III.

THE ETIOLOGY AND PATHOLOGY OF OTITIC CEREBELLAR ABSCESS.

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Although recent otologic literature contains many case reports of cerebellar abscess, there are but few carefully worked out pathologic examinations of this interesting condition.

Cerebellar abscess may complicate either an acute or a chronic suppuration of the middle ear and mastoid. It is, however, far more frequently associated with the chronic otitides. In the eighty-six cases which we have been able to collect from the literature since 1906 (the date of publication of Bárány's first important work on vestibular nystagmus), fourteen (16.2 per cent) have complicated the acute otitides, while seventy-two (83.8 per cent) have followed the chronic suppurations.

The reason for this undoubtedly lies in the fact that in the chronic cases there is greater destruction of bone, prolonged interference with drainage, and the presence of cholesteatoma. Thus in the vast majority of instances we find either polypi, cholesteatoma or small tympanic perforations, chiefly in Shrapnell's membrane. Furthermore, in chronic suppurative cases there usually occurs a sclerosis of the cortex which favors necrosis of the deeper lying bony structures. It is particularly in those instances in which cholesteatoma has eroded the bone and lies in direct contact with the dura that intracranial complications occur. The cholesteatoma not only seriously interferes with drainage, but also acts as an excellent medium for the growth of the invading microorganisms.

Males are attacked much more frequently than females, the proportion, in our series, being as three to one.

Neumann gives the following statistics on the relative fre-

quency of occurrence of cerebellar abscess at different periods of life:

Author.	0-10 Years.	11-20 Years.	21-30 Years.	Over 30 Years.
Koch	11	33	35	19
Okada	17	56	45	28
Körner	4	14	12	10
Heimann	16	57	38	26
Neumann	12	51	41	30

In the series here reported it occurs with greatest frequency in the second decennium, and with least frequency in the first and sixth decennium, while in the third it occurs about as frequently as it does in the fourth and fifth combined. Thus in the eighty-six cases, six were under ten years; twenty-nine were between ten and twenty years; twenty-two were between twenty and thirty years; twelve were between thirty and forty years; ten were between forty and fifty years, and five between fifty and sixty years. In two the age was not stated. It occurred forty-five times on the right side and thirty-seven times on the left. In four the side was not stated.

With regard to the etiology of cerebellar abscess Neumann differentiates between those which complicate the acute middle ear infections and those occurring with the chronic otitides. In the fourteen cases of cerebellar abscess complicating acute otitis media and mastoiditis, sinus thrombosis occurs in six. In only one, however, is there a direct connection evident macroscopically between the infected sinus and the cerebellar abscess. This proportion is probably too low, and in other statistics such evidence is more frequently met with. Although in the majority of instances the cerebellar infection is secondary to the sinus thrombosis, this does not seem to be invariably so.

Death from the rupture of a cerebellar abscess into the lateral sinus has been reported, and the fact that symptoms of sinus thrombosis occur late in some of the cases, notably those of Barr, Beck and others, brings to mind the possibility of the sinus infection being secondary to the brain abscess. Histologic proof of this etiologic relationship is not at hand. In four of the acute cases an epidural abscess in the posterior fossa was probably the pathway of the endocranial complication. So that in our series, too, the route which the cerebellar infection followed in the majority of cases complicating acute

otitis was either from a thrombosed sinus or an epidural abscess. In but one of the acute cases (Uffenorde) was the labyrinth the pathway of the intracranial infection. This was an acute middle ear infection complicating scarlet fever, and the cerebellar abscess resulted from an extension of a saccus empyema. Here, despite the virulence of the infection and the lack of resistance on the part of the individual attacked, the intracranial extension from the suppurative labyrinthitis was a circumscribed collection of pus—i. e., a brain abscess—and not a diffuse purulent meningitis.

From an etiologic point of view the cerebellar abscesses which complicate the chronic middle ear suppurations differ very decidedly from those which occur with the acute otitides. In Neumann's series there are one hundred and thirty-two cases of cerebellar abscess complicating chronic suppurative otitis media. Twenty are not sufficiently studied. Of the remaining one hundred and twelve, forty-nine—that is, 43.75 per cent—showed labyrinthine suppuration. In the series here reported, thirty-one of the seventy-two cases with chronic purulent otitis showed labyrinthine suppuration. These figures, startling as they are, are probably an underestimate rather than an overestimate. We base this judgment upon the following facts: If we examine individual reports included in our series, we find, first, eight cases reported by Michaelsen. Of these, six are cerebellar abscesses complicating chronic suppurative otitis media. In these six cases "dead labyrinth" was found five times. Again, Ruttin reports seven cases, all of them complicating chronic suppurative otitis. In six of the seven, labyrinthine suppuration occurred. Neumann reports five cases, all of them complicating chronic purulent otitis media. In these, labyrinthine suppuration occurred three times. These figures, we believe, are the result of careful clinical examination, and are not accidental. In many of the cases the labyrinthine suppuration was proven histologically.

Undoubtedly there is a small proportion of cases of cerebellar abscess in which, although the labyrinth is the seat of a suppurative process, it is not the pathway of the infection from the middle ear to the endocranial structures. In a case which the writer recently had an opportunity to examine postmortem, and in which the temporal bone was examined histologically, the following facts presented: Not a vestige of labyrinthine

end organs, auditory or static, remained. There was a fistula in the external semicircular canal filled with granulations and new formed connective tissue. From this focus the labyrinth probably became infected. The crura of the stapes were destroyed, but the footplate, although much eroded, was still present in the oval window. With the exception of this fistula in the external semicircular canal, the bony canals were everywhere intact. There was, furthermore, no erosion of the inner labyrinthine wall. The internal auditory canal contained no pus. The pathway of the intracranial infection could be traced through the inner (posterior) antral wall. In this situation there was an epidural abscess, and a perforation led through the bony wall and through the dura to the cerebellar abscess.

In our series suppurative labyrinthitis and epidural abscess occurred seven times. Thus suppurative labyrinthitis, epidural abscess and cerebellar abscess form a fairly common triad. The etiologic relationship between these three factors is not a constant one. While it is natural to suppose that the epidural abscess is secondary to the suppurative labyrinthitis, this is not invariably so. In his consideration of the relationship between deep epidural abscess in the posterior fossa and suppurative labyrinthitis, Grünberg reports a most carefully studied case. The labyrinth is the seat of a diffuse suppurative process. The windows, both the oval and round, are intact. The labyrinthine capsule is everywhere intact, with the exception of the common limb—i. e., the junction of the superior with the posterior semicircular canal. At this point there is an erosion from without inward, unquestionably caused by the epidural abscess in this location.

Ruttin has shown that in mastoiditis, particularly in those latent varieties due to infection by the streptococcus mucosus, there is a tendency to necrosis in the deep seated petrosal cells surrounding the labyrinthine capsule. Drainage from this region is particularly difficult, and an epidural abscess may result. It can readily be understood, then, how an epidural abscess in this location may give rise to a cerebellar abscess, on the one hand, and to a diffuse labyrinthine suppuration on the other.

Formerly, in many of these cases of suppurative labyrinthitis, epidural abscess and cerebellar abscess, a saccus empyema

was adjudged, upon the macroscopic findings, the pathway of the cerebellar infection. Macroscopic findings, however, are inadequate to establish such a judgment. The microscope alone is the final arbiter.

In eight cases suppurative labyrinthitis occurred with sinus thrombosis. In only one of these, however, is the labyrinth clearly indicated as the direct pathway of the sinus infection. In this instance, in addition to the sigmoid sinus, the bulb, inferior petrosal and cavernous were thrombosed. Here the infection, in all probability, started in the labyrinthine veins, extended to the inferior petrosal sinus, and thence forward to the cavernous and backward to the bulb and sigmoid.

Etiologically, pathologically and clinically, cerebellar abscess may be divided into: (a) Labyrinthogenic, (b) nonlabyrinthogenic.

In all probability at least fifty per cent of cerebellar abscesses complicating chronic suppurative otitis media are labyrinthogenic. In a small proportion of cases the labyrinth is the seat of diffuse suppuration and is destroyed, but is not the pathway of infection to the brain. On the other hand, of those cases of cerebellar abscess which complicate the acute otitides, from ninety to ninety-five per cent are nonlabyrinthogenic—i. e., they take origin either in a sinus thrombosis or an epidural abscess of the posterior fossa.

The pathway from the labyrinth to the cerebellum may follow the internal auditory canal, the aqueductus vestibuli, the aqueductus cochleæ, or a fistula in the bony capsule. In a general way, middle ear infections which progress to the intracranial structures through natural openings, lead to a diffuse suppurative process—i. e., meningitis—whereas, those that do not follow preformed paths, cause circumscribed collections of pus—i. e., either brain or subdural abscesses. The aqueductus vestibuli offers a notable exception to this rule. An infection traveling along this route is retarded probably because of the narrow lumen of the aqueduct, the facility with which protective adhesions are formed, and the fact that the ductus ends in a closed sac between the layers of a very resistant membrane—viz., the dura. Such infections, then, frequently lead to cerebellar abscesses, rarely through a saccus empyema, more often through an erosion of the bony wall of the aqueduct and the formation of an epidural abscess.

Whether the labyrinth infection leads to a diffuse meningitis or to a cerebellar abscess depends upon the virulence of the infection, the resistance of the individual attacked, and the pathway of the infection. In this regard the progress of the middle ear infection into the labyrinth is of considerable importance. Slow, erosive processes, destroying the labyrinthine capsule, are much more apt to arouse circumscribed intracranial complications than are labyrinthitides in which infection takes place suddenly through the windows. A notable example is the case reported by Thompson. In this instance cholesteatomata had eroded both labyrinths, and the latter were the seat of diffuse suppuration. Each lateral cerebellar lobe contained an abscess. The pathway of the intracranial infection was along the nerves in the internal auditory canal. That the pathway of the labyrinthine infection may be of importance in determining the character of the intracranial complication, even where the latter occurs with the acute otitides, the case reported by Uffenorde illustrates. Here the labyrinth became infected by an erosion through the promontory, and not by the windows.

Among unusual pathologic conditions associated with otitic cerebellar abscess, two cases in the series here reported are worthy of note. The one is an acoustic tumor reported by Dixon. The other is an abscess complicated by carcinoma-tosis of the temporal bone, reported by Hibler.

One of the most interesting problems connected with the pathology of brain abscess in general, but particularly with cerebellar abscess, is that concerning the process by which the brain substance itself becomes infected. All who have observed a number of cases of brain abscess have been impressed with the fact that in most instances the collection of pus is more or less remote from the cortex—i. e., that the majority of brain abscesses are situated in the white substance. To those who have performed postmortem examinations on a number of these cases, it has become evident that between the brain abscess and the source of the intracranial infection there is frequently a layer of brain substance which is macroscopically normal. In other words, there is a growing impression that the majority of brain abscesses do not occur through direct extension by continuity.

Even where there is a postpetrous epidural abscess, and

where the brain surface is grown fast to the dura, there is often interposed between the epidural abscess and the brain abscess a layer of normal brain tissue. Thus, Isemer, in his case report states: "It is remarkable that, although the brain surface is grown fast to the dura in the region of the epidural abscess, yet between the abscess in the white substance and the area of dura there is a distinct, if thin, layer of macroscopically normal brain." From this one might conclude that the brain abscess was not the result of an extension by continuity from the diseased labyrinth, but that the infection extended by thrombosis of small cerebellar veins which empty into the pial veins, or through the lymph sheaths around these veins.

With regard to this question, Michaelsen (quoting Uffenorde) states: "The origin of the brain infection is always the meninges—i. e., the dura and the piaarachnoid. At first the pial veins become involved and thrombosed. This thrombosis which, in the beginning, is not always septic, follows the venous channels backwards, leaving the cortex intact, until it reaches the ultimate ramifications of the veins in the white substance, where the stasis produces red softening. The infection then follows and an abscess results. The cortex is not involved because of its separate vascular system."

Even though the "origin of the brain infection is always the meninges," yet the appearance of the dura and its behavior during the postoperative treatment, often give absolutely no indication as to the presence of a brain abscess. Collections of pus within the brain, with a macroscopically normal dura, are not so rare. In a case of right temporosphenoidal abscess recently observed by the writer, the dura was disclosed at operation covered with cholesteatoma and large pussy granulations. During the postoperative dressings the dura returned to normal—i. e., it became coated with small red healthy granulations, and healing was uneventful. Two months after the radical mastoid operation the brain abscess ruptured into the lateral ventricle, and death ensued from diffuse purulent meningitis.

The observations of Heilbronn, although too isolated to be conclusive in themselves, throw a flood of light upon this question. He states: "There are few observations of brain lesions which have occurred through venous channels (Rück-

läufigem Wege). Such affections we would expect especially in disease of the great sinus, the chief return current of the brain's venous blood. With the most frequent form of sinus disease—i. e., thrombosis and thrombophlebitis—there have been few observations of brain disease."

Oppenheim in his "Lehrbuch der Nervenkrankheiten" states that he believes there are many opportunities in cases of sinus thrombosis to observe focal brain symptoms. Hemorrhage and softening are often the direct result of sinus thrombosis, and, although such observations are rare, they probably occur far more frequently than they are observed.

Macewen reported convulsions in children which were confined to one side of the body, and which he attributed to thrombosis of the longitudinal sinus and the veins of the motor area.

Heilbronn reports several interesting cases with general and focal brain symptoms complicating otitic sinus thrombosis. In the first case, a patient with acute suppurative otitis media and mastoiditis, symptoms of meningitis followed six days after a simple mastoid operation had been performed. With a gradual rise of temperature the patient became delirious and finally comatose. Postmortem examination showed no meningitis. There was a thrombosis of the left sigmoid sinus. There were hemorrhages in both optic thalami, thrombosis of the venæ Galeni and of other basal veins. The symptoms were due to the disturbance in the intracerebral circulation. He draws the following conclusions: 1. In sinus thrombosis, by the plugging of the small veins, lesions of the brain in the form of hemorrhages and softening may occur. 2. These lesions, like many other brain lesions, may cause general brain symptoms without any definite focal symptoms.

The second case was that of a young man in whom a sinus thrombosis complicated a chronic suppurative otitis media. He suddenly became unconscious. This condition, however, lasted but a few hours, and was followed by an aphasia. Postmortem examination showed extensive sinus thrombosis. The large pial vein which surrounds the left temporosphenoidal lobe, and has its origin in this lobe, was thrombosed. The aphasia was due to the occlusion of this vein, and stasis of the blood with the consequent nutritional disturbance.

The third case, probably the most significant, was that of a young woman in whom a sinus thrombosis complicated a

chronic suppurating ear. In this instance there were signs pointing to a bulbar lesion. Anatomically there was no gross obstruction of the veins leading from this region, but in the medulla there were changes which doubtless indicated a destruction of function of this part. There was destruction of fibers, a paucity of nuclei, while the remaining nuclei stained poorly and were deformed. The picture was one of anemic necrosis. The lesions were significantly distributed about the vessels. To the center of each focus ran a small vein.

These observations, while they are, as stated, too isolated to be conclusive, yet offer strong confirmation of Uffenorde's theory. Whether this theory, regarding the manner in which the brain tissue itself is infected, holds good only for those cerebellar abscesses which take their origin in a thrombosed sinus, or whether it can be applied to all cases, irrespective of their origin, remains to be proven. Only careful histologic research will clear this point.

In the eighty-six cases which we have collected the infecting agent is stated but eighteen times. The streptococcus was found eight times. The staphylococcus was found once. Among unusual bacteria may be mentioned the colon bacillus and Vincent's spirillum and bacillus. In their study of the bacterial agents in ten cases of intracranial complications of otitic disease, Turner and Reynolds found that streptococci were the virulent organisms in seven. In not a single instance of Michaelsen's series was the infecting agent in the cerebellar abscess found in pure culture. He found different bacteria at various times in the same brain abscess. Moreover, he found that similar changes occurred in the spinal fluid. Such results are unusual.

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