussion as to which was the best method of securing an abolition of vestibular function. The only cases in which he had divided the VIIIth nerve for Ménière's syndrome were those two which Dr. Howie would be talking about presently, one of which was shown this afternoon. The operation of intracranial division of the VIIIth nerve was not a difficult one but, as in all other procedures in which the posterior fossa is exposed and entered, there would, of course, be a certain amount of risk. It was probably less dangerous to a considerable degree than was similar section of the Vth nerve. The Vth nerve lay farther forward in the cerebello-pontine angle and it had been divided by the cerebellar route by Dandy, who had had an astonishing series of successes, with practically no mortality. It was a question, of course, whether otologists could secure the same results by perhaps a less drastic method. Mr. Paterson said there was one other method which he had been interested in but he had not had an opportunity of carrying it out. It seemed to meet the case. He understood one or two such operations had been carried out in America. The operation consisted in making an opening in the temporal fossa, and elevating the dura from above the petrous bone, exposing the projection of the superior semicircular canal and chiselling off the projecting dome of bone. A tiny stilette from a hypodermic needle was passed into the little canal and was connected with the diathermy apparatus and the vestibular mechanism was more or less cooked with the diathermy. Mr. Paterson said he would be interested to hear how the otologists regarded that operation. The point about it was that it was aseptic. It did not involve in any way entering the middle ear and, of course, *if* diathermy was not satisfactory, he thought alcohol could be injected

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in that way. He could only add his testimony to Dr. Mackenzie's and say how much he had appreciated Dr. Brand's excellent summary.

Dr. ADAM said that to prevent our becoming too surgically minded, he would like consideration of some facts afforded by some recent cases of Ménière's syndrome, facts pointing to (1) vasomotor spasm in the internal ear, and (2) to vitamin deficiency.

(1) Since finding in an asthmatic, aged 51, a normal pressure of 120/85 in one arm and only one of 40/20 in the other, although X-ray subsequently showed nothing abnormal in the chest and the pressure had become normal in both arms, he had been testing pressure in both arms. A woman of 28 who has had occasional Ménière attacks for three years, latterly more frequent and severe, had after her last attack a systolic pressure of 20 points less on the side of the affected ear. This suggested vasomotor spasm as does the picture of the typical Ménière attack. Is Ménière's syndrome the aural analogue of migraine? In migraine, spasm of the retinal vessels has been seen through the ophthalmoscope, spasm affecting only one eye, and sometimes only certain branches of the retinal artery in that eye.

(2) Again, in the case of the woman just mentioned, repeated examination of her urine revealed no ascorbic acid (vitamin C). She hardly ever ate raw fruit and, like two other patients with Ménière's syndrome, has improved since taking an adequate amount of vitamin, with special attention not only to C, but also to B and D. She was also given Betaxin intramuscularly. In this connection, it is worth remembering that vitamin C is essential for adrenal functions, as is vitamin B for the nutrition of the nerve cells. Therefore, one should keep both sets of facts in mind before cutting the VIIIth nerve.

Dr. DOUGLAS GUTHRIE said that no one who had seen Dr. McNally at work in Montreal could fail to admire the delicate technique of his operations on the frog's labyrinth. The results were not yet systematized for clinical application, and the usual tests were of little value in cases of aural vertigo. Dr. Brand had suggested that an undue mobility of the stapes might be responsible, but, of course, this could not apply in otosclerosis. Operative methods were not entirely free from risk, and even section of the auditory nerve or of its vestibular component did not cure every case. Mygind and Dederding had pointed the way to rational conservative treatment, and Furstenberg and his colleagues had gone one step farther in advocating a sodium-free diet and the use of ammonium chloride. The latter was probably the best form of treatment at our disposal and had yielded good results in the speaker's experience.

One essential point in the treatment of aural vertigo was to ensure the patency of the Eustachian tube. Inflation had cured many cases of temporary vertigo in aviators, and many other cases of longer duration. It was interesting to note that although Ménière's syndrome had long been recognized, the original case observed by Ménière was not accurately recorded. Indeed, Dr. Dan McKenzie, in a paper published some years ago, had referred to the "myth of Ménière's

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disease ". But whether accurate or not, Ménière had rendered great service by associating vertigo with a lesion of the semicircular canal.

Dr. DONALD said he had a personal experience with regard to this Ménière's disease. Six years ago he had been over in Toronto and, coming over at the beginning of *November*, there had been a pretty rough sea, and for a day or two he felt the movement of the ship though he had not been sick. About the second night after coming home, he wakened with a terrible vertigo ; everything moved up and down and he vomited. He had noises in the ear and deafness. He was thoroughly alarmed, but this passed off in twenty-four hours or less, and in another day or two the same thing came on again very violently. He had to lie on the floor and shut his eyes. He had, in all, about ten attacks, with a few days between. He went to his old chief, Dr. Galbraith Connal, whom he (Dr. Donald) was assisting at the time. Dr. Donald said that what interested him just now was—could Dr. Brand say what are the main causes of Ménière's disease ? Dr. Connal in those days did think there was such a disease, and he said that he (Dr. Donald) had had the symptoms without the disease, and his treatment was inflation of the Eustachian tube by catheterization. Dr. Donald wondered whether the movement of the ship could have caused these symptoms, as he was not troubled with his ear at all. Since then, the question had arisen in his mind as to whether there could have been anything in the drinking water, as Dr. Connal had considered the condition one of toxic origin.

Dr. FULTON CHRISTIE said he was interested to hear what Dr. Guthrie said. He, also, had found that examination of the VIIIth nerve in such cases often revealed no abnormality. This, however, was only to be expected when it was remembered that the vertigo and deafness usually occurred in attacks, and between the attacks the patient felt perfectly well. It was during this period that they were almost always seen. He was strongly of opinion that the history is the most important part of the investigation, and that a considerable time must be spent in getting a full and accurate history.

He said that the conditions causing " aural vertigo ", so called, were numerous, and he thought it might be of interest if he summarized the causes in 110 cases in which he had done functional tests during the past four years :

1. *Local Inflammatory.*
   1. Labyrinthitis.
   2. Labyrinthine irritation.

These conditions account for more of our cases than any other single condition. They are the result of suppurative disease of the middle ear with or without cholesteatoma. Tuberculosis of the middle ear was the cause in one case.

It is interesting to note that in a series of 117 cases of chronic suppurative otitis media in which the radical mastoid operation was performed, in the Western Infirmary, Glasgow, thirty-nine had vertigo as a symptom at some time prior to operation, i.e. one-third exactly.

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2. *Blood Vascular.*

1. Anæmia. We have had several cases with low hæmoglobin.

2. Arterio-sclerosis. When arterio-sclerosis and high blood pressure are present attacks occur on sudden change of posture, e.g. on rising in the morning, this probably causes a temporary ischæmia. I do not think that high blood pressure *per se* is a cause of vertigo. Low blood pressure may cause it.

3. Angio-spasm. Cf. Lermoyez syndrome.

In two of our cases, in which the symptoms suggested spasm of the internal auditory artery, the appearance of the drum membranes resembled that found in otosclerosis. I would be interested to know if anyone present has noted this association.

3. *Constitutional.*

1. Syphilis. Usually characterized by rapidly increasing deafness.

2. Endocrine dysfunction.
Hypothyroid (?)
Menstrual.

While we have noted endocrine upset as a cause in few cases it is probably a factor in a great many cases acting through the vascularity of the part. He agreed with Dr. Adam that the adrenals are probably of much greater importance in this connection than is generally thought.

4. *Toxic.*

1. Auto-intoxication. Dental caries and unhealthy gums I should rank first. Constipation, unhealthy tonsils and sinus disease follow close behind.

2. Drugs. Salicylates over a long period was the cause in one instance. Tobacco seemed to be a factor in some cases.

5. *Degenerative.*

Disseminated sclerosis affecting the cerebellum was the probable diagnosis in about three cases.

6. *New Growths.*

We have had no proved case of VIIIth nerve tumour or other new growth. Our abscesses of the cerebellum have been diagnosed without functional tests.

7. *Trauma.*

1. Mechanical. There have been several cases following fracture of the base of the skull. The original line of fracture has been doubtful in all. The symptoms in some suggest that the fracture has not been through the labyrinth but that the symptoms are due to adhesions affecting the VIIIth nerve.

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2. Post-operative. Occurring after radical mastoid operations in cases with well healed dry cavities.

8. *Disorders of Function.*

1. Functional deafness. In these cases the reaction to the caloric test is normal. We have had several such cases.

2. Worry is probably a factor in many cases.

3. Other psycho-neurotic causes.

9. *Miscellaneous.*

Affections of the eye, etc.

One case of " laryngeal vertigo " was investigated and, as expected, the reactions were absolutely normal.

He said that he had time to mention Ménière's syndrome only briefly. He had read four medical text books used by students and found that there was comparative unanimity in the description of the symptoms. The description of the deafness varied and loss of consciousness was mentioned by two only. When one came to the ætiology all resemblance ceased, and one might have been reading about four different conditions. He was quite sure that everyone at the Meeting knew exactly what he meant when he spoke of Ménière's syndrome, but he ventured to say that there were not two present who meant exactly the same thing when speaking of this syndrome. He thought that Ménière's name should be relegated to the history of medicine on account of the confusion at present existing.

In his article on Ménière's original case, Dr. Dan McKenzie said, " So stealthily has the story (I had nearly written myth) of Ménière's original case insinuated itself into our otological literature, that we are all in the habit of looking on its data, its traditional data, as proved, and have little or no hesitation in basing theories and explanations of various kinds upon them."

Dr. Albert Gray, in his text book, *Diseases of the Ear*, said, " The origin of this expression is to be traced to the common mistake of assuming the existence of a pathological and clinical entity from a paucity of observed facts."

Dr. Simson Hall said that he would like to thank Dr. Brand for the admirable way in which he had covered the ground and the wide scope he had allowed for discussion. He (Dr. Hall) would try to limit his remarks to one or two points which he would like to discuss.

In regard to the general examination of a patient, he did not share Dr. Brand's optimism with regard to the tilt-table test. Though he had been in the habit of using this test as routine for a good many years, Dr. Hall found that he got very little help from it. He had, therefore, abandoned it except for special cases, although he had had the privilege of seeing Dr. McNally's work in Montreal, and had watched him at work upon his experiments with frogs.

Ménière's disease, he said, was a subject which had interested him for a considerable period. In association with Mr. Norman Dott, he

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had seen a large number of these cases and had been able to note the effects of treatment upon them. Although the name "Ménière's disease" was an unfortunate one, he believed that there was a syndrome for which the name ought to be reserved. Time was lacking to describe exactly what comprised the syndrome; but there were certain features which, in his opinion, rendered it distinct from cerebellar and other lesions which were sometimes classed along with Ménière's Syndrome.

In Edinburgh, continued Dr. Hall, patients suffering from Ménière's disease were separated into two groups—the primary or essential form and the secondary form. The primary form usually provided a clear-cut clinical picture, while into the secondary form fell a certain number of variations and the more indefinite types of the disease. It was in this second group that occasional difficulty was found in classifying the patients.

Cause presented a very difficult problem and, regarding high blood-pressure, it was interesting to note that he had seen one case—that of a scientific medical man—in which the patient had attacks of Ménière's disease and at the same time suffered from low blood-pressure.

In treatment, Dr. Hall tried first of all the effect of anti-retentional régime. In doing so he covered the point raised by Dr. Adam, namely, that the diet of these patients was usually deficient in vitamins, for patients were given a diet sheet which he (Dr. Hall) believed adequately supplied what vitamins were necessary.

Inflation of the Eustachian tube was also carried out as part of the routine, but had rarely been found to have any influence on the disease.

With regard to the question of operation, several different types had been tried, and the operation upon the superior canal was now considered to be the easiest and most satisfactory. But in cases in which the hearing was fairly good, fractional section of the VIIIth nerve had been carried out by Mr. Dott, with the result that in one case only 18 per cent. of the hearing had been lost.

The problem which had engaged their attention recently was an attempt to destroy vestibular function and at the same time retain cochlear function. Several different methods had been tried but so far had met with no success.

Dr. ADAM said he would like to ask Dr. Hall a few questions. Had any of the cases lost consciousness? Had any of them migraine? Were they mostly cases with low blood-pressure?

Dr. HOWIE said that nobody had mentioned in connection with physiology, the instance of learning to waltz. At first one developed aural vertigo, with definite nystagmus. When one mastered the dance, the vertigo disappeared but actually nystagmus remained. The other symptoms became subconscious.

The other point was in connection with toxic nystagmus. One's first cigar, Dr. Howie believed, gave vertigo with nystagmus. Many of Dr. Christie's cases, Dr. Howie thought, were suffering from cold caloric reaction, from cold air entering through an enlarged meatus and striking against the horizontal semicircular canal, which was covered by only a thin layer of skin.

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CASES I and II (Drs. James Adam and E. A. M. Connal).

CASE I.—*Acute labyrinthitis following chronic suppurative otitis media; onset of meningitis; double vestibulotomy; pneumococcus persistent in cerebrospinal fluid: recovery.*

Wm. R., aged 39. Admitted to Stobhill 21.2.37, complaining of vomiting and giddiness for four days. He had a history of influenza a month before; later he had pain in right ear followed by discharge. Four days before admission vertigo and vomiting developed, becoming progressively worse until, on admission, he was vomiting incessantly.

Temperature 97° F. Pulse 100; nystagmus to left; Romberg's sign positive (falling to right). Past-pointing was present only during the attacks of giddiness (error to right) and then he tended to fall to the right. There was over the right mastoid slight swelling and tenderness but no œdema or redness.

Hearing—left ear normal; right, whisper at 2 in., conversation at 40 in.; Rinne negative; Weber to left; hearing present in right with noise-box in left.

Lumbar puncture—fluid clear, not under increased pressure (one cell per c.mm., globulin not increased, sugar normal, chlorides 670 mgm.).

As the cerebrospinal fluid was almost normal, the temperature not raised and some hearing existed in the right ear, operation was postponed in order to give the labyrinthitis a chance to settle. Two days later, however, the temperature rose to 100·2° F., nuchal rigidity and pain developed. Kernig's sign was absent. Lumbar puncture gave turbid fluid not under increased pressure—5,106 cells (mostly polymorphs), greatly increased globulin, no sugar. On films a few Gram-positive cocci were seen in chains and clumps, some failing to retain the stain, but culture gave a pure growth of pneumococcus, type III (pneumococcus mucosus). Pus from the mastoid gave a pure growth of the same organism.

Operation (by J.A.); radical mastoid and double vestibulotomy. Incision of tegmental dura; the facial nerve was exposed during the operation.

Lumbar puncture was repeated the same evening, done twice the next day (fourth day) and daily thereafter till the twenty-second day, except on days seven, fifteen and twenty-two. On days seven and twenty-two, owing to the difficulty of obtaining fluid by lumbar puncture, cisternal puncture was done. Lumbar puncture was also done on days twenty-seven and thirty. Altogether the theca was tapped twenty-two times. Fluid throughout tended to be scanty and on practically all occasions was not under pressure. After tapping, air was injected as routine in amounts varying from 6 to 20 c.cm. After fifteen of the tappings,

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fluid was sent to the laboratory and each time gave a pure growth of the pneumococcus. After the sixteenth day the colonies decreased until on the thirtieth only a few were got and then failed to retain Gram's stain. Sugar began to return to the cerebrospinal fluid on the nineteenth day and was normal on the twenty-seventh.

Antipneumococcal serum was given intrathecally on days four and five, that is, before the organism was typed. Transfusions of whole blood (300 c.cm.) were given on days nine and eleven. Prontosil was given intramuscularly twice or thrice daily from the first and sulphonamide also (two tabs.) four-hourly from the eleventh day. And, as Peters has given ground for believing that vitamin B plays an important part in the carbohydrate metabolism of the brain and as sugar was absent here from the cerebrospinal fluid, a c.cm. of Betaxin was injected intramuscularly daily from the seventh.

After the operation on mastoid and labyrinth, there was facial paresis. By the sixth day the patient's mentality was clearer, his nuchal rigidity less; these and the nystagmus were much less by the ninth day. The temperature, which had swung between 101° F. and 103° F. did not settle till the twenty-first day. The patient is now in excellent health but still has facial palsy.

CASE II.—*Case of double otitis media with acute flare-up on left; purulent meningitis involving first the base of left occipital lobe; death.*

David Stewart, aged 39. Admitted to Stobhill 5.3.37 with history of violent headache for a week and delirium for a few days; chronic otitis media on right for three years, acute on left of uncertain duration. Story of "epileptic fits" which had ceased three years ago.

Patient confused. Temperature 101·4° F., neck stiff, Kernig's sign positive, knee-jerks increased, plantar reflexes doubtful, spontaneous nystagmus on looking to left, no past-pointing; tenderness over left mastoid. Right ear—old perforation, some pus. Left ear—small perforation in upper posterior quadrant. Both labyrinths reacted to caloric test. Though too confused for accurate tests, patient heard conversation at 2 ft. and whisper at 8 in. on left; whisper at over 6 ft. on right.

Lumbar puncture—cerebrospinal fluid under pressure, slightly turbid (cells 351 per c.mm., mostly mononuclear; Fehling's solution normally reduced; globulin increased; a few degenerate cocci, not growing on culture).

The left mastoid was found to be acellular and ivory hard; a cholesteatoma and the tegmen were removed and an extradural abscess evacuated; lateral sinus looked healthy.

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6.3.37.—Temperature 100° F., pulse 110. Radical mastoid operation completed. No ossicles found; dura looked healthy. Lumbar puncture—10 c.cm. air injected; cerebrospinal fluid under pressure and with contents similar to above, chlorides 750 mgm.

7.3.37.—Temperature 101·5° F., motor aphasia. Dura incised. Lumbar puncture—fluid under pressure (cells 343, globulin increased, Fehling's solution totally reduced, no micro-organisms on films or culture).

8.3.37.—Distinct sensory aphasia, some weakness of right arm. Temperature 104° F., sinus wall healthy. Lumbar puncture—cerebrospinal fluid less turbid; cells 266, more mono- than poly-nuclears; Fehling's solution normally reduced; chlorides 758; culture sterile. (*This was the day before death.*) Through an opening in the squama the left tempero-sphenoidal lobe was explored with a Dandy's cannula in various directions but no pus was found. Next morning temperature rose to 106° F. and the patient died.

Prontosil was given twice daily.

Autopsy.—Except over the upper aspect of the left hemisphere, stinking, greenish, fluid pus covered the cranial surface of the cerebrum, and was present in the middle and posterior fossae of both sides, but more especially round the lower and inner aspects of the left occipital lobe. The floor of this lobe was covered by old, inspissated, greenish exudate and here on microscopical section the underlying brain showed considerable vascular reaction. *There was no temporo-sphenoidal abscess.* Through a needle passed into the ventricle purulent fluid was got which contained no sugar and yielded streptococci. There was some thickening of the meninges round the optic chiasma; also some clot in the left lateral sinus but no microbes were found in it by Gram's method. The paranasal sinuses were healthy.

Pathologist's comment (Dr. Marjorie Gillespie).—" The case was one of purulent meningitis affecting primarily the under surface of the left occipital lobe, spreading thence, and itself secondary to old chronic otitis media."

CASE XIII.—*Radical mastoid; purulent labyrinthitis; labyrinthotomy: recovery.* (Drs. D. Brown Kelly and T. O. Howie.)

Miss J.C., aged 28, bathing attendant. Admitted on 25.7.36 complaining of pain in left ear and left temporal region of two weeks' duration. Left ear had discharged for four months.

Examination showed granulations in the region of the left attic; foul discharge. Conversational voice heard at 2 ft.

20.8.36.—*L. radical mastoidectomy.*—Cholesteatoma in antrum; no fistula in canal.

23.8.36.—Complained of severe giddiness; vomiting; headache. Horizontal nystagmus to the right. Temperature 103° F., pulse 110.

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24.8.36.—*Lumbar puncture*—fluid turbid and under pressure; cells 2,672 per c.cm. No organisms on culture.

Mastoid wound re-opened. Poor granulation formation; dura exposed extensively above and posteriorly; sinus very far forward, making approach to labyrinth difficult; oval and round windows joined, promontory removed, and cochlea opened; horizontal canal opened up to vestibule. A large seeker could be passed from in front of the facial ridge to the opening in the canal posteriorly. Facial ridge intact. Drain inserted.

25.8.36.—Lumbar puncture—fluid still cloudy and under pressure; 1 gm. Solganal injected intrathecally.

26.8.36.—Cerebrospinal fluid clearer; 2 gm. Solganal injected. General condition improved.

Daily lumbar puncture with injection of Solganal until 1.9.36; fluid now clear. Patient rapidly improved; the operation cavity filling with healthy granulations.

29.10.36.—Plastic operation on meatus, which had become very narrow.

DISCUSSION.

Dr. BROWN KELLY, Junr., said that this patient upon whom Dr. Howie and he had operated, still had slight giddiness. One would have expected that since the operation was done nearly a year ago, compensation would have occurred. Dr. Brown Kelly asked why this giddiness should still be present, and if compensation had not yet occurred.

Dr. ADAM said that these two cases were additional to nine cases which Dr. Connal and he had previously published. The curious thing about the two cases shown to-day was that, judged by the cerebrospinal fluid alone, the case which died ought to have recovered, and the case which recovered ought to have died.

CASE III. (Dr. Gavin Young.)

CASE III.—*Lympho-epithelioma of right tonsil, right antrum and ethmoid.*

J.W., male, aged 50, admitted to hospital 14.4.37, with swelling over whole of right cheek, and ulcerated area in right tonsil. X-ray examination had shown the right antrum to be opaque.

Right antral and ethmoidal sinuses were radically cleared of a growth which filled these sinuses and the upper part of the nose, and biopsy of the tonsil and of the growth from the sinuses gave the following report by Professor Shaw Dunn:

"The tonsil is invaded by growth of the nature of lympho-epithelioma . . . I am of the opinion that the maxillary

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growth is of the same nature. The primary growth would most probably be tonsillar but of the relation between the two one cannot be certain."

Patient has been treated by deep X-ray therapy, but after eleven applications has refused more.

Patient will not be shown, but comments are invited as to the probable course of this metastasis.

DR. GAVIN YOUNG said there was nothing more to say about this except that it was an unusual combination. They did not know where the growth started, but presumed that it was from the tonsil. It was not a direct spread.

CASE IV.—*Cortical mastoid operation; lateral sinus injured; subsequent diplopia and bilateral optic neuritis; recovering.* (Dr. Gavin Young.)

E.C., female, aged 13, admitted to hospital 4.3.37. Otorrhœa (right) for 3½ weeks, following "cold".

Temperature 100° F.; œdema of right eyelids and pre-auricular region; cortical operation; lateral sinus, quarter of an inch behind posterior meatal wall and fairly superficial, damaged by gouge, and packed off; operation completed; very cellular mastoid with zygomatic extension.

10.3.37.—Sickness still; pulse slow; patient dull; headache.

11.3.37.—Packing removed; no hæmorrhage; no elevation of temperature since day of operation.

15.3.37.—Condition much more satisfactory; patient is brighter, and pulse more rapid.

22.3.37.—Headache rather troublesome; (?) a little dull.

23.3.37.—Lethargic; tendency to vomiting.

24.3.37.—Diplopia on looking straight ahead.

26.3.37.—Seen by Professor A. J. Ballantyne, who reports: "There is striking œdema of the optic nerve on both sides with whitish exudate over swelling, tortuosity of veins but no congestion; ripples in retina on temporal side of disc; the condition is one of optic neuritis rather than papillœdema."

V.A. Right 1/9; Left 1/6.

Examination by Dr. Gaylor, neurologist, who reported no evidence of localized intracranial lesion.

Condition improved gradually, diplopia and sickness diminishing, and patient was discharged 30.4.37, to report later.

Further reports will be made at the Meeting. The exhibitor's impression is that the condition was one of temporary hydrocephalus subsequent to ablation of the lateral sinus.

Query: Why does this phenomenon not happen oftener?

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DISCUSSION.

Dr. HUTCHISON said he had a case similar to this one four years ago. The sinus in this case also was very far forward. After operation the patient developed a temperature up to 103° F. for a few days, and Dr. Hutchison thought he had better open the sinus, which was perfectly normal. The symptoms subsided, and about ten days later there was double vision, optic neuritis and headache. The patient was examined by Dr. T. Kay Maclachlan who could find no sign of tumour, abscesses, etc. He did a lumbar puncture and the symptoms subsided almost immediately. Dr. Hutchison considered the possibility of back pressure due to an undeveloped lateral sinus on the opposite side, but X-ray of the jugular foramina showed both to be of normal size. The extraordinary rapidity of the recovery after lumbar puncture was interesting.

Dr. GAVIN YOUNG said that this girl was to have come on Tuesday for lumbar puncture, but she had moved and did not get the post card.

Dr. FULTON CHRISTIE said he had a similar case of a boy with sinus thrombosis; pus was present in the sinus when it was opened. The glands in the neck, on the same side, became enlarged and broke down forming a large abscess, which was drained. There was a high swinging temperature for several weeks. Headache, paralysis of the external rectus and papillœdema (3 diopters) developed. The intake of fluids was restricted. The headache and paralysis quickly cleared up, but the papillœdema has persisted. A lumbar puncture showed no increase in pressure or other abnormality. No cause has been found. Now several months later, papillœdema is still present with incipient optic atrophy. Dr. Christie asked if such cases were common in sinus thrombosis.

Dr. H. D. BROWN KELLY said he had recently seen a case of otitic hydrocephalus. The pressure of the cerebrospinal fluid was very considerable. It was quite clear, but it shot over the top of the manometer, and there was considerable bilateral papillœdema. They lumbar punctured the girl daily for about a week. She recovered completely, and the vision is now perfectly normal. That was following a Schwartze operation in which there was a perisinus abscess.

Mr. DONALD WATSON thought this condition was probably due to an encephalitis without abscess-formation. He had seen the condition on more than one occasion and they usually recovered.

Mr. PATERSON thought the condition was fairly well recognized since it was first described by Symonds, of London, who called it otitic hydrocephalus. One got this syndrome of increased intracranial pressure with papillœdema and sometimes VIth nerve paralysis. Dr. Kelly should have noted it was the contralateral VIth and not the VIth on the same side. It did not seem to be associated in any particular way with involvement of the lateral sinus.

CASES V and VI.—*Two cases of Ménière's syndrome treated by section of the auditory nerve.* (Shown by permission of Mr. J. E. Paterson.) (Dr. T. O. Howie.)

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Case V.—Robert G., aged 57, admitted 21.7.35.

History.—1929—onset of paroxysmal attacks of giddiness at long intervals, gradually becoming more frequent.

Deafness noticed in left ear for six years.

Vomiting once during attacks.

Tinnitus—thumping buzz in left ear of eight years' duration.

Headache—onset with dizziness in 1929; frequent attacks without dizziness; sometimes wakes him at 3-4 a.m.

Character of attack—no warning but gets time to get to a safe place; sits down, dizziness gets worse, objects seem to pass horizontally from left to right (of late tend to move vertically upwards).

Neurological Examination.—Negative except for slight hyposensitive left cornea and flattening over the left lower face.

Functional Tests VIIIth Cranial Nerves.—This shows marked nerve deafness on left side; very reduced response to cold caloric test on left side.

Diagnosis.—In view of prolonged history of repeated attacks—Ménière's disease.

Operation (16.10.35).—By left cerebellar exposure the left auditory nerve was completely divided.

Further progress. 22.10.35.—Complains of dizziness, especially on opening eyes; irregular spontaneous nystagmus, horizontal and especially to left.

23.10.35.—Nystagmus now to right; right pupil dilated and fixed.

25.10.35.—Complains of dizziness; nystagmus marked in both directions, labyrinthine in type to right, cerebellar to left. Past-pointing 2½ in. to right both sides.

Disturbances gradually subsided and patient reports himself free from vertigo.

Case VI.—Henry C. T., aged 31, admitted 9.5.36.

History.—September, 1934, developed a severe attack of giddiness while walking; had to support himself to prevent falling; attack lasted three hours; vomited; slight headache; objects seemed to move horizontally to left.

For six months after *September,* 1934, has had three attacks of giddiness a day, not so severe as the initial attack. Occasional severe one with slight headache. In *February,* 1935, very severe attack, with objects coming up vertically and to right; unconscious. Since then one in two days; five weeks ago, six in six days. Between times feels light-headed.

Tinnitus is gradually decreasing; occasional hum; never vomits now, perspires during attacks.

Previous Ear History.—In 1928, coryza followed by noises in head and dullness of hearing in both ears which passed off and

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returned on two to three occasions for a period of six months. In 1932 submucous resection. Deafness cleared for three months but has been deaf in right ear since; left ear seems to have returned to normal.

Neurological Examination.—Apart from right VIIIth, the cranial nerves seem to be normal.

Cerebellar Functions.—No ataxia, no dysdiadokokinesia; slight past-pointing to right with right forefinger (not constant on later tests).

Gait and Station.—Romberg—swaying but does not fall; walks heel toe easily; falls to right standing heel toe.

Functional Test Auditory Nerves.—Incomplete nerve deafness on right side. Slight reaction of right labyrinth to cold caloric test and rotation.

Diagnosis.—Partial destruction and loss of function of both portions of right auditory nerve. History of onset tends to exclude Ménière's disease and give diagnosis of an early acoustic tumour.

Operation (20.5.36).—By right cerebellar exposure; no evidence of acoustic neuroma; auditory nerve completely divided.

Further progress.—Severe vomiting—vertigo, coarse horizontal nystagmus in both directions, past-pointing to right. Emaciation; depression; no cerebellar signs. Complains of deafness in good ear; still perceives high sounds.

Gradual improvement in hearing and vertigo and since has reported free from attacks.

CASES VII and VIII.—*Two cases of "short œsophagus" with development of fore stomach—originally diagnosed as epigastric hernia.* (Dr. T. O. Howie.)

CASE VII.—Mrs. T., aged 51, admitted 21.3.35.

History.—Quite well until three months ago when she developed hiccough and vomiting; unable to swallow solids; gradually found fluids returning; choking sensation but no pain. Vomitus brownish and had acid taste.

On rectal feeding for fourteen days before admission. Has lost 1½-2 stones in weight.

X-ray Examination suggests epigastric hernia (films shown).

Endoscopic Examination (25.3.35).—Negus tube passed to 27 cm. and quantity of brown fluid had to be aspirated before view of lumen was obtained. Fluid had appearance of gastric juices with altered blood. At 27 cm. the lumen narrowed and an irregular prominence on posterior wall suggested cancerous involvement. (Specimen removed, reported by Dr. Anderson, the pathologist, to be a chronic inflammatory invasion of submucous tissues; no signs of malignancy.)

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Further endoscopic examination showed a moderate stenosis, at the junction of a shortened œsophagus with gastric mucosa (which was definitely identified at 30 cm.).

The dysphagia and vomiting completely disappeared after the passage of œsophagoscope, and patient rapidly regained weight.

It has been necessary to dilate the stenosis at intervals of three to six months, to allow of swallowing of solid food.

CASE VIII.—Mrs. McK., aged 52, admitted 16.1.37.

History.—Good health until two years ago, when she noticed a choking feeling, followed by regurgitation of burning fluid, after every meal. Swallowing became more difficult one year ago, since then has been on semi-solids. Disability more marked during past six months. On attempting to swallow solids, after fluids, she experienced a gripping feeling at the lower end of the sternum and all food taken was regurgitated. Has lost 1 stone in past six months.

X-ray Examination.—Films shown.

3.2.36.—Semi-solids only, held up at the lower end of the œsophagus but ultimately pass on.

Diagnosis.—Spasm.

21.12.36.—Narrowing noted at junction of middle and lower thirds, smooth walled.

At lower end, œsophagus has a bent course and presents dilated appearance, (?) diverticulum. Examination in upright position inconclusive.

Films show herniation of portion of stomach through diaphragm, rugae are visible. In prone position herniation recurs.

Diagnosis.—Epigastric hernia.

Endoscopic Examination, 21.1.37 (Dr. G. B. Brand).—Between 20-3 cm. slight varicosity of veins on the posterior wall. Bleeding occurred from these. Gullet otherwise normal until 30 cm. was reached. Here a definite sphincter was acting. On passing through this the gullet seemed to pass forwards and to the left and gastric mucosa was identified at 32 cm. It was not possible to enter the main stomach through the diaphragm.

DISCUSSION.

Dr. HOWIE said he just showed these cases from the point of view of interest. They were diagnosed by X-ray examination as epigastric hernia, and on œsophageal examination the œsophagus was found to be shortened. In the first case the œsophagus was normal to 27 cm.: between 27 and 30 cm. it was stenosed. In the next case the œsophagus was measured at 30 cm. and then a displaced cardia was seen to be acting. Below this, normal stomach mucosa appeared. The point

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Dr. Howie wished to make was that one could not diagnose the condition by X-rays only; the œsophagoscope must be passed.

Dr. A. Brown Kelly said that Dr. Howie's two cases showed that we had still a great deal to learn with regard to shortening of the œsophagus and the resulting thoracic stomach. The symptoms in his cases (as in many adults) were indefinite, viz. vomiting, sickness and some discomfort in the epigastrium while only slight dysphagia pointed to the œsophagus. Radiography generally gave the first clue as to the presence of œsophageal obstruction which was usually situated about the level of the VIIth dorsal vertebra. Previous to seven years ago that condition, when found in infants, was commonly termed "congenital stricture of the œsophagus", but the supposed stricture in most cases was probably in the cardiac canal situated at a high level, and more or less closed by spasm. The dilated viscus below the canal could be proved to be part of the stomach when the X-ray examination was made with the patient in the Trendelenburg position. The barium mixture then gravitated in the stomach to the lower end of the canal, and the rugae passing through the hiatus became visible. The speaker agreed with Dr. Howie that a reliable diagnosis could not be made without endoscopy. This revealed dilatation of the œsophagus above the cardiac canal. The latter appeared either closed, or rhythmically opening and closing, or sufficiently open to give a view of the interior of the stomach. Any doubt as to the lining membrane below the cardiac canal being gastric mucosa could be dispelled by removal and microscopic examination of a snippet. If a record of the exact position of the upper end of the stomach were wanted a radiograph should then be taken while the lower end of the œsophagoscope was at the gastro-œsophageal junction. It was usually found that an endoscopic examination improved the patients considerably by enabling them to swallow with comparative ease. After several weeks or months, gradually increasing obstruction might set in, but that was easily remedied by passing a bougie. In treating cardiospasm one could not get the benefit desired without overstretching the cardiac canal, which in cases of short œsophagus must be avoided because injury to the canal is apt to lead to fibrosis with permanent narrowing of the lumen. The rare *post mortem* material which came his way demonstrated the important part played by fibrosis. This began at the cardia where glands were situated which were susceptible to injury and infection. The resulting fibrosis travelled upwards and had been traced to the level of the bifurcation of the trachea, producing in its ascent both shortening and narrowing of the œsophagus. The speaker said that the cases he had referred to were those of young children, in whom the shortening was apparently congenital, whereas, in Dr. Howie's cases the symptoms did not arise until the patients were over 50 years of age. Dr. Brown Kelly appealed to the members to obtain when possible *post mortem* material, so that further knowledge might be acquired regarding ascending fibrosis of the œsophagus.

Cases IX, X and XI.—*Three cases of severe external nasal deformity treated by hinged cartilage graft.* (Plaster casts before and after operation were also shown.) (Dr. T. O. Howie.)

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CASE IX.—Julia M., aged 23.

Six months before admission had injury to nose leading to septal hæmatoma. This became septic and erysipelas developed. Deformity was severe, with a " pug " nose.

Operation was performed in two stages—submucous resection and, later, hinged cartilage graft.

CASE X.—Alex. R., aged 25.

Severe injury to nose in childhood ; had undergone at least five previous operations for the deformity.

Operation was performed in three stages—submucous resection ; subcutaneous reduction of hump and prominence on left of nasal ridge, by rasping ; finally, a hinged graft of cartilage was introduced.

Hæmatomata developed in the nose and epigastric wound. Some cartilage was exfoliated, but the graft was retained in spite of the development of frank pus in the graft bed.

CASE XI.—John A., aged 23.

Footballer who sustained a blow which drove the nasal bones downwards and outwards. The septum was severely crumpled.

Operation was performed in three stages—submucous resection, subcutaneous osteotomy of frontal processes (both maxillary) and the nasal bones were lifted inwards to reduce breadth of nasal bridge ; hinged cartilage grafting.

Dr. HOWIE said that the point he would make from these was that the best cases for treatment are those injured in adult life, in whom there is no sepsis. When injury has taken place in childhood, as in the middle case (Case X), who had an injury in childhood and then five operations of some description on his nose, the tissues were very bound down and it was difficult to raise them sufficiently to smooth out all the depressions. He had a good deal of extra bone growth, probably stimulated by the original injury and subsequent operations. The last case was the best ; he had an injury some six or eight months before he was operated upon and no sepsis, and he was very much more easily treated.

CASE XII.—*Bleeding polypus of the septum.* (Dr. D. Brown Kelly.)

Mrs. J.W., aged 50. Attended 9.3.37 complaining of obstruction in right nostril and epistaxis.

Examination revealed a polypus growing from the right side of the septum. This was removed with the cold wire snare and the base cauterized with the galvano-cautery.

Pathological report (Dr. Cameron).—A vascular cellular polypus with attempts at new gland-formation and with many hæmorrhagic areas. There is also some proliferative change in the epithelium

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although not of such a degree and character as to suggest malignancy.

1.5.37.—Recurrence of symptoms. Polypus found to have grown again in the same situation. It is proposed to elevate the septal muco-perichondrium underlying the base of the polyp, and remove it together with a wide margin of healthy tissue.

DISCUSSION.

Dr. LAND said that about eighteen months ago he had had the opportunity of showing a case of a tumour in this region. Originally a papilloma, it became malignant and was treated by diathermy. Although local recurrences had had to be treated, there had been no glandular metastases. Recently he had dealt with another case, clinically resembling the present one. Histologically it was a melanoma. There was no evidence of a primary growth in the retina or elsewhere. After excision and biopsy, the region was irradiated.

Dr. GAVIN YOUNG said that this seemed a very marked recurrence in a little over two months. There seemed to be a fair amount of growth present, and in spite of the pathological report, it was very suggestive of malignancy to his mind.

Dr. BROWN KELLY, Junr., said he had never heard of a case of actual carcinoma of the septum.

Dr. BRAND remarked that the growth of this polypus had been very rapid in the last three weeks.

CASE XIV.—*Case for Diagnosis.* (Dr. D. Brown Kelly.)

A.B., aged 34, chemical worker, complains of hoarseness of nine months' duration. No pain, cough or dysphagia ; no loss of weight ; general condition good.

Examination shows immobility of the left vocal cord, which is fixed slightly to left of mid-line. Both cords are red. The left arytenoid is red and slightly swollen. There is a perforation of the nasal septum (chronic).

X-ray examination of the chest shows slight fibrosis at the lung roots and enlargement of the left ventricular shadow.

Is this purely a fixation of the crico-arytenoid joint, or is there involvement of the recurrent nerve ?

Extra Case No. I. (Dr. Howie.)

Male, aged 27. Has always had " port-wine " stain on forehead and over root of nose. During the past ten years he has noticed small areas of dilated blood-vessel in the skin over " Adam's apple " and on the lips. Thickened patches have appeared on the insides of the cheeks adjacent to the corners of the mouth. During the past two years these have been increasing in size and occasionally catch between the upper and lower teeth, while chewing.

On examination, there is an extensive cavernous hæmangiomatosis under the mucous membrane of the cheeks and inner side

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of the lips. The condition is also present to a minor degree on the fauces and on the palate. A similar dilatation is seen at the skin-mucous membrane junction on both sides of the nasal septum. Suggestions are invited as to the safest method of treatment.

DISCUSSION.

Dr. HOWIE asked if any member had experience of a similar condition. Diathermy seemed rather dangerous and it was a question of treatment of the infiltrated area on the inside of the cheeks on which advice was sought. He felt himself that radium or electrolysis might be used.

Dr. BROWN KELLY, Junr., asked if carbon dioxide snow would be of any value in a case like this.

Dr. GAVIN YOUNG strongly deprecated the use of diathermy in such a case. He once nearly drowned a patient in his own blood.

Dr. BRAND suggested seeking the advice of the skin specialist.

Extra Case No. II. (Dr. Howie.)

Joe W., aged 47. Cranesman.

Six years ago he first became aware of slight noises in the right ear. These were of the nature of hissing steam, or the sound of a waterfall. At first they were intermittent, but are now constant and have developed a throbbing character, synchronizing with the heart-beat.

Four years ago he experienced an attack of giddiness. Outside objects seemed to pass from left to right. He had to lie down to prevent himself from falling. He felt drunk. He vomited. The attack lasted for two hours. Since then he has experienced somewhat similar attacks at intervals of two to three weeks, with occasional intervals of three months. He is always slightly giddy on assuming an upright position in the morning. He experiences no difficulty in walking. He has developed a gradually increasing deafness in the right ear. He can influence the state of the noise and hearing by pressing in the tragus of the right ear.

Functional Tests.

	Right ear.		*Left ear.*
Spoken voice (N.B.)	3 ft.		18 ft.
		W⟶	
+ −	+ − ve.	R.	+ve.
	− 4 sec.	Schw.	− 1 sec.
	nil	32	
	nil	64	
	− 20 sec.	128	
	− 18 ,,	256	Normal.
	− 23 ,,	512	
	− 25 ,,	1,024	
	− 12 ,,	2,048	

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Vestibular Apparatus.

Spontaneous nystagmus especially to the left. (?) slight horizontal nystagmus to the right.

Spontaneous past-pointing with left hand 2-3 in. to right. Very slightly to right with right hand.

Cold Caloric Test.

Response not obtained in 3 mins.	Response in 1 min. ↙ for 2 mins.
No change in nystagmus or past-pointing.	Rotatory nystagmus. No change in past-pointing.

Dr. HOWIE said he showed this as an interesting case in connection with the discussion. The condition appeared to be Ménière's syndrome. The only difficulty was that the tuning fork tests were not very clear, but he (Dr. Howie) intended going over the case again.

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