and it will be interesting to know how long Africa and Oceania will remain free from contamination, and particularly the West Indies, which would seem to be more exposed to the infection, since they are inhabited to a great extent by Mulattoes who often visit the American coast.

Now, if we are allowed to draw certain conclusions from the observations we collected from all quarters of the globe, we may say that ozæna is an infectious disease and is found in all races. Yet the disease is less frequent among the blacks than among the whites, and is most prevalent among the yellow race. The marked deviation of the septum on these latter, to which we must add their state of habitual uncleanliness and nasal irritation, produce in their mucous membrane a splendid breeding ground for the microbes.

The blacks of Africa, Oceania, and the West Indies are not yet infected, although they are susceptible to the disease, and the small number of Europeans who came under our notice—and they were not afflicted with ozæna—does not allow us to express an opinion as to the future apparition of the disease in those countries.

SOCIETIES' PROCEEDINGS.

ROYAL SOCIETY OF MEDICINE—OTOLOGICAL SECTION.

November 17, 1916.

H. J. Marriage, *President of the Section, in the Chair.*

A Further Case of Otosclerosis associated with Otitis Media.—J. S. Fraser—(*continued*).

Mr. C. Ernest West: I am obliged to Mr. Fraser for showing us these specimens. I should like to ask a few questions with regard to the case of otosclerosis. This woman was aged something over sixty when she met with her death. How long had she been deaf? How long does Mr. Fraser think the condition had been going on—if it had been going on at all? If it had not been going on, what was the exact nature of the resting condition? And what is it that leads to progress in one case and to arrest in another? Histologically, are these patches of so-called osteitis vasculosa inflammatory tissue, or are they merely altered tissue—post-inflammatory tissue? I think that one very important point which Mr. Fraser brought out in connection with his previous demonstration was the anatomical feature respecting the existence of a layer of what one might call cancellous bone really surrounding the true capsule of the labyrinth, and lying between it and what one might call the adventitious capsule, which is really a part of the petrosa. That seems to be the central part of his work so far as it affords a basis for the spread of an inflammatory and presumably infective process, whether it has penetrated from the tympanum or has originated by deposition, by blood infection. This case, in which there

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are apparently typical otosclerotic lesions on the one side and manifest results of past suppuration on the other, does lend colour to the theory that otosclerosis is essentially an infective or post-infective process originating in the tympanum. I reluctantly admit that I have been struck by Mr. Fraser's demonstration in that sense. I think it is important that we should arrive at a definition of what we mean by otosclerosis—whether a pathological process demonstrable histologically, or a clinical condition of a certain type as regards appearances, symptoms, and the like. At present we are a little liable to be at cross-purposes in our discussions, because we do not know whether we are

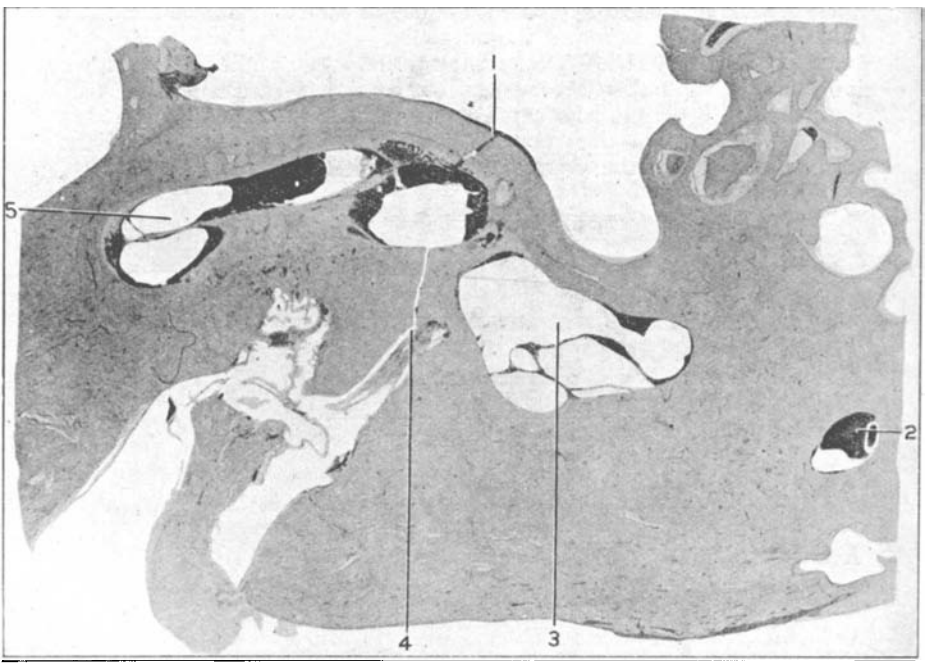


FIG. 19.—Fracture of cranial base with injury to labyrinth capsule. P. B ——, male, aged forty-one. Horizontal section through right ear just above round window. No. 235.—1. Fracture of promontory. 2. Both peri- and endo-lymph spaces of posterior canal contain blood. 3. Vestibule with slight hæmorrhage. 4. Line of fracture reaches canal for nerve to ampulla of posterior semi-circular canal. 5. Basal coil of cochlea with hæmorrhage in all three scalæ. (× 8.)

talking of a pathological process demonstrable under the microscope, or of clinical types of deafness familiar to all of us. Before I sit down I should like to make a protest on the ground of accuracy in the use of terms. I am an upholder of exactness of phrasing as far as possible. The term I object to is "chronic adhesive process." I do not know what it means. I do not see any evidence for it in Mr. Fraser's slides. I see evidence of post-inflammatory conditions, but I see no reason why this should be called a chronic thing, any more than one's boot is chronic because it is always on one's foot. The condition is a resting one, and "chronic" to my mind implies a process which is going

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forward. It may be that there is a cicatrised tympanum—that there has been post-suppurative cicatrisation. That is the way I should like to see it described. Ultimately, it seems to me, the turning-point of the discussions will hinge on the question of ætiology. When we have arrived at some clarified ideas with regard to ætiology, it is conceivable that we may arrive at some ideas as to treatment. I do not mean treatment when the patient has a developed otosclerosis, but treatment rather in the preventive or the abortive sense. It is extremely important that we should have laid before us, in as categorical a manner as possible, the views of those who are working on the subject as to

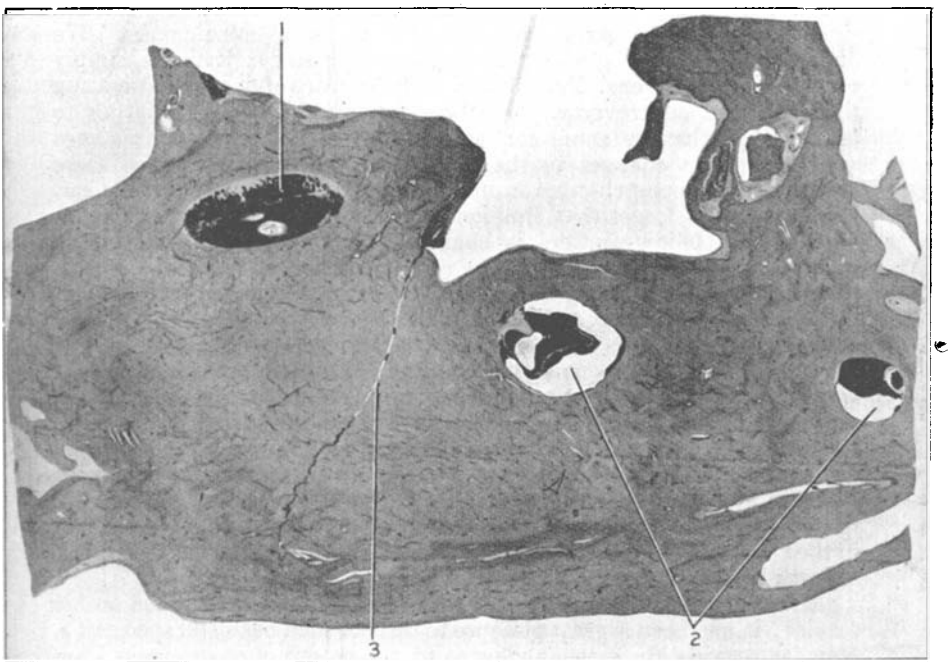


FIG. 20.—Fracture of cranial base with injury to labyrinth capsule. P. B——, male, aged forty-one. Horizontal section through right ear below round window. No. 300.—1. Extensive hæmorrhage in spiral ligament in lowest part of basal coil of cochlea. 2. Both ends of posterior canal show blood in endo- and peri-lymph spaces. 3. Line of fracture which does not quite reach dura *mater of cerebellar fossa*. (× 8.)

whether this is a definitely infective condition—as to whether the process is due to some toxic or degenerative change taking place in tissue naturally prone to it, or whether we have to do with a chronic osteomyelitis. If so, there ought to be changes taking place progressively, and such appearances as Mr. Fraser has shown this evening in this woman aged sixty ought to be different from those in a patient aged twenty-five who is a progressive otosclerotic. That seems to me a point which Mr. Fraser should take up. There is one other poin that appeals to me, and that is the light which is thrown upon the formation of labyrinthine sequestra. One is familiar with the way in which the labyrinth is sometimes shelled out, with the inner

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layer of the capsule intact, over wide areas, not only in septic but in tuberculous cases, and we see a reason for that demonstrated in this cancellous sheath which runs round and over the ossified cartilage of the primary labyrinthine capsule. I have been leading nowhere, I fear, except to ask questions, but I am very grateful to Mr. Fraser for wha he has shown us.

Dr. DAN MCKENZIE: I have always listened with pleasure to what Mr. West has to say, and it has been very interesting to hear from him that he has been convinced by to-day's demonstration that the inflammatory changes in the ear may lead to osteitis vasculosa. We have only seen two cases, however, in which this has occurred, and one would like to see more before arriving at definite conclusion, since both of these cases may simply have been coincidences. We do not know anything about their previous history: they are simply *post-mortem* specimens. This should make us wary about overestimating their value in controversy. On the other hand, it is difficult not to believe that there is some connection between inflammatory changes and these bone changes in the ear. It is not surprising that there should be such bone changes in inflammation of the mucosa of the ear. We must never forget that Politzer's typical otosclerosis, according to him and his fellow-workers, is characterised by the fact that in the mucosa of the middle ear there are no changes to be seen, and in the patient there is no history of former attacks of middle-ear trouble. It is difficult, I admit, to exclude all middle-ear trouble of infancy. I suppose very few of us have escaped such attacks, and yet the cases of otosclerosis among the generality of us would be insignificant. In other words, we have the association of a common occurrence and an uncommon sequel, and when this is the case there must be a third factor intervening to explain the sequel. It is true that another point in favour of the local cause is—what seems to be the case here—that one meets with cases of otosclerosis without any evidence of rheumatoid or arthritic trouble elsewhere. We get bone change precipitated locally in the ear in the temporal bone, and we naturally look for a local cause. The most obvious local cause is an inflammatory one.

Dr. A. A. GRAY (Glasgow): Mr. Fraser is to be congratulated on his results; it has been a great pleasure to inspect such beautiful specimens. Now, as regards Mr. Fraser's view as to the nature of otosclerosis, I am afraid I must say I am not entirely in agreement with him. I have examined half-a-dozen specimens of otosclerosis, and I have never seen any inflammatory change in the bone. I have never seen any round-cell infiltration nor any sign of inflammatory activity. It is difficult to accept the view of the disease being inflammatory when such a sharp line of demarcation is present. I mean that I can hardly conceive an inflammatory change stopping abruptly with the sharpness of the otosclerotic focus. It rather gives one the impression of a tumour—a new growth. With regard to infection from the middle ear being the cause of otosclerosis, it is quite true that Mr. Fraser has shown us two cases apparently illustrating this; but one must remember the number of cases which show the contrary. I have made pathological investigations of several cases of suppurative middle-ear disease, and I have not found otosclerosis in any of them; at the same time I know that the two conditions may occur coincidently, and I think that Mr. Fraser has happened to light upon two such cases. In these one must look for some antecedent tendency. Cases which fall into this category are not

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necessarily chronic suppurative cases; they may be cases of acute middle-ear inflammation. I recall such a case of middle-ear inflammation; it was very painful and acute, but healed without suppuration. The patient, however, to her physician's perplexity, continued to get more and more deaf. The explanation of the condition was that there was a marked tendency to otosclerosis in her family. The left ear—which was the side affected—gradually became more and more deaf, the right ear remaining perfectly healthy all the time. But, sixteen years later, she had an attack of appendicitis, and at the same time the right ear became slightly deaf and there was tinnitus; this was followed a little later by a second attack of appendicitis, and later by a third attack, after which the appendix was removed. Then the tinnitus disappeared, the degree of deafness remaining approximately the same. This was a case of acute middle-ear inflammation exciting otosclerotic change in the bone on one side; then, after the lapse of sixteen years, the other ear began to be affected. One might say that this was a case due to infection. It may have been infection from the appendix, but it was not from any acute or chronic middle-ear inflammation. In most, if not all, of the cases of otosclerosis I have clinically examined that have apparently started from suppurative middle-ear disease, on going carefully into the patients' histories and eliciting the facts and getting information as to deafness in relations, such as uncles and aunts, as well as parents and grandparents, I have almost always found evidence of some family deafness. I do not believe that otosclerosis can occur in the majority of individuals. It is true that we never know what we carry about with us in our body, but the majority of people could not be otosclerotics by any process whatever; and, on the other hand, there are a certain number of individuals who can never be prevented from becoming otosclerotics, because the tendency is so strong. And between these two classes of persons there are a number who will become otosclerotic under certain conditions which induce changes in the bodily constitution —*e. g.*, typhoid fever, pneumonia, exhaustion, or various other conditions of that nature. In these cases otosclerosis is contingent upon this immediate condition, but the essential factor lies in the personal quality of the individual. The organ of hearing will withstand damage from various causes up to a certain point, but beyond this in some individuals it will give way, and in others it will not give way.

Mr. R. LAKE: At the International Congress of Otology, held at Boston in 1912, Prof. Siebenmann (I think it was) showed a large number of photographs, and among them were some from patients who had suffered from suppurative otitis, in which these patches of osteitis were extremely large. I am inclined to think the case we are discussing this afternoon is due to a similar condition.

Sir WILLIAM MILLIGAN: I am not prepared to argue the point from microscopical appearances, because I have done so little work on otosclerosis along this line. But I think it will be difficult for Mr. Fraser to prove that otosclerosis is secondary to a septic condition of the middle ear. The number of cases of suppuration of the middle ear is legion, but the number in which otosclerosis is present as a complication is very small. We have, I think, to look to an entirely different ætiological factor as the causative agent; probably it will be found to be some anomaly in one or other of the internal secretions of the body. I have always been struck with the relation between anomalies of menstruation and otosclerosis. The time at which the otosclerosis first

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manifests itself in young girls is about that at which menstruation commences. I am not prepared to say it is a question of cause and effect; it is only a striking fact. There is a deficiency of lime salts in the blood of many of these otosclerotics; is that connected with the production of the disease? Against Mr. Fraser's view there are also the objective evidences from clinical examination. In many of the vast number of cases of otosclerosis there is no sign of there having been any middle-ear disease, nor any history of such either. I quite agree with Dr. Gray that we have closely to investigate the family history of these patients; if we do, we shall find some hereditary tendency, and most frequently on the female side. It may be a case of hereditary transmission of defect in internal secretion. There will have to be a great deal of work done by Mr. Fraser before it will be possible for him to convince the Section that otosclerosis is the outcome of a previously existing, or actually existing, suppurative otitis media.

Mr. Fraser (in reply): I am very much obliged to the members of the Section for the way in which they have received my communications. The President's remarks emphasise the need for the co-ordination of research. General surgeons tell us that in cases of fracture of the base the deafness only comes on after the patient has more or less recovered from his injury. I maintain that this is not so. The deafness is present all the time, but only becomes manifest when the patient recovers control of his mental faculties and finds that in one—more rarely in both—ears the hearing has been lost. I feel sure that if such cases were examined by otologists these views would be found correct.

With regard to otosclerosis, the theory that I support—namely, that in many cases otosclerosis is due to an inward spread of otitis media into the labyrinth capsule—has met with a good deal of criticism.

In answer to Mr. West, I can only say that I am sorry that the clinical history of my case is so imperfectly known. Mr. West objects to the term "chronic adhesive process." It is quite possible that a better term might be invented, but the words serve to indicate the conditions which exist in the middle ear when a catarrhal or purulent otitis media results in the thickening of the tympanic mucosa and the formation of adhesions. In some of these cases, at any rate, the old term "chronic catarrhal otitis media" is really more accurate as an inflammatory process is still going on. In other cases the term "cicatrised tympanum" would be more correct.

Sir William Milligan and Dr. Gray do not agree with me as to the inflammatory nature of the changes in otosclerosis. I have never maintained that otitis media is the "be all and end all" of otosclerosis. We have all suffered from head colds and a certain amount of tracheitis and bronchitis, and yet we do not all have chronic bronchitis or bronchiectasis. Most of us have had some indigestion and hyperchlorhydria at one time or another, and yet we do not all suffer from gastric or duodenal ulceration. Every case of catarrhal rhinitis does not settle down in the middle meatal region and result in nasal polypus formation. In the same way very many children have attacks of otitis media, and yet they do not all develop otosclerosis later in life. Undoubtedly there is another factor—the hereditary tendency to the disease. In susceptible people the region of the oval window is a *locus minoris resistentiæ*. If a child with a tendency to otosclerosis gets an attack of otitis media, I believe that otosclerosis is very likely to develop. Dr. Gray made some remarks last May which appear to me to show that he also was inclined

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to this view. There is also a growing amount of evidence to support the view that in certain families cases of otitis media are specially likely to develop intra-cranial complications. The only way to get rid of the hereditary tendency would be to forbid people to marry if there was a strong tendency to otosclerosis in the family. Koerner takes this line, but it seems rather a "large order."

If Sir William Milligan will read the conclusions to the article by Mr. Muir and myself in the November issue of the JOURNAL OF LARYNGOLOGY, RHINOLOGY, AND OTOLOGY, he will see that we do not claim that all cases of otosclerosis are secondary to otitis media. We believe that there is a "primary" variety which occurs with an apparently normal middle ear, and is usually seen in young women. In such cases the infection probably comes by way of the blood-stream, and the condition appears to be allied to osteomalacia. In the second class of case the otosclerosis is "secondary," and is due to an inward extension to the labyrinth capsule of an otitis media. In this variety the sexes are probably affected more or less equally. Lucae was of opinion that in chronic progressive middle-ear deafness it was impossible to draw a sharp line between cases of chronic adhesive process in the tympanum and those in which otosclerosis was present, and I believe that subsequent observations will still further support that view.

The most important question to decide is this: Is otosclerosis an inflammatory condition, or is it not? I believe that it is, but my opinion is not of great value. It is a very different matter with regard to the opinion of Mr. Richard Muir, who is one of the best known pathologists in this country. Mr. Muir holds that otosclerosis is undoubtedly of inflammatory origin. Dr. Gray can see "no sign of inflammation," and yet in the osteoporotic areas we find rarefied bone, in the spaces of which there are dilated blood-vessels surrounded by numerous small cells. Giant cells are also found in many cases. These appearances bear strong evidence of an inflammatory character.

I have been asked why, if otosclerosis follows otitis media, it is always located in the anterior margin of the oval window. I have shown you that in the present case the area of otitis vasculosa in the left ear was much more extensive, and affected not only the anterior margin of the oval window, but also the bony prominence of the external canal and the apex of the cochlea. I think it was Politzer who showed that at the anterior margin of the oval window there is a vascular connection between the vessels of the middle ear and those of the labyrinth capsule. Bruehl has also pointed out that this area is liable to special stress on account of the movements of the anterior part of the footplate of the stapes, and the presence of the tendon of the tensor tympani muscle in this region.

Sir William Milligan believes that there is some connection between the occurrence of otosclerosis and disorders of internal secretion. Well, of course, the ductless glands are all "the rage" at present. After failing with iodides, phosphorus, fibrolysin, otosclerol, and radium, I am now doing my best to be in the fashion, and am treating cases of otosclerosis with extracts of the ductless glands—pituitary, thyroid, and suprarenal. As yet the duration of treatment has been far too short to enable me to give a report on the results. My own view, however, is that it is *most important to make sure that cases of catarrhal and suppurative otitis media in children really do clear up completely.* Such cases should be carefully watched and treated for a long time after the acute

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symptoms have passed off. It is to be hoped that the attention now being paid to the operative treatment of the nose, nasopharynx and fauces of young children may result in fewer cases of otitis media, and a better recovery from this affection when it does occur. If this is so, we may expect to see a decrease in those cases of otosclerosis which are secondary to middle-ear inflammation. Vaccine therapy in cases of recurrent catarrhal otitis media may also be of use.

Since I have adopted the President's method of immediate skin-grafting at the end of the radical mastoid operation, and have observed the excellent results obtained, I have come to the conclusion that it would be quite justifiable to operate on cases of otosclerosis. I suggest that after the completion of the radical operation, the bony cap of the lateral canal should be removed without injuring the endosteum and a skin-graft applied at once. In this way a movable window would be supplied to replace the ankylosed oval window. The operation should, of course, in the first place be performed only on the more affected or deafer ear.

Dr. Dan McKenzie suggests that it may merely be coincidence that in these two cases otosclerosis was associated with otitis media. If this is his opinion, I do not agree with him. In the present case the patient had only slight remains of otitis media on the right side, but on the left there was a marked adhesive process in the upper part of the tympanum, while a perforation was still present in the lower part of the drumhead. It was on this left side also that the osteoporotic changes in the labyrinth capsule were most marked. The bony ankylosis is also more extensive on the left side.

(*Note.*—January 11, 1917: Since my return from London I have examined microscopically a *third* case of otosclerosis associated with chronic purulent otitis media. I hope to show this case to the Section at an early date.—J. S. F.)

There does seem to be a loss of trophic nerve influence in cases of otosclerosis, as evidenced by diminished sensibility of the external meatus and drumhead, and diminution or absence of the secretion of wax. The question is, Which is the cause and which the effect? If otosclerosis is an inflammatory condition, it cannot be entirely due to loss of trophic influence, but it is quite possible that weakening of this trophic influence may be an important factor in the pathology of otosclerosis; indeed, it may be that this is the way in which the hereditary tendency to the disease manifests itself.

A Note on the Monochord, with some Illustrative Figures.—W. M Mollison.—Ear tests are of doubtful value, but some of those in common use are at least interesting, and serve to differentiate cases of deafness into two main classes. Of these tests, that for determining the *upper tone limit* is perhaps the most interesting; it has always been doubtful what is actually the highest note appreciated by the normal ear, and I do not think it is important. What is of interest is that the upper limit is fairly constant and can be very accurately determined. The instrument which appears to me to give the most constant results is the monochord. It consists of a length of steel piano wire stretched between two points on a steel frame on which runs a bridge; this bridge grips the wire and so alters the effective length of wire which is to be thrown into vibration. Longitudinal vibrations are produced in the wire by rubbing it with resined leather (Tyndall) or with cotton-wool

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soaked in spirit. The great advantage of the monochord is that the note produced depends on three factors only: (1) The length of wire thrown into vibration; (2) the calibre of the wire; (3) the material. These three factors are easily standardised, and so an instrument is produced which gives always the same notes and requires no complicated standardising when finished as in the case of Galton's whistle.

The monochord has another advantage pointed out by Mr. Scott some few years ago; not only can the upper tone limit through air be tested, but also that through bone; the possibility led to the discovery that Rinne's test with high notes is always negative. The accuracy with which the upper tone limit can be defined is most striking; a difference of $\frac{1}{2}$ c.m. in a length of 20 c.m. is quite easily appreciated by the patient.

The results of the tests are expressed in lengths of wire the note from which the patient hears as a squeak; the result for hearing through air is placed above that through bone (the frame of the monochord being held on the mastoid process); thus for normal individuals the result is $\frac{13}{11}$ as an average.

The following are cases of shell shock or gun deafness and are chosen as typical of such cases.

	Age.	Hearing for whisper or conversation speech.		Monochord.		Notes.
1	21	4 to 5 ft.	3 to 4 ft.	$\frac{16}{14}$	$\frac{17}{14}$	In France fifteen months.
2	37	1 to 3 ft.	1 ft.	$\frac{17}{14}$	$\frac{16{\cdot}5}{14{\cdot}5}$	In the artillery.
3	40	2 to 4 ft.	3 to 5 ft.	$\frac{16{\cdot}5}{15{\cdot}5}$	$\frac{16{\cdot}5}{15{\cdot}5}$	Royal Field Artillery; always tinnitus after shooting.
4	35	8 in. conversation	3 to 4 ft.	$\frac{30}{18}$	$\frac{18}{14}$	Shell shock and gun fire.
5	26	A. c.	3 to 4 ft.	$\frac{36}{30}$	$\frac{31}{21}$	Shell shock.
6	38	3 in.	4 in.	$\frac{0}{45}$	$\frac{26}{17}$	Buried by shell one month ago; previous middle-ear catarrh.
7	24	8 to 9 ft.	2 ft.	$\frac{0}{0}$	$\frac{0}{0}$	Left eye destroyed by bullet; tinnitus before war; upper tone limit, ? 4000.
8	40	8 to 10 ft.	9 to 11 ft.	$\frac{25{\cdot}5}{16}$	$\frac{26}{17}$	Eight months ago a high explosive shell destroyed both eyes.
9	20	6 in.	3 in.	$\frac{30}{14{\cdot}5}$	$\frac{28}{18}$	Lost both eyes from shell explosion.
10	24	1 ft.	12 ft.	$\frac{18}{14}$	$\frac{15}{14}$	Tinnitus; knocked down by sandbags.
11	44	1 in. conversation	6 in. conversation	$\frac{0}{18}$	$\frac{32}{18}$	Previous deafness; no shell shock; Rinne negative.
12	46	4 ft.	2 in.	$\frac{22}{16}$	$\frac{42}{22}$	Tinnitus four years; much quinine in India.
13	21	3 to 8 ft.	10 ft.	$\frac{13{\cdot}5}{11}$	$\frac{13{\cdot}5}{11}$	Had been exposed to very severe shelling; thirty a minute for half an hour.
14	24	0	0	$\frac{34}{16}$	$\frac{34}{16}$	Functional dumbness and blindness cured by suggestion; with an electrical aid could hear quite well with left ear

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Traumatic Dislocation of the Incus, which was found lying in the Antrum in the Course of a Radical Mastoid Operation.—W. M. Mollison.—C. P——, aged thirty-eight, was exposed to a violent explosion in April, 1916. The explosion occurred about twelve yards from him on his left-hand side. He was unconscious for twenty-four hours; he then found he was deaf in the left ear. Soon after discharge began and has continued. He has some pain in the ear and slight tinnitus.

On examination in July, 1916, there was found a large perforation in the left membrana tympani posteriorly; there was foul pus coming from the middle ear. Hearing in the ear was *nil*.

On account of the foul otorrhœa, the radical mastoid operation was advised. This was done in October. The mastoid process was cellular, but contained no pus; the antrum was, perhaps, a little larger than normal, but it contained no pus. Whilst curetting was being done in the antrum and towards the aditus to remove small spicules of bone due to the gouging, the incus was found lying quite loose and bare, and stained by powder (?). The radical operation was completed, the malleus was in position, and the anterior half of the membrane affixed to it; a particle of powder (?) on a small granulation was lying over the stapes. The cavity was skin-grafted. The malleus and incus are shown.

A Case of "Cerebellar Storm."—W. M. Mollison.—Dr. Fawcett has kindly allowed me to bring forward these notes on a case which was under his care in Guy's Hospital.

Nurse P——, aged twenty-four, had a mild attack of German measles on March 7, 1916; she was apparently well in about a week, except for severe rhinitis. On March 16 she was walking in the hospital grounds when she felt sick and vomited. She had vertigo and vomited several times on March 16, 17, 18, and 19, and was quite unable to take any food at all. At the request of Dr. Fawcett I saw her on March 20. She lay in bed with her eyes closed, and through the lids the eyeballs could be seen moving. She felt very giddy, complained that objects moved from left to right, and that she herself felt as if she were moving. There was marked vertical nystagmus, with deviation of the eyes upwards and the quick movements of the eyes downwards.

Examination of the ears showed a little wax in the right, but a normal membrane with ? slight retraction; on the left side the membrane was a little pink in its posterior part. There was no mental clouding; she resented being turned over for examination as it made her more giddy. She retched frequently. The optic discs were normal.

Mr. Sydney Scott kindly saw her with me, and the following points were noted: The patient heard a whisper at least 8 ft. from either ear; the appreciation of low tones was normal (sixteen vibrations per second being heard). Rinne's test was positive, and the upper tone limit normal (monochord 14·5 cm.). There was no pain in the ears, and she had had none. The patient yawned frequently; knee-jerks were very brisk, Achilles jerks normal, plantar reflexes flexor; abdominal reflexes present and a well-marked *white tâche*; there was slight impairment of movements of pronation and supination of the right forearm compared with the left. There was a *pointing error* in the vertical plane; the arm passed much too far upwards, especially on the right side; in the horizontal plane the fingers fell below the starting-point on both sides, but perhaps this was due to lack of effort on the part of the patient.

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Lumbar puncture showed the fluid to be under very decidedly increased pressure, and one and a half test-tubes were drawn off; the fluid was clear and normal. It was not considered wise to apply the caloric tests on account of the vertigo already present.

Dr. James Taylor also saw the patient. No definite diagnosis was arrived at, but it was agreed that the condition was due to some cerebellar irritation of unknown origin. Potassium bromide was given *per rectum*. The patient could only take small amounts of lemonade and peptonised milk for the next two or three days.

On March 24 objects still seemed to move from left to right. Vertical nystagmus was still present, combined with lateral nystagmus to the left when looking to the left. There was no vomiting.

March 27: Patient was much better; lateral nystagmus to the right.

March 28: Nystagmus was now right rotatory on looking to the right, but lateral when looking to the left; no vertigo; rhinitis considerable.

The patient continued to improve, and by April 12 could sit up, though this made her feel somewhat giddy; there was then slight vertical and right rotatory nystagmus. After that the patient made a perfect recovery, and she is now back at work.

Labyrinthine Irritation in a Patient on whom a Complete Mastoid Operation had been performed some years previously.—Somerville Hastings.—M. J——, a young woman, aged thirty-one, had a left complete mastoid operation performed for chronic ear discharge eight years ago. The wound healed well, and for two years she had no trouble at all. Six years ago the discharge began again and was accompanied by tinnitus, and both have continued ever since. Three years ago she began to be troubled by giddiness, and this has also continued. The giddiness comes on at all times of the day and night, and she tends to fall to the left. When first seen on November 7, 1916, the left ear and mastoid cavity were filled by foul-smelling epithelial *débris* and pus. On removal of this, ulceration was seen on the inner wall of the middle ear, immediately below the promontory. The hearing in the right ear was good, but nothing was heard on the left by air conduction. On the left mastoid C_{64} and C_{512} forks were heard when vibrating strongly. The fistula symptom was well marked, and increased pressure produced rotatory nystagmus to the right with falling to the left. The left labyrinth reacted readily to cold, and rotatory nystagmus to the right was produced in ten seconds.

I should be glad of the opinion of members as to whether a labyrinthectomy would be desirable should symptoms still persist now that the ear is being kept clean.

A Doubtful Case of Labyrinthitis.—E. D. D. Davis.—A married woman, aged forty-six, attended the hospital seven and half years ago, February, 1909, for left middle-ear suppuration of twelve months' duration, with a large perforation in the lower and posterior part of the drum. She complained of tinnitus, which was arrested by compression of the carotid. She returned six weeks later with a mass of granulation tissue in the meatus, which suggested a malignant growth. The section merely showed granulation tissue, but a radical mastoid operation was performed by Major Waggett. The facial nerve was

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exposed and a temporary paralysis occurred, but the face recovered within two months, when the ear was healed and dry. After the operation the patient complained of attacks of vertigo, which have persisted up to the present.

When she was seen a fortnight ago the gait was staggering, and there was a tendency to fall to the left. There was no nystagmus, the pupils were equal and reacted to light; the disks were normal. The knee-jerks were exaggerated. Rombergism was well marked. Irrigation of the ear with lotion at 100° F. produced neither nystagmus nor giddiness. Irrigation with lotion at 105° F. for twenty seconds resulted in giddiness, with a tendency to fall to the same side, but there was no nystagmus. The mastoid area showed slight Eustachian discharge and *débris*. Hearing on the left side was considerably diminished, and bone conduction was reduced.

Mr. J. F. O'Malley: In Mr. Hastings's case I advise the performance of labyrinthectomy: the patient seems much incapacitated by the attacks of giddiness and vertigo which occur in the most unexpected manner. At present the ear is useless as an organ of hearing. With regard to the tests, the disturbance of equilibrium points, in my opinion, to the labyrinth on that side being under-active. There is sufficient life in it to cause irritation, but the disturbance of equilibrium is associated with the over-action of the good side. There seems to be a difference between the fistula test response here and that which one usually gets in fistula of the external semicircular canal, which suggests a fistula, not in the canal, but somewhere round the stapes, and this may explain the altered direction of the nystagmus. I think this degree of labyrinthine defect may become dangerous at any moment by failing to indicate the spread of sepsis, and, therefore it would be well to perform labyrinthectomy.

Dr. H. J. Banks Davis: Is Mr. Somerville Hastings certain that the patient has not got cerebellar symptoms? She is in a miserable condition, and before so serious an operation is undertaken, I think cerebellar abscess should be excluded.

Mr. Somerville Hastings (in reply): I thank members of the Section for the expression of their opinions. I thought of letting the case go a little longer, and then, if the patient did not improve, of performing labyrinthectomy. I think it unlikely that she has a cerebellar abscess. The symptoms have been going on for three years, and have varied very little, which is not like the behaviour of a cerebellar abscess.

Case of Malignant Disease of the Ear.—Sydney Scott.—The patient, H. R——, aged forty-nine, an engineer's fitter, had had malignant disease of the external ear, which was first noticed eight and a quarter years ago.[1] Histologically the disease was a small spheroidal celled carcinoma like rodent ulcer, but contained cell-nests and involved lymphatic glands (Prof. F. W. Andrewes). Three operations had now been performed during this period, and the whole of the left auricle with the bony meatus, the mastoid process, the parotid salivary gland, the whole of the sternomastoid muscle, and all lymphatic glands on the left side of the neck have been removed. The last operation was performed in May, 1916, when there was a small recurrence in the middle of the

[1] The patient was first shown in January, 1914; see *Proc. Roy. Soc. Med.*, 1914, vii (Sect. Otol.), p. 23.

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scar in the neck. Since this the left vocal cord has become paralysed, but there has been no sign of returning disease until the last few weeks, and now several nodules have appeared in the scar along the clavicle and in the posterior triangle. There is also a large nodule which has commenced to ulcerate over the carotid sheath close to the angle of the jaw. An expression of opinion is sought as to whether any further attempts should be made to remove the disease.

The patient is presented in conformity with the recommendation of the Council of the Section that the after-history of cases shown in the past should be recorded.

The PRESIDENT: I think it would be well to excise the outlying nodules, which are all quite small. It must be a chronic type of case. If the ulceration can be stopped, a great advantage will be gained. Are there any signs of secondary growths anywhere?

Mr. SCOTT: None, so far.

A Post-mortem Specimen of a Temporal Bone from a Case of Cerebellar Abscess.—W. M. Mollison.—The left temporal bone from a patient who died with a cerebellar abscess which gave rise to no symptoms. The posterior surface of the petrous bone shows a small collection of pus lying in the pocket of dura mater over the meatus vestibuli.

Dr. H. BANKS DAVIS exhibited Anatomical Specimens of the Petrous Bone.

Abstracts.

PHARYNX.

Excision of the Retro-pharyngeal Gland.—Norman Patterson. "Lancet," 1917, vol. i, p. 487.

Describes two cases and mentions a third, in which the author has performed this operation. He states that nearly all cases of retro-pharyngeal abscess in children especially, are due to suppuration taking place in one of the two retro-pharyngeal glands.

The operation is described. *Macleod Yearsley.*

Pharyngeal Pouches.—N. S. Finzi. "Proceedings of Royal Society of Medicine," February, 1917. Section of Electro-Therapeutics, p. 33.

The author is concerned only with the radiographic examination, appearances, and diagnosis of these cases.

The method used is the administration of a thick paste of bismuth oxychloride and water of such a consistency that, when heaped up, it has practically no tendency to flow back to its level.

The fluorescent screen examination of these cases is most important, as they may be very difficult to distinguish from stricture unless it is seen how the bismuth leaves the pouch. A pouch can be observed to fill, and when full, the bismuth passes into the œsophagus, going past the upper end of the pouch with ease, but being held up lower down in those cases in which the pouch is big enough to press on the œsophagus: in some cases the pouch can be observed to empty into the œsophagus when the patient contracts the neck muscles.

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