# SOCIETIES' PROCEEDINGS

## ROYAL SOCIETY OF MEDICINE—SECTION OF OTOLOGY

*December 6th, 1963*

### Discussion on Certain Aspects of the Anatomy, Radiology and Surgery of the Temporal Bone

DR. RUTH E. M. BOWDEN, M.B., B.S., D.Sc., of the Royal Free Hospital School of Medicine, said that several questions are raised by Dr. Proctor's elegant demonstration. It would be of interest to know if he found variation in the size and incidence, and time and mode of development of the middle ear compartments. His paper suggests that they arose by an active invasive process from the Eustachian tube, but is it not possible that condensation, rather than invasion, of the gelatinous tissue of the middle ear cleft is responsible for their development? Is there evidence of their continued post-natal development and of their relationship to the various groups of tympanic air cells? Furthermore, would Dr. Proctor support the view that there is marked regional variation in the structure of the healthy mucosa?

The scattered and focal distribution of early otosclerotic changes shown in Dr. Compere's beautiful films also poses several questions. Do the changes initially arise in periosteum, cartilage or bone and are these foci related to the sites of the centres of ossification? The latter have been shown to be associated with blood vessels and is there any evidence of changes in the nerves or their blood supply in cases with nerve deafness?

Is it possible that the blood supply of the nerves was implicated in cases of otosclerosis and some of Mr. Potter's cases of facial paralysis? (Some slides illustrating Professor M. J. Blunt's work on the blood supply of the facial nerve were shown.) Has Mr. Potter been able to investigate Nerve Conduction Times and Strength/Duration curves for muscles in those patients who suffered from facial palsy?

DR. F. G. M. ROSS, M.B., D.M.R.D., F.F.R., (Bristol) said that Dr. Compere's impending visit had stimulated him to perform tomography of the temporal bone on some patients suffering from otosclerosis. He had been able to show the thickening of the bone round the oval window and sclerosis round the semi-circular canals and cochlea. In one patient who had bilateral sensorineural loss, the turns of the cochlea were narrowed by sclerotic bone and in another patient with similar symptoms there was evidence of bone destruction or less dense bone extending out from the cochlea into the surrounding dense bone. Mr. Angell James had performed unilateral Schuknecht's operation on one of these patients and had inserted a metallic piston into one oval window of the other patient. The



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radiological appearances of the post-operative state were illustrated. Obliteration of the normal translucency of the oval window on the lateral aspect of the vestibule by the thickened footplate of the stapes was also demonstrated by tomography. The tomographic features of these patients seemed to support the findings that Dr. Compere had depicted so clearly on his slides. Dr. Ross drew attention to Ruedi's description (1963) of the histology of otosclerosis which suggested that the early lesion was in fact one which produced bone destruction. He asked Dr. Compere whether he had been able to demonstrate any bone destruction in the temporal bones in patients with otosclerosis.

REFERENCE

RUEDI, L. (1963) *Arch. Laryng., Chicago*, **78**, 469.

MR. I. B. THORBURN (Blackpool) said that Dr. Bruce Proctor had made an important contribution to the minute surgical anatomy of the tympano-mastoid cleft. In the course of stapes surgery Mr. Thorburn had often seen fine mucosal folds but had simply assumed that they were adhesions resulting from an old healed inflammation. Dr. Bruce Proctor had given to most of these mucosal folds and spaces new and precise anatomical names. It would make for greater clarity if proper names, e.g. for the spaces of Prussak and von Troltsch, were discarded and replaced by similar descriptive anatomical terms.

Clearly the minute clefts permitting ventilation between the epitympanum and mesotympanum could easily be closed off by an extension of the mucosal folds, possibly provoked by mild inflammation. This might have an important bearing on Eustachian tube function in the ætiology of attic disease. Certainly at operations for attic cholesteatoma one commonly saw, and took care to preserve, an intact layer of mucous membrane apparently resisting the spread of disease into the mesotympanum. Now one must consider whether this mucous membrane layer might be the cause and not the result of the attic disease.

MR. MILES FOXEN (London) complimented Dr. Bruce Proctor on the superb drawings and paintings which had been made of his specimens and asked if Dr. Proctor would describe briefly the method of fixation used in the preparation of the specimens.

**Discussion on Facial paralysis following head injury**

MR. J. D. K. DAWES, B.Sc., M.D., F.R.C.S., (Newcastle) said that although not an advocate of decompression in cases of idiopathic facial paralysis there is a definite place for decompression in cases of delayed facial paralysis following head injuries. After surveying 20 patients, 7 of whom had had a decompression, the impression gained was that those cases which had been decompressed made a better functional recovery. The longitudinal fracture lines which were not always demonstrated by routine radiology of the mastoid either crossed the facial nerve in its descending portion or close to the geniculate ganglion.

The facial paralysis developed between 1 hour and 15 days after injury. When the onset was delayed for 6 days or more recovery was good with or without operation. If the onset of the paralysis occurred within the first three days the outlook was unfavourable.

Compression of the facial nerve in the cases of early onset resulted from bone



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fragments and bleeding into the facial canal and communicating cells. Paralysis of later onset followed œdema (traumatic or inflammatory in origin) in the neighbourhood of the fracture line. As prolonged or rapid compression of the nerve may lead to permanent damage, surgical decompression of the affected region would be expected to help recovery.

After onset of the paralysis, the rate of progression is variable and those cases progressing rapidly will need an early surgical exploration, possibly an emergency operation. Frequent clinical observation will determine the need and the timing of any operation. Electrical tests contribute nothing to the making of this decision.

REFERENCE

ROBSON, F. C. and DAWES, J. D. K. (1960) *J. Laryng.*, **74**, 275-89.

**Discussion on the Development of the Middle Ear Spaces and their Surgical significance**

MR. S. W. ALLINSON, F.R.C.S., D.L.O., (Boston) said that he would hesitate before removing the body of a normal incus which was part of a normal intact ossicular chain so as to improve the drainage from the mastoid.

Heath's type of cortical mastoidectomy is so satisfactory for draining the mastoid and the ossicular chain is not disturbed.

**Discussion on Radiological appearance of the Temporal Bone in Otosclerosis**

MR. J. ANGELL JAMES, M.D., F.R.C.S., (Bristol) said that he congratulated Dr. Ross on the remarkable skiagrams he had shown and his clear interpretation of the details which were visible in them. With the help of such excellent pictures as these it was possible to visualize otosclerotic and other changes in the inner ear and internal auditory canal and even sometimes in the windows. This information was important in the diagnosis of cases of otosclerotic perceptive deafness without other clear signs of otosclerosis. However, the interpretation still remained extremely difficult even after prolonged and detailed study of the type of films Dr. Ross had shown and those obtainable by polytomography. In the future, otologists would certainly be relying more than before on these examinations.

PROFESSOR RUTH BOWDEN

*Question:*

Do mucosal folds appear regularly?

*Answer:*

DR. BRUCE PROCTOR in replying told Prof. Bowden that mucosal folds can be found in most middle ears if certain precautions are taken. The dissection should be undertaken as soon after autopsy as possible. The specimen must be preserved in deep freeze immediately after its removal. Fixatives should not be used. During the dissection the middle ear must be kept moist and undue heat from lamps avoided. With these precautions these various folds can all be found in nearly all specimens. They vary somewhat as to whether or not they are complete.

Some of the important and frequent variations that occur have been described in the text. In a complex structure such as the middle ear with a complex



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embryology one encounters frequent variabilities in the position of these folds. To consider this problem further, in a clinically oriented text, would complicate the picture enormously.

The embryological concept presented herein is based on the extensive wax reconstructions undertaken by the embryologist, Hammar of Upsala. Fraser of London opposed Hammar's concept of the manner in which the entire middle-ear cleft developed. Speaking as an amateur anatomist it appeared to Dr. Proctor that Fraser's contention is that the middle ear develops in a passive sort of way by condensation and absorption of the gelatinous fluid of the middle-ear cleft. Hammar's explanation appears more active and dynamic with emphasis on active progression of an endothelial pouch up the eustachian tube and into the middle ear. The latter concept seems to fit in better with what we see in the adult ear.

The middle-ear development is most rapid during the 6th and 7th fœtal months. This is precisely the time when the otic capsule approaches its adult size.

Terms such as Prussak's space and the anterior and posterior pouches of Von Tröltsch have been used for many years. There has not been any confusion in regard to what they designate. We must first standardize the nomenclature of the middle-ear structures to avoid confusion. Later perhaps descriptive substitutes may be worked out for proper names attached to structures.

Mr. Thorburn states that he has been somewhat puzzled at times by the folds as seen in attic cholesteatoma and particularly so in natural tympanoplasties.

One must first accept some concept of how the middle ear is compartmentized. The text presented this morning is an attempt to define the structures encountered in the middle ear. The concept will undoubtedly be modified by other workers from time to time.

Once a system of orientation is adopted it then becomes much easier to seek and identify the anatomical structures which have been altered by the disease process.

The complimentary remarks of Mr. Miles Foxen concerning the quality of American art in medicine will be transmitted to Mr. Grant Lashbrook who is responsible for the art work. As soon as a specimen was dissected in the presence of the artist and the drawings sketched and photographed the specimen was gently sprayed with a plastic material for preservation. These specimens were not sectioned.

*Remarks:*

Concerning partial removal of the incus I regard this as a very dangerous procedure.

*Answer:*

In reply to Mr. Allison he agreed that partial removal of the incus is dangerous if pains are not taken to be gentle. The long crus is cut off with a small bone clipper taking care that any force is forward in the direction of the long axis of the stapes. The stapedial tendon will help prevent undue displacement of the stapes.

JOHN M. POTTER, M.B., F.R.C.S., (Oxford) in reply to discussion, said, that "I



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agree with Professor Bowden that disturbance of the blood supply to the facial nerve is likely to be an important factor in these delayed palsies, but the actual mechanism is uncertain. Ischæmic compression by bleeding or by swelling of the nerve itself in its bony canal is, I think, the usual conception, and one which is supported by direct observation at operation. Thrombosis is hard to verify; but another possible factor is arterial spasm such as one sees in the cerebral arteries and others in response to trauma and extravasated blood. Moreover, a locally-acting vasoconstrictor substance, such as serotonin, might aggravate any tendency to ischæmia.

"I did not have time to say anything about electrical reactions, but I do not feel that they are sufficiently helpful in the delayed palsies. By the time they show degeneration, it is, I suspect, too late to remedy this; and when they do show degeneration, they cannot tell us to what extent acceptable facial movement will recover.

"Mr. Dawes feels that his surgically decompressed facial nerves recover better than those left alone, but I should need to be convinced that a really satisfactorily controlled trial had been done before accepting this, and, as I said, I think such a trial would be difficult to carry out. Of Turner's 34 cases of delayed facial palsy good spontaneous recovery occurred in all except two."



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