Pooley (T. R.)

CONTRIBUTION TO THE PATHOLOGY

OF THE

TEMPORAL BONE.

By THOMAS R. POOLEY, M. D., of New York.

[Re-printed from the TRANSACTIONS OF THE AMERICAN OTOLOGICAL SOCIETY, July, 1879.]

NEW BEDFORD:
PRINTED BY THE MERCURY PUBLISHING COMPANY.
1879.

CONTRIBUTION TO THE PATHOLOGY OF THE TEMPORAL BONE.

By Thomas R. Pooley, M. D., *of New York.*

Case I. *Fracture of the Petrous portion of the Temporal Bone.*

The history of the case from which the specimen to be described was obtained is furnished, as follows, by my brother, Dr. J. H. Pooley, of Columbus, Ohio: "On the evening of Oct. 29, 1878, I was called in consultation to see Mr. Lindsey, aged about 30, a teacher in the Blind Asylum, himself partially blind, who a few hours before had been thrown from a wagon upon a hard asphalt pavement. He was able to get up and walk a short distance, after the accident, only complaining of feeling slightly shocked and confused. He was conveyed to the asylum, and soon afterwards became soporose and partially unconscious. This condition gradually increased until coma became complete. When I saw him, he was lying on his back in a condition of perfect unconsciousness and collapse. His pulse was slow, 40 in the minute and labored: breathing slow and irregular, not stertorous, but at intervals, and for a minute or two at a time puffing, almost whistling; pupils neither contracted nor dilated, but insensible to light. All attempts to rouse him to the least exhibition of consciousness were in vain. There was complete relaxation of the whole body, so that the presence or absence of paralysis on either side could not be determined. He had had before I saw him some convulsive movements on the right side.

From his right ear there was a free and continuous flow of bloody serum. After gently syringing out the ear with warm water, I was able with the speculum and mirror to

observe a rupture of the anterior inferior portion of the membrana tympani. I made the diagnosis of fracture of the base of the skull, with extensive hemorrhage. It was proposed to administer croton oil, but to this I objected that it was incapable of doing any good, and any perturbation would be likely to increase the hemorrhage. As he was capable of swallowing, though slowly and imperfectly, very small doses of brandy and water were directed. A catheter was introduced into the bladder, but very little urine was found. He died before morning."

The post-mortem was made 24 hours after death by Dr. O. Frankenberg, Prof. of Pathological Anatomy in Starling Medical College, assisted by my brother and myself. There was slight extravasation of blood under the scalp; immediately under the right temporal bone there was a fresh clot as large as the spread hand of considerable thickness, and producing a marked depression of the cerebral substance. Upon removing the brain, a fissure fracture was found extending from the vertex directly through the temporal bone and across the base of the skull as far as the foramen magnum. This fracture was joined just above the mastoid process by another and shorter one only two inches in length, running upwards and backwards. The fractured portions were very slightly movable upon one another.

The temporal bone, which was carefully removed by Lucae's method, was given to me for dissection. It was put in Müller's fluid, and examined by Dr. Spitzka and myself several months later. The fracture is a fissure. A single fissure starts about $\frac{1}{2}''$ behind the groove for the middle meningeal artery, runs towards the latter, and there divides into two. The posterior one of these fissures runs through the pyramid of the temporal bone on its anterior superior face and nearly parallel with the petrosal crest. This fissure is a serrated one. The other fissure is but a continuation of the main one. A sector comprising the posterior inferior portion of the membrana tympani remains attached to the

posterior fragment. The other and greater portion with the intact malleus to the anterior fragment, in which the greater part of the Eustachian tube is contained. The chorda tympani nerve has been torn through, a portion about $\frac{1}{8}''$ long protrudes from the posterior fragment. The articulation of the malleus with the incus is cleanly separated, otherwise the chain of ossicles are in their normal position and connections. Stapes is freely but excessively movable. The tear runs along the axis of the auditory canal, in its deeper parts, both on its upper and lower walls. In the anterior aspect of the deeper part of the canal, the soft tissues are separated from the bony walls. The lower scala of the cochlea was laid open and found to contain no blood. The superior semicircular canal being opened, a clear fluid issued therefrom, but it contained no blood. Furthermore, the other semicircular canals were opened into by a section transverse to the axis of the bone, and all found to be filled with a perilymph, but contained no blood. Bristles were passed from the canals into the vestibule.

The special interest which attaches to the specimen, it seems to me, is the immunity from injury of the structures of the inner ear, which, as is clearly shown, were not involved in the line of fracture. Of course the injuries which are found upon examination of the fractures of this bone are naturally very variable. In this instance the damage, so far as the structures of the ear were concerned, were mainly expended upon the middle ear. Especially interesting was the complete disarticulation of the malleo-incudal joint, and the injury done to the chorda tympani. As we all know, the usual result of fracture of the petrous portion is death, either immediately from hemorrhage or compression, or later from inflammation of the brain or its meninges. That recovery may occur we also know; although union of the fracture seldom occurs by osseous consolidation, but more frequently by fibrous union.

If the case should not terminate fatally, total deafness is

the rule, and this has usually been in such instances attributed to the destruction of the inner ear. It is questionable whether in such a case as the one now described (if recovery had ensued) there would be total deafness. Of course the amount of violence done to the auditory canal and tympanic cavity would have resulted in a more or less protracted purulent inflammation of the middle ear, which would have seriously compromised hearing; but with the nervous apparatus still intact, we cannot conceive that the deafness would have been absolute.

I regret that the opposite side was not examined, to see whether the fissure extended through to the other temporal bone. For such a case is recorded by Voltolini, (M. f. O., 1869, s. 110,) to which Schwartze refers in his book.

Case II. *Chronic Otitis Media. Necrosis of Inner Ear, Abscess of the Cerebellum.*

September 22, '78, I was consulted by Miss L., 23 years of age, on account of an offensive otorrhœa. She had suffered from earache and otorrhœa since childhood. The discharge would cease for a time, then after severe pain recur. The last attack of this kind, which she says began about four weeks ago, was accompanied by distinct cerebral symptoms, nausea, vomiting and vertigo. S. P. Face pale and very much emaciated, with a chronic cough, and physical evidences of phthisis pulmonalis. Her mother died of phthisis. She complained of constant pain in the ear and vertex. There was a slight but most offensive otorrhœa, which suggested by its smell the presence of diseased bone. She complained of a very distressing vertigo which was always aggravated when assuming a recumbent posture. There was facial paralysis on the same side as the diseased ear. Examination of the ear after cleansing, showed a large linear-shaped perforation of the anterior inferior quadrant of the membrana tympani. The mucous membrane of the promo-

tory, which was fully exposed, pale, and there were several spots of hemorrhage upon it, as well as upon the remnant of membrane. There was no evidence of any inflammatory action in the appearance of the middle ear, nor was the mastoid region either painful or swollen. h=o. v. $\frac{5}{60}$.

I ventured the diagnosis of extension of the ear disease to the brain, abscess of the cerebellum, and made an unfavorable prognosis. The propriety of trephining the mastoid was considered, but the operation rejected as not likely to do any good, as the extension was evidently not by this way. All the symptoms became rapidly worse. The pain was intense, the vertigo such that the patient thought the bed was going round, and although she was excessively weak, she assumed an upright posture most of the time for the relief to this symptom thus afforded. She retained her consciousness until only a few hours before her death. Vomiting was throughout a very distressing symptom. Death took place on the 29th inst., just one week after I first saw her. Treatment was merely palliative. Steaming the ear gave her more relief than anything else.

The post-mortem was made 24 hours after death, by Dr. Spitzka and myself, in the presence of Drs. Knapp, Chamberlain, Wright and Partridge.

Upon removing the calvarium, an abscess about the size of an English walnut, with a well formed pyogenic membrane, was found in the left hemisphere of the cerebellum. When opened, it gave issue to the most offensive pus. No abnormality of any other parts of the brain was found. The abscess itself was distinctly circumscribed, and there was no softening of the brain substance in its vicinity.

The temporal bone was removed for dissection. No evidence of necrosis of the inner surface of the bone could be found, but the part which lay in apposition with the abscess was blackened. The temporal bone, which was kept in Müller's fluid, was examined by myself and Dr. Spitzka, several months afterwards.

No evidence of necrosis of the surface of the bone; that portion corresponding to the eminences of the cochlea and superior semicircular canals, perfectly normal in consistency and appearance. Perforation of the membrana tympani in its anterior inferior quadrant. The portion of membrane still existing seemed to be only newly formed connective tissue (probably healed perforation). The auditory canal was opened by sawing in an anterior posterior direction, just in front of the attachment of the drum-membrane, when a small purulent cavity, situated above and behind the tympanum, and communicating with the latter immediately behind the attachment of the membrana tympani, was laid open. No ossicles were present. The mucous membrane was thickened and covered the promontory. The latter was smooth and healthy. At the posterior portion of the necrotic bone and purulent mass lies the facial nerve, which is softened and diminished in volume, while above this point it is quite healthy. The tendon of the tensor tympani was attached to the thickened part of the mucous membrane in its upper portion. The cochlea was opened into by a strong knife. The modiolus was entirely destroyed except at its base; the septum between the three coils was preserved. The soft parts of the vestibule were entirely wanting. The horizontal semicircular canal was absent; the anterior vertical one contained pus, but was still lined by membrane. The superior vertical contained pus, but no lining membrane.

The walls of the vestibule and the bony canals were smooth and surrounded by ivory healthy-like bone. Both the saccule, utricule and superior and horizontal canals absent; membranous part of the anterior canal present, but bathed with pus, which extended into the vestibule. A bristle passed into the aquæduct of the vestibule failed to reach the surface of the bone. The internal auditory meatus was examined; both nerves at this point healthy, all the other parts of the canal being healthy.

No trace of a necrotic path communicating with the cavity of the skull could be found. The abscess of the cerebellum was probably not of recent origin, since it had such a well marked thick pyogenic sac. Is it not probable that each of the oft recurring attacks of pain, to which the patient referred, were due to cerebral irritation which lasted for a few days and then passed away, but finally by repeated attacks gave rise to the formation of an abscess?

Another point of interest is the simultaneous existence of labyrinthine and cerebellar disease. I made the diagnosis of the seat of the intracranial disease from the vertigo and unsteadiness of gait. May this, however, not just as well have been caused by the disease of the semi-circular canals? I am sorry that I did not notice more particularly whether the staggering in walking was towards the affected side, as this might have enabled me to have included the probability of the inner ear being disorganized in my diagnosis. But I only saw the patient once before she took to her bed, and then forgot to make the inquiry.