SOCIETIES' PROCEEDINGS

ROYAL SOCIETY OF MEDICINE—SECTION OF OTOLOGY.

President—Mr G. J. Jenkins, F.R.C.S.

March 6, 1926.

Hæmorrhagic Types of Ear Disease occurring during Epidemics of Influenza, by Sir William Milligan, M.D. The Paper appears in the *Journal of Laryngology and Otology*, vol. xli., No. 8, August 1926, p. 493.

Discussion.

Mr G. J. Jenkins (President) said that during the last few years in this country there had been epidemics of influenza associated with otitis externa hæmorrhagica; three years ago he (the President) had had five cases of this kind within a short time and had believed that he had met with a new disease, but had subsequently found that there was a clinical description of the condition in Politzer's work. In most of the cases he (Mr Jenkins) had seen the hæmorrhagic blebs had been quite large, almost filling up the meatus, though usually one could see the tympanic membrane beyond, almost normal in appearance. In some cases in this group, however, he had found all the signs and symptoms of the condition, without the hæmorrhagic blebs in the meatus; but in the latter cases there was always a redness of the meatal wall, often with slight injection of the margin of the tympanic membrane. In addition to the meatal changes, there was commonly pain and tenderness in the mastoid region; in a few the patient complained of vertigo, and there was definite nystagmus to the same side with indefinite Rombergism. The most striking feature was that with all these sometimes severe symptoms and marked changes in the meatus, there had been the slightest, if any, affection of the hearing. Occasionally the condition was associated with cranial complications. In two of his (the speaker's) cases there had been cerebritis, and in one of these the patient died from thrombosis of the longitudinal and lateral sinuses. The suggestion was that some cases of persistent nerve deafness or labyrinthine disorder were due to the general affection of the region. The hæmorrhagic blebs, if kept clean, often dried up without suppuration. His (Mr Jenkins') method was to protect the ear or, at most, to put a few drops of perchloride lotion into the meatus. As the condition was associated with influenza and the blebs had not an inflammatory character (never went on to suppuration), he thought the condition was not due to infection with the ordinary pyogenic organism, and that it might be caused by one of the filter-passing organisms. An attempt had been made to make cultures of ordinary pyogenic organisms from the blood of the blebs in the three epidemics; in some the results were negative, but in others streptococcus or pneumococcus was found. He (Mr Jenkins) had consulted with Dr Perdrau, an authority on filter passers, who injected some of the fluid from a bleb under the skin, and some of the cerebro-spinal fluid

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of an affected patient, into the brain of a rabbit. The injection did not produce the reaction caused by an injection of the herpetic virus, establishing the fact that the disease was not a herpetic eruption in the ordinary sense, and that there was no relationship to herpes labialis. He regarded the suppurative process associated with this condition as secondary, just as in the case of antrum infection after influenza. Among the cases being watched at King's College Hospital last week there was one of typical otitis externa hæmorrhagica, and at a later stage it was decided that it was an ordinary suppurative mastoiditis; it was difficult to know when the process became suppurative. In the last three years there had been a number of cases at King's College Hospital.

Mr SYDNEY SCOTT said that he had observed that the bullæ which Sir William Milligan had described occurred in both the mildest and most grave forms of otitis media. There were cases in which the patient would come to the out-patient department with a hæmorrhagic bulla on the tympanic membrane, or in the meatus, which cleared up under the simplest treatment. On the other hand, they met with the same condition in otitis media with fulminating leptomeningitis. He (Mr Scott) had been particularly interested in Sir William's reference to the nerve deafness sometimes following the influenzal otitis media cases which showed signs of true nerve deafness. It might well be, as Sir William had suggested, that effusions took place in the cochlea similar to those seen in the drum membrane or meatal wall.

Mr E. D. D. DAVIS said that two definite clinical types were under discussion. One was the acute streptococcal otitis media, of which there had been a considerable epidemic this year, with a high temperature, an infection of nose and throat, and occasionally tonsillitis. When examining the ear in these cases, blebs were seen on the drumhead, and after puncture there was a profuse blood-stained serous discharge. This otitis media was considered to be caused by a hæmolytic streptococcus. In the other type the inflammation seemed to be confined to the auditory meatus and was an otitis externa. Sometimes a bleb would burst and the serous discharge appear to come from the middle ear, resulting in the mistaken diagnosis that the drum had ruptured and that puncture was unnecessary. In cases of the otitis media described above, the drum should always be punctured to enable free drainage to take place, and he (Mr Davis) had never known puncture do harm. When pain persisted after puncture it was difficult to decide whether a mastoid operation should be done or not. He (the speaker) usually waited for forty-eight hours, and if in that time the patient's condition had improved, the mastoid was not opened.

Mr J. F. O'MALLEY said he remembered that otitis media met with during an influenza epidemic was associated, in many cases, with hæmorrhage. Up to the severe epidemic of 1918, which had been marked by pneumonic complications of a fatal type, he (the speaker) had been at a loss to account for this hæmorrhage. Then it had been reported that there was a common association of *streptococcus hæmolyticus* with the bacillus of influenza, and he (Mr O'Malley) thought that that association had been perhaps the chief factor in an epidemic of ear disease with hæmorrhage occurring at Woolwich at the end of 1918. He had sought,

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by examination of the fluid, for a proof of this theory, but only in two or three cases could he get the *streptococcus hæmolyticus* in secretion drawn directly from the middle-ear cavity.

Sir JAMES DUNDAS-GRANT said that the appearance of the tympanum in this condition was apt to be unduly alarming ; it was out of all proportion to the actual mischief, for on testing the hearing it was found to be almost perfect. This disproportion should put the observer on his guard. He had sometimes experienced difficulty in diagnosing between the bullous condition and disease in the external attic. Sometimes there was a bulging from the attic which looked like a sac, but it was infiltrated tissue. If pricked it still remained there. It could be snared off, though even if that were not done it often subsided. There was, however, always at least some slight loss of hearing in attic disease. It was very important to remember the apical infections in which the mastoid might be opened and at first nothing be found. Yet one knew something was there, and it was not until one came to the apex that one found a large cell containing a considerable amount of pus. On evacuating that, all the symptoms subsided. Politzer had found the same condition prevalent in an epidemic in Vienna. Why should cortical or apical cells be selected? It depended largely on the anatomical construction of the individual mastoid process. There was less surface for phagocytosis on the walls of one large cell than on those of a number of small ones with dissepiments. Bezold had suggested, and it was quite possible, that the narrow communication between the antrum and the large cells had become sealed up by inflammation. He (the speaker) brushed out the meatus with spirit to render the outside aseptic, and then introduced drops such as the late Mr Hunter Tod had described, namely, morphia 1 part, cocain 2 parts, phenol 3 parts, to a drachm of almond-oil or glycerine, above all keeping the patient at rest, to prevent the condition from becoming a major one.

Mr NORMAN BARNETT said that after an epidemic of influenza a case was sent to him in which there was apparent hæmorrhage into the semi-circular canals ; there was no symptom of hæmorrhage elsewhere. All other possible causes of hæmorrhage in this region were excluded ; under treatment absorption gradually took place and the symptoms passed off.

Mr W. M. MOLLISON said he had seen epidemics of so-called influenzal otitis externa, and he had never had the courage to leave the membrane unincised. He had seen another condition in which blebs occurred, namely, in the terminal stages of very severe illnesses ; a few days before death he had found the same variety of bleb in the meatus.

Mr A. R. TWEEDIE said that he looked upon this condition as essentially a middle-ear otitis, illustrating the point that the subperiosteal space of the bony meatus was in anatomical continuation with the subperiosteal space of the tympanum, and that thus, in this respect, the bony meatus really represented an extension of the middle ear. This point was also demonstrated in the case of acute otitis media with distension of the posterior meatal wall and adjacent membrane ; it supported the old instruction as regards paracentesis, to the effect that the knife, after incising the membrane, should be carried backwards along the posterior meatal wall. The method of applying a local anæsthetic to the middle ear, as suggested by Neumann, was also dependent on this anatomical condition.

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Mr G. J. JENKINS (President) said that an attempt should be made to distinguish whether it was a suppurative or a non-suppurative process, or whether a suppurative process was engrafted on to another condition.

Sir WILLIAM MILLIGAN (in reply) regretted that no speaker had dealt with the theory of nerve-infection which he advanced in the paper. He considered that in influenza epidemics much nerve damage was frequently caused ; there was a weak point in the sensory nerve tract ; it could be seen developmentally, it was evident histologically, and experimentally it could be proved by the introduction of capsules containing septic material in the neighbourhood of sensory nerves, when the degeneration was found to start at that particular point where the neurolemma was absent, as he (Sir William) had described, and where β-neuroblasts ceased. What he wished to suggest was that those cases of nerve deafness were really toxi-hæmorrhagic in origin, and that though the organism could not be found (it might be a non-filtrable virus) there was evidence that it could produce much toxæmia. If the commencing degeneration at the vulnerable spot was to be avoided, it was necessary to be more alert in recognising early internal ear disease. He wished particularly to urge the use of pilocarpin to absorb the rapid exudate into the cochlea.

Sir James Dundas-Grant's observation on the anatomical condition of the bone was probably correct. It was difficult to explain why there should be such a predilection for the apex, but such there certainly was. He (the speaker) had not diagnosed hæmorrhage into the semicircular canals as a separate entity. He did not think the temporal bone was developed in the way Mr Tweedie had indicated. He (the speaker) believed that there was a squamo-zygomatic and a petro-mastoid segment, and that between the two the middle-ear cleft ran up to what should be called the tympanic antrum.

The condition he (Sir William) had described was, he believed, a disease *per se*, due to a micro-organism which was difficult to find, but the manifestations of which were very evident. If otologists were on the *qui vive* for these cases and took active measures by early drainage, depletion, and repeated lumbar puncture, meningitis would often be avoided as well as much nerve deafness.

Gradenigo's Syndrome, by H. G. ESTCOURT, M.B., B.S. The paper appears in the *Journal of Laryngology and Otology*, vol. xli., No. 8, August 1926, p. 499.

DISCUSSION.

Mr SYDNEY SCOTT said that the cause of ocular paralysis in these cases had been considered at the National and other hospitals for nervous diseases. One point Dr Estcourt had not mentioned was the anatomical arrangement of the blood-vessels in relation to the sixth nerve in that part of the nerve between the pons and its entrance into the walls of the cavernous sinus ; nor had he referred to the fact that a branch of the cerebral arteries sometimes looped beneath the sixth nerve in that situation. With an excess of cerebro-spinal effusion into the basal cisterna the brain-stem was displaced, causing a kink in one or both sixth nerves. For a time,

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he (the speaker) had thought, with Dr Howell, that this was the explanation of Gradenigo's syndrome. Rapid recovery from paralysis in a few hours after lumbar puncture, the recurrence of the paresis with return of headache favoured this view. Few cases in comparison with the frequency of Gradenigo's syndrome proved fatal. In some, such as Mr Just's, there were apical petrous cells causing a localised leptomeningitis, but these belonged to the relatively uncommon fatal group. He (Mr Scott) considered that there were two quite different causes of the syndrome in each group.

Mr J. F. O'MALLEY said that he had had two cases of the kind, one in 1916, the other during the present year. The first was an acute mastoiditis on which he had operated, in the other there were no mastoid symptoms and he had not operated. The second patient was a child, aged 9, with two months' discharge from the right ear. There had been diplopia for five days; the discharge had yielded a pure growth of *Streptococcus hæmolyticus*. Three days later the temperature had suddenly risen to 104° F.; albumin and pus had been present in the urine; and the only organism found had been *Staphylococcus albus*. Later on, although the right rectus was regaining power, there had been another rapid rise of temperature to 103° F. Next day, however, the temperature fell again. Albumin had then been present in the urine. Two days later a specimen of urine obtained by catheter had contained neither cells nor organisms, and the ocular movements had become normal. He (Mr O'Malley) had not considered it justifiable to open the mastoid, as there had been no indication of mastoiditis. The syndrome might be due to a blood infection, and explicable on these lines.

Mr T. JUST said he also thought that if the patient was not seriously ill there was no reason to urge an operation. He (the speaker) had had such a case, the patient being a boy who had subacute unilateral mastoiditis which had necessitated a Schwartze operation. A week later sixth nerve paralysis had developed on the same side, and he (Mr Just) had considered the case to be one of Gradenigo's syndrome. He had examined the wound again to assure himself that he had not left any cells undrained. A week later contralateral facial paralysis had developed. He had found it very difficult to associate the two conditions. A neurologist had then seen the boy and had diagnosed encephalitis lethargica. The patient had recovered from the cranial nerve paralysis. Encephalitis should be remembered when there occurred paralysis of a cranial nerve or nerves for which it was difficult to account.

Mr G. J. JENKINS (President) said he thought that eventually two distinct conditions of intracranial trouble would be recognised: the first connected with the cerebro-spinal fluid; the second coming from the petrous bone. The latter may affect the fifth cranial nerve first.

Specimen from Case of Cerebellar Abscess shown at the Meeting held 5th December 1925 — T. H. JUST, F.R.C.S. — Specimen from a child, a girl, aged 7. Chronic suppurative otitis media left side for years; granulations in the tympanum, free discharge; child admitted to hospital because she was "seedy"; no cerebellar

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signs present; no nystagmus or inco-ordination. The child walked up to hospital.

While in hospital the pulse- and respiration-rates and the temperature were normal. Patient complained of slight intermittent headache. Lumbar puncture produced cerebro-spinal fluid clear and not under increased pressure. Patient died suddenly in bed, thirty-six hours after admission.

Left temporal bone shows three erosions exposing the dura mater; one into the middle fossa, and two into the posterior fossa, one behind and one in front of the lateral sinus. That in front of the sinus communicated with the abscess. At this point only was the dura mater adherent to the skull and to the brain.

I am indebted to Dr J. G. Greenfield for the preparation of the specimens and the pathological report.

Cerebellum.—The edge of the abscess is five-eighths of an inch (1.5 cm.) from the dentate nucleus. The tissue between shows practically no evidence of inflammation, only slight neuroglial hyperplasia, but there is some perivascular infiltration at a distance from the abscess, even internal to the dentate nucleus. This is altogether a *mononuclear* infiltration. In the actual wall of the abscess there is considerable formation of fibrous tissue, the meshes of which are filled with inflammatory cells, polymorphonuclears, lymphocytes and plasma cells, as well as many larger phagocytic and fat-containing cells.

The abscess is evidently of comparatively recent formation since (1) there is nowhere a dense fibrous tissue wall, and (2) although the pus-cells are degenerated they have nowhere lost their nuclear staining. At the same time the amount of new connective tissue and the neuroglial reaction suggest that it has been forming for more than a week.

It is an interesting case, as the patient died symptomless. Dr Gordon Holmes, in his work on cerebellar localisation, found that with a purely cortical wound of the cerebellum, except for an immediate giddiness which passed off in a few hours, there were no cerebellar signs later on. The abscess in this case is purely cortical, and on section there is seen to be five-eighths of an inch distance between the abscess and the dentate nucleus.

Mr G. J. JENKINS (President) said this case was of such great interest that it deserved full discussion, but the hour was late. Was it certain that this condition was not tuberculous? (Mr JUST: I have not tested it.) It would be well worth while to make the investigation. There was a tendency to speak rather loosely of raised pressure, and there should always be the statement whether the pressure was measured with a manometer.

Mr JUST (in reply) said that the house surgeon had measured the pressure of the cerebro-spinal fluid in this case, and had reported that there was no increase.

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