PROGRESS IN OTOLARYNGOLOGY

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

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INTRADURAL COMPLICATIONS OF AURAL AND NASAL ORIGIN

A Survey of Recent Literature

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THE RÔLE OF EAR AND NOSE IN INTRACRANIAL LESIONS IN GENERAL

Trauma.—A suppurating ear following a fracture through the petrous pyramid is the cause of death in about 8 per cent of cases of intracranial lesions, while a depressed fracture through one or other of the accessory sinuses gives rise to septic meningitis much more frequently—possibly months later—although a linear fracture through the anterior fossa of the base is usually exempt from meningitis.

Surgical treatment of meningitis from a complicating suppurative otitis or sinusitis gives a better prognosis in fracture cases than in primary otitis or sinusitis, because a protective zone of adhesions is built up in the pia-arachnoid prior to the advent of the suppurative process. Consequently, drainage of the infected area (if performed early) results in a cure in a considerable proportion of cases.

Rand and Nielsen [1] report seven cases of meningitis in 171 skull fractures. The portal of entry was through the ear in four instances, and through the upper nasal passages by way of the cribriform plate or ethmoid cells in two others.

One patient (mucosus capsulatus of Friedländer) recovered after intravenous injections of mercurochrome-220 soluble and repeated lumbar punctures.

These authors emphasize that a bleeding ear must never be irrigated, but the canal plugged with sterile cotton.

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* Reviewer's Note: In this survey I have attempted to apply the philosophy of intradural suppuration to the unusual and unexplained cases of recent literature whenever it is applicable; leaving the real advances to stand for themselves because of my lack of personal verification or experience, and often adding, I regret, a too scanty word of praise.

1. Rand, Carl W., and Nielsen, Johannes M.: Fracture of the Skull: Analysis of One Hundred and Seventy-One Proved Cases; Diagnosis and Treatment of Associated Brain Injury, Arch. Surg. **11**:434-458 (Sept.) 1925.

The Nasal Cavity and Accessory Sinuses as the Route of Nonsuppurative Infections in the Brain.—Bronzini,[2] under the title "Vascular Relations Between Subarachnoid Spaces and the Healthy or Inflamed Nasal Mucosa," points out that inflammatory conditions of the nasal mucous membrane prevent the drainage of the cerebrospinal fluid through the lymphatics of the mucosa.

According to Yates and Barnes,[3] the infection in lethargic encephalitis is possibly a symbiotic process. At first the nasal mucous membrane is damaged by one of the catarrhal group of organisms; the subsequently inhaled virus of encephalitis passes through the damaged mucous membrane, and is absorbed into the brain by way of the perineural lymphatics.

Disease of the Nose and Ear in the Etiology of Intradural Suppuration.—(*a*) Congenital Defects: That *congenital defects* in the sphenoidal sinus may be the explanation of the sudden appearance of meningitis after a coryza without suppurative sinusitis, is anatomically established by the work of Gilse,[4] who also found two cases in the literature (Meyers [5]) in which a prolapse of the dura mater into the sphenoidal sinus is recorded. All these defects occur at one developmental site.

(*b*) Abscess of Hypophysis: Cases of acute suppurative hypophysitis complicating purulent sphenoidal sinusitis have been reported by Harvey, Meyer, and Boggs and Winternitz. Glinski, Smoler and Thom and Plaut have reported abscesses in the anterior lobe of the hypophysis as an accompaniment of purulent leptomeningitis. Simonds [6] reviews them and adds two of his own, both following otitis.

CASE 1.—Bilateral suppurative otitis media and mastoiditis in a boy were complicated by purulent leptomeningitis. Postmortem examination revealed an abscess about one-fourth the size of the entire lobe, beneath the capsule on the front of one lateral half of the anterior lobe of the hypophysis.

CASE 2.—Acute suppurative otitis media was complicated by purulent leptomeningitis. Necropsy showed that almost one entire lateral half of the anterior lobe of the hypophysis had been destroyed by an abscess.

Simonds says that hypophysial abscesses, complicating purulent leptomeningitis, are not infrequent, and are interesting because of the mechanism by which the infection reaches the gland. They are most commonly situated immediately beneath the capsule in the anterolateral

2. Bronzini, A.: Arch. ital di otol. **36**:824, 1925.

3. Yates, A. L., and Barnes, S.: Nasal Sinuses as Route of Infection in Lethargic Encephalitis, Lancet **2**:130, 1925.

4. Gilse, P. H. G. van: Investigation on the Development of the Sphenoidal Sinus, J. Laryng. & Otol. **41**:137-144 (March) 1926.

5. Meyers, A. W.: Ann. Otol., Rhin. & Laryng. **24**:257, 1915.

6. Simonds, J. P.: Studies on the Pathology of the Hypophysis: V. Abscess of the Hypophysis, Endocrinology **9**:117-121 (March-April) 1925.

quadrants of the anterior lobe. Here two fairly large arteries enter at a little distance from the sagittal plane. These vessels penetrate the glandular portion and pass to the rich plexus of vessels in the core of connective tissue which is located at about the center. The infection appears to enter the hypophysis along one or the other of these blood vessels, perhaps by way of the perivascular lymphatics.

Fink[7] also reports an abscess of the hypophysis, the result of a thrombophlebitis of the cavernous sinus from suppurative ethmoiditis and sphenoiditis.

(*c*) Brain Abscess and Bronchiectasis: Adam[8] discusses "The Connection of Brain Abscess with Bronchiectasis," and, after calling attention to the fact that abscess of the brain is a known cause of death in bronchiectasis, says the association has been puzzling, but it seems probable that the abscess may result (like the bronchiectasis itself) from a nasal sinusitis rather than be a metastasis from the lung. Corroboration of this view is afforded by his two cases of bronchiectasis with brain abscess; in both there was nasal sinusitis.

(*d*) Intracranial Pneumatocele: All recorded cases of intracranial pneumatocele are reviewed by Dandy,[9] who says that while there are two sources of extracranial aerogenous tumors, (*a*) the mastoid cells and (*b*) the frontal sinus; intracranial pneumatoceles receive air (1) from a break in the wall of the skull, through which this air is forced, or (2) by the product of gas producing organisms after their entrance into the brain.

In one of his cases the air entered through a fracture of the frontal bone; in the second, through a fracture of the temporal bone, and in a third, it probably followed an operative defect of the mastoid cells.

A man entered the hospital in a state of semicoma, with a temperature of 107.2 F., and a pulse rate of 110. One week before admission, he complained of severe headache and pain in the left ear. Repeated chills and stiff neck followed. The left mastoid was tender. The leukocytes numbered 12,000; polymorphonuclears, 85 per cent. The spinal fluid was not under marked pressure (70 mm. of water), but was of ground glass appearance and contained 2,000 cells (96 per cent polymorphonuclears); there were no organisms. Pressure on the left jugular vein caused a rise of spinal fluid to 140 mm. and on the right to 160 mm.

The mastoid antrum contained a collection of pus of foul odor; the mastoid abscess was walled off but communicated with an extradural abscess. From the pus three organisms were grown: (1) *Bacillus coli*, (2) *Staphylococcus albus* and (3) the pneumococcus, type IV.

A second collection of pus of a foul fecal odor, with many bubbles of gas, lay immediately beneath the dura.

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7. Fink, E. B.: The Intracranial Complications of Posterior Sinus Infections: Report of an Unusual Case with Autopsy Findings, Surg. Gynec. & Obst. **41**:689, 1925.

8. Adam, J.: J. Laryng. & Otol. **41**:93, 1926.

9. Dandy, Walter E.: Pneumocephalus (Intracranial Pneumatocele or Aerocele), Arch. Surg. **12**:1-982 (May) 1926.

The patient died three days later.

Necropsy showed a large abscess of the left temporal lobe with a fistula in the descending horn of the left lateral ventricle; diffuse meningitis; thrombosis of the left lateral sinus (presumably a recent development, since Queckenstedt's test on admission had indicated the sinus to be patent).

Dandy feels that the air was forced through the mastoid defect into the intradural abscess cavity by swallowing, coughing or sneezing.

His conclusions are: 1. Pneumocephalus may follow in the wake of any artificial communication between the paranasal or mastoid sinuses and the cranial chamber. The opening may result from a fracture, from an operative defect, from the erosion of a chronic infection, or from destruction of the floor of the skull.

2. Pneumocephalus also results directly from infections of gas producing organisms—*Bacillus aerogenes-capsulatus* (of Welch) and possibly the colon bacillus.

3. The source of air may be the frontal, ethmoid, sphenoid and mastoid sinuses.

4. The symptoms are mainly those of increased intracranial pressure. A discharge of cerebrospinal fluid is usually present; its occurrence should always cause suspicion of pneumocephalus. Sneezing is a frequent symptom. Rhinorrhea after sneezing or after change of position is almost pathognomonic of this condition.

5. In fistulas through the frontal sinuses, the dural tear if located may be covered by a transplant of fascia lata.

Differential Diagnosis in the Presence of Suppurative Otitis or Sinusitis.—Hofer [10] reports a case of tuberculous meningitis in a child, aged 2, combined with otogenic thrombophlebitis.

On admission, the child presented the typical picture of otogenic thrombophlebitis, with intermittent temperature and bilateral middle ear suppuration. Left sided trephining of the mastoid showed suppuration under pressure (undoubtedly the cause of the intermittent fever). The temperature curve became continuous. There was no rigidity at the nape of the neck or turbidity of puncture fluid, but an increase of the cellular contents, at first slight, later considerable. Necropsy showed extensive tuberculous meningitis and diffuse miliary tubercles in all organs of the body.

Schlander [11] emphasizes a case involving the difficulty of a decision as to the meningitis being otogenic or due to other causes when a middle ear suppuration is present.

The patient's appearance suggested a nonotogenic cause of the meningitis, which was confirmed by well marked rigidity at the nape of the neck, not usually

10. Hofer, J.: Monatschr. f. Ohrenh. **60**:80, 1926.

11. Schlander, E.: Cerebrospinal Meningitis in Bilateral Chronic Middle Ear Suppuration, Monatschr. f. Ohrenh. **60**:70, 1926.

seen in otogenic meningitis, and the absence of all signs of an acute exacerbation of the ear suppuration. On the other hand, the adherent crusts in the aural attic, the sudden onset of deafness and the negative findings in the cerebrospinal puncture fluid were suggestive of an otogenic complication. A bilateral mastoid operation was performed with negative findings. Spinal puncture fluid showed meningococci.

Influence of Trauma in the Liberation of Septic—or Nonseptic—Emboli into the Brain.—That embolic septic—or nonseptic—particles must frequently be thrown into the circulation by the trauma of an operation is a mechanical necessity; the importance of this has only recently received the attention it should (especially in infections of the valveless facial veins—when squeezing or incisions are frequently followed by fatal retrograde thrombophlebitis).

Di Vestea's [12] contribution from the University of Pisa refers to the case of a woman (aged 37) on whom an operation for acute mastoiditis was followed by an apoplectic seizure (hemorrhage of the internal capsule) four days later.

Aids to Differential Intracranial Diagnosis.—(*a*) Cerebrospinal Fluid: Ohnacker,[13] under "Investigations on the Value of Sugar Determination in the Cerebrospinal Fluid in Otological Practice," says that the diagnostic value of the ratio of blood sugar to the sugar contents in the cerebrospinal fluid, for the differentiation of brain abscess, brain tumor, sinus thrombosis and meningitis, has not yet been investigated sufficiently. In hyperacute otogenic meningitis, the sugar content was diminished in 86 per cent of his cases, normal in 14 per cent, but never increased. In meningitis of a slower course, the corresponding proportions were 31, 33 and 6 per cent, respectively. A fairly constant diminution was noted only in very acute cases of meningitis.

Karbowski [14] studies the "Prognostic Value of Spinal Puncture in Intracranial Oto- and Rhinogenic Complications." He reports forty-five cases; of which thirteen were cases of cerebral abscess, three of intrameningeal abscess and seventeen of meningitis. Four patients with turbid fluid had meningo-encephalitic abscess, the suppuration having extended by continuity from the mastoid process to the brain. In 69 per cent of the brain abscess cases the fluid was clear; consequently, Karbowski thinks that a clear fluid is characteristic of brain abscesses. This, however, adds to the difficulty of the differential diagnosis between brain abscess, tuberculous meningitis and syphilis. Of the three cerebellar abscess cases, the fluid was turbid in two and clear in one. The same applies to intrameningeal abscess. Of the seventeen cases of meningitis, the spinal fluid was clear in four; the Nonne-Apelt test was

12. Di Vestea, D.: Apoplectic Contra-lateral Syndrome After Operation for Acute Meningitis on the Opposite Side, Rinasc. med. **2**:278, 1925.
13. Ohnacker, P.: Ztschr. f. Hals- Nasen- u. Ohrenh. **11**:350, 1925.
14. Karbowski, B.: Acta oto-Laryng. **7**:356, 1925.

positive; cytologically, there were from 15 to 20 mononuclear cells per cubic centimeter; these patients recovered. All the meningeal cases with turbid fluid, except one, died.

Yerger,[15] from seventeen personally observed cases of brain abscess, believes that the most important diagnostic method for the discovery of suspected intracranial pus collections is the examination of the cerebrospinal fluid. In his seventeen cases, the cell count was below 400 in 65 per cent. A complicating septic meningitis must be suspected when the count rises suddenly. Certainty is afforded only by the demonstration of micro-organisms. Of thirteen necropsies, 70 per cent showed meningitis.

In a later communication,[16] he reports eighteen cases which came to necropsy. He believes that early diagnosis can be made only on the basis of a combination of symptoms, not on a single change. Early spinal puncture usually reveals a "sympathetic meningitis."

(*b*) Papilledema: White's[17] contribution is based on 184 cases of intracranial complication of otogenic origin. He concludes that papilledema is a useful diagnostic sign in meningitis, brain abscess or infection of the lateral sinuses, but its absence does not exclude an intracranial complication. Three stages can be distinguished: pronounced papilledema, mild incipient papilledema, blurred contours or optic neuritis. Papilledema was noted in 38 per cent of the cases of cerebellar abscess, in 60 per cent of the cases of temporosphenoidal abscess, in 44 per cent of the cases of meningitis, and in 40 per cent of sinus infections.

In 50 per cent of the cases of meningitis with increased spinal pressure, the ocular fundi were normal. From the bacteriologic point of view exclusively, streptococci were found in the cases with papilledema. Increase of papilledema in the course of the disease is an unfavorable prognostic sign.

Lillie,[18] on the other hand, had four patients with choked disk, associated with mastoiditis, who recovered completely. From these he came to the conclusion that while there was no evidence of intracranial extension, it could not be definitely excluded. Three patients had parasinoidal abscess; the other a pachymeninigitis and a large cholesteatoma formation pressing on the sinus and cerebellum. One patient was subjected to intradural exploration, without result.

15. Yerger, C. F.: Left Temporal Lobe Abscess with a Report of Two Cases, Ann. Otol. Rhin. & Laryng. **33**:1364, 1925.

16. Yerger, C. F.: Case Histories of Otitic Brain Abscess Observed at Cook County Hospital, Ann. Otol. Rhin. & Laryng. **34**:258, 1925.

17. White, L. E.: Papilledema of Otitic Origin, Arch. Otolaryng. **2**:371 (Oct.) 1925.

18. Lillie, Harold I. and Walter I.: Choked Disc in Association with Surgical Mastoid Disease Without Apparent Intradural Involvement, Tr. Am. Otol. Soc. **17**:268-282, 1925.

Lillie believes that while the incidence of choked disks in surgical mastoiditis without intradural extension is low, it is sufficiently frequent to be considered in the differential diagnosis.

Hesse [19] asks: "Is the incidence of complications in acute inflammation of the middle ear dependent upon bacteriological or anatomical factors?"

He observed intradural complications in six of fifteen cases of infection from *Streptococcus hemolyticus;* nine of twenty-one from *Streptococcus nonhemolyticus;* seven of eight from *Streptococcus mucosus;* and one pneumococcus infection without complication.

(*c*) The Pulse in Suppurative Lesions: Lund [20] says that the pulse rate and spinal pressure in otogenic brain abscess may vary independently of each other. The slight increase of intracranial pressure in acute intracranial affections—such as meningitis—does not, as a rule, diminish the frequency of the pulse; but conditions differ with localized unilateral pressure, as, for example, in brain abscess and in acute cerebral hemorrhage. The latter is often associated with bradycardia, which is undoubtedly referable to the sudden rise of pressure. The opposite behavior—a sudden rise of pulse rate—is observed occasionally as an immediate sequel to opening a cerebellar or cerebral abscess. Lund thinks that localized pressure with unilateral action may be the most essential cause for the bradycardia in otogenic brain abscess, as his cases showed a marked bradycardia as often in abscess of the cerebrum as of the cerebellum, in spite of the close vicinity of cerebellar abscess to the vagus nucleus. The pressure of the abscess is not the only cause of the slow pulse; as it is relatively common in otogenic meningitis, in sepsis combined with pleocytosis in the cerebrospinal fluid, as well as in brain abscess and in acute otogenic encephalitis. Subdural abscess was associated with well marked bradycardia in one out of four cases.

Prognosis: Altogether, of 151 patients, 65 had a slow pulse, with a mortality of 81 per cent, whereas the mortality in 86 cases with a pulse proportionate to the temperature did not exceed 32 per cent; in other words, in acute otogenic intracranial complications, the mortality was high in cases of bradycardia. As Lund believes that the principal cause of slow pulse is apparently referable to a toxic irritation of the vagus nerve, the resulting bradycardia is therefore an unfavorable sign, and can be considered as analogous to the dangerous bradycardia (described by Traube) which is occasionally noted at the height of acute infectious diseases.

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19. Hesse, Walter: Arch. f. Ohren- Nasen- u. Kehlkopfh. **113** (June) 1925; abstr. J. Laryng. & Otol. **40**:675-676 (Oct.) 1925.

20. Lund, R.: Behavior of the Pulse in Labyrinthine Affections and in Otogenic Intracranial Complications, with Special Reference to Brain Abscess, Ugesk. f. Laeger 1926, no. 10, p. 246.

BRAIN ABSCESS

Relative Frequency of Suppurative Intracranial Complications.—Hirzinga [21] reviews the cases of brain abscess and purulent meningitis seen during the last fifteen years at the University of Groningen, Holland.

Of 28 cases of brain abscess, 6 originated as the result of acute otitis, and 22 from chronic otitis. The 28 cases of brain abscess included 4 of cerebellar abscess (one recovery); 24 of cerebral abscess (nine recoveries). In 14 cases, the correct diagnosis of brain abscess could be made prior to the operation. Contralateral paresis was present in 7, and aphasia in 5 cases. Of the ten patients cured, 2 had acute and 8 chronic cases.

Clinical Pathology.—The future advancement in the early diagnosis of local intracerebral collections of pus must depend on a better understanding of the blood supply of the different areas uniting the primary focus with the intradural contents. This can be obtained only by painstaking injection and corrosive cadavar work, such as found its first—as far as I know, its last—expression in the atlas of Adamkiewicz.[22] For it is now generally admitted that the vast majority of brain abscesses are adjacent and occupy rather definite sites in the temporal lobe or cerebellum, depending on the original focus in the ear or in the frontal lobe when from the nasal sinuses. However, when the pathologic process which causes the lesion is from a vascular embolus, the abscess may be situated far distant from the original focus.

Bleyl [23] reports an unusual "frontal lobe abscess as a sequel to otogenic sinus thrombosis," and calls attention to the fact that the latter may lead to the formation of brain abscess in one of three ways: (1) by continuity, (2) by the metastatic-embolic route (after passing through the pulmonary circulation), (3) by retrograde transportation of infectious material from the sinus into a meningeal vein. He believes that continuity should be considered especially in cerebellar abscess; that metastatic embolic abscess is usually situated in the marrow substance and the retrograde type in the cortical substance. He presents a case with intracranial manifestations two months after evacuation of the sinus and ligation of the jugular vein. The cerebellum and temporal lobe were explored without success. Necropsy revealed an extensive cortical abscess at the upper pole of the frontal lobe on the

21. Hirzinga, E.: Intradural Complications of Acute and Chronic Otitis Media, Geneesk. Gids (Holland) **3**:561, 1925.

22. Adamkiewicz, A.: Die Blutgefässe des menschlichen Rückenmarkes, Sitz. d. k. Akad. d. Wissensch., Math.-naturw Cl. **84**:469-502, 1881; **85**:101-130, 1882. Die Arterien des Verlängerten Markes vom Uebergang bis zur Brücke, Vienna, 1890.

23. Bleyl, R.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:16, 1925.

same side, the cause of which was a thrombotic vein which passed from the abscess backward to the vein of the sylvian fissure and communicated with the transverse sinus through the anastomosing vein of Trolard.

Hofmann,[24] in an important communication, reports the result of a pathologic study of the course within the brain itself of six otogenic temporal lobe abscesses. He found that the pus takes a rather typical direction. When the abscess originates from a diseased point in the tegmen, it invariably has a uniform tendency to extend toward the inferior horn.

This is of great surgical importance and should lead to more exact exploration, in much the same way that studies of the limitation of pus by the fascia of the neck have improved the surgery of the upper cervical region.

His first case was a large abscess of ovoid shape, which extended forward almost to the anterior end of the lower horn and backward to the beginning of the posterior horn. Below, the abscess reached to the base of the brain. In its mesial direction, it communicated with the lower horn. However, the *rupture had remained localized through agglutination of the choroid plexus with the wall.* The abscess wall was formed by the greatly infiltrated, softened and hemorrhagic medullary substance. The portions around the abscess were edematous, the edema extending forward and upward to the upper temporal convolution.

The second temporal lobe abscess extended forward to near the temporal pole and backward into the occipital lobe beyond the posterior horn. The entire abscess was composed of two parts, the posterior portion being of simple structure, extending from the base, in a curve, toward the inferior horn and reaching close to it. The anterior portion consisted of several recesses; the largest, at the level of the middle temporal gyrus, extended forward nearly reaching the temporal pole. The abscess wall was formed by infiltrated medullary substance, which was very much changed. Locally, the abscess had invaded the cortex and in part destroyed it. There was no capsule formation.

In the third case there were two temporal lobe abscesses; one mesially situated, reaching close to the lower horn; the other lateral, much larger, reaching below to the base, while its upper end closely approached the mesial abscess, as if they were just about to unite. The abscess wall was formed by infiltrated, proliferating medullary tissue. The brain was extensively softened; the inflammation had locally involved the cortex and caused partial cortical atrophy. The ventricle contained pus; the choroid plexus was infiltrated and adherent to the wall at a place near the abscess.

His fourth case was a temporal lobe abscess, larger than a walnut, extending into the lateral ventricle.

A fifth brain showed a left sided temporal abscess, with circumscribed fibrinous internal pachymengitis in the immediate vicinity of the field of operation. There was a loss of substance in the course of purulent disintegration, which reached to the vicinity of the lower horn. There was also a small abscess in the upper convolution of the temporal lobe. This abscess was distinctly bounded anteriorly and above, passing gradually backward and downward into a region of inflamed, softened and necrotic medullary substance. The diseased portion comprised a large part of the temporal lobe and approached the inferior horn, the wall of which was likewise considerably involved, leading to the formation of pyocephalus.

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24. Hofmann, L.: Otogenic Temporal Lobe Abscesses, Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:93, 1926. (From the Vienna University Clinic for Diseases of the Nose, Throat and Larynx.)

How a cortical abscess of the brain develops from a septic thrombophlebitis by nutritional death of the tissue and may thus remain sterile, while suppurative leptomeningtis is caused by a discharge of bacteria into the pial meshes, which leads to general bacterial leptomeningitis if the bacteria are not locally limited, is described microscopically by Turner and Reynolds [25] in a case of "Furuncle of the Right Nasal Vestibule: Septic Thrombosis of the Cavernous Blood Sinuses."

The brain presented a red area, 1 cm. in diameter, situated in the right frontal contiguous to the temporal lobe, on the one hand, and the caudate nucleus, on the other. Microscopic examination showed this to be an abscess. The brain was necrosed and infiltrated with cells, many of which were polymorphonuclear leukocytes. Liquefaction was just commencing in the center. The pus was continuous with that lying in the meshes of the sylvian fissure and along the pial sheaths of the larger blood vessels entering the brain in this area.

Relation of Brain Abscess to General Infections.—That brain abscess should at times be regarded from the point of view of a local manifestation of a general infection—as a metastatic embolic process—and not simply as an evidence of direct infection from a local focus in the ear or nose (although when such a condition coexists, it doubtlessly has some bearing on the etiology), is shown by the case of Siegmeister,[26] in which the route of the infection was apparently from a carbuncle in the right lumbar region, emboli from this area causing the temporosphenoidal abscess. This assumption was confirmed by the demonstration of staphylococci in pure culture in the pus of the brain abscess, as well as of the carbuncle.

Cases of Temporosphenoidal Abscess with Unusual Symptoms.—(*a*) Spontaneous Nystagmus: Tweedie [27] reports a case of cerebral abscess with spontaneous nystagmus.

A child entered the hospital with bilateral otorrhea of eight days' duration. Three days later the temperature and pulse were below normal, and bilateral optic neuritis appeared. Paralysis of the left external motor oculi supervened, with spontaneous nystagmus. Spinal puncture yielded a clear cerebrospinal fluid. There was frontal headache, without vomiting. Incision of the tense dura mater of the left temporosphenoidal lobe was followed by hernia of the brain. Exploration of the temporosphenoidal lobe liberated an ounce and a half of pus. The patient died. Necropsy was not performed.

The unusual appearance of the spontaneous nystagmus in a small temporosphenoidal lobe abscess calls for comment. In view of the absence of necropsy, the question must be asked, Was the nystagmus not a manifestation of an associated cerebellar abscess, especially as there

25. Turner, A. Logan; and Reynolds, F. Esmond: J. Laryng. & Otol. **41**:73, 1926.

26. Siegmeister, W.: Temporosphenoidal Abscess and Mastoiditis of Doubtful Origin, Laryngoscope **35**:787, 1925.

27. Tweedie: Otogenous Brain Abscess, Right Temporal Lobe with Normal Appearance of Tympanic Membrane, J. Laryngol. & Otol. **40**:467 (July) 1925.

was an accompanying (1) external ocular paralysis, (2) intense papilloedema, and (3) cerebral herniation? Statistics demonstrate that cerebral and cerebellar abscesses coexist in nearly 6 per cent of the cases, although up to the present not diagnosed during life.

(*b*) General Hyperesthesia, Except of the Arm of the Contralateral side: Carrasco [28] reports an otitic cerebral abscess, with hyperesthesia.

A man with a history of progressively severe headache followed by right sided ear suppuration of one month's duration, was deaf in the right ear. There was also a slight facial paresis and extreme hyperesthesia. No part of the head could be touched without producing extreme pain. This cutaneous hyperesthesia exended over the entire body. Light and noise likewise caused severe suffering. When the patient sat up in bed, the headache increased and nausea supervened. The pupils were narrow and rigid, the pulse slow (60); temperature was normal. On the seventh day after admission, he became more quiet, the somnolence being referable to morphin. *The cutaneous hyperesthesia persisted, except in the left arm, which was not very sensitive.* On the following day, the patient was in a state of deep coma, insensible to all stimuli. Mastoid exploration was negative. Two brain punctures for pus were without result. Necropsy showed softening of the right temporosphenoidal lobe, and an abscess containing greenish and offensive pus, without a capsule, the walls being formed by softened brain substance.

Cases of Intradural Involvement in which Accompanying Serous (Protective) Meningitis Contributed Outstanding Symptoms.—That localized arachnoiditis—protective or septic meningitis—is a frequent complication of extradural suppuration, I have ample clinical evidence. In several of my middle fossa cases it has produced hemianoptic indentations of the visual fields; and in one, a complete hemianopsia.

In the posterior fossa cases, I have had numerous examples of a complete disappearance of intense vertigo—dizziness, nystagmus—and papilledema after the evacuation of fluid from the subarachnoid meshes. Cisternal arachnoiditis simulating cerebellar tumor was first described by Horrick,[29] and, I think, is the explanation of Boenninghaus' [30] case of "Acute Serous Meningitis After Otitic Labyrinthitis with the Picture of Cerebellar Abscess; Cure by Incision of the Cerebellar Dura; Subsequent Development of a Meningocele in the Auditory Meatus, Probably from the Occluded Lateral Pontine Cistern."

The subsidence of the symptoms of a brain abscess following simple incision of the dura causes Boenninghaus to assume that there existed a serous extravasation in the ventricles. His explanation is that the incision of the dura allowed brain prolapse which led to diminution of the pressure. About a month after the incision a meningocele developed, which ruptured three times in the course of some months, but ultimately

28. Carrasco: Siglo méd. **75**:531, 1925.
29. Horrick: Arachnoiditis, Tr. A. M. A., Section of Nervous & Ment. Dis., 1923, p. 210.
30. Boenninghaus, G.: Ztschr. f. Hals- Nasen- u. Ohrenh. **13**:1, 1925.

closed of its own accord. During the entire period the patient felt perfectly well, which Boenninghaus thinks indicated that the meningocele was closed off from the remaining meninges. The origin of the meningocele is thus explained by Boenninghaus: The original serous meningitis produced an agglutination of the pia and arachnoid at the base of the skull, interrupting the communication with the fourth ventricle. A cyst formed in which cerebrospinal fluid accumulated, because the pontile cistern has a choroid plexus of its own. External influences (the first time by extraction of a sequestrum from the labyrinth) led to an increased secretion and rupture.

Sensory Aphasia from Extradural Abscess.—Thormann [31] reports a case of verbal amnesia and occasional paraphasia with an extradural abscess over the left temporal lobe, drainage of which was followed by recovery. The cerebrospinal fluid was always normal; consequently Thormann thinks temporal lobe abscess could be excluded. He asserts that verbal amnesia occurs often with extradural abscess in the left middle cranial fossa, but sensory aphasia rarely. The speech defect is explainable as a result of pressure in a large abscess; but in small ones by a collateral meningo-encephalitis. He thinks that his case teaches that sensory aphasia may occur with extradural otogenic abscess in the left middle cranial fossa, and consequently cannot be regarded as pathognomonic of temporal lobe abscess.

In reviewing Thormann's case, it might be suggested that (1) an incapsulated abscess still exists—a far from unknown condition—to which Ott also calls attention when he says: "Even though the symptoms of intracranial infection subside spontaneously, these patients should be regarded as abscess suspects, especially so if an occasional attack of headache, vertigo and loss of weight persist." [32]

More probably, however, the aphasia in Thormann's case was the result of (2) an incapsulated pia-arachnoid accumulation of cerebrospinal fluid over the temporal lobe, which was relieved by the evacuation of the extradural abscess.

The motor cortical area for speech is susceptible to slight and transient pressure, such as pial hemorrhages, but sensory aphasia without an intradural complication is almost unknown. It is, however, a frequent occurrence in localized cortical pia-arachnoid collections of pus. In Thormann's case, I would suggest that the disappearance of the aphasia after the evacuation of the extradural abscess is best explainable by the supposition that the accumulation of protective intradural fluid near the extradural collection was not infected, or but slightly so.

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31. Thormann, H.: Sensory Aphasia with Otogenic Extradural Abscess in the Left Middle Cranial Fossa, Ztschr. f. Hals-, Nasen- u. Ohrenh. **11**:429, 1925.

32. Ott, W. O.: Intracranial Complications of Ear and Accessory Nasal Sinus Infections, Texas State J. Med. **21**:12, 1925.

MacCuen Smith,[33] in reporting "Three Cases of Otitic Brain Abscess," shows that in all these cases there was almost entire absence of neurologic and ophthalmologic symptoms, while in one of the cases the probability is that the patient had a brain abscess for three years before he came under Dr. Smith's care. The abscess did not seriously interfere with the patient's work until a few weeks before he was admitted to the hospital.

FRONTAL LOBE ABSCESS

Jessaman [34] states that abscess of the frontal lobe is not of frequent occurrence, the most common causes being infection secondary to frontal sinus suppuration and trauma. The mortality is very high, a large factor being the great difficulty in making an early diagnosis. Persistent headache is probably the most common symptom, and its continuance after the drainage of infected sinuses should be regarded with suspicion. Optic neuritis is rare. The occurrence of convulsions or paralysis in the presence of infection of the accessory nasal sinuses is strong evidence of an intracranial complication, and demands prompt attention.

Jessaman reports a case of persistent right sided headache and obstructed drainage from the right frontal sinus; the anterior part of the right middle turbinate was removed and the anterior cells opened. Improvement followed, but two days later the patient had a convulsion, became rather drowsy, and complained of severe pain in the head. A large opening was made in the posterior wall. The dura was rather dark and appeared under tension. Incision of the dura did not show any *pus beneath it, but the brain was discolored.* An incision through about one-fourth inch of the brain cortex opened an abscess, from which a large amount of dark colored pus was evacuated. This abscess must have been very close to the lateral ventricle. Subsequently there was considerable trouble in keeping drainage, and finally a curved cautery was employed to burn down the granulations from the frontal sinus to the nose.

Arnoldson and Bostroem [35] also report a frontal lobe abscess successfully treated after a second abscess had been drained; it was suddenly manifested by convulsions.

Cowan [36] reports a case of an intrapia-arachnoid abscess. The patient died as the result of a collection of pus in the left lateral ventricle.

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33. MacCuen, Smith S.: Ann. Otol. Rhin. & Laryng. **34**:109, 1925.

34. Jessaman, L. W.: Abscess of the Frontal Lobe Secondary to Suppuration of the Frontal Sinus: with Report of Case, Boston M. & S. J. **192**:739, 1925.

35. Arnoldson, N., and Bostroem, C. G.: Cerebral Abscess of Rhinogenic Origin, Acta oto-laryng. **8**:339, 1925.

36. Cowan, H. Alexander: Intradural Abscess Secondary to Frontal and Ethmoidal Sinusitis, Lancet **2**:648, 1925.

Frequency of Multiplicity.—For some unknown reason the cerebellum is much more frequently the site of several separate foci of suppuration than the large brain.

Many of the supposed separate abscesses on microscopic examination will be found to have a small communication with the larger primary cavity. These abscess prolongations are undoubtedly caused by compression from the dural envelop, as stressed by Fraser,[37] who at postmortem found three abscess cavities in the left cerebellar hemisphere, only one of which had been opened at the operation. The pressure effect was shown by a well marked herniation through the foramen magnum.

Fremel [38] in a contribution on the "Morphology and Growth of Cerebellar Abscess," reports two multiple out of three cases of cerebellar abscess.

The first patient with chronic left sided otorrhea was admitted with fever and headache in the left frontal and parietal region. The dura was found to be exposed over the tegmen tympani. The sinus was thrombotic. Two weeks later puncture of the cerebellum evacuated a large abscess. Death followed at the end of nine days. Necropsy showed a very tense dura with flattened walls, smooth and free from exudate; the left cerebellar hemisphere was enlarged, and a seropurulent exudate of a greenish color was seen in the vicinity of the incision.

The second patient had suffered from left sided ear suppuration, with partial loss of hearing; the otorrhea returned after a cold bath. No vomiting, fever, vertigo or aural tinnitis was present. Radical operation was followed by the formation of a *retropharyngeal abscess.* Meningitic symptoms supervened. A later operation revealed a granulation on the dura behind the labyrinth; puncture yielded discolored pus. Death occurred two hours after operation. Necropsy showed a large opened abscess in the left cerebellar hemisphere, filled with blood clot. The abscess occupied the entire covering of the cerebellar hemisphere, and the encephalitis reached the ependyma of the fourth ventricle. The pus contained *Streptococus pyogenes.* Histologic sections of the cerebellum showed two abscess cavities.

The third patient had chronic bilateral suppurative otitis media. There were attacks of severe headache, especially severe at night, followed by vertigo, nausea and vomiting. Temporary improvement occurred without operation. Necropsy showed three cerebellar abscesses the size of a nut, separated by the cortex.

Comment: The multiplicity of abscesses can perhaps be interpreted as individual processes of a common abscess cavity, especially as the abscesses were separated by the cortex and pia.

Massesi [39] contributes a good anatomic description of an abscess situated in the anterior one third of the cerebellum—the usual position when the infection originates from caries of the petrous pyramid.

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37. Fraser, J. S.: Chronic Middle Ear Suppuration; Cholesteatoma; Latent Labyrinthitis; Cerebellar Abscess; Death, Proc. Scottish Soc. Otol. & Laryngol., Twenty-First Meeting, J. Laryngol. & Otol., no. 9, **40**:598-599 (Sept.) 1925.

38. Fremel, F.: Morphology and Growth of Cerebellar Abscesses; Monatschr. f. Ohrenh., Laryng. Rhinol. **60**:128, 1926.

39. Massesi, B.: Cerebellar Abscess of Otitic Origin, of Unusual Localization, Gior. di chir. med. **5**:164. 1926.

Necropsy on the transverse section of the cerebellum showed that a large portion of the *right* cerebellum was replaced by an enormous cavity filled with pus. The destroyed nervous substance comprised a large part of the right hemisphere and a portion of the vermis.

Diagnosis of Cerebellar Abscess.—(*a*) Indefiniteness of Localizing Symptoms: Gavello,[40] of the University of Turin, observed five otogenic cerebellar abscesses in 1918-1919. Recently he recorded three more cases in which the diagnosis was rendered difficult, or impossible, because of the indefinite symptoms and the serious condition of the patient, which prevented an examination. The site of the abscess was always on the side of the affected ear. He says that from the diagnostic point of view the location of the headache is of slight importance, as it was altogether absent in some patients during certain periods of the disease, and in other cases, was diffusely distributed, and was limited to the nape of the neck only in a few cases. Rigidity of the neck and tenderness on percussion of the bones therefore have no special significance. Vomiting may be present or absent. Changes of the ocular fundi were observed but were not invariably present; neither did spinal puncture furnish an accurate guide. The most regular and noteworthy symptom consists in more or less marked spontaneous nystagmus; which may be produced by pressure on the vestibular nuclei (Deiters' nucleus), as well as from the cortex of the hemispheres. Gavello thinks the nystagmus does not differ from labyrinthine nystagmus as its direction may be toward the diseased, as well as toward the normal ear. When a labyrinthine cause of the nystagmus can be excluded, it is a valuable diagnostic sign of cerebellar affection. The vertigo is parallel with the nystagmus. Disturbances of equilibrium are not dependent on definite positions of the body or the head as distinguished from labyrinthine affections. Associated facial paralysis is directly connected with middle ear disease. The tests of cerebellar localization, such as disturbances of the coordinated muscular function on which modern cerebellar diagnosis is based, could not be carried out by the author on account of the grave condition of the patients.

On the other hand, Nühsmann [41] states that the spontaneous nystagmus of cerebellar abscess is at first to *the same side,* but within a few hours after the destruction of the vestibular end organ, it is to the healthy side, from the unopposed action of the opposite labyrinth. When the head is turned toward the sound side, the patient falls forward instead of backward to the diseased side as in cerebellar abscess. Some time between the eighth to the twenty-eighth day the roof nuclei become

40. Gavello, G.: Cerebellar Abscesses of Otitic Origin, Arch. ital. di chir. **12:** 567, 1925.

41. Nühsmann, T.: The Differential Diagnosis Between Inflammatory Labyrinthitis and Otogenic Cerebellar Disease, Arch. f. Ohrenh- Nasen- u. Kehlkopfh. **43,** no. 4 (July) 1925.

compensated to the altered condition, and with this adjustment the nystagmus, past-pointing, falling, and locomotory disturbances disappear.

(*b*) Conflicting Vestibular Manifestations: Jones [42] contributes a case which shows how misleading the vestibular findings may be when regarded entirely from a localizing standpoint; as associated with an aphasia (perhaps due to arterial thrombosis), they caused an exploration of the temporal lobe, when in reality the abscess was situated in the cerebellum.

The patient had otitis media and mastoiditis. The vestibular findings were: Prompt response to nystagmus, with total absence of vertigo; tendency to conjugate deviation on stimulation of both horizontal canals; the presence of response from left vertical canals on first examination, followed at a later date by absence of response, incoordination of ocular movement, which came into evidence from ear stimulation—the deviation of the left eye inward on douching the right ear, and the deviation of the right ear inward on douching the left ear.

Necropsy revealed in the region of the left cerebral cortex, covered by the bone flap, two small areas of necrosis extending into the subcortical white tissue. (These areas apparently corresponded to the points into which the exploratory needle had entered at the operation.) There was enlargement of both lateral ventricles. There was a large abscess, which occupied practically the entire right cerebellar hemisphere, with a pouting area of pus directly adjacent to the mastoid cavity (making it evident that there had been a direct extension through the dura at this point from the mastoid).

(*c*) Localizing Vestibular Symptoms: That the *induced* vestibular manifestations, however, may be of the greatest localizing value, especially in *acute* lesions of the cerebellum, is shown by Mauthner,[43] who reports a case of hemorrhagic encephalitis and cerebellar abscess after measles and subsequent otitis, with histologic findings.

The patient had a mastoditis as a sequel of measles and acute otitis media. After operative intervention she lost consciousness. A few hours later, the eyeballs were immovable, the extremities rigid (in the sense of Sherrington's decerebration rigidity). *Cold irrigation of the right ear was followed by raising of the right eye and lowering of the left.* Necropsy revealed encephalitic foci in the medulla oblongata, an abscess of the central portion of the right cerebellum and a small thrombotic blood clot in the third ventricle.

Vestibular Tests in Nonsuppurative Lesions.—Wishart [44] summarizes the present status of the diagnostic localizing value of the ear tests, and states that his evidence permits of the following postulates:

1. Tumors of the frontal lobes do not interfere with cochlear or vestibular function.

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42. Jones, Isaac H.: Cerebellar Abscess with an Unusual Complication, Laryngoscope **35**:893-895 (Dec.) 1925.

43. Mauthner, O.: Only Apparently Otogenic Cerebral Complications in Acute Infectious Diseases, Monatschr. f. Ohrenh. **59**:672, 1925.

44. Wishart, D. E. S.: Neuro-otological Examination in Seven Consecutive Verified Cases of Brain Tumor in the Posterior Fossa in Children, Tr. Am. Otol. Soc., Fifty-Eighth Annual Meeting, pt. I, **17**:86-147, 1925.

2. Subtentorial tumors which are completely outside the brain stem and the cerebellum affect homolateral cochlear and vestibular function.

3. Subtentorial tumors which are in the midline and which do not involve cerebellar tissue do not affect cochlear function. They do interfere with vestibular function. The interference tends to be bilaterally symmetrical, and the functions of both vertical canal reflex arcs are affected before those of either horizontal canal.

4. Unilateral intracerebellar tumors do not interfere with cochlear function. They do interfere with vestibular function. The function of the reflex arcs of the vertical canals will both be affected before those of the horizontal canals. The tumor will be found on the side opposite to the vertical canals the function of which is most interfered with.

Fisher,[45] however, would add that vertigo and past-pointing were found to be reliable indications of the condition of posterior fossa contents. A cerebellar lesion, with very few exceptions, should show impaired vertigo and past-pointing. However, the presence of good vertigo and past-pointing is even more strongly indicative of a normal cerebellum. Another feature of cerebellar lesions is the lack of susceptibility of those patients to vestibular stimulation. Nausea, pallor, sweating and vomiting as a result of douching and turning, were practically never encountered in cases of cerebellar disease, and their presence was used almost as a definite indication that the lesion was not cerebellar.

Treatment of Brain Abscess.—The only real advance in the surgical technic of brain abscess is contributed by Cahill,[46] who reports the largest number of successive cures of brain abscess ever recorded. His method may be summarized as (1) attempting to produce protective adhesions of the pia-arachnoid prior to dural incision, and (2) later establishing drainage by a wide-mouthed wire cone sutured into the dural opening.

Cahill says:

> When the mastoid route is used, the dura of the middle fossa is exposed to a diameter of three centimeters. The cavity is packed with dichloramine T, or mercurochrome gauze, for forty-eight to seventy-two hours. Then the dura is opened with a crucial or three-limbed incision, after passing silk sutures through the tip of each of the dural flaps. The operator seeks the abscess with the grooved director or Cushing needle. The anterior portion of the exposed area should be used for exploring the anterior portion, and the posterior area for exploring the posterior portion of the temporal lobe. If the abscess is found, without

45. Fisher, Lewis: The Present Status of Vestibular Tests in Intracranial Conditions, Tr. Am. Otol. Soc., Fifty-Eighth Annual Meeting, pt. I, **17**:148-156, 1925. (Record of 103 suspected cases of brain lesion. Cases selected were verified by operation or necropsy; operative findings.)

46. Cahill, Harry P.: Twelve Cases of Cerebral and Cerebellar Abscesses Drained by the Mosher Wire Gauze Cone, Tr. Am. Otol. Soc., Fifty-Eighth Annual Meeting, pt. I, **17**:42-66, 1925.

permitting the grooved director to shift to the slightest degree, the drain with its plunger in place is guided along the grooved director into the abscess.

The cone should be held in place by granulations after the first week. If it becomes blocked, it may be gently curetted out. In the third week the skin and dural sutures may be removed and the brain permitted to expel the drain.

Errecart [47] drains by means of a glass tube with perforations 5 mm. in size, projecting from the surgical wound in such a way that the gap in the dura mater remains well separated. The tube is not expelled by the pulsating movements of the brain, and there is a protection against the occurrence of cerebral hernia.

Beck [48] describes Neumann's method for exposure of the floor of the anterior portion of the middle fossa.

The patient, with a history of chronic left sided middle ear suppuration, was admitted in an unconscious condition, with symptoms of typical meningitis. Spinal puncture revealed a purulent fluid with innumerable polymorphonuclear cells. The bone at the base of the skull, which appeared softened and discolored, was removed. The exposed dura of the middle cranial fossa showed thick fibrinopurulent deposits and in one place appeared necrotic. Exploratory puncture of the temporal lobe yielded pus. Because the purulent deposits on the dura extended into the anterior portion of the middle fossa, the temporal muscle was detached in its fascia from the zygomatic arch and turned upward. It was thus possible to resect a large portion of the squama and the zygomatic process; this was followed by the removal of the bone of the middle cranial fossa to within 1 cm. of the foramen ovale. The meningeal symptoms subsided after eight days.

Results of Surgical Treatment of Cerebellar Abscess.—That marked advance in the surgical treatment of cerebellar abscess has been made during the past few years is shown by the lowered mortality in the Halle clinic.[41] From 1911 to 1920, of twenty-six cases of brain abscess, eight were cerebellar, with only one survival; while from 1921 to 1924, of twenty-one abscesses, ten were cerebellar, with seven survivals.

After-Treatment of Brain Abscess; Rest and Selection of Type of Work.—Fraser,[49] in reporting a case, emphasizes the importance of a prolonged period of enforced rest after evacuation of an abscess. Failure in such after-care has been followed by death in numerous instances.

The patient did very well for six days, and was allowed to get up, but at the end of half an hour he complained of headache and vomiting, and his temperature rose. Postmortem examination revealed an abscess of the right temporal lobe which had ruptured into the lateral ventricle; there were red softening and hemorrhages round the abscess, basal meningitis and corking of the cerebellum into the foramen magnum.

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47. Errecart: Drainage of Otitic Cerebral Abscess, Rev. españ. y Americ. de laringol., 1925, p. 25; l'oto-rhino-laryng. internat. **11**:467, 1925.

48. Beck, O.: Extensive Brain Abscess—Labyrinthitis—Operation—Recovery, Wien. klin. Wchnschr. 1926, no. 11, p. 514.

49. Fraser, J. S.: Acute Middle Ear Suppuration (right); Extradural Abscess; Temporal Lobe Abscess; Ventricular Meningitis; Death, Proc. Scottish Soc. of Otol. and Laryngol., Twenty-first Meeting, J. Laryngol. & Otol. **40**:593-595, no. 9 (Sept.) 1925.

Dr. Ewart Martin, in the discussion of Fraser's case, raised the question as to how long after recovery patients should be allowed to return to work.

In a patient operated on by him, recurrence took place six months after drainage of the abscess, and it was again opened. Shortly afterward the patient had a fit of the jacksonian type. Three months afterward a decompression operation was performed and a blood clot removed from the area of the abscess. The patient worked as a gardener which entailed a certain amount of stooping, and developed further attacks of jacksonian epilepsy. He then acted as a valet, without any stooping, with no recurrence of epilepsy.

MENINGITIS

Surgical Anatomy.—Future progress in the treatment of meningitis must largely depend on a better understanding of the anatomy and physiology of the pia-arachnoid. Not sufficient importance has been attached to its minute anatomy since Key and Retzius' [50] work in 1875 until Locke and Naffziger [51] in 1924 and Karlefors [52] in 1925 published their important papers.

Boss [53] says that since the publication of Bárány's complex (which was explained through dropsy of the cisterna pontis lateralis), these spaces do not seem to have acquired the importance to which they are entitled in otologic surgery, especially in the therapy of otogenic meningitis. He investigated the topographic relations of these cisterns to one another and to the petrous pyramid by the injection of a methylene blue and agar mixture.

He summarizes as follows: 1. The cisterna magna (or cerebellomedullaris) is bounded anteriorly by the pons and the fourth ventricle, above by the lower vermis of the cerebellum, behind by the falx cerebelli. Ventrally, it extends to the medulla oblongata, where it communicates with the anterior basal cisterns. 2. The cisterna pontis media contains the basilar artery. 3. The cisterna pontis lateralis is the only cisterna in relation with the petrous pyramid. It is directly applied to the anterior portion of the mesial pyramidal surface, and practical importance is attached to the fact that the labyrinth, the meatus acusticus internus and the cochlear aqueduct open into it. It is traversed by the glossopharyngeal, vagus, facial, acoustic, trigeminal and abducens

50. Key, Axel, von; and Retzius, G.: Studien in der Anatomie des Nervensystems und des Bindegewebes.

51. Locke, Charles Edward, Jr., and Naffziger, Howard C.: The Cerebral Subarachnoid System, Arch. Neurol. & Psychiat. **12**:411-418 (Oct.) 1924.

52. Karlefors, J.: The Meningeal Spaces of the Cerebellum; Communications of the Fourth Ventricle with Subarachnoid Spaces and the Cochlear Aqueduct in Man, Acta oto-laryng., 1925, suppl. 4.

53. Boss, L.: Topography of the Arachnoid Cisterns, Zentralbl. f. Chir. 1926, no. 9, p. 542.

nerves. In rare cases, it passes with the pontile cerebellar peduncles into a larger subarachnoid space of the horizontal cerebellar sulcus.

Types of Otitic and Rhinitic Meningitis.—Wharry [54] states that Milligan has differentiated three types of meningitis of otitic origin: (1) meningitis benigna (or serous); (2) meningitis semimaligna (in which pus cells are present in the cerebrospinal fluid); (3) meningitis maligna (in which both pus and organisms are present). Of patients with type 3 cases, only 2 or 3 per cent have been known to recover irrespective of any treatment. From what Wharry has seen, the majority of the few patients with meningitis maligna who recover, appear to have staphylococcal cases.

Wharry reports the case of a patient with staphylococcic meningitis of otitic origin, who was operated on and recovered after transthecal irrigation. A large fistula was found in the lateral semicircular canal. The infection had evidently passed through the labyrinth and lamina cribrosa into the internal auditory meatus and thus to the basal meninges. The lamina cribrosa was removed to allow as free drainage as possible of the basal cisterns along the route of infection (Scott-West method). The right internal ear was irrigated through the spinal canal with Locke's solution at body temperature.

Pathology.—The microscopic studies of Turner and Reynolds [25] show how infective thrombophlebitis gives rise to leptomeningitis—the usual terminal process. The leptomeninges showed diffuse purulent meningitis with septic thrombosis of branches of the middle meningeal and superficial middle cerebral veins. In some instances, the wall of the vein had broken down and pus had escaped into the pial meshes.

Pathologic Origin of Cortical Symptoms.—Přecechtěl [55] describes three cases, which may be summarized as follows: In all, the earliest symptoms of meningitis were localized cortical signs; in one, irritative signs and convulsions followed by paralysis; in the other two, paralysis without any preceding stage of irritation.

He says that cortical signs have a different significance in the two classes of cases; when appearing suddenly in the course of an acute otitis media, they suggest the onset of a "hood" meningitis, and the prognosis is accordingly unfavorable.

Rhinorrhea and Its Relation to Meningitis.—That a large proportion of patients with rhinorrhea die from a complicating meningitis, and those who live become blind, was demonstrated by St. Claire Thompson a quarter of a century ago.

The etiology is obscure, but Beck, in discussion, said he had seen two specimens dissected by Chiari which showed deficiency in the cribri-

54. Wharry, H. M.: Three Cases of Middle Ear Disease with Intracranial Complications, Brit. M. J. **1**:85, 1926.

55. Přecechtěl, A.: Disturbances of the Motor Cortex in Otogenic Meningitis, Zentralbl. f. Hals- Nasen- u. Ohrenh. **7**:797, 1925; abst. J. Laryng. & Otol. **41**:259 (April) 1926.

form plate, with a fistula that had never closed. I have seen a rhinorrhea following a fracture of the skull, with death from meningitis a year later, which at postmortem showed that the watery discharge from the nose originated from a chronic traumatic pial cyst of the brain which drained through the frontal sinus. Up to the present time, no progress has been made in its treatment, although the suggestion of placing a strip of fascia lata was suggested to the patient.

However, Stein,[56] in reporting a case, states that the discharge almost ceased for two weeks after the administration of benzyl benzonate solution, 20 drops, three times a day, and after three weeks it entirely disappeared and has not recurred in five months. Atropine internally had no effect, and alcohol injection of the left Meckel's ganglion was performed without result. At the time of the report, the patient had a feeling of wellbeing with no increase of headaches.

Kuemmel[57] reports a case of a girl, aged 18, who had been suffering from atrophic rhinitis of two years' duration, with frontal headache and a mucopurulent nasal discharge. An injection of her own blood was made under the mucosa of the right lower turbinal and in the right side of the septum. Some days later, another injection of about 3 cc. of blood was made under the left lower turbinal and in the left side of the septum. Both underwent complete absorption. The third injection of about 5 cc. of blood under the mucosa of the lower turbinal and the septums of both sides led to the formation of an abscess in the left frontal brain, hemolytic streptococcic meningitis and death.

Meningitis following Gradenigo Syndrome.—The statistics of Sears[58] demonstrate that death from meningitis supervenes in more than 15 per cent of the cases of Gradenigo syndrome.

Gradenigo[59] of the University of Naples says that while numerous theories have been advanced as to the origin of his syndrome, he is of the opinion that the abducens paralysis is only of extradural origin. The infection may spread from the small peripheral pneumatic cells of the petrous bone and by a lymphatic route, or between the carotid artery and the wall of the cavernous sinus. The lymphatic path was demonstrated by his pupil Papele.

Engelhardt[60] observed that in one instance abducens paresis did not develop until the onset of terminal purulent meningitis, although at

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56. Stein, Otto J.: Cerebrospinal Rhinorrhea, Chicago. Laryng. & Otol. Soc. Proc., April 6, 1925; Ann. Otol. Rhin. & Laryng. **34**:1269-1271 (Dec.) 1925.

57. Kuemmel, W.: Two Unusual Cases of Rhinogenic Meningitis, Acta otolaryng. **8**:209, 1925.

58. Sears, W. Hardin: Otogenic Paralysis of the Abducens, with Especial Mention of Isolated Palsy Associated with Irritation of the Gasserian Ganglion, Tr. Am. Laryng. Rhinol. & Otol. Soc., 1925.

59. Gradenigo, G.: The Syndrome of the Apex of the Petrous Bone, Rev. otoneurol. ophth. **2**:158, 1925.

60. Engelhardt, G.: Abducens Paralysis in Extradural Abscess at the Apex of the Petrous Bone, After Acute Middle Ear Suppuration; Death from Meningitis, Ztschr. f. Hals,- Nasen- u. Ohrenh. **11**:194, 1925.

necropsy there were changes at the petrous apex. He thinks that the value of the symptom of abducens paresis is variable, as it is often absent in extradural abscess at the apex of the petrous bone until the meninges are affected. On the other hand, it may be present in middle ear suppurations which heal without major intervention.

Ulrich [61] reports a case in which the entrance of infection to the meninges was demonstrated only on microscopic examination. The postmortem examination showed that the original focus was extradural and affected the tip of the petrous bone. The gasserian ganglion was invaded and materially damaged by suppuration, and the sixth nerve was reduced in size to a mere thread.

There was an acute exacerbation of a chronic middle ear suppuration, calling for paracentesis and mastoid operation. The symptoms subsided with the exception of the abducens paralysis. Two months later there came on suddenly giddiness, vomiting and diffuse headache, followed by stupor, stiffness of the neck, Kernig's sign and exaggerated reflexes; with *Streptococcus mucosus* in the cerebrospinal fluid.

Recurrence of Meningitis After Apparent Cure.—Experimental evidence [62] demonstrates that the micro-organism of suppurative meningitis may remain dormant—hidden as it were—in the subarachnoid prolongations of the cranial nerves as they leave the skull, and then suddenly take on renewed activity.

Lannois and Jacod [63] report a case of "Otogenous Suppurative Meningitis with Remissions," with purulent but sterile cultures.

The meningitis lasted altogether three months, with two remissions, one of short duration before operative intervention, during which the patient resumed his work, another, of about one month, after a mastoid evacuation. During the remissions, the cerebrospinal fluid was clear.

Treatment of Meningitis.—Slowly the treatment of septic meningitis is resolving itself into (1) early diagnosis while the process is still limited to an area of the pia-arachnoid adjacent to the primary focus of infection, (2) the drainage of this area; to which I [64] have added the necessity of (3) putting the parts at rest by ligature of the internal carotid artery, and (4) attempting to increase the immunity of the cerebrospinal fluid by repeated blood transfusions from an immunized donor, associated with massive lumbar punctures.[65]

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61. Ulrich, K., Zurich: Otogenous Abducens Paralysis, Ztschr. f. Hals-Nasen- u. Ohrenh. **9**:403 (Jan.) 1925.

62. Weed, L. H.; Wegeforth, P.; Ayer, J. B., and Felton, L. D.: Experimental Meningitis, Monog. Rockefeller Inst. Med. Res., March 25, 1920, no. 12, pp. 57-112.

63. Lannois and Jacod: Rev. de laryngol., Bordeaux **45**:1 (Jan. 15) 1924.

64. Eagleton, W. P.: The Surgical Treatment of Meningitis (Third Communication), J. A. M. A. **83**:1900-1906 (Dec. 13) 1924.

65. Eagleton, W. P.: Meningitis of Otitic Origin (Fourth Communication), presented to the Section of Otolaryngology, A. M. A., 1926.

Goerke [66] would open wide the dura mater of both cranial fossae, because he believes it is only in this way that the paths of infection can be followed and the primary foci of intracranial infection be removed.

He does not think that the question when to open the labyrinth is as yet definitely settled. Total loss of labyrinthine function does not always call for opening, as it may be caused by a serous or a past fibrous inflammation. On the other hand, in the presence of a functioning labyrinth it should be opened when the clinical picture is suggestive of a circumscribed labyrinthitis. With meningitic symptoms, even a functioning labyrinth should be sacrificed. After the radical removal of the source of infection, the next step is the eradication of the invading organisms and the neutralization of their toxins by medicinal therapy. He states that the majority of the many recommended remedies, such as vucin, optochin, eucupin, protargol, collargol, electrargol, argochrome, dispargen, and others, have been found to be inefficient. He has some faith in trypaflavin and methenamine. He does not recommend intradural introduction of methenamine by means of spinal puncture as it reaches the infectious focus only in very weak concentrations, but thinks that the best results are obtained by intravenous introduction. Spinal puncture is an important adjuvant, as it leads to the lowering of the brain pressure, which is followed, in its turn, by the production of a new bactericidal cerebral fluid, and by an improved absorption through the pachionian granulations.

The last and most important step is drainage of the subarachnoid space. A permanent drainage is not secured by the mere opening of the dura mater, or by the introduction of a cannula. The large cisterna cerebromedullaris is not accessible from the ear, whereas the cisterna pontis lateralis with its ramifications is within easy reach. After wide exposure of both cranial fossae, the dura is raised with a spatula from the posterior surface of the petrous.

Nühsmann [67] reports forty cases of purulent meningitis seen during the last four years in the Halle University ear clinic. He thinks the principal treatment is the elimination of the primary pus focus in the mastoid process and repeated spinal punctures. He regards an increase of the polymorphonuclear leukocytes in the cerebrospinal fluid as generally indicative of an extension in the meningitis and as an unfavorable sign even with a temporary improvement of the clinical symptoms. On the other hand, a diminution of the polymorphonuclear leukocytes in the puncture fluid or an increase of the lymphocytes is a favorable prognosis even with a discouraging clinical picture.

66. Goerke, M.: Treatment of Otogenic Meningitis, Zentralbl. f. d. ges. Chir. u. i. Grenzgeb. **33**:512, 1925.

67. Nühsmann, T.: Treatment of Otogenic Purulent Meningitis, Arch. f. Ohrenh.- u. Kehlkopf. 1925, 113, p. 16.

He thinks that the injection of vucin solution into the spinal space is not only useless but often harmful on account of the injury to the spinal meninges and abscess formation in the skin.

Kepes' [68] case of cured otitic meningitis is important because it shows the more favorable prognosis of meningitis when the source of infection of the meninges can be determined, such as in sinus thrombosis, and points the way to the routine method of operation, namely, by following the path of infection into the meninges, whether from the sinus, a small vein, the labyrinth or a brain abscess.

A boy had acute middle ear suppuration followed by symptoms of sinus phlebitis. The turbid cerebrospinal fluid contained *Streptococcus longus*. Operation revealed no thrombus, and consequently Kepes thinks that the initial symptoms were presumably caused by Koerner's osteophlebitis. There was a brain abscess in the middle cranial fossa.

Treatment by Septicemin.—Mavens [69] reports two cases of otogenic meningitis cured by septicemin after operation, one from a chronic otitis, the other from acute labyrinthitis. In the first case, with bidaily spinal punctures, he injected septicemin intraspinally (an iodine-tropin compound) first 1, then 2 ampules of 4 cc., until the cerebrospinal fluid, which had contained staphylococci and streptococci, was free from bacteria. Septicemin was also administered intravenously. The second patient was cured by intravenous injections of septicemin and local irrigation of the operative wound.

Results of Treatment.—Of fifty patients with meningitis treated in the Breslau ear clinic during the last five years, sixteen were cured; nearly one third of the cases. These favorable results are referred by Hinsberg [70] to (1) early recognition of the meningitis and removal of the pus focus in the ear or in the nasal sinuses; (2) the subsequent treatment by large doses of methenamine (6 to 10 Gm. daily, in part intravenously, in part by mouth). Hemorrhagic cystitis almost invariably supervenes, sometimes within a few days, often delayed until over 200 Gm. of the remedy has been administered. He is in favor of suboccipital puncture, because it does not involve the danger of a flow of cerebrospinal fluid like incision of the cisterna pontica.

Vaccine and Serum Treatment of Meningitis.—In one of the clinics in France, cases of brain abscess (following evacuation) are treated as a routine with an autogenous vaccine to prevent, if possible, the secondary staphylococcic infection, which so frequently occurs. Treatment of the

68. Kepes, P.: Pathology and Symptomatology of Otogenic Meningitis: Cured, Zentralbl. f. Hals,- Nasen- u. Ohrenh. **8**:54 (Oct. 25) 1925.

69. Mavens: Curability of Septic Purulent Meningitis of Otitic Origin, Ann. d. mal. de l'oreille, du larynx **44**:28, 1925.

70. Hinsberg: Results of Treatment of Purulent Meningitis, Klin. Wchnschr. **3**:1648, 1924.

meningitis by all forms of vaccine and serum therapy alone has been uniformly unsuccessful, as is shown by Barajas'[71] case.

A woman, aged 50, had a right sided ear suppuration of twenty-four hours' duration, and some pain in the corresponding side of the head. Simple antiphlogistic and antiseptic treatment was instituted, with reactivation of the general defensive forces through 0.5 cc. of the antipyogenic polyvalent Bruschetti vaccine. A comatose state suddenly supervened on the following day, deepening into absolute unconsciousness, with convulsions, conjugate deviation of the eyes and a rise of temperature. The patient when seen by Barajas presented the picture of diffuse purulent meningitis. Operation was refused. Spinal puncture yielded a purulent fluid with abundant polymorphonuclear cells and a large quantity of streptococci. The patient died a few hours later.

However, vaccine treatment combined with operation, removal of the source of infection, undoubtedly has had a favorable influence in a few reported cases of meningitis; but, as in the case of Ferretti,[72] they are open to the suspicion that the patient has suffered from meningismus only—without micro-organisms in either the spinal or cerebral fluids.

A man with acute otitis media, after seven days, presented the signs of labyrinthine involvement, with vertigo, headache and tremors. On the tenth day, he had retraction of the head and Kernig's sign. An extensive mastoid operation was performed. Spinal puncture four times daily, with evacuation of large quantities of turbid fluid (30 to 40 cc.). An autogenous vaccine was prepared and injected intramuscularly. Immediate improvement followed, and the patient was discharged in good condition at the end of a month. A recurrence took place three weeks later, but a complete cure was obtained by spinal puncture and irrigation of the mastoid cavity with surgical solution of chlorinated soda (Dakin's solution).

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71. Barajas, y de Vilches, J. M.: Hyper-acute Otogenic Meningitis, Siglo méd. **76:**345, 1925.

72. Ferretti, C.: Leptomeningitis Due to Acute Otitis, Arch. ital. di otol. **36:** 628, 1925.