THE SYMPTOM OF VERTIGO.

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INTRODUCTORY.

Ecclesiasticus exhorts us "to let our speech be short, comprehending much in a few words." I trust that I may not ignore so venerable a dictum; and therefore, as time is short and my subject a vast one, with your permission I will go straight to the point.

I have chosen the subject of to-night's discussion, partly because it is of common interest to the Services, and partly because it has presented me personally, in my clinical capacity, with the most baffling problems that I have yet been called upon to assess. My object to-night is not to be original or epoch-making, but rather to clarify my mind; and, in the course of that process, I hope to clarify some of yours as well. In short, I propose to think aloud.

DEFINITION.

What is vertigo? Well, the word itself implies rotation, and indeed, the symptom has been well defined as "a sensory disturbance with hallucinations of rotation, either of the patient, of his surroundings, or of both." But inasmuch as rotation is not always present, even in auditory vertigo, I prefer Russell Brain's definition, that is to say, "the sensation of a disordered orientation of the body in space." For, as he observes, "the common factor in these hallucinations is the abnormal feeling of spatial disorientation, no matter what plane they occur in" [1].

THE STRUCTURES CONCERNED AND THEIR FUNCTION.

I trust, gentlemen, that you will forgive me if for a few moments I take you back with me to school (I might almost say to sea), for I am about to turn my gaze on those dread gyroscopes of mal-de-mer, the cerebellum and the semicircular canals.

There exists in the mid and hind brain a whole system of ganglia, linked and connected up by the stem of the brain; that is to say, the crura cerebri, the pons and the cerebellar peduncles. In front one finds the optic thalamus, the corpora quadrigemina, the geniculate bodies, flanked by the grey corpora striata; behind is the cerebellum. In the brain stem one finds the nuclei of the oculo-motor muscles, the facial nuclei, the auditory and vestibular nuclei, and, closely adjacent to these, the nuclei of the bulb or medulla. This system of ganglia in the primitive mid and hind brain, has been thrust back in man into unconsciousness; but there

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1 A paper read before the United Services Section of the Royal Society of Medicine, April 9, 1934, and published by permission of the Royal Society of Medicine.
is a definite suzerainty of the all-dominant and conscious cerebrum above in the regulation of the body's position in space.

All these ganglia and nuclei are *en rapport* in the most intimate manner; their interplay is easy and harmonious; they all take part in the tone and position of the body, both static and kinetic. They are all in touch, one way and another, with the ear and the eye, and with the cornua of the spinal cord. They have two sets of intermediaries between themselves and these outer structures; the red nuclei in front and the nuclei of Deiters behind. These may well be pictured as the Willesden and Clapham junctions of this ancient metropolis. For to-night's purposes we are concerned chiefly with the posterior part of this system, the cerebellum and the nucleus of Deiters, and their afferent vestibular influences.

**THE CEREBELLUM.**

Co-ordination means the maintenance of muscular tone. That is essential for balance or equilibration; and, as Purves-Stewart reminds us, the cerebellum is essentially a co-ordinating centre for equilibration. "It receives its afferent impressions from the skin of those parts on which the body happens to be resting, from muscles and joints concerned in maintaining our balance, from the muscles of the head and eyes; but, most important of all, from the semicircular canals" [2] via the nucleus of Deiters. It exercises its influence on the limbs of the same side, and that influence passes through the nucleus of Deiters.

**THE NUCLEUS OF DEITERS.**

We have seen that the vestibular nucleus, or that part of it known as Deiters, receives afferent impulses from the ear and transmits them to the cerebellum. It receives and transmits to the spinal cord, and further it has forward connexions with the oculo-motor nuclei, with the opposite nucleus of Deiters, and indeed with all adjacent nuclei. Far and away its most important connexion is the vestibular one, with impulses ascending from Scarpa's ganglion in the vestibular nerve-trunk, i.e. static impulses from the otolith, sending up messages of the head's position in space, and kinetic messages of movement from the semicircular canals.

**THE VESTIBULAE, SEMICIRCULAR CANALS AND COCHLEA.**

Finally, a word on this specialized nerve-ending—the labyrinth. It consists of the semicircular canals and otolith and Scarpa's ganglion on the one hand, and the auditory cochlear nerve-ending and the ganglion spirale on the other. Between them is the vestibule with the otolith. These run so far together in the eighth nerve-trunk, and part company near their entrance to the pons and medulla. The canals form a sensitive register of stability and equilibrium, and indeed of healthy harmonious comfort. If they are absent there can be no vertigo. When present their range of sensitivity varies enormously among normals. Some people get sick in a
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train, or in a ship in harbour, or at the suggestion of going to sea, or even when walking from pavement to soft grass or earth. The normal labyrinth can be stimulated in health or experimentally. You are all acquainted with the rotation test for both ears together, and with the caloric and galvanic tests for each individual organ. When so stimulated the labyrinths respond by the phenomena of vertigo, nystagmus, forced movements of the head and body, and by what is called mispointing.

There are varying combinations of these, according to the strength of the stimulus and the position of the head at the time of testing. It is all a matter of reflexes. The reflex of nystagmus, for instance, reduced to its simplest form, is comparable to a flexor plantar response. If the stimulus for the latter is strong enough you get wriggling and writhing movements travelling up the trunk. If the stimulus for nystagmus is strong enough you find head inclinations, forced movements, mispointing and vertigo.

So that, gentleman, we see the semicircular canals passing on their impulses to Deiters; Deiters most profoundly impressing the cerebellum and adjacent nuclei; and they, in turn, influencing head and limbs and eyes in the matter of tone and of balance or equilibration.

Vertigo as the Physician Sees It.

With these preliminary reminders of structure and of function, the clinical study of vertigo is vastly simplified. There is a firm ground for logic to exercise itself upon. But, even so, there are many cases to challenge and defy the diagnostician. No physician forgets that Gower referred 90 per cent. of cases of vertigo to disorders of the ear; nor does he forget that there is a vertigo of the eyes. He should let no case leave his ward without an aural examination, and, in the majority of cases, an ophthalmic one as well. In any case he must examine the optic discs in all cases of vertigo.

In his general examination, the physician will note the age of his patient. In the younger he will inquire into epilepsy, migraine, disseminated sclerosis or hysteria. In the older he will think of cardiovascular lesions and arteriosclerosis. He may see evidences of neurasthenia, though his instinct makes him loathe that label as a substitute for diagnosis. As regards hysteria he will be wary, because he knows that vertigo can induce hysteria. Therefore, he excludes all other causes before he is reduced to that; and if so reduced, he looks for other indications of hysteria, e.g. deafness, loss of smell, gross anaesthesias, &c. He will be careful with the nervous system, recalling possibilities of intracranial lesions, tumours and cerebral abscesses. In these he will look for focal signs or a rise in intracranial pressure.

Congenital conditions, e.g. Friedreich's ataxia and familial vertigo, and cerebral degenerations like syringo-bulbia, will not be forgotten; nor inflammations like encephalitis lethargica and the various types of meningitis. Physiological states of vasomotor and endocrine unbalance
will run through his mind, e.g. the menopause or the climacteric; and the reflex causes of vertigo from the pelvis, bowel or bladder; and the psychical reflexes like anxiety or fear. Blood diseases, notably leukaemia, are possible sources of hæmorrhage and atrophy of labyrinthine nerve-endings. Endocrine and vegetative disorders with their varying degrees of unbalance between sympathetic and parasympathetic, e.g. Addison’s disease, diabetes, or a disordered thyroid; all these will be thought of and excluded. Finally, he recalls that enormous group of toxic causes of vertigo, be they mineral, vegetable, gaseous, bacterial or metabolic.

Some Clinical Notes on These Disorders.

A word or two in passing on some of these conditions:

Epilepsy sometimes has an aura of vertigo. If that precedes petit mal, unconsciousness must be established for the diagnosis. The interest of the aura lies in this: epilepsy is almost certainly a cortical brain storm; and it is now known that stimulation of the interparietal sulcus causes intense vertigo. May not this explain the cause? Again, latent epilepsy is sometimes brought to light through an acute middle ear; obviously a reflex affair and the starting trigger of fits. The use of small doses of quinine helps to diagnose between auditory vertigo and the epileptic aura. It relieves the vertigo, but not epilepsy. Migraine sometimes has an aura of vertigo, but here one notes the slow deliberate march of the aura, and the optical and sympathetic upsets. Vertigo sometimes alternates with migrainous attacks. Now migraine is almost certainly due to spasm of the cerebral vessels. We can actually see the spasms in the retinal vessels; and we believe that spasm of the internal auditory arterioles is a fruitful source of vertigo.

Cardiovascular Causes.

It is well to speak of dizziness where there is no objective stagger; giddiness, when the patient tends to fall; and to reserve vertigo for the paroxysmal manifestations. Sudden cerebral hyperæmia causes dizziness especially on stooping, for instance, over a billiard table or in tying one’s shoe laces. If there is hyperpiesia or arteriosclerosis, as for instance in renal disease, that effect is enhanced. In the cerebral hyperæmia of the menopause we notice the same thing. Sudden anæmia causes giddiness as in syncope. Sometimes there is premonitory giddiness in older people, before a thrombosis or a stroke. Giddiness with headaches in atheromatous old men is always a danger sign. If there is fixation of the stapes, the local safety valve for pressure is lost in that ear, so that vascular pulsations bear direct on the sensitive labyrinthine cells and make things worse. In organic heart disease, when the heart is beginning to fail, some feel giddy in the morning on rising. That indicates vasomotor failure. Some feel giddy at night when going upstairs. That is failure of the myocardium. If blood-pressure is low, as in convalescence, we see giddiness. This is also true for the blood diseases. Certain arrhythmias, like the effort
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syndrome, manifest dizziness or giddiness; these may be quite severe in the higher grades of heart block—the old Stokes-Adams syndrome, for example. Again, we see it in paroxysmal tachycardia, in flutter, and in the palpitations of hyperthyroidism. A cathartic purge occasionally induces vertigo, especially in persons with some circulatory disorder.

Neurasthenics will tell you that they feel as if their legs were giving way under them, that there is a terrible thumping of the heart, a lump rising up through their chests into their throats and that they are gasping for breath. This may be menopausal or digestive; more frequently it is anxiety or fear; the fear of sudden death from what they conceive to be heart disease. They never, however, lose consciousness in these attacks.

Disseminated sclerosis is a disease of the young. Such patients complain frequently of a dizzy swaying, almost certainly pontine in origin; but sometimes they get severe and paroxysmal vertigo in the early stages; the vestibular form of onset which the French describe. These may land with the aural surgeon, in the first instance, as cases of vertigo; or they may be mistaken for hysteria. The associated signs should help however: intention tremor, absent abdominal reflex, retrobulbar neuritis or optic atrophy, or an extensor plantar response, &c., &c.

Some of the digestive causes (so-called) are really the effects of vertigo; acute gastric and persistent vomiting for instance. Gastro-jejunostomy has been performed before now, and even appendicectomy in such cases.

Intestinal Parasites.

Intestinal parasites may be a reflex cause, but there may be a toxic element as well. The associated eosinophilia helps one here.

Toxic Conditions.

Finally, in the group of toxic conditions: among the drugs tobacco has a high place: alcohol of course everyone knows; and among the alkaloids, quinine and salicylates are prominent. The atropine and hyoscine groups are fairly notorious; and among the minerals one should not forget chronic arsenical poisoning. Carbon monoxide is a cause also—in ordinary illuminating-gas poisoning.

The bacterial groups of toxins are legion, and so are their foci of infection: teeth, tonsils, sinuses, ears themselves, gall-bladder, appendix, colon, and \( B. coli \) infections. I would mention one in particular, and that is syphilis, both congenital and acquired. There is a selective action; sometimes it is the fibres of the eighth nerve, a neuritis, or again a labyrinthitis; it may be a gumma of the petrous portion or a meningeal gumma; or a fistula of the external semicircular canal. There is no pain in such cases. Other pairs of nerves may be involved, notably the third nerve. The deafness is bilateral and may come on as early as the seventh week of the disease.

Herpes zoster of cranial origin is sometimes associated with vertigo.
Any acute toxic disease can cause this symptom; I would mention mumps as a cause of severe vertigo.

Metabolic disturbance accounts for the giddiness of gout. One sees this in the hypoglycaemia following an overdose of insulin, or even in Addison’s disease where the blood-sugar is also low; but, of course, the anaemia and low blood-pressure might be enough to account for it in this disease.

**Aural Vertigo.**

With such reflections in his mind and in his notes, and possibly some suggestive hints as well, the physician transfers the case to his aural colleague, the state of the optic disc and the Wassermann report accompanying him.

What may the otologist find? Well, he can find many obvious causes in the outer and middle ears, e.g. wax, a blocked Eustachian tube, nasopharyngeal catarrh, that most fruitful source of vertigo, middle ear disease, cholesteatomata, mastoiditis, or disease of the mastoid antrum. These are the commonplaces of his consulting room. Further, by watch and tuning fork he can test the cochlear auditory apparatus, and by means of the caloric and galvanic tests he can measure the sensitivity of the labyrinths. If he does that in all requisite dimensions he will produce some bewildering data, enough to tax the memories of the gods, let alone those of men! I remember Dundas-Grant used to help us with a mnemonic, a copy of which I always keep; but, even so, I am glad that this is exclusively the realm of the otologists. Verily “They have chosen that good part which shall not be taken away from them,” at all events by me.

Now auditory vertigo has these characteristics, none of them pathognomonic, but together conclusive [1]: (a) There is a sense of rotation of the patient or of his surroundings. (b) There is diminished excitability in the semicircular canals, as shown by the caloric tests. (c) It is frequently associated with deafness and tinnitus—cochlear disturbance. (d) It is associated with nystagmus and a tendency to what is known as forced movements. (e) It is sometimes accompanied by diplopia. (f) If severe, prolonged or paroxysmal, there will be pallor, sweating, disturbance of the pulse-rate and pressure, nausea and vomiting. (g) Very rarely there is loss of consciousness. (h) In labyrinthine lesions the occiput is tilted towards the shoulder of the affected side.

The matter, however, is not so simple and clear cut as this. The signs are often atypical. Sometimes there is little loss of sensitivity, as indicated by the tests. Occasionally unconsciousness occurs (Russell Brain). Sometimes the auditory symptoms are wanting. Both labyrinths may be affected in a varying degree, and so on. An acute inflammation of the middle ear can modify labyrinthine sensibility to the various tests. Moreover, there are differences in the responses to caloric tests in different diseases. Asymmetry between the conditions of the two ears, and the resulting heterogenous stimuli should never be lost sight of.
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On this matter hear Scott [4]. He finds that lack of patency of the Eustachian tubes and invagination of the tympana, unequal on the two sides, a most fruitful cause. If both tubes are equally inefficient, and both tympana equally depressed, one finds deafness but not vertigo. If, however, one side is worse than the other one finds severe vertigo and the deafness is not noticed. He attributes many defective air landings and many fatal spins to this cause, because vertigo in the air is associated with reflex forced movements. In consequence, a pilot with a blocked Eustachian tube thinks he is landing on an even keel when in reality one wing is dipping very noticeably. He says that all pilots should be able to inflate both Eustachian tubes by swallowing or Valsalva, or else give up high altitude flying. The same thing occurs with sailors in submarines and with divers in high atmospheric pressures.

Scott records the case of a lady with early otosclerosis in one ear, who suddenly got intense vertigo in a shop. She was thought to be tipsy and sent home in a cab. He tried to inflate the bad ear. Matters did not improve. When he inflated the Eustachian tube on the sound side she got well almost at once. That tube had become temporarily blocked.

Spasm of the tensor tympani or stapedius can cause vertigo by disturbing the stapes. When the stapedius is at fault the facial muscles may twitch and quiver.

A number of troublesome cases occur with old chronic otitis media, or where a mastoid operation has been done and everything seems quite well. They are subject, after a time, to giddiness which is accentuated by walking on a narrow plank or on going aloft. I had such a case. He was sent in to hospital as a seaman who refused to go aloft. After exhaustive collaboration between the aural surgeon and myself, we could find no special reason for his giddiness beyond a long standing chronic bilateral otitis media, now dry. He was sent back to his ship after reassurance, and with the alternative of changing his rating if he did not succeed in going aloft. He declined to do either, and was sent back to hospital and finally invalided. At the time I thought he was trying to get out of the Service, but I have since read in the literature of many such cases; and it appears that they are genuine enough.

From all this we see that the auditory vertigo is most commonly produced, not by disease in the labyrinth itself, but by stimulation from the neighbourhood without. It may be inflammatory, or a matter of pressures, or of hyperæmia or ischæmia; it may be a reflex phenomenon.

The Ménière Syndrome.

But suppose no demonstrable lesion is found, and the vertigo is severe and definitely auditory in type, then the semicircular canals are suspect; the labyrinth, the cochlea or both; and the otologist has to ask himself what is going on in that bony petrous invisible fastness; or again, is the lesion behind the petrous bone, that is to say is it central or peripheral? The
answer is not always easy. He now finds himself in that perplexing maze known as the Ménière syndrome. Two things may help to clear his mind at the onset: (a) He can compare the labyrinth, which is an invisible and inward nerve ending, with the outward and visible signs of the retina and optic discs. For instance he can ask himself “Is there such a thing as glaucoma or papilloedema in the labyrinth?”—A rise of endolymph tension? It is plausible. Why not? The intravenous drainage system from the vestibule is more intimate with the intracranial circulation than is even that of the retina. Again, is there a labyrinthine equivalent of tobacco amblyopia? He notices that spasm occurs in the migrainous retinal vessels. Why not then in the arterioles of the internal auditory vessels? (b) He must be struck (indeed he has good reason to be) by the gross vegetative or sympathetic upsets so manifest in severe vertigo. Portmann [5] of Bordeaux has experimented freely on the question of spasm and stasis of the labyrinthine vessels, by vasoconstrictors and dilators; by pressure upon the vertebral vessels and common carotid; by section of the cervical sympathetic; and by pericarotid sympatheticotomy. He finds that paroxysmal vertigo can be produced either by stasis and edema or by angiospasm and ischemia in the cochlear and vestibular branches of the auditory vessel; the first producing hyposensitivity and the latter hypersensitivity in the labyrinthine cells; and this he compares ingeniously and somewhat convincingly with the digital ischemia of Raynaud’s disease. Either extreme of sensitivity may induce vertigo.

The Original Ménière Case, which I have taken the trouble to look up, came to autopsy; and it was found to be a plastic lymph exudate, which Scott [4] has convinced himself was a serous meningitis due to acute infective labyrinthitis, where the drum had not perforated. Before Ménière had found that vertigo originated in the labyrinth itself, the symptom was always looked on as a forerunner of apoplexy.

Hæmorrhage.

Hæmorrhages as a cause are very rare. Fraser [6] has found one or two old organized hæmorrhages in cases of leukæmia, and of course we can surmise small hæmorrhages after concussion. In that connexion one should mention those traumatic cases of persistent cerebral contusion, so usual nowadays after motor-car accidents, with giddiness and headache and loss of concentration and general irritability.

Grading of Ménière’s Syndrome.

Ménière’s syndrome in its acutest form consists of paroxysms of vertigo of the auditory type, giddiness, reeling, deafness, and tinnitus, with bulbar and sympathetic phenomena, nausea, vomiting, cardiac and pressure changes and cold clammy sweat. The attacks strike the patient down suddenly in a paroxysmal manner; but he is not unconscious. He falls away from the side of the affected ear. Nystagmus, even diplopia, may be
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seen whilst the paroxysm lasts. Headache and vomiting may persist for some time after the attack. The complete paroxysm is very rare. In practice we see modified editions of the syndrome. Fraser [6] divides it into three degrees:

(a) Apoplectiform, with total deafness at the onset and loss of vestibular function on the side of the lesion. Causes: haemorrhage, purulent labyrinthitis, mumps.
(b) Sudden onset, but not complete loss of vestibular response. Causes: toxic neuritis, herpes, glaucoma.
(c) A gradual onset. Causes: cerebral arteriosclerosis, tumours of the eighth nerve, acquired syphilis, occasionally otosclerosis. Some of the latter cases may have only a transient giddiness with a tendency to recur. There may be some deafness and tinnitus between the attacks.

As a general rule the vertigo of cerebral lesions is less severe than that in the labyrinth itself. It may be exceptionally severe or very slight in cerebellar abscesses, depending entirely on their position in that organ. Any tumour may present vertigo as a symptom, but it is commoner in tumours of the posterior fossa. Tumours in the cerebello-pontine angle may present some nasty attacks, but they are not so sudden or paroxysmal as, say, that of labyrinthitis proper. Further, they are accompanied with tinnitus and deafness. In vascular cerebral lesion a thrombosis will cause paroxysmal vertigo, whilst in local arteriosclerosis the onset is gradual. A focal lesion affecting Deiters' nucleus would cause paroxysmal vertigo with facial pains due to its proximity to the fifth nerve nucleus and its roots.

Differential Diagnosis between General and Peripheral Vertigo.

I regret I have no time, in a survey of this character, to go into the differences between central and peripheral lesions. Generally speaking, central lesions present focal signs, and later, a rise of intracranial pressure. One finds ocular signs, insensitive cornea, crossed anaesthesias or crossed pareses, hemiataxias and the like, and possibly optic neuritis, together with deafness, tinnitus and vertigo in a varying degree, and nystagmus.

Dundas-Grant advises careful readings with the galvanic tests rather than the caloric, when central lesions, so called, are suspected.

Ocular Vertigo.

As I have already remarked, some cases of vertigo are ocular in origin. How then can we distinguish auditory from ocular vertigo? Well, in the first place, the usual causes of ocular vertigo are (a) low degree of astigmatism, (b) a strabismus of paralytic type with false projection of the visual fields and diplopia, (c) anomalies of muscle balance-heterophoria. Now, shutting of the affected eye in a paralytic strabismus will stop
vertigo. Indeed, the simplest test is to open and close the eyes. If vertigo is present when the eyes are open and not when closed, the vertigo is ocular, not aural. Take the example of the superior rectus muscle. I know of such a case which took months before it was fully tracked down. The patient had to keep his eyes open and look up to obtain the diplopia tendency. If he kept his eyes closed, the vertigo resulting from his diplopia disappeared. If he looked upwards with the sound eye closed, a very unpleasant sensation of false projection was experienced. Objects in the upper field were displaced too far up.

The ophthalmic surgeon must first discover the diplopia which occurs on looking up and then decide which eye is affected. In aural vertigo the patient has vertigo when looking straight ahead, which a patient with a paresed ocular muscle never has. Moreover, diplopia is rare in aural cases. “Then one notes in the aural case the jerky movements of objects, a distinct erroneous projection in the direction of the object, so that if the patient tries to touch it his hand goes too far in the direction of the object-mispointing, and also associated tinnitus” [7].

Finally a word on treatment.

TREATMENT OF VERTIGO.

The first maxim is treat the cause; that is if you can find it, which I think I have shown you is sometimes more easily said than done. Take auditory peripheral vertigo first. Luminal is the drug par excellence, 1/2 to 1 gr. t.d.s. Larger doses can be given subcutaneously during an attack. I believe alcoholic injections have been tried in very severe cases; and recently Cairns and Brain [1] have had marked success on five cases by section of the auditory nerve. In Eustachian cases inflate by catheter. Some use bougies. Tweedie[3] has used small doses of iodides (1 or 2 gr. t.d.s) very successfully when inflation or dilatation was not permanent in its effect. In some cases small doses of quinine are very effective. In concussion cases 1/36 of a gr. of perchloride of mercury will relieve severe tinnitus and vertigo. Amyl nitrite may give relief in the giddiness of arteriosclerosis. Iodides are the real sheet anchor in that disease. In the neurotic cases bromides give relief. Then there is the whole group of vaso-dilators and vaso-constrictors. Adrenalin can be used locally or internally, the nitrites, e.g. mannitol and the like, can be used internally. The belladonna group is useful in seasickness; and so on.

In all cases look to the nasopharynx and nasal passages, the sinuses, the tonsils, and the teeth. An x-ray may show an offending unerupted wisdom tooth which is causing vertigo reflexly. Various operations can be done on the outer and middle ear. Wax can be removed. In acute cases fenestration of the tympanic membrane can be performed. The ossicles, if fixed, can be removed. Mastoid operations are constantly being performed. Pericarotid sympatheticotomay may be helpful in angiospasm. In
central cases, where tumour or abscess is diagnosed, intracranial surgery must be resorted to. There is a class of cases where neuropathic treatment is essential for success. I remember one such case so cured, who, when congratulated on the result by his friends, promptly went deaf. Lumbar puncture does good in certain cases, especially if the lateral or pontine cisterns are under pressure. Uncapping the external canal and draining the labyrinth by catgut has been successful in Peter's hands.

CONCLUSION.

In conclusion, Mr. President and Gentlemen, vertigo is a sensory expression of disordered function, produced in so many ways from such a variety of afferent impulses, all bearing on the labyrinth, that I make no apology for the broad acres of medicine I have had to traverse to-night in this very rapid and, I fear, superficial survey.

The immediate stimuli, both reflex and direct, would appear to be in the nature of hypo- or hypersensitivity in the affected labyrinth, the result of varying pressures, either circulatory or inflammatory, resulting in tension changes in the labyrinthine perilymph and endolymph. Stasis of the arterioles or spasm of these vessels determines the onset of vertigo and tinnitus and deafness in a large class of cases. Lesions of the middle ear and Eustachian tubes are a fruitful source of trouble. Asymmetry, both ears being affected in varying degree, should always be considered. The labyrinth is sometimes the direct cause of the disorder and sometimes incidental in this disorder.

Only by a sound knowledge of structure and function, and by close co-operation between the physician, especially the neurologist, and the aural surgeon, is there a reasonable hope of success.

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OTHER REFERENCE WORKS.

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