

1935

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Robert A. Lovell

University of Nebraska Medical Center

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SENIOR THESIS

THE INTRACRANIAL COMPLICATIONS OF MASTOIDITIS

ROBERT A. LOVELL

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THE INTRACRANIAL COMPLICATIONS OF MASTOIDITIS

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THE INTRACRANIAL COMPLICATIONS OF MASTOIDITIS

I Introduction

In writing this paper I will attempt to review the literature available on intracranial complications of mastoiditis with emphasis on their development and diagnosis, but will disregard a discussion of their treatment.

Because of the largeness of the subject, mastoiditis as a disease will not be discussed. The intracranial complications alone will be taken up with regard to their relation to mastoiditis.

However, in speaking of the complications of mastoiditis, the writer realizes he is speaking of the complications of middle ear diseases as the former is a direct complication of the latter.

The direct object in mind in this paper is to bring out the frequency of intracranial complications following mastoiditis and the importance of recognizing their presence and early diagnosis.

It was not until recent years that the part mastoiditis plays in intracranial complications was really known and even today this field furnishes a fertile field for further work.

The serious intracranial complications of mastoiditis

with their devastating results make the general practitioner as well as the trained specialist in otology fearful when confronting these cases.

Hassler(39) compiled the intracranial complications from a total of 31,684 cases of diseases of the ear, from which number there were 116 deaths from intracranial extension, classified as follows:- Meningitis, 40; Sinus thrombosis, 48; Cerebral abcess, 28.

Komer(39) compiled the results of 115 autopsies where death was due to intracranial complications of aural disease, and found meningitis in 31 cases; sinus thrombosis in 41 cases, and brain abcess in 43 cases.

Pitts(39) reported covering nine thousand consecutive autopsies at Guy's Hospital, London, showing 67 cases wherein death was due to intracranial disease of otitic origin, or one in every 158 autopsies.

Gruber(39) investigated the findings reported on 40,073 autopsies covering deaths from all causes and found intracranial complications of aural disease as the cause in 232 cases or one in every 273.

Burkner, Randall, Dench, Phillips and others reported findings in accordance with those just given.(39)

In reading over such figures one sees sufficient reason for fear of intracranial complications due to aural disease and realizes that it is not a rare condition but rather a common one. This makes it one of great alarm and importance in regard to knowledge of development and early diagnosis.

II General Consideration of Intracranial Complications.

In a case of mastoiditis, intracranial complications should always be suspected and watched for until positively disproven or until the case is dismissed as cured. In doing this there are numerous points to be considered and watched for. These same points hold true in intracranial complications due to any cause.

In this regard, according to Coates(22), it is of prime importance to determine the gross pathological process present, whether it be sinus thrombo-phlebitis, a meningitis or a brain abscess. Upon deciding this an attempt should be made to analyze the symptoms, distinguishing those due to general toxemia, to local toxemia, to pressure and to death of tissue.

It is only possible to determine the probable path of the advance of the lesion through the dura by a combination of careful analysis and history and examination, along with visualization of pathological sequences.

The chief requisite to the diagnosis of an intracranial

complication of mastoiditis is a careful chronological history and a record of all findings from the beginning of the condition. Thus in operating a mastoid, the exact findings should be recorded as they may be of value at a later date in diagnosing a complication. The finding of a petrosal tip abscess at operation, followed later by intracranial symptoms is of diagnostic assistance.

Careful, frequent neurological examinations repeated at close intervals may result in diagnosis of an intracranial complication otherwise unrecognized.

In the presence of suppuration in the ear, the development of cerebral or toxic symptoms such as headache, vomiting, chills or irregular temperature, is presumptive evidence that an intracranial complication has arisen. Too much importance must not be attached to one symptom, but the clinical picture as a whole considered, as in the early stages all cerebral symptoms may be very vague. It is at this point that the careful chronological history and repeated examinations, with a visualization of pathological sequences is of greatest value. The symptoms and chronological sequences will be discussed more fully with the individual complications, but at this point the symptoms common to all intracranial complications will be discussed.

Pain is an important symptom which must be carefully

weighed and followed in suspected intracranial complications. Careful history as to the location, type and any changes are of utmost importance in diagnosis. The different types of pain as to location, type, severity, changes in, et cetera, will be discussed later with their respective conditions.

General malaise is frequently the only symptom during the early period of suppuration within the cranium and, in the presence of mastoid involvement, must be carefully considered. Any vomiting should be suspiciously regarded in the presence of mastoid affliction. Chills are one of the vaguest but also one of the most important symptoms in intracranial complications. They are not only often disregarded but sometimes not elicited without careful questioning. The importance and significance of this symptoms will be brought out later in the discussion of specific conditions and in the review of cases.

Temperature is not only a symptom common to intracranial complication but because of its difference in the various conditions is of great value in differential diagnosis.

It is the conclusion of most men, Babcock(1), Wechsler(50), Coates(22), and others, that a combinations of severe headache, vomiting and general malaise not warranted by the manifest process in the mastoid, is an indication of intracranial involvement, especially if preceded by a vague chill and asso-

ciated with an increased cell count on lumbar puncture.

III Sinus Thrombo-phlebitis

Hooper(20), as early as 1826, correctly recognized both sinus phlebitis and sinus thrombosis. Abercrombie followed in 1835, Bruce(20) in 1840, Virchow(20) in 1845 and Sedillot (20) in 1849. Lebert(25) in 1856 published an accurate description based on personal observations and accompanied by a citation of cases. Although we are not herein interested in therapy, it is interesting to know that Zanzal(8) in 1880 was the first to advocate internal jugular vein ligation. In 1884 he reported a case in which he opened, washed and drained the diseased sigmoid sinus but in which case death resulted because of metastatic lung abscess. In 1886, Hawkins (17), unaware of Zanzal's work, reported a spontaneous cure and also described and advised the same treatment as Zanzal.

In 1888, Lank(33) performed the operation in England and was followed by Ballance(3) in 1890, both reporting cases. At this same time, Vrerick(41) reviewed the whole subject and reported five additional cases. At this time 170 cases had been reported in the literature, in which operation had been performed.

Gifford(41) operated the first case in Nebraska and a year later, 1905, reported five cases. He was assisted and

followed by Potts(41) a few years later who reported an interesting group of cases.

At this point a brief review of the sinuses of the dura will be made. The sinuses, sixteen in number are the venous channels which carry blood from the brain. Each lies between two layers of dura and is lined by a layer of epithelial cells continuous with that lining the veins. Of these sinuses, the lateral sinuses are the largest. One, usually the right, is a continuation of the superior sagittal sinus. The other is a continuation of the straight or tentorial sinus. A communication is established between them at their origin by a small branch from the distal end of the straight sinus to the torculi Herophili.

Starting at the internal occipital protuberance and contained between the attached layers of the tentorium cerebelli, the lateral sinus runs outward, forward and downward, resting first on the inner surface of the occipital bone, then crossing the posterior inferior angle of the parietal bone, it reaches the petrous portion of the temporal bone, where it lies in a groove on the inner surface of the mastoid process. This last relationship to the mastoid process accounts for the secondary involvement of the lateral sinus in purulent otitis media with a purulent osteitis of the mastoid.

The lateral sinus can be divided anatomically into two

parts, the lateral or horizontal portion, and the descending or sigmoid portion. The anatomical landmark which divides the sinus into these divisions is the point where the horizontal portion turns downward and forward. This point is termed the knee of the lateral sinus.

The tributaries of the lateral sinus are: 1)-The superior petrosal sinus, which empties into the sigmoid portion at the base of the petrous portion of the temporal bone near its posterior end. 2)- The mastoid emissary vein, which joins it at a point slightly below the opening of the superior petrosal sinus and emerges through the mastoid foramen. 3)- The posterior condylar veins which unite with the sigmoid portion at the posterior condyloid foramen. 4)- The inferior cerebellar and cerebral veins. 5)- The petro-squamous sinus, which is inconstant, but which runs along the petrosquamous suture when present, and empties into the lateral sinus. 6)- The inferior petrosal sinus which joins the sigmoid sinus when the latter rests on the jugular process of the occipital bone. The junction of these two forms the beginning of the internal jugular vein.

Just as in all other anatomical arrangements in the human body, there is considerable variation in the position of the sinuses in different individuals. Trautman(25), Whiting(51), and Held(18) have made an interesting group of observations of these variations.

Sinus thrombo-phlebitis is the most frequent of the aural complications. Because of its frequency and seriousness, it is essential that the condition be watched for and diagnosed early. Thrombo-phlebitis of the lateral sinus may occur through several routes in acute or chronic mastoiditis. Briefly the routes of infection are:- 1)- Through dihsences of the bone tissue which cover its parietal surface, thus affording easy acces to the pathological process. 2)- Through the direct extension of the active purulent lesion in the bone to the walls of the sinus. 3)- Through involvement of the smaller veins in the diseased bone, or through the involvement of intermediate anastomotic veins in the thrombotic area.

Two factors are necessary for the production of an infect-thrombus in a vein:- 1)- a slowing down of the rate of flow of blood through the vein, and 2)- the presence of pathogenic microorganisms in the blood. In addition there must also occur a trauma to the endothelial lining of the venous channel.

Where a thrombo-phlebitis occurs by direct extension of the purulent process there is a gradual progression of the inflammatory mass toward the large veous channels. When a vessel is finally reached there is produced a comprssion of the wall by the encroaching infection, causing a narrowing of the lumen and a slowing of the blood stream. On account of the contact of the wall with the infected mass, the wall soon

becomes involved in the inflammation and becomes a part of the inflammatory process. We then have the additional element of trauma to the vessel wall and a thrombus is formed. As the process advances the wall soon becomes necrotic because of the lack of blood supply and the infection gains access to the interior of the vein. The organisms find a barrier against their invasion of the general circulation in the form of a thrombus and as a result they invade the mass. The previously sterile thrombus now becomes infected and begins to disintegrate, eventually resulting in an intravenous pus collection.

In the type of infection which extends by small thrombi in neighboring vessels into the sinus the small veins are already the seat of thrombi. These thrombi are produced in the same manner as those just mentioned in the extravascular extension.

In the sinus thrombus resulting from a tributary thrombus the two factors necessary for thrombus formation are produced in a way which differs markedly from that produced by extravascular extension, as just considered. There is already a small thrombus present in the smaller vessels of the infected focus, the mastoid. When this thrombus extends into the larger vessel, there are at once produced all the factors necessary for the formation of an infected thrombus. The blood stream is slowed by the protruding mass and the endothelial lining

is attacked by the pathogenic organisms contained in the thrombus. The infective thrombus is not produced here by a succession of factors as in the thrombus of extravenuous extension. The vessel wall does not have to undergo necrosis for the extension of the infection through its substance. Such a thrombus is more apt to grow with the blood stream and form a mural thrombus in contradistinction to the obliterating thrombus which occurs more often in the first variety.

The thrombus produced by extravenuous or direct extension is to be viewed as a protective barrier against invasion of the general circulation by pathogenic organisms. The thrombus of intravenous extension differs in that it represents the progression of infection into the large blood channels from the smaller ones. The continued enlargement of the thrombus is merely a futile attempt on the part of the body to localize the infection to that portion of the blood stream.

Because of the anatomical peculiarities of the venous sinuses of the dura, intracranial thrombus is of greater frequency than extracranial. The cranial sinuses all have at least one of their wall resting against a boney structure and they have a complete intercommunicating anasomosis.

The route of the extension depends upon, very largely, if not completely, the type of mastoiditis present. The coalescent of acute purulent type of mastoiditis usually produces

the thrombosis by extension extravenously while the hemorrhagic meningitis or fulminating septic type produces the thrombosis more often by intravenous extension.

The dangerous types of chronic purulent mastoiditis also usually result in thrombosis produced by the direct extension or extravenous extension.

Sinus thrombosis complicating mastoiditis are lateral sigmoid sinus thrombosis, primary bulb thrombosis, and cavernous sinus thrombosis. Of the three types, only the sigmoid sinus thrombosis is a direct complication of mastoiditis. The cavernous sinus thrombosis of mastoiditic origin is always the result of the extension of a sigmoid sinus thrombosis to the cavernous sinus through the petrosal sinuses. It is very rarely primarily affected and is not viewed as a complication of otitic disease.

A primary bulb thrombosis occurs independently of a sigmoid thrombosis, although the latter may follow. This condition is usually produced without the presence of a surgical mastoid. It is only mentioned here because of the conflict that arises in a clinical thrombo-phlebitis without mastoid involvement. In the subsequent development, mastoidal symptoms absent at the beginning make their appearance and little can be done. The symptomatology in sinus thrombosis naturally varies with the route and type of infection.

It is believed that the symptomatology as to route of

Kopetzky(29) reviews the symptomatology as to route of infection, while Guernot(16) groups them as general, brain symptoms, ear symptoms and findings of other organs as metastatic lung abscess, splenic abscess and positive blood culture. However, it is too late for any help when the last of these symptoms appears.

Phillips(40) feels that in the typical case fever is the most important finding. He groups his symptoms into local and general.

Fever is a constant symptom of sinus thrombosis, being rarely absent. The fever is the result of the invasion of the blood stream by bacteria. During the early stage of the attack the fever is characteristically pyemic. Usually the patient has a distinct chill, during which the temperature rises to 103 or 105 degrees, receding after a short time to normal, or below, only to rise with a subsequent chill. Vomiting may accompany the chill, but is not a constant symptom and also occurs in the other intracranial complications.

Papilloedema is rather a common finding, although it is slight and usually without hemorrhages. Headache is usually present and is located about the mastoid, parietal and occipital regions of the affected side.

- Sepsis appears later as a prominent symptom, its time of

onset and seriousness dependent upon the route of infection.

Local symptoms are the Griesinger sign of swelling behind the mastoid, and one which is generally absent. This swelling is painful to the touch, especially at the foramen of the mastoid emissary vein. This condition is marked in those thromboses which occur by way of the local blood stream, especially with thrombi of the mastoid vein.

Many other symptoms may appear such as gastric irritability, sweats following drops in temperature, petechial hemorrhages, and extreme prostration. These symptoms are variable and often confusing.

Boeminghaus(40) has laid down four cardinal points in the diagnosis of lateral sinus thrombo-phlebitis which are rather conclusive. They are: 1- When after an acute mastoiditis, in spite of adequate drainage, the fever recurs after having dropped, then we should be suspicious of a sinus thrombosis. Especially if the temperature continues to persist or run a septic course. 2- When or if a fever appears after an interval of normal temperature which has followed the procuring of adequate drainage from operation. 3- When fever suddenly appears after the infection has apparently subsided for some interval of time. 4- When in cases of chronic middle ear suppuration, with an accompanying chronic mastoiditis, there is a sudden appearance of fever, then sinus thrombosis is to be suspected.

Blood count with the characteristic leucocytosis and high polymorphonuclear percentage is a positive finding of help. The value of repeated blood cultures in all suspected cases is stressed by Todd(48), Kemler(23), Phillips(40), Kopetzky(30) and others as a very important procedure.

In typical cases the diagnosis is not difficult to make but unfortunately too few of the cases are typical. In atypical cases the diagnosis is difficult and most careful observation must be made with an exhaustive consideration of the symptoms. The problem in most of these is to eliminate other conditions as the cause of the findings. Blood culture and blood examination are most helpful in these cases. In way of prophylaxis, to enable easy treatment, all cases should be watched very carefully following operation for any early signs of thrombosis, as any changes in temperature, chills, blood findings, et cetera.

Little need be mentioned about the complications of sinus thrombo-phlebitis. During the later stages if unrelieved, the thrombus may invade the contributing branches, the torcular, the jugular bulb or vein and result in metastatic abscesses in the brain, lungs, spleen, joints, or any place in the body, with an extremely high mortality.

IV Meningitis

Meningeal involvement complicating mastoiditis is not only one of the most dreaded conditions but one of the most

frequent. Korner(25) and Hassler(40) reported that as a cause of death it rates equal with sinus thromb-phlebitis. As to its occurrence it is much greater as it accompanies many cases of thrombo-phlebitis and brain abcess as a complication.

Before considering the classification, route of development, pathology, symptoms and diagnosis, I will give a brief summary of the anatomy of the meninges.

The brain and spinal cord are covered by three membranes: 1- an external fibrous membrane termed the dura mater; 2- an internal, the piamater; 3- the arachnoid membrane which lies over the piamater. The dura lines the interior of the skull and forms a loose sheath which envelops the cord in the spinal column. The pia is composed of loose areolar, vascular tissue and closely invests the cord and brain. The arachnois, the intermediate layer or membrane, is non-vascular in nature. It is closely connected with the piamater in some places, while in others a considerable space intervenes between the two. These spaces form the cisterns of the arachnoid.

The dura is composed of fibrous and elastic tissue, traversed by blood vessels which run principally between the outer layer. It is supplied with nerve branches from the fourth, fifth, and twelfth cranial nerves, and its lymph supply is very abundant. The subdural space is in communication with the dural veins. The dura is adherent to the inner surface of the cranial bones, representing their internal periosteum.

The connection between the bone and its dural covering is dependent upon numerous blood vessels and fibrous processes which pass from the bone to the dura. The dura is loosely attached to the bone especially over vertex and closely attached at the suture lines. At the base of the skull with its irregularities and many foramina, the dura is continued through the foramina to be continuous with the pericranium, or its fibrous tissue is lost in the nerve sheaths. As previously mentioned, the dural sinuses are formed by the separation of fibrous layer of the dura.

Toward the brain and in the interstices between its component parts, there are reduplications of the dura designated as the falx cerebri, falx cerebelli, and the tentorium cerebelli.

The arachnoid situated between the dura and the piamater, is a thin delicate membrane of interlacing fibers containing a few blood vessels. In the cerebral portion it passes from one convolution to another. It bridges the sulci and in places is closely applied to the underlying pia, especially at the upper and lateral portion of the cerebrum. Where it is not adherent to the pia, a space is formed between the arachnoid and the pia known as the subarachnoid space, and containing a greater part of the cerebro-spinal fluid. This space also lodges the greater part of the blood vessels which supply the brain.

The subarachnoid space is unevenly distributed over the brain surface, in places being scarcely demonstrable and in others presenting a wide interval separating the pia from the arachnoid. The widest part of this space extends over the medulla, pons, and interpeduncular recess, as far forward as the optic nerve.

At the foramen of Magend, the subarachnoid space is in communication with the ventricles of the brain. Although there is no direct communicating channel between the subdural and subarachnoid spaces, it is believed that fluid can pass through the meshes of the arachnoid into the subdural space.

The pia mater is a delicate, highly vascular, fibrous tissue, closely investing the brain and the spinal cord. It entirely covers the cortical surface of the brain, dipping into and lining the sulci. It becomes invaginated at the lateral and third ventricles, forming the tela choroides superior and the choroid plexus, and at the fourth ventricle it forms the tela choroides inferior and choroid plexus of the fourth ventricle. The pia contains many blood vessels.

Diseases of the meninges of otitic origin are best classified in two large groups: in inflammation of the meninges of a protective type, and inflammation of the meninges of a dangerous type.(11)

In the meningitis of the protective type the inflammatory process does not involve the subarachnoid space. This type

of meningitis is often discussed and commonly known as meningitis serosa and meningitis sympatheticus which occur as the result of inflammation in the vicinity of the meninges, as in sinus thrombo-phlebitis, brain abscess and extradural abscess.

This form of meningitis is due to an increased exudation of cerebro-spinal fluid with a consequent stoppage of the pathways and is the result of an effort on the part of the meninges to prevent the infect of the subarachnoid space by the inflammatory process adjacent to it.

The second group or suppurative meningitis as it is most commonly called, consists of those cases in which there is suppuration within the meninges. These suppurations may be localized, diffuse, acute or chronic, fulminating or exudative. They result from an extension of a purulent focus of otitic origin which has gained entrance to the meningeal spaces.

Localized pachymeningitis externa, an inflammation of the parietal layer of the dura, is the most common of all infections involving the intracranial tissues secondary to mastoiditis. It occurs at all ages and is found as a complication of both acute and chronic mastoid suppurations.

The only difference in a localized pachymeningitis externa and a subdural abscess is that the latter is characterized by a localization of pus while the former is merely an inflammation.

The development of either of these conditions is through the same route as the development of a sinus thrombosis, extravenous or intravenous, and may be part of the picture of a thrombosis or complicate it.

In the cases with pus formation, those of extradural abcess, the mode of development is nearly always extravascular, by bone necrosis or eburnation. In those of pachymeningitis externa, they are generally of intravenous origin or by way of dehiscences in the bone.

In those cases of chronic mastoiditis with suppuration which develops extradural abcess, there is often a fistulous tract leading to the dura. Extradural abcesses are usually found over the cerebellar region and often take the form of a perisinus abcess. A more rare condition is petrous tip abcess. This condition is being recognized more commonly today and is not considered as rare as previously. Considerable work has been done on the problem with a resultant increased amount of literature on the subject in recent years.

Druss(10) in studying the anatomy of the mastoid process of the temporal bone in relation to pathology, found a marked, but rather consistent variation in the structure . Belinoff(10) and Balan(10) of Bulgaria studied grossly the petrous pyramids of forty consecutive cases giving particular attention to the anatomic structure and found the apex pneumatic in 35 percent, diploetic in 27.5 percent, and mixed in 42.5 percent.

Zuckerhandel(10) in studying 250 temporal bones found the mastoid process pneumatic in 36.8 percent, diploetic in 20 percent and mixed in 42.5 percent.

Comparison of figures showed the percentage of each type was practically the same for the mastoid process and the petrous pyramid or apex. On the contrary, these men found that in the study of the process and petrous pyramid of the same temporal bone, the structure varied in 62.5 percent of cases. While it is the rule for the process to be more pneumatic than the petrous pyramid, nevertheless the reverse condition sometimes exists. This anatomical variation plays a large and important role in the spread of infections to the apex.

Infections of the petrous pyramid may break through the cortex of the bone along its course and produce localized pachymeningitis externa, an extradural or subdural abcess, or a brain abcess or generalized purulent meningitis.

Taylor(47) has stressed the importance of roentgenology in diagnosing this condition and preventing fatal complications. The work of Kopetzky(28) on this condition has helped to clarify the diagnosis and will be discussed later.

The role played in sinus thrombo-phlebitis in causing a subdural abcess, extradural abcess or purulent meningitis can easily be understood from the previous discussion of that complication. Where the purulent process of an extra-

dural abcess or a pachymeningitis externa, or sinus thrombosis disintegrates the dura and penetrates into the intradural space an intradural abcess or intrameningeal abcess is formed and if the lesion is permitted to advance far enough, the brain surface will eventually become ulcerated or the brain abcess may rupture into the cerebro-spinal circulation and produce a suppurative leptomeningitis.

Leptomeningitis is a diffuse infection of the pial and arachnoid membranes and is produced by either the method just mentioned, direct extension, or by metastatic infection through the blood stream. The inflammatory products are deposited in the subarachnoid space.

Where a purulent leptomeningitis results from an otitic suppuration, the reaction of the meninges depends upon the resistance of the patient, the virulency of the organisms and the duration of the primary infection. Where the meninges have had time to develop protective barriers, infection by contiguity is more apt to result in what is termed the exudative type of purulent leptomeningitis. Here the exudate present in the subarachnoid space is the result of both bacterial activity and the protective mechanisms of the meninges in their endeavor to counteract the infection. This type often shows brief periods of recession during which time the patient appears to be on the road to recovery. Such remissions are due to the temporary overwhelming of the infection by the pro-

tective forces of the meninges. This is the type so often referred to as chronic or subacute purulent meningitis.

Labyrinthitis as a path of infection in otitic meningitis has not been discussed here but will be mentioned in regard to brain abscess. The frequency of this route of infection should be mentioned, however. Because of the frequency of a para-labyrinthitis and labyrinthitis in mastoiditis, it is a common route of infection. Coates(22) found thirty percent of otitic meningitis to be by this route, 27 percent by thrombophlebitis of a large sinus, and ten percent of thrombophlebitis of a small vein of the bone. In 27 percent of the cases the route was not known but was probably from labyrinth disease or small vein phlebitis. Some of these cases are undoubtedly due to petrositis and direct extension.

The symptomatology of meningitis may best be analyzed as to those of general toxemia, symptoms of cerebral irritability and localizing symptoms. The symptoms in the advanced case are most marked and diagnosis is thus easy but because of the importance and difficulty of early diagnosis, this point will be stressed more.

The general toxemia symptoms are rather constant and become more marked as the condition progresses. Temperature, an ever-present symptom, may be slight at first and may remain so as long as the process is limited, but with further invasion the temperature is a septic one. As the condition becomes

worse the temperature becomes continuously high. It is not characterized by any regular exacerbations or remissions except that it frequently shows a marked rise following a chill. Chills are not a constant symptom being rarely present in exudative meningitis. In the fulminating type, repeated chills with marked remissions of the temperature are rather characteristic and generally significant of blood stream infection.

The pulse, which is at first slow, soon becomes fast and follows the temperature. If the pulse rate is too rapid in proportion to the temperature, the prognosis is poor.

Vomiting is a variable symptom though usually marked when present. The vomiting is cerebral, being projectile and not preceded by nausea. Vomiting is most common in children and in infants is replaced by convulsions, an ever important symptom here.

Albuminuria and glycosuria may be prominent in septic cases but are not important in early diagnosis.

Symptoms of cerebral irritability are prominent and are present from the onset. The first symptom which arouses suspicion following mastoiditis, is restlessness and sleeplessness. These are accompanied by or followed shortly by irritability and excitability. These help to distinguish meningitis from sinus thrombo-phlebitis as in the latter a state of composed demeanor and optimistic attitude are the rule early.

Delirium is often marked and continuous and accompanied

by sharp maniacal cries, or hysterical outbursts. This symptoms also aids in differentiating from the somewhat somnolent dreamy state or mild toxemic delerium or brain abcess and the dreams associated with sinus thrombo-phlebitis.

Stiff neck and opisthotonus are usually among the early symptoms but should not be waited for to make a diagnosis and institute treatment. They are due to irritations of sensory and motors nerves as they leave the base of the skull and are present only when the base of the skull is involved. In meningitis limited to the cortex, these symptoms are not present.

Suboccipital pain and general spinal tenderness is generally present as soon as the process has become diffuse. Kernig's sign is also a symptom of basilar involvement and is one of the earliest menifestations.

The pain in the head, usually frontal or occipital, is agonizing as a rule and usually is associated with the sharp cry mentioned.

Pain above the temple or limited to one of the branches of the trifacial nerve, especially when deep in the eye of the affected side, should be investigated for petrousitis of the same side with a localized meningitis of the floor of the middle fossa. This pain is constant, usually beginning as a nocturnal pain and following a quiescent period of five to twenty days. Other constant and diagnostic findings here are sixth nerve involvement on the same side with external rectus para-

lysis, the so-called Gradenigo symptom-complex.(50)

Localizing symptoms, although of little importance in diagnosis because of their late appearance, are often helpful in atypical cases, especially those of a subacute or chronic nature.

Anosmia, although rarely observed, is an early sign of basal involvement and should be continuously sought for.

Ophthalmological findings are absent early but examinations should be made repeatedly to note any change. There may be a slight dilatation of the veins and any indication of early obstructive internal hydrocephalus will be noted in the progressive papilloedema.

Unequal pupils are frequent in the meningites but early in the course they react to light and accommodation. Laziness of reaction is an important early sign, however. A widely detailed non-responsive pupil is indicative of involvement of the intrameningeal portion of the nerve from exudate in the basal cisterns.

Motor ocular paralyses occur only in the late stages of the disease, although the third is frequently involved early and the sixth in cases of petrositis. With the seventh and eighth nerve involvements, facial paralysis and total deafness, due to effusion in the internal auditory meatus, are often terminal symptoms.

Because of the importance of early diagnosis of meningitis and its frequent occurrence complicating mastoiditis, all

such cases should be followed very carefully.

The important early symptoms, irritability, restlessness, insomnia, headache and neck pain should be watched for. With the appearance of any early symptoms, the most important aids to diagnosis should be used, blood examination, blood culture and spinal fluid examination.

Blood culture is very important as it often shows the presence of organisms before the spinal fluid is altered. Repeated blood cultures should be made upon the least suspicion. The blood examination shows a fairly high leucocytosis of from 25,000 to 40,000 and is very helpful aid. Here again repeated examinations should be made as the leucocyte rise is rapid and the normal count may be followed very shortly by a most rapid rise.

Lumbar puncture has been the most important diagnostic aid of all contributions and should be performed at the least suspicion. Although when performed early, nothing may be found, it is very worth while, especially if confirmatory negatives are found on blood culture and blood examination. Lumbar puncture fluid should be examined carefully macroscopically, chemically and microscopically. It should be observed for pressure, color, cloudiness, and consistency.

Positive findings of increased pressure, pleocytosis, increased protein, decreased sugar and bacteria give a positive diagnosis of meningitis. The finding of a marked pleocytosis in itself, with no other findings is very important,

as it is indicative of neighboring pathology without actual development in the meninges. Repeated cultures in this instance are important to notice any change and help govern the treatment.

V Brain Abscess

A brain abscess is a localized, intradural collection of pus, usually referred to by its anatomical location as frontal, temporo-sphenoidal, or cerebellar abscess.

Hippocrates(25) recognized the coincidental occurrence of ear disease and brain abscess. At a much later date, Morgagni apparently recognized the fact that ear disease stood in casual relationship to brain abscess. In 1856 Lebert,(25) as the result of independent investigation, fully established the etiological role played by ear suppuration in the production of brain abscesses.

Successful surgical treatment of brain abscess, with recovery was first recorded by Morand(25) in 1768 and second not until eighty years later by Roux(25). The middle of the nineteenth century marked the introduction of the surgical treatment of brain abscess. An added impetus was given to the field of otitic brain surgery abroad by the work of von Bergmann(25) and Korner(25) and in this country by Bacon(25), Burnett(25), Knapp(25), Phillips(40) and Lederman(40).

Intracranial abscesses occur most frequently in persons between the ages of ten and thirty years.(40) According to most authors, cerebellar abscess is not as common as abscess in

the temporo-sphenoidal lobe.(5) Koerner(5) found 69 cases of temporo-sphenoidal lobe abcess to thirty of cerebellar. Growers(5) found 186 temporo-sphenoidal lobe abcesses to 41 cerebellar. Le Fort(5) and Lehman(5) found 327 temporo-sphenoidal lobe abcesses to 113 cerebellar. Hunter Todd(40) found in 100 cases of intracranial abcesses among children under ten years of age that the temporo-sphenoidal abcesses occurred in 87 percent and cerebellar abcess only in thirteen percent.of cases. Among adults on the other hand, he reports cerebellar abcess in 65 percent and temporo-sphenoidal in 35 percent of cases.

Aural suppuration, whether acute or chronic, is potentially capable of producing a localized collection of pus within the brain substance by virtue of its close proximity to the endo cranial structure. This mode of extension, as discussed in sinus thrombo-phlebitis, is one of direct extension from the ear spaces to the brain. It necessitates the destruction of some portion of the inner table by the erosion action of the disease in the mastoid process.

Brain abcesses of mastoiditic origin can also be caused through the medium of the blood stream in which instances it is more often and extension of an osteo-thrombotic phlebitis to the veins of the pia or cerebrum, with the formation of an obliterating thrombus.(Kopetzky)(26)

Brain abcess following chronic otitic suppuration are t in the majority of instances the result of destructive action

of a cholesteatoma and a direct extension of the infection to the brain tissues. Since the necrotic process of long standing attacks the mastoid process most commonly, the pneumatic spaces disintegrate in the lines of least resistance. The mastoid cortex being the most usual sight of eburnation, the line of least resistance is toward the cranium. An acute infection of the mastoid process interposed on a chronic middle ear suppuration thereforr progresses toward the cranium.

Brain abcesses are further calssified as acute and chronic. This classification is a pathological, and not a clinical one, and is based on the presence or absence of a limiting capsule. When the abcess formation has occurred rapidly and the brain tissue has been unable to set up protective barriers against further destruction, no limiting membrane is demonstrable and the abcess is termed acute. These are usually the result of metastatic infection, though thrombo-phlebitis, but are occasionally produced by direct extension.

The chronic brain abcess is one wherein a definite limiting capsule is present. This capsule is the result of the activity of the cerebral protective mechanisms. There first occurs an increased fibroblastic proliferation, eventually resulting in the formation of new connective tissue which tends to limit the progress of the abcess and is the reason for the so-dalled latent intervals so characteristic of the type. The micro-organisms present in the chronic abcess are not so virulent as in the acute. The chronic abcess is commonly

the result of infection by direct extension.

As previously reviewed, the temporo-sphenoidal lobe is the most frequent site of the complicating abscess, in approximately 65 percent of the cases. The common occurrence of location here and in the cerebellum is due to the frequency of infection by direct extension. Where the abscess is the result of metastatic infection, the location may be in any part of the brain.

The symptoms of brain abscess may be divided into those which are due to increase intracranial pressure or the so-called general symptoms, and those which are the result of injury to the brain substance or focal symptoms.

The general symptoms of brain abscess may best be divided into those due to pus retention and those which are the result of pressure on the brain. The symptoms due to pus retention are headache, chills, vomiting, subnormal temperature and evidence of protective meningitis in the cerebro-spinal fluid.

During the initial stage, that of invasion, the symptoms of the abscess formation are obscured by the disease within the ear. On close questioning however, one is able to elicit a history of either a chill or chilly sensations. This is generally overlooked at the time of occurrence because of the predominance of aural symptoms. When, however, suspicion is aroused of the presence of brain abscess, it is advisable to inquire as to the time and occurrence of the chill, as one is — thus able to determine the approximate duration of the disease.

Headache is present in all cases of brain abscess. The severity of the pain may vary to a great extent but the patient will usually complain of the presence of a constant headache throughout the development of the abscess. The persistence of the headache over a considerable time following a mastoid suppuration should arouse the suspicion of some intracerebral suppuration. Because of the frequency of this symptom on various and numerous conditions it is too often disregarded. Every effort should be made in these suspected cases, in fact in all cases, to trace the cause of the headache and to exclude an intracranial condition at least. More attention to this symptom will lead to the earlier discovery and relief of a brain abscess which may have been overlooked for the want of the so-called classical picture.(28)

The vomiting which occurs during the early stages of the development is a manifestation of cerebral suppuration. It is not the characteristic projectile vomiting seen later in the course of the disease and meningitis due to sudden increase in intracranial pressure. It is more apt to be sporadic in its appearance and associated with vague headaches.

The finding of a subnormal temperature which continues over a period of time is, in the presence of the other signs mentioned and with previous evidences of possible development, a definite indication of the presence of a brain abscess. Grile(25) considers that the brain tissue has an internal

secretion which acts in conjunction with the other endocrines in controlling the temperature. According to his theory, any destruction of brain tissue would upset the normal balance. It is advisable in suspected cases and to prevent a missed diagnosis to keep careful temperature records on post-mastoiditis cases where there is any suspicion of intracranial complication. The finding of a more or less constant subnormal temperature is definite evidence of brain abscess.(13)

As mentioned in the discussion of meningitis, there is present in the brain abscess developing from the extension of a purulent otitic focus, a protective meningitis early in the disease. Examination of the spinal fluid at this time will give the findings of a protective meningitis, but if examined after localization, the fluid may appear normal. Later when the abscess again approaches the meningeal covering, the evidence of a protective meningitis reappears. This shows the importance of repeated spinal fluid study.

In the early stages of the extension abscess, the spinal fluid will be found to contain a predominance of polymorphonuclear cells. Later, as the abscess becomes localized, the mononuclear lymphocytes make their appearance in large numbers, acting as removers of the detritus in the subarachnoid space. Bacteria are never demonstrable in the spinal fluid during the protective stage of the meningitis associated with the formation of a brain abscess. When present, they are indicative of either a superimposed suppurative meningitis or a rup-

ture of the abcess cavity into the general cerebro-spinal circulation.

The symptoms of increased pressure are slowing of the pulse rate, projectile vomiting, change in the blood pressure, ocular muscle palsies, coma and choked disc. The appearance of these signs is always an indication of a progression of the abcess and diagnosis is best made before their appearance.

A pulse rate which is distinctly retarded over a prolonged period of time is definite evidence of the progress of the abcess. Schwartz(44) speaks of cases where the rate dropped to 44 and to ten to fifteen beats to the minute. According to Koch(44), the pulse rate is not dependent upon the size of the abcess, since he has not seen cases in which the pulse remained slow after the evacuation of the abcess. Other men, notably Macewin(37), feel that the pulse rate decreases as the abcess increases in size. In some cases the slowing of the pulse is observed for a few days, the rate rapidly returning to normal and at times becoming accelerated. This may be due to an accompanying meningitis.

Projectile vomiting is more often a symptom of brain tumors and meningitis than of brain abcess. When present it is of the usual type, not associated with gastric disturbances or nausea. Cerebellar abcesses are more prone to be associated with projectile vomiting due to the pressure exerted on the brain stem with irritation of the vomiting center in the dorsal

nucleus of the vagus.(14) With the acute abscesses there is generally associated a marked rise in blood pressure because of the circulatory efforts to overcome the sudden obstruction to the free circulation of blood within the cranium. In the chronic type of abscess, wherein the increase in intracranial tension is gradual, there is no such characteristic rise in pressure. Nature has had time to compensate for the resistance offered. When such rapid rise does occur in the chronic type of abscess, it is always indicative of a sudden increase in the size of the abscess or of a rupture of the abscess into the cerebro-spinal fluid.

Where the abscess cavity has reached considerable size, and pressure is exerted upon the brain stem, ocular muscle palsies may result. The nerves most commonly affected are the third and sixth. These palsies are by no means characteristic of brain abscess, but they may result from the cerebral compression.

In discussing the coma of brain abscess, it is well to outline briefly the prodromal symptoms which are due to the derangement of cerebral function by the pressure of the abscess and which in a way are forerunners of the comatose state. The first manifestation of compression of the cerebral tissue is a clouding of the patient's intellect. The abnormal deviations are scarcely noticeable at first, but gradually there appears increasing drowsiness, changing at times to extreme restlessness.

The response to stimuli is markedly retarded, being especially noted in the patient's response to question because of slow cerebration and delayed speech. The memory is gradually impaired. As the disease progresses the patient may be depressed at times and at others, irritable. Delerium is a frequent forerunner of coma, and the patient may become maniacal, necessitating forceful restraint. Toward the terminal stages of the disease, coma develops as a result of the marked increase in the cerebral compression and consequent interference with the cerebral functions.

The causes of papilledema of brain abscess and the stress placed upon its importance is not founded upon, or confirmed by, physiological reasoning. The optic nerve is enclosed in a prolongation of the sheaths of the dura and the sheath contains fluid which is in direct communication with the cerebrospinal fluid. The most common causes of papilledema are interference with the return flow of blood from the cranium and an increase in the amount of fluid in the subarachnoid space or to an interference of the free flow of fluid. In the acute type of abscess there there is apt to be a sudden increase in the intracranial tension with a consequent obstruction of the venous and arterial blood supply within the cranium, the presence of a papilledema is of rare occurrence. The obstruction to the venous return is compensated for by the obstruction to the arterial supply and the optic nerve head is therefore not

affected.

In the chronic type of abscess, wherein the signs of increase intracranial pressure are absent, the eye grounds will be normal. However, when the abscess reaches a size large enough to impede the free circulation of cerebro-spinal fluid, or when it is so situated as to cause a blocking of the spinal fluid, the fundic changes will be observed. The severity of these changes depends on the amount of increased tension and may vary from a mild papilledema to a marked choked disc. Marked choked disc is seldom found in brain abscess complicating mastoiditis and when found in this complication is a grave sign. It is indicative of a beginning meningitis or an acute internal hydrocephalus due to a complete blocking of the ventricles by pressure or of a rupture of the abscess into the ventricles.

The time of the appearance of the papilledema varies to a great extent with the location and character of the abscess(5). Those within the cerebellum are apt to be associated with papilledema earlier in the course of the disease than abscess located within the temporo-sphenoidal lobe. This is due to the proximity of the pus pocket to the foramina in the fourth ventricle, with a consequent blocking of the ventricles by pressure. Abscesses of the temporo-sphenoidal lobe rarely show the presence of papilledema, except late in the disease, on account of the location of the lesion. When the abscess reaches the

stage where its size is sufficient to obstruct the spinal fluid circulation by pressure, the optic nerve head may then show signs of the increase in pressure. If the abscess has caused a secondary protective meningitis, which is so often the case, the nerver may be the seat of changes due to neuritis caused by the irritant present in the cerebro-spinal fluid. This is, however, an optic neuritis and not a real papilledema.

While the eye grounds should be constatnly watched for the appearance of papilledema, the observer should not plave too much reliance upon this sign as the determining factor of the presence or absence of intracerebral suppuration.(25) The other symptoms are of greater diagnostic aid. Papilledema, when found, is to be regarded as a sign of impending danger, and immediate remedial measures msut be instituted for the relief of the cause.

In discussing local symptoms of temporo-sphenoidal abcess, it is to be remembered that the temporo-sphenoidal lobe is one of the areas of the brain which have been designated as silent areas. As a result, very often local symptoms are absent and the diagnsois must be made from the general symptoms. and the findings at the mastoid operation.

The local symptoms of temporo-sphenoidal abcess are of two kinds: those due to interference with the function of the temporal lobe and those due to pressure upon adjacent structures.

The former, when present, are definitely diagnostic in the presence of the general signs of brain abcess, They consist of an aphasia, hemianopsia and partial deafness. Aphasia is more apt to be present when the left side is the seat of the lesion in a right-handed person. It is difficult however to detect the presence of aphasia even when present as the sensorium is frequently so clouded that one is unable to differentiate between real aphasia and that produced by the impaired intellect. When present, it is more apt to be of the sensory type, namely, word deafness. The inability to name objects is likewise observed as a feature of the sensory aphasia.

In the examination of the eyes, a definite search should be made for the presence of hemianopsia as it is of great diagnostic aid in localizing the lesion in the temporal-sphenoidal lobe.(14) It is produced by the involvement of the tract which runs from the optic center in the cuneus through the temoro-shenoidal lobe to the geniculate bodies. The hemianopsia is always of the homonymous type, that is, the opposite sides of both retinae are affected, there being loss of either the right or left field of vision, depending on the side of cerbral involvement.

Because the center of hearing is localized within the temporo-sphenoidal lobes, there will be a loss of hearing.

— This loss is not total as the auditory function is controlled

by both hemispheres resulting in bilateral innervation. It is difficult to determine the amount of loss unless a record of the hearing has been taken previous to the onset of the disease.

The symptoms due to pressure exerted by the abcess are of two varieties: the motor symptoms, which include the palsies produced by pressure on the motor area or internalcapsule, and the sensory symptoms, which are caused by an involvement of the sensory fibers of the internal capsule. Usually the first sign of motor involvement is the appearance of a facial paralysis on the side opposite the lesion. This may be at first manifested as a weakness of the muscles of one side of the face. With the increase in pressure on the motor area, the paralysis becomes more evident. The facial palsy is of the supranuclear type which involves only the lower two-thirds of the face in contradistinction to the peripheral type which involves the whole face. This differentiating point is important in determining the origin of the facial palsy in cases where mastoid surgery has previously been done.

As the abcess increases in size and more pressure is exerted upon the motor area, a weakness of the upper extremity will be noticed on the same side as the facial paralysis of the spastic type, which later becomes complete. By degrees one notices the involvement of the lower extremity and if the disease is permitted to proceed unchecked, complete hemiplegia of the side opposite the lesion develops.

These findings are significant in localizing brain abcess in the temporo-sphenoidal lobe in a broad sense, but for more exact location of the focus within the lobe a thorough understanding of the functions of the temporal lobe is necessary.

The temporal lobe has three different types of cortex: the cortex embraced by the two transverse gyri of Heschel, the cortex of the superior temporal convolutions and the cortex which invests the remainder of the temporal lobe from the angular gyrus above to the pole of the temporal lobe.

The function of the transverse gyri of Heschel is the primary reception of sound stimuli. Here auditory impulses are made known to consciousness. Destruction of the region, if unilateral, will result in the diminution of the acuity of hearing.

The function of the superior convolution is in all probability the understanding of words. A lesion of this portion will result in word deafness.

The third type of cortex functions from the association of the auditory impulses with the visual centers. It enables various sounds to be identified with certain definite objects, and also acts as the supervisor over the use of words when speaking. An abcess of this area will cause a disassociation of the auditory impulses with the visual and speech centers. The patient will hear words but be unable to understand them. or repeat them. He will also be able to see objects but will be

unable to name them. These symptoms are very important in the diagnosis of the area of the temporo-sphenoidal lobe involved and in determining thus the area to be explored.

In discussing local symptoms of cerebellar abcess it is to be remembered that the cerebellum can be the site of a large lesion without showing the least sign of cerebellar involvement. The manifestations of cerebellar disease are never complete until the terminal stages. In arriving at the diagnosis of cerebellar abcess during the early stages of the disease, one must depend upon the location of the causal lesion in the temporal bone and the general signs of brain abcess. Most complete observation of the case and careful repeated neurological examinations from day to day should be made to note and estimate any finding as to change and significance. The signs of cerebellar involvement are asynergia major and minor, nystagmus and vertigo.

When fully developed asynergia major manifests itself by the characteristic ataxic gait, a positive Romberg's sign and spontaneous past pointing.(5)

Asynergia minor is demonstrated by the presence of adiadochocinesis, a coarse, irregular tremor, difficult movements and difficulty in mastication.

The nystagmus caused by cerebellar disease is due to interference with the vestibular mechanism and is apt to be irregular, appearing when the patient looks in all directions. Very

often a vertical nystagmus is observed as an early sign in cerebellar abcess and its presence is apt to be transient.

Speech is often disturbed, slow and interrupted, the so-called asynergic speech. It differs from the aphasia noted in temporo-sphenoidal abcess.

Vertigo is always present in cerebellar abcess and is usually from the affected side toward the sound side, being just opposite from any vertigo which may appear in extra-cerebellar tumors.

Dysmetria is also noted in fully developed abcesses of the cerebellum and is due to a disturbance of the patients' judgement of distance and the time and force of muscular movements. It is best demonstrated by the heel to knee test nad asking of the patient to grasp an object.

In the search for signs of cerebellar involvement, the functional tests of the vestibular apparatus are of distinct value. Too much reliance must not be placed on the findings elicited from the tests of the vestibular apparatus on one examination. They are of value only before signs of pressure have developed as the appearance of the latter cloud the picture presented by the functional tests.

The abnormal past-pointing following labyrinthine stimulation in cases of cerebellar abcess is almost always transient. It may be present at one examination and absent at the next, only to reappear during subsequent tests. The

bizarre character of the reaction is due to what Eagleston(14) terms the establishment of compensation on the part of the cerebellum. The slow growth of the abcess permits the cerebellum to readjust itself to the decrease in size of the intracranial space. Because of the advisability of arriving at an early diagnosis in cerebellar abcess, as in all other intracranial complications, it is best not to wait for these most manifest symptoms to develop.

In addition to the early general symptoms of brain abcess, a cerebellar abcess should be suspected when the following conditions are present:

1)- When slight spontaneous cerebellar signs are present which upon repeated examination are apt to show variations as to intensity and character. These may be a nystagmus which comes and goes, spontaneous past-pointing which is present one day and absent the next, or a temporary falling reaction.

2)- When the lesion in the temporal bone is so situated that the route of progression naturally leads toward the cerebellum.

3)- The presence of variable findings on repeated labyrinthine tests.

4)- Frequent attacks of projectile vomiting, which speak more for cerebellar abcess than for a temporo-sphenoidal abcess.

5)- When there is a distinct localization of the headache to the occipital region.

VI Conclusions

1. Intracranial suppuration complicating mastoiditis is more frequent than is generally thought.

2. Early diagnosis is most important in intracranial complications.

3. The pathways for intracranial complications are extra-venous, direct, or intravenous.

4. Visualization of the possibilities, route of spread and pathology is most important in early diagnosis.

5. Most careful chronological histories are most important in all ear disease.

6. Careful observation of all mastoid cases with repeated examination is essential.

7. The value of blood culture, and spinal tap can not be overemphasized for its importance in early diagnosis of suspected intracranial lesions.

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