Glandular abscess behind ear	6
Plastic operation	7
Eustachian tube curetted	1
Aural polypi	21
Foreign bodies removed from ear	3
	278
Injection of galyl	2
Injection of salvarsan	2
	4

Anæsthetics administered.

Chloroform followed by ether	273
Ethyl chloride	1041
Local anæsthesia	650
	1964

Number of new patients attending the Department during 1915 = 3233.

SOCIETIES' PROCEEDINGS.

ROYAL SOCIETY OF MEDICINE—OTOLOGICAL SECTION.

February 18, 1916.

President: Dr. ALBERT A. GRAY.

Tumour on Concha after Radical Mastoid Operation (shown at the last Meeting).[1]**—W. M. Mollison.**—The tumour on the concha proved to be skin, with some fibrous tissue underneath; there was no cartilage in it.

The PRESIDENT: Is it keloid? It does not yet present that appearance. One views these growths which appear in or near a scar with suspicion lest ultimately they may become malignant, however innocent they appear to be. When there is a recurrence, the second growth is more rapid than the original one.

Sir WILLIAM MILLIGAN: Is the tumour keloid in structure? The importance of the case is from the practical point of view. My experience is that if one removes a keloid it returns, and generally becomes worse than before. Keloid in the neighbourhood of the ear is a disappointing condition with which to have to deal.

Mr. HERBERT TILLEY: Some twenty years ago a patient of mine had a very extensive keloid after the mastoid operation, and the question arose many times in the subsequent years whether it should be removed. Ten days ago, however, it was impossible to see any keloid in that patient, but its place was taken by a widened scar, as opposed to the usual narrow linear scar. I think it is wiser not to operate on keloid. As an alternative treatment I would suggest radium; we have had many

[1] See JOURN. OF LARYNGOL., RHINOL., AND OTOL., March, 1916, p. 91.

https://doi.org/10.1017/S1755146300016152 Published online by Cambridge University Press

cases of keloid at the Radium Institute, and I know of no pathological condition for which radium is more successful than in a case of keloid.

Mr. MOLLISON (in reply): For a keloid it had a very narrow base; one could not tell whether it started in the scar or not; probably it must have done so. Re-operation has since been performed on the mastoid, and the meatus is already tending to contract so much that gauze cannot be got into the cavity[1]; I do not know whether this may be due to the same cause as the "keloid."

Audible Tinnitus in a Boy (shown at the last Meeting).[2]—**W. M. Mollison.**—Since he was here the boy has been operated upon for removal of his tonsils and adenoids, but the tinnitus continues. His tubes have been inflated without causing any alteration in his tinnitus.

Dr. H. J. BANKS DAVIS: At the last meeting I saw Mr. Lake compress the carotid and stop the noise; but when the pressure was withdrawn the sound returned, which seems to point to a vascular condition as the cause. It would be interesting to ascertain whether the noise stops when the boy is under an anæsthetic. That would settle the point as to whether it is a "habit" or not.

Mr. E. D. DAVIS: Two months ago I had a case in a Belgian, who produced the noise by contraction of the temporal muscles. I taxed him with it, and told him that it was of no importance.

Mr. CLAYTON FOX: On nipping the boy's nostrils and making him blow out his cheeks to fix the soft palate the noise ceased, so possibly it was caused by the contraction of the tensor palati.

Dr. DUNDAS GRANT: I had a similar case, in which the noise ceased when I passed a Eustachian catheter and bougie. I think the noise was due to sudden separation of the glutinous walls of the Eustachian tube. It remained absent for some minutes, and then recurred. I think this should be treated as a nerve condition, being, in my opinion, of the nature of a habit spasm.

The PRESIDENT: If the case is of the nature of *tic convulsif* the outlook, according to some authorities, is not satisfactory, for such cases are said ultimately to develop mental trouble.

Mr. SYDNEY SCOTT: Last September I saw a patient, with normal hearing, who appeared to have voluntary control over the tensor tympani muscle. She was a lady with an extraordinary neurotic family history and was personally extremely introspective. I could hear a sound through the auscultatory tube which corresponded with the patient's effort to move the drum of the ear. I was able to exclude jaw movements, and could actually see a change in the direction of the light reflex from the membrane which indicated movement. During the apparently increased tension of the membrane, the lowest tones on the Bezold-Edelmann series were inaudible. At first the control was experienced in the right ear only, but after practice the patient was able to produce the same effect in the left ear. The examination was made with eyes closed, and the signs were repeatedly consistent with the patient's statement of her sensations.

Mr. MOLLISON (in reply): My assistant and I have looked at the palate repeatedly, but could not see a trace of movement. The noise stops momentarily when the patient opens his mouth wide. I see no reason why it should not be caused by the contraction of the tensor tympani.

[1] *Note.*—March 18: The cavity was completely dry.

[2] See JOURN. OF LARYNGOL., RHINOL., AND OTOL., March, 1916, p. 91.

https://doi.org/10.1017/S1755146300016152 Published online by Cambridge University Press

Sections through the Footpiece of Stapes from the Case of Otosclerosis shown at the last Meeting.[1]—**A. A. Gray.**—The sections are from four cases of otosclerosis of one year's, three years', twenty-five years', and sixty years' duration.

In the first of these the change in the bone was different from the ordinary otosclerotic changes; there was only absorption of bone, no deposit of it, so that the diseased parts scarcely stained at all. All that was left were a few blood-vessels and broken-down bone cells. The patient had fairly advanced phthisis, which may account for the absence of deposit of bone.

The second case was also the subject of phthisis, but it was not advanced. Here the new-formed bone stained deeply, and was sharply defined; there was no fixation of the stapes.

In the third case the stapes was united by bone from the anterior margin to the oval window. The bridge of bone which united the stapes to the oval window was exceedingly fine. It was not one-tenth part of the diameter of the cross-section of the footplate of the stapes.

The fourth case was that of an old lady, aged eighty. Rarefaction in one portion of the wall of the labyrinth had gone so far that there was practically no bone left. Osteoclasts were absorbing the bone. Manasse was of opinion that the bone is not absorbed by osteoclasts but by simple pressure.

An interesting feature in all the cases is the position of the anterior line of demarcation. It was almost identical in all of them, despite the great differences in the duration of the disease. I think it is a mistake to suppose that the bony changes are necessarily progressive. There was no sign of inflammatory activity in any of the sections. There was no evidence of change in the organ of Corti, nor in the ganglion spirale, except for the ordinary *post-mortem* changes. I am satisfied, however, that there is a change in the nerve structures, but our methods are not sufficiently refined to enable us to detect them.

Dr. Dundas Grant: Sometimes the clinical conditions and the tuning-fork responses are not typical, and yet the osteoporotic changes are found *post-mortem*. I should like to know whether Gellé's test was tried, and whether there was raising of the lower limit of audition.

Histological Preparations of the Labyrinths and Tympana of a Soldier suffering from Deafness due to a Bullet Wound of the Vertex of the Skull.—Sydney Scott.—C. T——, aged forty-two, a private in the Lincolnshire Regiment, was wounded on November 13 near Ypres, and died in France from meningitis on December 14, 1914. The autopsy showed the fracture was confined to the vertex of the skull. The histological examination reveals extensive intra-tympanic hæmorrhage on the right side. The serial sections demonstrate that the tympanic membrane, the footplate of the stapes, the annular ligament, and the membrana secundaria are perfectly intact and there is no hæmorrhage within the labyrinths.

Clinical notes.—The patient was admitted into the base hospital three days after being wounded; he was able to walk, was quite rational, but very deaf. He remembered being wounded, and stated he was standing in a trench at the time; when hit he fell down, and noticed loud buzzing noises in the ears immediately, with deafness. There was a lineal suppurating wound of the scalp 3½ in. long over the right parietal bone,

[1] See Journ. of Laryngol., Rhinol., and Otol., March, 1916, p. 92.

https://doi.org/10.1017/S1755146300016152 Published online by Cambridge University Press

running from the lambda to the parietal eminence. The X rays showed comminution and fissured fracture of the skull beneath the wound. There was no paralysis of the face or limbs, and the superficial and deep reflexes were normal.

Ears.—Some cerumen was present in both ears, but no blood or clot or cerebro-spinal fluid. The tympanic membranes were normal, but the light reflex on the right side was dark blue, an appearance which suggested the probable presence of blood in the tympanic cavity.

For several days after admission he could only hear shouting close to his ear, then he improved somewhat and could hear an ordinary clear voice about a yard away. He always spoke loudly, as if he could not hear his own voice.

	Right ear.	Left ear.
Edelmann-Galton whistle, No. 473	2·5 × 0·8 mm. = 19,000 d. v.	4·5 × 0·8 mm. = 13,920 d. v.
Tuning-fork, 256 d. v.	Rinne neutral Schwabach −6 sec.	Rinne negative Schwabach −2 sec.
Watch normally heard at 3 or 4 ft.	Just audible close to but not touching either auricle.	

Unfortunately, owing to the lack of instruments, we do not know what the low tone-limit was.

A fortnight after admission the patient became hemiplegic on the left side. The skull was explored, and fragments of the fractured parietal bone removed. A small tear in the dura mater was discovered, and some bone splinters were removed from the superior parietal lobule between the angular gyrus and the great longitudinal fissure. The hemiplegia was not relieved, and the patient succumbed to meningitis a fortnight later.

The necropsy showed extensive softening of the right cerebral hemisphere. The pia-arachnoid over the external and mesial surfaces of the right hemisphere were infiltrated with pus, which was limited to this region. There was no basal meningitis, and no fracture of the temporal bones or base of the skull. The temporal bones were fixed in formalin and decalcified with nitric acid, and embedded in celloidin in the ordinary way. The sections, which are arranged in series, were stained with iron hæmatoxylin, and show extensive blood-clot throughout the right tympanum, and only traces of blood-clot in the left tympanum.

The PRESIDENT: We have to thank Mr. Scott for showing us those sections, beautiful as his always are. Why is it that the hæmorrhage came into the tympanum without any sign of injury in the deeper parts?

Sir WILLIAM MILLIGAN: I should like to ask Mr. Scott why this patient was not operated upon a fortnight before the case actually was? I should have thought, from a general surgical point of view, immediate operation was called for; he ran every risk of having septic meningitis of his vertex. In regard to the deafness, I cannot understand how a patient who has a hæmorrhage into one tympanum and an intact labyrinth could be so deaf as the tests show. I ask whether there has not been some disturbance in the interior of the cranium which accounted for the profound deafness; whether the cortical centres for hearing were not injured by a commotio cerebri.

Dr. DUNDAS GRANT: In support of what Sir William Milligan has said, the tuning-fork on the mastoid was diminished to −6 sec. on one side and −2 sec. on the other. In the right ear the Galton whistle was

https://doi.org/10.1017/S1755146300016152 Published online by Cambridge University Press

heard at a higher pitch than in the left (apparently normal) ear. I therefore think there is justification for supposing there was some disturbance of the auditory centres. I have had at least one case in which deafness followed shell concussion, with similar hæmorrhages on the surface of the membrane. There were hæmorrhagic bullæ, which discharged into the meatus, from which came a discharge of blood. In some of the septic cases there has been previous traumatic rupture of the membrane, and infection has followed on account of the circumstances in which the patient was placed.

Mr. MOLLISON: In answer to Sir William Milligan, surely it is not necessary in the case of a man who has had a head injury to suppose he has necessarily anything wrong with his cortical areas because he has a high degree of deafness? This man had been exposed to shell shock, and had had a bullet wound of the scalp. I myself—and, no doubt, Sir William also—have seen men in whom complete deafness has resulted for a time from a wound in the head which had no relation whatever to the ear. Recently I saw an officer who was shot through from the right temporal region to the left malar bone; he was completely blind and deaf. But, during three or four weeks, his blindness gradually passed off and his hearing was restored; he could hear an ordinary conversation without trouble. These sections are of great interest as showing there is nothing wrong with the labyrinth, but it is unfortunate that they do not shed any light on the causation of cases of deafness from shell shock.

Dr. KELSON: The interest of the case is increased by the fact that there is no fracture of the base of the skull. Probably Mr. Scott has noticed that many patients who have been shot about the face or jaw speak often spontaneously of a discharge from the ear which commenced after the injury, and has persisted ever since. This occurs too often to be a mere coincidence; I think we must consider these as injuries due to indirect violence.

Sir WILLIAM MILLIGAN: I was not referring to the psychic cases of deafness in which there had been no injury to the skull. I have seen many cases of profound deafness due to shell shock, and have found the most severe deafness in men who had been buried for the time being.

Mr. J. F. O'MALLEY: The tests show the deafness here to be of the middle-ear type, although emphasis has been laid by some speakers on it having been a nervous injury. It is not due to shell shock. With regard to the deafness sometimes mentioned by soldiers who have had wounds of various parts of the head, my experience has been that many of them have evidence of old ear lesions. I have notes of several who manifestly have had loss of tympanic membrane, dating, perhaps, from a serious lesion in early childhood, and although the deafness preceded the injury, it only assumed importance afterwards.

Mr. SYDNEY SCOTT (in reply): The records of this case are too fragmentary to show what was the cause of the bilateral deafness. The damage to the right cerebral hemisphere should be taken into account. The chief feature of the case is that an injury to the vertex of the skull was associated with hæmorrhage into one middle ear without any fracture of the base of the skull, or of any injury to the tympanic membrane, labyrinth, membrana secundaria, or stapes. The source of the hæmorrhage cannot be found in any of the sections (of which there are about 200 in series on each side). Considering all the circumstances of the case, it is doubtful what would have been the result of an earlier intervention.

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Removal of a Piece of Shell from the Cerebellum of a Soldier wounded at Ypres.—Sir William Milligan.—The piece of shell (specimen shown) passed through the auricle, cut a groove in the soft tissues, and entered the anterior portion of the left cerebellar lobe. Severe hæmorrhage resulted, possibly from the lateral sinus. When seen a suppurating sinus led down to the foreign body. Removal was easily effected and complete recovery ensued.

Tuberculous (?) Septic (?) Meningitis following Chronic Suppurative Otitis Media; Operation; Repeated Lumbar Punctures; Prolonged Illness; Recovery.—Sir William Milligan.—Male, aged. twenty-seven, admitted to the Manchester Royal Infirmary (Throat and Ear Department) on December 28, 1914, complaining of discharge from left ear, violent headache, and vomiting.

Previous History.—Had severe attack of broncho-pneumonia as a boy, followed by pulmonary tuberculosis. In February, 1912, had a second attack of right-sided pneumonia, followed by extensive breaking down of the lung and formation of a large apical cavity. Enormous quantities of secretion were expectorated. Shortly before this second attack of pneumonia the left middle ear had become infected and had discharged freely. In November, 1914, he joined the R.A.M.C.; he was sent to Southport and was vaccinated there. Was very much upset by vaccination (?), violent pain in the head and profuse discharge from the ear. Was sent home. Discharge from ear profuse, violent headache, vomiting, dull lethargic condition, gradually deepening into semi-unconsciousness. Admitted to Royal Infirmary on December 28, 1914. Temperature 99° F., pulse 80, respirations 23. Left-sided facial paralysis, pupils unequal. December 29: Complete post-aural operation and decompression over temporo-sphenoidal and cerebellar areas. December 30: Evening temperature, 104° F. December 31: Mid-day temperature, 99·5° F.; evening temperature, 102° F. Retraction of head; severe headache. Kernig's sign present; patient delirious, very noisy; swore immoderately when touched. Lumbar puncture performed: fluid under pressure; very turbid. Report *re* cerebrospinal fluid: "The fluid contains pus; no tubercle bacilli or other organisms found. Culture yields no growth of organisms. The condition may therefore be tuberculous. Ice-bags applied to head. Urotropine, 20 gr., every four hours. During the following ten days repeated lumbar punctures; fluid still turbid. January 11: Condition critical; saline enemata with brandy. January 14: Temperature came down from 102° F. to 98·4° F.; pulse from 138 to 120, and respirations from 36 to 24. Temperature remained about normal until January 19, when it went up to 101° F., pulse 112, respirations 32. Repeated lumbar punctures. From this date a gradual improvement set in. February 7: Morning and evening temperature normal; patient rational. February 22: Patient allowed out of bed. March 8: Wound healed; complete epidermisation of antro-tympanic cavity. March 16: Discharged. Patient has remained well; his mental condition good; pulmonary symptoms not so marked. Attention is specially drawn to the value of repeated lumbar puncture.

Mr. J. F. O'Malley: I would add a third query to the two propounded in the title—namely, as to the possibility of epidemic cerebro-spinal meningitis. I have now had a case for several weeks, and the patient was under my care before the present attack, as three months ago I removed an aural polypus from him. He had a rash. The cerebro-spinal fluid was examined the second day of the disease, and though pus

https://doi.org/10.1017/S1755146300016152 Published online by Cambridge University Press

was found there were no organisms after three days' incubation. This was so until the eighth day, when no organisms were found by direct examination by two bacteriologists, but there were two colonies of meningococci on culture. The early symptoms of Sir William's case and mine were almost identical, except that my patient did not vomit, but had headache all the time. The medical officer at the isolation ward was very expert, and felt confident about the diagnosis, though it was suggested by the bacteriologist that the rash might be septic. As the cerebrospinal fluid finding was so often negative it was thought it might be otitic meningitis. Lumbar puncture is performed on alternate days, and the patient is recovering.

Dr. H. J. Banks Davis: I do not think a patient who appears to have been so ill could live with cerebrospinal meningitis from December 31 to March 16, and the history is against this diagnosis. As a rule, when extremely ill, the patients succumb to cerebrospinal meningitis within a week or ten days.

Mr. Sydney Scott: Was there any evidence to show whether the infection was from the ear, and if so was it by the translabyrinthine path? I recollect a child who died of meningococcal meningitis after pneumonia, from whose tympanic cavities two kinds of organisms (on each side) were isolated—namely, intracellular cocci and capsulated extracellular cocci, the first corresponding to the meningococcus which had been isolated from the spinal fluid during life, and the second resembling pneumococci.

The President: I presume the pus from the case was not injected into a guinea-pig, as that might have solved the problem as to whether it was a case of tuberculosis?

Sir William Milligan (in reply): It never occurred to me that the case might be cerebrospinal meningitis; I regarded it as an ordinary septic case, and expected that an organism would be found. Only two bacteriological examinations were made, and no organisms were found. Still, I should be rather sceptical of its being cerebrospinal meningitis; there was no rash. The other day a case was sent in with symptoms of meningitis, and I diagnosed septic meningitis; but when the fluid was reported on by the bacteriologist, he said he had discovered one single meningococcus. I expressed my doubt as to that being sufficient to support a diagnosis of cerebrospinal meningitis, and it proved later to be an ordinary septic case. I do not know what the path of infection in this case was: it was not the labyrinth; it was a pure middle-ear case. What ought we to consider septic meningitis? Is the presence of this established without demonstrating organisms? I have been in the habit of rejecting the diagnosis unless organisms are found. With regard to injection of material into a guinea-pig, I still do that occasionally, but did not do so in this case. I do not think the decompression did him much good; what I regarded as most beneficial were the repeated lumbar punctures which kept the intracranial pressure down, thus preventing death from compression of his own vessels and respiratory paralysis.

Malignant Disease of Middle Ear following Chronic Suppurative Otitis Media; Extension to Mastoid Cells; Operation; Rapid Recurrence; Employment of Radium; Temporary Relief.—Sir William Milligan.—J. B——, male, aged fifty-eight, seen on April 3, 1913, suffering from right-sided suppurative middle-ear disease of many years' duration. Right ear discharging freely; perforation of

https://doi.org/10.1017/S1755146300016152 Published online by Cambridge University Press

posterior segment of membrana tympani; polypus; slight tinnitus; no vertigo. April 7, 1913: Polypus removed; chromic acid applied. June 5, 1913: Ear quite dry; perforation not healed. July 8, 1914: Recurrence of polypus—rather vascular. July 21, 1914: Removal of polypus; malignancy suspected, but not proved microscopically. August 6, 1914: Discharge slight, but fœtid; slight hæmorrhage. Antiseptic treatment ordered. March 8, 1915: Radical mastoid operation; granulation tissue very vascular. April, 1915: Antro-tympanic cavity unhealed; definite microscopic evidence of malignancy. May, 1915: Post-aural cicatrix broken down; fungating granulation tissue; slight pain; no glandular involvement. July, 1915: Patient losing weight: several fairly severe attacks of hæmorrhage from ear; slight enlargement of glands; severe pain. August, 1915: Radium emanation tube (25 mgrm. element) inserted into fungating mass. September, 1915: Pain and hæmorrhage much relieved; growth shrinking; free discharge from the meatus. November, 1915: Growth half its former size; recurrence of pain; glandular involvement greater than before; facial paralysis. Radium emanation tube (25 mgrm. element) again inserted. December, 1915: Local relief, but systemic decline; evidence of toxæmia. January, 1916: Patient much weaker; no great alteration in local condition. February, 1916: Patient rapidly losing ground; glandular involvement about the same; no increase of local growth, but progressive loss of strength and weight. Condition hopeless.

The PRESIDENT: I think cancer arises more frequently in, and is more common in, a suppurating middle ear than in an ear which is not so affected. I saw a case in which a growth was deep in the meatus there was no suppuration, and it was secondary to malignant disease in the breast. Radium was employed, and the growth completely disappeared and did not return; but the patient died of a lesion in the lung, secondary to the breast cancer.

Sir WILLIAM MILLIGAN (in reply): With regard to a remark made by a member as to radium increasing pain, my experience has been the opposite—namely, that it relieves pain. It also stops the hæmorrhage, which is a very important point. I do not know who made the report on radium to which reference has been made. Its action in cases of sarcoma is extremely beneficial, but in carcinoma its benefit is doubtful. However, we are only just beginning to understand about the action of radium, which is generally used only in the cases which are otherwise hopeless; it has never had a proper chance in cases which we speak of as operable. To condemn radium at the present stage is absurd. Radiologists seem to agree that embedding is the best way in which to use it.

The Significance of Unilateral Sero-mucous Catarrh of the Middle Ear in Persons over Middle Age.—Sir William Milligan.—Unilateral sero-mucous catarrh of the middle ear in persons over middle age should always be regarded with suspicion. A diagnosis of simple catarrh is often made and is just as often wrong. Some incipient malignant process or syphilitic manifestation should always be thought of, and a guarded prognosis given.

In many cases malignant ulceration of the naso-pharyngeal mucosa or a malignant growth of the basi-sphenoid is discovered if careful rhinoscopic examination is made, or some syphilitic process is found to be the underlying fact in the case.

https://doi.org/10.1017/S1755146300016152 Published online by Cambridge University Press

A careful rhinoscopic examination by mirror or naso-pharyngoscope should be made as a matter of routine in all such cases, and frequently leads to important and far-reaching clinical discoveries.

Mr. TILLEY: I wish to support emphatically what Sir William Milligan has said. In the last fifteen years I have seen eleven cases of endothelioma of the naso-pharynx, and one of the saddest of these I saw three years ago. The patient had seen two well-known aural surgeons in this country, and three on the Continent. He complained of deafness, which varied in degree, and he was told it was catarrhal deafness. One surgeon catheterised him every week for two months. I saw him rather late in his illness, when one nostril was blocked, and there was a bluish, congested-looking mass in the naso-pharynx. It eventually invaded his skull, and he died of cerebral symptoms and in great pain. In these cases, when the deafness has been present for a matter of weeks, you generally find some difficulty in the movement of the levator palati on the same side; this is due to mechanical impairment rather than to a nerve lesion. Then follows anæsthesia of the second division of the fifth nerve, evidenced by loss of sensation on the outside of the lower jaw and over the lower portion of the cheek. This anæsthesia and the weakness of the levator palati are almost pathognomonic of the condition. If in such a case 100 mgrm. of radium are embedded for twelve hours, it is extraordinary how the condition will disappear. Last week I saw a man in whom the growth was as large as the end of one's thumb, and the patient could not blow down the right nasal fossa. Radium was embedded a month ago, and when I saw my patient last week no trace of the growth was to be seen; the nasal breathing was free, and the pain in the temporal region had disappeared.

Dr. DAN MCKENZIE: Possibly the reason why these cases are overlooked by those who see the patient earlier is that the growth is not then sufficiently large to be visible by the post-rhinoscopic mirror; therefore all cases of deafness should be examined with the naso-pharyngoscope as a matter of routine. Such cases would not be missed by this means, even at the earliest stage.

Dr. JOBSON HORNE: I have always practised and taught a routine examination of the post-nasal space and of the posterior nares with a mirror wherever it is possible, in all cases of diseases of the ear, the nose, or the throat. The importance of that practice and of that teaching has been fully established not only by the clinical surprises from time to time observed and by the aids to diagnosis afforded, but also by the calamities at times thereby prevented.

Mr. SYDNEY SCOTT: It is sometimes difficult to decide whether some cases of middle-ear catarrh are secondary to malignant disease or to syphilis. Last November I saw a patient at the National Hospital for Paralysis, who for six months previously had been treated at another hospital for unilateral middle-ear catarrh and Eustachian obstruction. He then developed optic neuritis and paralysis of the left sixth and seventh cranial nerves. The left eighth cranial nerve was unaffected. In the naso-pharynx was an ulcer which involved the infundibulum of the left Eustachian tube; it looked like malignant disease, but the nerve lesions pointed to syphilis. (I shall refer to this case more fully another time.) More recently I saw another patient whom I had examined three weeks previously, and found nerve deafness in the right ear. During the interval he had awakened with loud tinnitus and great deafness in the left ear, which had been previously normal. I found the signs of nerve deafness with great loss of bone conduction and of high and low tones,

https://doi.org/10.1017/S1755146300016152 Published online by Cambridge University Press

but the left Eustachian tube was impervious even to bougies. His history of syphilitic infection was very definite; he had been treated intramuscularly with salvarsan two years ago, followed by a course of mercury for one and a half years. A week later I found the Eustachian obstruction had gone, the membrane returned to the normal, but the hearing was still worse.

Cases illustrating Otogenic Facial Paralysis.—Dan McKenzie. —Case 1: *Post-operative Paralysis.*—Male, aged twenty-nine, shown before this Section on October 12, 1912, after recovery from lateral sinus thrombosis and serous meningitis, for which translabyrinthine drainage had been adopted. The patient had two operations in all. At the first a huge cholesteatomatous cavity was disclosed, in the depth of which the facial nerve was found slung across the space like a thread and devoid of all bony protection from the genu to the stylo-mastoid foramen. But no paralysis was observed until several hours after the second operation, at which the labyrinth was penetrated to reach the internal auditory meatus, so that the injury to the nerve must have been inflicted during this procedure. The course of the paralysis since its inception illustrates the striking tendency to recovery, but often only to partial recovery, manifested after severe injuries to the nerve in the temporal bone. For six months no change could be detected in the face, but at the end of that period a little fibrillary movement in the fibres of the orbicularis palpebrarum was perceived when the patient was made to close the eyes tight, and the improvement has gone on continuously, but very slowly indeed, during the three years that have elapsed since then. Now he can close the eye, the ala nasi shows movement, and the naso-labial fold appears when he smiles. But none of these movements is equal to that corresponding to it on the other side of the face. The patient himself is of opinion that the improvement is still continuing, and with this I agree, but progress is extremely slow. The reaction of degeneration has never been present.

Case 2: Male, aged thirty-two, shown before this Section on May 14, 1915, as a case of recovery after purulent meningitis. In this, as in the first case, translabyrinthine drainage was effected, but the paralysis was observed on the patient recovering from the anæsthetic. The first sign of recovery was seen on December 17, 1915, thirteen months after the operation, in slight movement of the orbicularis palpebrarum, the dilator naris, and the levator anguli oris. The improvement, in this case, unlike the last, is proceeding rapidly.

Case 3: *Concussion (?) Paralysis.*—This case is included in the series, as it seems to suggest the explanation of facial paralysis following mastoid operation in which no sign of exposure of the nerve at the operation has been detected. A male, aged twenty-five, received what was supposed to be a shrapnel wound of the mastoid on May 1, 1915. He was rendered unconscious for a couple of hours, but no facial paralysis was noticed until two days later, and in four weeks the paralysis began to get well. There is now no sign of it. A small fragment of metal which had lodged in the tympanum was removed by the exhibitor eight weeks ago, by means of a radical mastoid operation, and the route of the missile was then seen to pass through the mastoid and the posterior meatal wall close to the tympanum, and within a few millimetres of the Fallopian canal. As far as could be made out the nerve itself had not been exposed.

Case 4: *Paralysis from Otitis Media Purulenta.*—Woman, aged

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thirty-two, who came to hospital with chronic suppuration in the left ear and complete facial paralysis of the corresponding side of the face of one week's duration. The radical mastoid operation revealed cholesteatomatous disease, which had exposed the facial nerve for about ¼ in. in the tympanum. The facial paralysis began to improve six months after the operation, but in this case, as in the first, complete recovery delays its coming.

CASE 5: *Herpetic Paralysis.*—Male, aged thirty-two. This case was published *in extenso* as one of herpes zoster oticus combined with recurrent laryngeal paralysis in THE JOURNAL OF LARYNGOLOGY, RHINOLOGY, AND OTOLOGY (September, 1915, p. 339). It is now shown here with the facial paralysis nearly well. (The laryngeal paralysis lasted a few days only.)

CASE 6: *Paralysis from Otitis Media Catarrhalis.*—Boy, aged eight. After an attack of acute pain in the right ear, facial paralysis suddenly appeared. There has been no discharge from the meatus. When first seen on December 8, 1915, the membrana tympani showed some dull redness in the upper hemisphere, but it did not rupture. There was no herpes. Consequently this is evidently one of those rare instances of paralysis from simple catarrh of the middle ear, several cases of which have been shown before this Section. The paralysis is rapidly recovering, having been complete for a month only.

The most important cases of facial paralysis from the surgeon's point of view are, of course, those which follow mastoid operations. In my experience, severe facial paralysis from this cause is very rare, and when it does occur it nearly always undergoes great improvement, if not complete recovery, sooner or later. Years, indeed, may elapse—according to Vohsen as much as ten years—and yet recovery take place. I suggest that the reason for recovery, after even complete section of the nerve, lies in the fact that in the facial nerve, passing as it does through a fine canal in the bone, section may occur without the ends of the nerves being separated from each other, and as the intervening gutter or bridge of bone may be assumed to serve as a guide to the sprouting nerve fibres, their ultimate union is probable. On the other hand, as the cases now exhibited show, complete recovery may be delayed for a very long time; indeed, it may never be reached. This, also, I ascribe to the narrow channel through which the nerve passes. If the nerve is severed the scar tissue which forms will block the passage and prevent the full reunion of the nerve.

I have said that in my experience, and I believe in all otologists' experience, complete facial paralysis from operative trauma is extremely rare. On the other hand, I find paresis to be relatively common. This paresis varies in severity. In the mildest cases a slight lagging behind, or delay in closing the corresponding eyelid as compared with the movement of the opposite eyelid in the ordinary action of the involuntary winking of the eyes, is all that appears, and it is so slight as to escape all except the most jealous observation, and so transient that it is visible for but two or three days only. Then there are the more obvious and familiar cases, in which the paresis slowly spreads from the eyes to the rest of the face, and after four or five weeks slowly disappears. In many of those cases the surgeon is prepared for the paralysis by the appearance of a facial twitch at the operation. In others, again, no warning has been given, and the paralysis comes as a surprise. It is in such cases that I suggest concussion or contusion to be the cause of the nerve lesion—by contusion I mean rather hæmorrhage into the

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canal—the effects of which are not immediately apparent after the operation. Case 3 illustrates this type, and I can recall another in which the paralysis followed the use of a blunt set of chisels at the operation.

When the nerve is exposed by natural dehiscence in its bony envelope, or by caries of the bone, post-operative paralysis, when it appears, is probably due to septic or traumatic neuritis. It appears about three days subsequent to the operation, lasts about six weeks, and finally gets well.

We frequently come across cases at which a twitch during the operation reveals the exposure of the nerve, but in which, for some unknown reason, no paresis or paralysis ensues.

In the radical mastoid operation one great difficulty we have to contend with is to clear disease from the posterior or postero-external wall of the tympanum (the sinus tympani), where caries often lurks. Curetting here is liable to injure the nerves, and yet to neglect this corner is to run the risk of prolonging the disease and losing the benefit of the operation.

The PRESIDENT: This question of facial paralysis is a very practical one. I have had my share of cases of post-operative facial palsy. I had one case in which the nerve had been exposed by cholesteatoma, and the operation was the ordinary radical mastoid. In these cases I have found that the quicker the suppuration ceases, the quicker is the repair of the nerve. That is what one would expect on *à priori* grounds.

Dr. H. J. BANKS DAVIS: In the first case the note says: "The reaction of degeneration has never been present." I suppose that this is an error and means that it has always been and is still present. I think it is there in every case of paralysis after the first week or ten days following injury to the nerve below the nucleus. It may, of course, only be "partial reaction of degeneration" and not complete, but the responses to electrical stimulation cannot be normal if facial paralysis is present, unless it be "functional."

Dr. DUNDAS GRANT: I wish to support what Dr. McKenzie says with regard to the jamming of the nerve by cicatricial tissue in the tube. I would remind you of a paper which was read by Ferdinand Alt at the International Congress in London in 1913. It was on old-standing cases of facial paralysis which he had operated upon by deliberately opening the Fallopian canal, and liberating the facial nerve from the pressure. I should feel some diffidence in doing it, but there may be others who have experiences of the kind to offer. I agree it is very rare for the paralysis to remain complete for any great length of time. I think, with Dr. Gray, that recovery is hastened by using the continuous current. I should like to hear what results have been obtained by the anastomosis of the facial nerve with one of the other nerves. I have not had the opportunity of seeing the ultimate results of that procedure; operators do not seem anxious to record them. In opening the aditus I use a guard, and I think it has helped me to avoid facial paralysis. But the guard must be held by an experienced person, otherwise it may defeat its object, especially if there is anything like dehiscence in the canal. Anatomical investigations have shown that the course of the facial nerve varies; in some cases it runs vertically downwards from the inner wall of the aditus; in others it runs for some distance horizontally outwards, and then makes an almost right-angled turn downwards; and in the latter cases it is specially liable to injury. By careful operating one can chip off the corner of the bend without cutting through the facial nerve.

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In using the chisel, I find it best to turn the bevel of it over, so that the tendency is for the tool to work towards the surface rather than the deeper parts. I ask whether it is common for the facial nerve to be affected when translabyrinthine drainage is carried out. It is such a fascinating procedure that one would like to be assured it is free from that risk. Of course, a freer drainage can be secured by making an opening in another place, though the ensuring of asepsis is then probably more difficult.

Sir WILLIAM MILLIGAN: If Dr. McKenzie will do me the honour of reading Milligan and Wingrave on "Diseases of the Ear," he will find a chapter devoted to facial nerve paralysis. I rise, however, to draw attention to the injury which is often done by curetting the middle ear. If it is to be done at all, it should be carried out from behind forwards; it is then less risky. If, unfortunately, one divides the nerve in the course of an operation, one should have the courage to open up the aqueduct immediately as far as possible, and endeavour to twist a little piece of foil or indiarubber tissue round the nerve, so as to conduct it to the distal opening in the bone, and so encourage repair. I have seen two cases of bilateral facial paralysis in middle-ear disease. I agree that there is generally some recovery in these cases, and the lapse of years does not make the case hopeless. It is rare to find a patient suffering from absolute and permanent palsy of the nerve. We in this country do not see anything like so many cases of this kind as are to be met with in many Continental clinics.

Dr. JOBSON HORNE: I agree with what has been said about the importance of care in curetting the middle ear in the mastoid operation, and apart from any mastoid operation I would urge particularly the advisability of confining all curettage of the middle ear to patients indoors and in bed. Referring to Dr. Grant's point about reversing the chisel when removing the bridge in the mastoid operation, so that the bevel of the chisel is on the under surface, at that stage of the mastoid operation I work with a curved chisel with the bevel on the under surface. With the use of that instrument and Hartmann's punch forceps I have always escaped injuring the facial nerve. Apart from traumatism, there have been cases of ear disease in which facial paralysis was inevitable, such as tuberculous disease of the ear.

Mr. SYDNEY SCOTT: We do not find that facial paralysis always follows the translabyrinthine operation for meningitis,[1] although it occurred on the third day in the first case Mr. West and I described. One should be specially careful to keep close to the floor of the internal auditory canal.

Dr. DAN MCKENZIE (in reply): I will not reply at length at this hour, but suggest the subject for a discussion at another session. Unless you curette the ear very thoroughly in some cases, you will not get it to heal up. And in the cases of caries in the posterior part of the tympanic cavity, the spot where the nerve is most liable to be exposed and where facial paralysis is produced, one must always curette there if the case is to be cured. I think we should decline to remove a polypus from the middle ear except through the mastoid. I have twice seen a polypus attached to the exposed trunk of the nerve, and if it had been removed in the ordinary way, facial paralysis would have been inevitable.

[1] *Proc. Roy. Soc. Med.*, 1908–9, ii (Otol. Sect.), p. 11.

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