Brain abscess

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BRAIN ABSCESS

BY

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SENIOR THESIS
1935

UNIVERSITY OF NEBRASKA COLLEGE OF MEDICINE
OMAHA
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BRAIN ABSCESS

INTRODUCTION

Of all of the pathological entities, brain abscess is perhaps one of the most intriguing and possibly the most difficult to solve from a clinical standpoint. We are here dealing with an involvement of the most highly specialized body tissue with destruction and subsequent damage to the body welfare. Possibly the worst feature is the fact that actual nerve tissue will not regenerate, so as a rule there must be some residual left.

It is a relatively new subject, not from the standpoint of recognition, for it was recognized years ago, but from that of early symptoms, early diagnosis and treatment. It is still confused with meningitis, encephalitis, sinus thrombosis and acute anterior poliomyelitis. Each individual case seems to present a set of different symptoms even with the abscess located in the same areas.

This thesis has been handled from the points of historical interest, types of abscesses, location, symptoms and diagnosis. Mortality figures have been quoted from cases found in the literature. It has not dealt with the treatment, as this in itself would constitute a thesis.

HISTORICAL EVENTS

Brain abscesses, their etiology and clinical
symptoms, have not been clearly known for many years. A search through the literature does not show a case report linked with the clinical findings earlier than 1776. The records of this case were more or less vague. It was not until the year 1800 that diagnosis was made of this condition before post mortem. A very excellent and vivid description of this case taken from the Lancet (1) which was quoted from a Middlesex hospital report of March, 1825, was as follows: "B. J., a carpenter, age 31, was admitted to the hospital March 6, 1825, complaining of (1) tingling pain in the sole of the right foot accompanied by numbness of the leg and thigh; (2) convulsive action of all the muscles lasting for about ten minutes, (3) on the day following, convulsive paroxysms of the right arm."

"Examination showed right leg and thigh deprived of all powers of motion, the temperature less than the opposite, no loss of sensation. The patient was perfectly rational giving a circumstantial account of his disease and with no loss of memory. He stated that whilst employed in building about 15 years since, he fell from a considerable height upon his head and from his account laboured under symptoms of concussion of the brain.

"At this time he has no pain in the head although he has been subject to headaches. He is a man
of very temperate habits, but any occasional indulgence in stimuli would affect the brain so much as to cause a temporary insanity. His pulse is regular, tongue clean.

"8th. Had two paroxysmal attacks yesterday and one of long duration in the evening. There is no loss of consciousness.

"9th. Two paroxysms yesterday; these are less violent. The right arm is becoming paralytic. A diffused dark-coloured inflammation is appearing upon the right leg.

"10th. Patient is now hemiplegic but rational.

"13th. Sensorium affected, starts suddenly when spoken to, answers questions in a sharp, petulant manner, disposed to sleep.

"15th. Lies in a state of stupor; pulse slow. The sore on the leg appears gangrenous.

17th. Breathing anxious, pupils not obedient to light, laboring under symptoms of effusion in the brain, has remained the same for four days. Death on the 25th.

"Post mortem. No fracture line noted. On lifting the dura, large quantities of greenish coloured pus was poured out of the posterior part of cerebrum, the right hemisphere. The cerebellum appeared free from disease".
This description impresses one as being exceedingly good in describing the symptoms present. These medical men had quite a good understanding of the nervous system and its affliction. Previous to 1800 abscess was not generally recognized.

Wernicke's Lerbuche d. Gehunkiankheiten (2) in 1883 outlined a division which was ultimately to become marks of infectious and non-infectious abscesses.

During 1883 another London physician reported an abscess at post mortem and remarked to the effect that an abscess was an occurrence of once in the course of a lifetime. This report was observed in Lancet, London, May 1883, by William Macewen, a Scottish surgeon, who did not quite agree to the statement and who decided that brain abscess was far more common than suspected. Macewen's studies in 1893 gave a great impetus to the understanding of the ports of entrance to the brain. His text, "Pyogenic Infectious Diseases of the Brain and Spinal Cord" (3), is a very well written book and probably opens the largest and newest channels to the conception of this pathology.

Korner's studies (1902 - 1908) have set in clear relief the importance of aural disease. Brissaud and Soquer, in Bouchard and Brissaud's "Traite de Medicine" in 1904 also bring out the importance of trauma and aural disease primary to abscess. Southard in
Osler's "Textbook of Medicine" (1915) (4), Lewandowsky in his "Handbuch der Neurologie" (1912) (5) have both improved on symptoms of brain abscess. Warrington (6) brings out some interesting work on early diagnosis and treatment. The study of this disease and its relation to primary origin with consideration of treatment, seemed to have had a good start during Dr. Macewen's time. At the present time there are a number of renowned English, French and American surgeons and neurologists who now have fit methods of diagnosis and treatment and are putting the mortality rate far below the recovery percentage.

ETIOLOGICAL SURVEY

Abscess of the brain is secondary to a primary focus elsewhere in the body. In general, conditions which may lead to brain abscess are direct extensions of trauma to head, infective conditions of the ear, eye, nose and its accessory sinuses, and infection of the face and scalp. Metastatic abscesses may develop from endocarditis, osteomyelitis, bronchiectasis, abscess of lung, pneumonia, empyema, streptothrix origin, puerperal infection, liver abscess and cerebro-spinal meningitis. Septic diseases of the ear are the most common causes of brain abscess and are responsible for 33 to 50 per cent of the cases. Chronic aural diseases cause more brain abscesses than acute conditions. Otitis
disease may extend into the brain along thrombosed vessels or lymphatics. Sequestra in the temporal bone may be regarded as exciting causes (7).

Trauma of the head is a rather frequent cause. The basis for this is trauma to the part causing lowering of tissue resistance plus a primary focus in the body. Trauma probably plays a more important part in the production of frontal lobe abscess than abscess in any other part of the brain (1). Trauma followed by increased intracranial pressure due to edema suggests the possibility of abscess. The brain is attached to the dura by blood vessels and nerves, the dura to the bone by fibrous connective tissue. Direct or contre-coup violence may produce tearing of the vessels with hemorrhage and edema, and with this sort of pathology laid down plus lowered resistance, a potential abscess is possible.

Other anatomical and physiological factors favoring less abscesses in this area are the facts that the nasal and paranasal sinuses are protective in nature and guard against infection extending intracranially. If this barrier is broken down, then a pachymeningitis externa or a subdural pachymeningitis interna may be started, but both of these must be present before an abscess develops (8). Turner attaches most importance to this route of infection for intracranial complication (9),
but Eagleton does not regard it in an important light. One of the physiological peculiarities favoring abscess is the fact the frontal lobe is known as the "silent area", giving few symptoms. If the abscess is large, rupture into the ventricles is rapidly followed by death. In the Cumulative Index 1900 - 1934 the larger percentage of frontal lobe abscesses went to post mortem before diagnosis accurately was made (10).

Abscess of the middle fossa, secondary to otitic origin is the most common. The usual sites are in the pia-arachnoid directly above the tegmen of either the antrum or middle ear or in the substance of the temporosphenoidal lobe. As thrombosis of the lateral sinus is often the immediate mode of transmission to distant portions of the cerebellum, away from the petrous, so thrombosis of the superior petrosal is in rare cases the transmitting agent of the temporosphenoidal lobe.

In a number of post mortem analyses, it has been found that an abscess primarily situated in the pia-arachnoid will remain localized but by pressure effect may cause brain necrosis at this area, but never resulting in actual abscess. An extension abscess or a so-called temporosphenoidal abscess with a "stalk" is located in the second convolution of the temporosphenoidal lobe, directly above the tegmen (11).
Another group which must be differentiated from the adjacent type already discussed are the metastatic abscesses, purely hematogenous in origin. An adjacent abscess is adjacent to the primary focus, such as extension from the middle ear into the cerebral tissue (10). An infected embolic particle circulating in the blood stream with later occlusion of the vessel with tissue death is the beginning of an abscess. A metastatic affair may be so sudden as to produce apoplectiform symptoms whereas adjacent abscesses are always slow in producing symptoms (7). Abscesses of metastatic origin from the ear are not common. Eagleton reports only one case. It is believed that about 12 per cent are due to suppurative disease elsewhere. Two cases with primary origin of amebic dysentery of the liver have been reported.

In cerebellar abscess the larger number of them originated from aural infection. In 117 post mortems (12), 99 were of otitic origin, 2 from sphenoid sinus suppuration, 2 from metastasis, 2 traumatic and 2 tuberculous, 1 carcinoma and 1 syphilis. Acute exacerbation in chronic suppuration is generally the immediate cause while trauma during mastoidectomy may also be a factor.

The accompanying chart is worked out on the principle of metastatic and direct extension forms.
SCHEME OF THE PRIMARY FOCUS, THE INITIAL PATHWAYS
OF EXTENSION, THE SUBSEQUENT PROGRESS OF THE INFECTION AND
THE RESULTANT (27)

Subsequent Process of Extension and the Result
Of a series of 120 cases recorded (Cumulative Index 1920 - 1930) the predominant organism in 66 was of the staphylococcus group, 30 of the streptococcus class, 4 of the influenza bacillus, 14 of the pneumococcus types, 3 of meningococcus and 2 of Endamoeba Histolytica.

The following table shows the route of invasion to the cerebellum in 125 cases (26):

<table>
<thead>
<tr>
<th>ROUTES OF INVASION TO CEREBELLUM</th>
<th>No. of cases</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Labyrinth</td>
<td>19</td>
<td></td>
</tr>
<tr>
<td>Semicircular Canals</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Vestibular aqueduct</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>Retrograde thrombosis</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>from empyema of the vestibular aqueduct</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Ductus endolymphaticus or sacculus endolymphaticus</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Internal auditory meatus</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>Facial nerve</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Subarcuate hiatus</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Fold passing from dura to mastoid cells, probably subarcuate hiatus</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Undetermined, but thought to be by &quot;pre-formed way&quot;</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Caries of Petrous Bone (There can be no doubt that many of these were labyrinth cases)</td>
<td>22</td>
<td>17.6</td>
</tr>
</tbody>
</table>
Sinus Thrombosis
(Associated sinus thrombosis and labyrinthitis.)

Of 97, the total number of cases of infection from the labyrinth and sinus thrombosis, 13 (or 13.3%) had both labyrinthitis and sinus thrombosis; showing the intimate association of the blood stream infection with cerebellar abscess. Cerebellar abscess with labyrinthitis need not necessarily be situated in the anterior 1/3 of the cerebellum, as an associated sinus thrombosis may be the immediate origin of the abscess.

Necrosis of Mastoid 3 2.4
Metastatic 1 0.8
(From Pulmonary Suppuration)

Unclassified
Fistula of dura from extradural abscess, probably perisinus from description 1
Fistula of neck 1 2 1.6

125 100.0

HISTOPATHOLOGY IN CEREBRAL TISSUE

Considered on a histological basis, cerebral tissue is composed of neurons, the functioning cells of the brain and the most highly specialized tissue in the body. Lying between the neurons is the supporting tissue of the brain, the neuroglia, glial cells and the fibres. The neuroglia plays an entirely inactive part in the nervous mechanism in that along with the small amount of connective tissue from the pia and alongside
the blood channels it forms a framework in which the nerve cells functionate.

Under pathological conditions certain cells of the neuroglia take on ameboid activity and are instrumental in the removal of waste products of the brain.

The more highly specialized the cell, the more it depends upon nutrition for its life, and the less able it is to regenerate itself. Nerve cells thus die easily and do not regenerate; glial tissue itself, not brain tissue proper, regenerates. This regeneration, however, plays no active part in restoration of nerve function. Thus in any pathological condition which is not relieved at an early date there is a proliferation of glial tissue which replaces the active neurologic tissue.

PATHOLOGY OF CHRONIC BRAIN ABSCESSE

The pathological process in chronic brain abscess is the same as in abscess formation in other parts of the body; that is tissue death and changes due to the tissues' protective reaction, the latter being governed by the special tissue involved.

A microscopic examination of a chronic brain abscess with a capsule shows it to be composed of: (1) a varying sized cavity filled with pus and detritus, the final result of complete death of tissue from bacterial action and nutritional disturbances, (2) a
limiting capsule formed by the irritation of the bacteria and their toxins on the tissues and the tissue's protective and reconstructive action. The capsule may be divided into 3 zones, these three being hardly distinguishable, the destructive bacterial action having predominance nearest the cavity while in the external zones the tissue's reconstructive and protective powers are chiefly manifest, although far beyond the capsule bacterial action can always be demonstrated.

External to the cavity and projecting into it is the "necrotic zone" in which the tissue now dead is undergoing liquefaction by the toxins. Merging into the necrotic zone is the granulation tissue zone with connective tissue developing from it. External to this there is a round cell infiltration in the perivascular sheath of the vessels, showing that the inflammatory or the protective process is still active well beyond the capsule.

The capsule of connective tissue is the result of two factors; the virulence of the infection and the resistance of the tissues. By a high grade resistance and low virulence nature attempts a complete encapsulation of the abscess by a wall of new connective tissue, which grows denser with time (13).

The connective tissue reaction of the body is simply the attempt of the organism to limit the extent
of the inflammatory process. In most parts of the body a low virulent infection is usually encapsulated by connective tissue because connective tissue is easily available, is a low order of cell and proliferates rapidly. In the brain, however, the tissue is so highly specialized, so compact, that connective tissue formation takes a longer time; also because practically all of the new cells making up the capsule must be brought to it by the newly formed blood vessels. These blood vessels consist at first of only an endothelial covering, the connective tissue later arising from the endothelial lining to invade the brain substance. The glial cells being a highly specialized connective tissue play little or no part in the encapsulation.

Microscopically the formation of connective tissue can be demonstrated in a short period; the development of a capsule firm enough to be appreciable macroscopically is a slow process. Some clinical observers contend that a capsule is not formed until between the third and eighth week, others contend it may form as early as the beginning of the third week.

Intralabyrinthine suppuration, of which there are four types, may be followed by cerebellar suppuration: (a) caries of the compact bony walls of the semicircular canals associated with labyrinthitis with granulation tissue replacement of the membranous lab-
yrinth; (b) necrosis of the petrous pyramid; (c) infective labyrinthitis and (d) suppurative labyrinthitis with closed empyema of the bony labyrinthine cavity.

Abscesses of the different varieties may be situated in part in the anterior one third of the cerebellum: (1) adjacent, meningeal, chronic or acute, limited by meshes of the pia-arachnoid and the inner surface of the dura, the intradural suppuration resulting from an adjacent infective process by direct extension from infection of the petrous pyramid; (2) adjacent intracerebellar without macroscopical evidence of cortical involvement; (3) adjacent intracerebellar with capsule or stalk (3). Suppuration may be due to a retrograde thrombosis of the cerebellar vein entering the sinus. The abscess is then situated in the lateral lobe and if very large may include any portion of the cerebellum (12).

The opposite may hold true in which the cerebellar abscess may be primary and not secondary to sinus thrombosis. Walbach (13) found that with increased intracranial pressure the sinuses were obliterated and small areas of brain tissue are herniated into the yielding points of the entrance of the arachnoid villi within the sinuses. The herniation may be followed by occluding thrombus which may undergo septin degeneration.

Traumatic and metastatic abscess similar to cerebral abscess has already been discussed.
Courville and Nielsen (28) have found that in the cases of 10,000 autopsies, 76 cases of brain abscess were found. The sources of these abscesses are given in the following table:

**SOURCES OF INFECTION IN SEVENTY SIX CASES OF BRAIN ABSCESS**

A. Adjacent Infection

1. Otitic
   - Temporal Lobe  17
   - Parietal Lobe  3
   - Frontal Lobe  3
   - Occipital Lobe  0
   - Cerebellum  12

2. Rhinogenous
3. Infection of scalp, skull and spine

B. Metastatic Infections

1. Lungs (abscess, 5; empyema, 5; carcinoma, 1; tuberculosis, 1)
2. Heart (endocarditis)
3. Liver (abscess)
4. Bladder (cystitis)
5. Teeth (apical abscess)

C. Origin Undetermined

Total 10

Of the otitic abscesses 23 were cerebral and 12 cerebellar. This corresponds to statistics in general (of 1,950 otogenic abscesses 1,281 were cerebral and 669 cerebellar).

Of the 23 cases of otogenous cerebral abscesses 17 were temporal lobe, 3 in the parietal and 3 in the frontal. Of the abscesses in the temporal lobe 8 were on the right side and 9 on the left side. The average age of the patients with abscess of the temporal lobe
was 34 years. Ten were males, 7 females. Of the 3 abscesses of the parietal lobe 2 were on the right side, 1 on the left. The ages were 32, 37 and 45 respectively. Of the 3 abscesses of the frontal lobe 2 were on the left and one was on the right. The ages were 9 months, 1½ years and 41 years, suggesting a tendency to occurrence in early life. Two were females, one was male.

Of the 17 patients with temporal lobe abscess, 7 had meningitis alone (in 3 of which the abscess ruptured into the ventricle), one had meningitis with thrombosis of the cavernous sinus and lateral sinus, one had an extradural abscess with thrombosis of the lateral sinus and one had septicemia. The remaining 6 had no other intracranial lesion, death resulting from respiratory failure in the cases of the larger abscess. Of the 3 abscesses of the parietal lobe, one was uncomplicated, one was associated with thrombosis of the lateral sinus, and one patient dies of septic meningitis following operation. All 3 patients with abscess of the frontal lobe had associated septic meningitis.

Pitt (29) found in 9,000 autopsies 56 encephalic abscesses of which 18 were otogenous.

OTOGENOUS CEREBELLAR ABSCESS

The usual concept of this lesion is a more or less well defined cavity in the white substance of the
cerebellum, lined with a pyogenic membrane. On the basis of location Friesner and Braun divided them into superficial or cortical and deep or central abscesses (30).

In a series of 12 of 35 otogenous abscesses of the brain located in the cerebellum, 7 were males and 5 were females. The infection in the middle ear was chronic in 9 cases, acute in 3. Six abscesses were found on each side. The average age of the patient was 24½ years.

Multiple cerebellar abscesses are not uncommon for 3 reasons:

(1) Owing to formation of abscesses along the course of an infection which has spread between the folia;

(2) Owing to multiple thrombosis of the cerebellar veins;

(3) Owing to multiple points of invasion from a diffuse exudate about the peduncles.
SITUATION OF ABSCESS IN CEREBELLUM
IN 125 AUTOPSY RECORDS (26)

No. of Cases

On or near Anterior Surface of Cerebellum 30
Whole of Lateral Hemisphere 11
Not stated 36
In Posterior Two-Thirds of Cerebellum 23
On or near Lower Surface 6
On or near Lower and Mesial Surface 3
On or near Posterior and Mesial Surface 1
On or near Upper Surface 1
Lateral Surface 1
Opposite Lobe 2
In middle Lobe 3

Total 117

Multiple Abscesses 10
Bilateral 2

SYMPTOMATOLOGY AND DIAGNOSIS
IN GENERAL

The earlier writers depended on signs such as papilloedema, medullary signs such as slow pulse, coma, etc., for a diagnosis of abscess. This seemed to be the time at which little could be done as these might well be said to be the graver signs. Much of the literature has been recorded as showing a number of cases which could not be diagnosed as signs of abscess were lacking. It has been understood that cerebral compression does not show up until the later stages of the abscess and in the case of frontal lobe abscess may not show at all.

In the presence of known focus of infection from either nose, ear, lungs or other organs, with
cerebral symptoms brain abscess must be differentiated from (1) bloodstream infection, (2) meningitis, (3) vascular lesions and (4) brain tumors.

The question of cerebral suppuration must always be considered. A primary focus must be present where there are cerebral symptoms. If not a primary focus, a primary factor has to be present. If there is not a focus in the ear, nose, throat, teeth, lungs, etc., although cerebral symptoms are present it remains the possibility of cerebral abscess. With the presence of cerebral symptoms with a focus as described above, cerebral suppuration is quite probable, while as yet no number of diagnostic signs are present. Too much importance cannot be attached to a single absent symptom, but all signs, minor or major, must be considered.

PROOF OF CEREBRAL SUPPURATION

An initial vague chill indicates the beginning of an intradural suppurative process (26). Macewen (3) does not consider this factor in his early diagnosis. This mild symptom was not utilized until the 1900s. Since that time, textbooks dealing with nose and throat (31) regard the initial chill as a definite sign of beginning intradural suppuration. This is of utmost importance as the duration of virulence, length of duration of abscess and the presence or absence of an encapsulating membrane may be found.
Headache must be considered as very important evidence of abscess. It is present quite constantly in almost every case. Out of 25 cases picked at random in the literature 21 gave headache as one of the first symptoms of suppuration. In one case headache did not appear until vomiting appeared. The other three cases gave headache as one of the last signs of cerebral suppuration.

Vomiting of the projectile type was once regarded as a good sign of suppuration. This, though, is quite a late sign, basing this on the fact that cerebral compression must have already occurred. Intracranial pressure increase is irritating to the vomiting reflex center, therefore setting up this cycle. The toxic condition present though may be sufficient to set up spells of irregular vomiting. Other symptoms may consist of dry coated tongue, loss of appetite, general malaise, etc., but these initial symptoms may also be present in mastoiditis, sinusitis, and beginning meningitis, so these other types of pathology must be ruled out in beginning suppuration.

Lumbar puncture has come into prominence in the last few years as an aid to diagnosis of brain abscess. Protective meningitis demonstrated by lumbar puncture proves this. This protective meningitis may be evidence of a serous meningitis from the irritation
of a neighboring infected focus, but if the adjacent suppuration in the ear or nose has been fully removed, the persistence of a protective meningitis is absolute evidence of intracranial suppuration (26). A high percentage of polymorphonuclears in the early stage of the disease indicates that the process is an early one. If there is a larger percentage of mononuclears present, the process is subsiding, as the function of the mononuclear cells is to remove the debris from the intraleptomeningeal spaces (14). It is quite well understood now that there will sometimes be no fluctuation in the blood picture in abscess. It has been shown that there may be no polynuclear rise in abscess due to the fact that the abscess has touched no portion of the meninges. For example, this might be shown in case of metastatic abscess carried by the blood stream, causing no meningeal irritation, hence no blood picture change.

Convulsions may be more frequent in the metastatic type although no meningitis may be present. Convulsion may occur in intradural abscess, probably expressed as cortical instability or as in a metastatic type of abscess (15).

There has never been an adequate explanation for the cause of subnormal temperature in cerebral abscess. This fact is of the greatest diagnostic importance. Our theory to explain this is that the
normal temperature is disturbed because of the interference with the heat regulatory center by cerebral suppurative. It is believed that the brain possesses two sets of cells which cause an increase or a loss of heat according as one or the other is stimulated (16). This idea is possibly erroneous and is not believed in by the physiologists as being the factor. Experiments have shown that the heat regulatory centers are located in the mesencephalon and that by removal of the cerebral hemisphere and the thalamus the mechanism for regulating body temperature is destroyed (16, 17).

Other theories are those of an endocrine hook-up, the brain itself possessing endocrine substance which helps to add to itus to the heat regulatory center. There may be a distant relationship between the brain and other endocrine centers which has some connection with the heat regulatory center (17).

In a comparison between meningitis and brain abscess we find that the meningitis temperature curve is exceedingly high, this being due to the microorganisms passing into the bloodstream and the irritation to the meninges. When there is no suppuration near the meninges in abscess plus thrombosis of vessels and autolysis in the region of the abscess, the temperature findings may be accounted for (18).

Papilloedema may occur in abscess, but never
with the frequency and intensity as found in brain tumor. Frontal lobe abscess may never show any degree of papilloedema but cerebellar abscess may. This is due to a disturbance in the cerebrospinal circulation (19).

The abscess, when once proven to be present, must next be localized. A determination has to be made whether it is present in the frontal, temporo-sphenoidal or the cerebellar lobes. A definite history of primary suppuration is of importance, whether there has been a chronic discharging ear, mastoid, frontal sinusitis, empyema, etc. If the labyrinth is involved, the abscess is liable to be cerebellar; if not involved, it is more apt to be temporo-sphenoidal. In ear disease as the primary focus, the absence of nystagmus and negative cold caloric test aid the early diagnosis of temporo-sphenoidal lobe abscess (20).

In temporo-sphenoidal abscess, two pathognomonic signs may be found, aphasia (naming) and hemianopsia. The aphasia in the early stages is apt to be transient. The transverse temporal gyri contain the primary centers for the reception and analyses of the auditory stimuli (31). Pressure by abscess here may well give rise to anaphasia, i.e., word deafness (21).

A hemianopsia may occur due to an involvement of the association fibers running from the cortical
optical center in the cuneus to the geniculate bodies, the cuneopulvinar tract. It has been found that tumors as well as abscesses will affect this tract (22).

Facial paresis of the opposite side may be found at times. This is a cortical type of involvement. Other signs which may be elicited are (a) paralysis of contralateral arm, (b) contralateral hemiplegia, (c) pain in the teeth on the side of the affected hemisphere (34), (d) pain behind the eyes, (e) convulsions (23), (f) occasional dreamy states and (g) psychic manifestations.

Cerebellar abscess may be much more difficult to diagnose as here we are dealing with a comparatively silent area. Symptoms here may not manifest themselves until late in the disease. There is probably a factor here which has to do with masking the symptoms. It is known that there is a compensation of function in the cerebellum which is established far earlier than in any of the higher centers.

CLASSIFICATION OF CEREBELLAR SYMPTOMS

Regarding the cerebellum as an inhibitor, co-ordinator and regulator, we find that abscess will definitely disturb these properties. Lateral deviation of the eyes occurs with destruction or compression in the cerebellum. When there is an infection, the posterior
fossae should be regarded as the site of infection. Vertigo may be present and with caloric and rotating tests establish the lesion in the cerebellum. According to Eagleton (26) there is no set formula for diagnosis of cerebellar abscess. Due to compensation in the cerebellum, no diagnostic signs may be present while the vestibular manifestations of the increased intracranial pressure overshadow and obscure all others. Barany (24) believes that the cortex of the cerebellum contains areas, the destruction of which causes alterations in the ability to execute certain voluntary movements properly.

The following are some suggestive symptoms of cerebellar involvement:

1. Suboccipital tenderness (25),
2. Rigidity of neck,
3. Yawning,
4. Rapid loss of weight,
5. Alteration in knee jerks,
6. Projectile vomiting, and
7. Psychic disturbances.

The yawning may be due to disturbance of reflex. Loss of weight may be due to disturbance in metabolism. Loss of knee jerks would not hold true unless pyramidal tract involvement was present. According to Eagleton (26), vomiting of the projectile type is more suggestive
of cerebellar abscess.

Internal hydrocephalus by mechanical pressure may be caused in cerebellar abscess. This gives rapid signs of medullary involvement with a fatal outcome.

**DIAGNOSIS OF FRONTAL LOBE ABSCESS**

There is a closely related symptom complex in ethmoid, frontal sinus disease and frontal lobe abscess. These involvements may have such symptoms as to make it extremely difficult to diagnose. The frontal lobe must be regarded as a "silent area" making it harder to make a diagnosis. In the number of brain abscess cases, University Hospital 28 - 31, one diagnosis of abscess with findings of abscess on operation was made. The literature shows few cases diagnosed before death. Unusual reasoning must be acquired to help make the diagnosis. It would depend on the history of invasion of a chronic suppuration, presence of a known focus of infection and general symptoms of cerebral suppuration.

Symptoms of Frontal Lobe Abscess. Coated tongue, dry skin, loss of flesh, high leucocyte count, subnormal temperature, headache (may be only symptom), vomiting and high cell count in spinal fluid indicate that exploration should be done.

Convulsion is regarded by Turner (27) and Macewen (3) as positive evidence of cerebral involvement.

Localizing signs may be loss of smell, aphasia,
loss of abdominal reflexes on the same side. A pathognomonic sign is one of paralysis of the arm of the opposite side. Hemiplegia of the contralateral side may sometimes be seen (26).

Encephalitis always has to be ruled out and from readings of case presentations it would seem to be very difficult. It is a septic process, unlimited, while abscess is limited. According to Eagleton (26), a slowly developing compression signifies a refilling of the abscess while exhaustion denotes encephalitis.

The following are a series of cases taken from the files of the University Hospital, 1928 - 1931. These cases quoted all came to autopsy. One or two case histories are missing and only the autopsy reports can be given.
Case I. G. M.  
Hosp. No. 39713  

A - 32-69

Admitted 7/14/32 with complaint of pain and swelling of right ear and mastoid.

July 14  Bilateral paracentesis done.
21  Mastoidectomy - right side
24  Mastoidectomy - left side; left lateral sinus explored
31  Spinal puncture - showed 40 mm. pressure.

Bilateral choked disc.

Aug. 3  Right mastoid explored; post fossae explored
5  Right jugular vein tied and right lateral sinus explored

Spinal fluid cell count 72,000; fluid purulent and under 35 mm. pressure

Temp. 7/14/32 105 degrees. 7/15/32 to 7/18/32 100-101
Temp. to 8/28/32 (death) septic temp., highest 105, lowest 97 degrees.

Pulse 140 - 70 - 160
Presp. 40 - 25 - 45

Leucocytosis - varied from 10,000 to 15,000 throughout course.

Neurologic - varied considerable throughout course.

On admission: Right facial weakness
Positive Kernig 5 days later.
2 ½ Diopters choking
Reflexes diminished

Post mortem. Purulent exudate found scattered over entire surface of brain. Abscess cavity in right temporal lobe.
Case II. G. S.  
Hosp. No. 29788  
A - 29 - 144

Age 15.

Admitted to Hospital 9/24/29. Details of onset of illness vague.

Onset July 1, 1929. Double frontal sinusitis.

August 9, 1929 - symptoms of brain abscess.

Operation and drainage of frontal lobe abscess.

On admission, temperature and pulse; 9/24 - 9/26 P110, 90  
9/26 -99 - 98.5, P 75

Leucocytes 11,000

Neurologic - negative

Operation 9/25/29. Over roof of orbit. About 6 cm. in 
brain tissue pus found.

Operation 10/13/29. Abscess cavity not found - second 
exploratory operation. Temp. and pulse both elevated.

Operation 11/1/29. No abscess found.

Patient became stuporous. Symptoms of meningitis.

Died Nov. 4, 1929.

Post mortem.

1. Purulent generalized meningitis.

2. Large left frontal lobe abscess.
Case III.  O. M.  
Hosp. No. 32978
A - 30 - 93

Admitted to hospital Sept. 12, 1930.

On admittance patient comatose, had severe headache day before.

Temp. 104 - 105 degrees.
Pulse 100 - 20

Septic Temp. 105 - 106; Pulse 165; Respiration 50 - 60.

Diagnosis at first between tuberculous meningitis and poliomyelitis. Temperature and pulse wan rampant on the 13 - 16 day, and child died 9/16/30

Autopsy:
1. Acute vegetative endocarditis.
2. Metastatic abscesses to occipital region of brain, kidney and spleen.
3. Localized area of meningitis over the occipital region of the brain.
Case IV. A. F. 

Admitted 3/3/31

Complaints (1) Rigidity of neck
(2) Headache - frontal - 1 week
(3) Earache

Condition since February 24. No history of chronic running ear; has had pain in ear two years ago for a period of several weeks.

Neurologic: (1) Positive Kernig
(2) Positive Brudzinski
(3) Hyperirritable to small stimuli

Temp. 3/3/31 to 3/8/31 102 - 100 - 104

Pulse 90 - 14; Respiration; 30 - 35

Impression (1) Meningitis
(2) Encephalitis

Death 3/8/31

Post mortem
(1) Ethmoiditis (Possible primary focus?)
(2) Small abscess in left frontal lobe.
Case V. G. W.  
Hosp. No. 25065  
A - 28 - 44

Admitted to hospital 12/5/27

Onset; severe aching of right ear on Nov. 23, 1927.  
Headache following day. Has had headache since  
onset with most of pain over frontal sinus.

Temperature and pulse

Dec. 5 - 9  T & P subnormal, Rsp. normal  
Operation on 9th.

Dec. 9 - 12  T 101.4  P 90  R 26

Dec. 12 - 28  T and P subnormal

Dec 28 - 30  Operation on 28th.  T 100, P 80

Dec. 30 - Jan 1  Normal T, P, and R.  Temp. subnormal  
until 1/24/28  White blood count about 14,000;  
average spinal puncture 25 mm. pressure; high  
cell count.

Patient became stuporous.

Physical examination: Fields normal.

Sluggish closing left eye.

F. N. T. normal.

No ataxia, no past pointing.

Weakness left face; loss of left abdominal reflex,  
exaggerated on right.

Disturbed sensory on left.

Slight hypalgesis.

Diagnosis: Right temporo-sphenoidal lobe abscess.
Case V., Concluded

Operation: Trephine - abscess drainage

12/24/27  Headache, projectile vomiting

Physical 12/27/27  Choked discs R and L, 2 Diopters

Hypalgesia and hypesthesia of face.

Upper extremities: motor power less on right

Hypesthesis and hypalgesia of left arm

Partial astereognosis left

Lower extremity: Sensory loss on left

Summary: left hemianesthesia, motor paresis and partial

loss of deep sensibility.

Opinion: extension of abscess forward, involving post

and prerolandic areas.

12/29/27  Operation - recovery

Dismissed Mar. 2, 1928

March 17, 1928 - Right and left temporal region pain.

Vomiting, severe headache.

March 24, 1928 - Third operation - evacuation of pus.

March 25, 1928 - High temp. 105. Severe headache,

delirium, positive Kernig.

March 26, 1928 - Death.

Post mortem:

1. Purulent meningitis

2. Right temporal lobe abscess
Case VI. V. C.  Hosp. No. 36186
A - 31 - 108

Admitted to hospital 9/8/31
Onset; following septic abortion of 8/20/31, patient
three weeks later was admitted to hospital in
comatose condition.
Findings: 1. Neck rigidity
2. High fever
3. Vaginal discharge

Temperature on admission, 102; eleven hours later, 105
Pulse 130 - 140; later 150
Rsp. 25 - 30; later 40

Neurologic: 1. Neck rigid
2. Eyes react sluggishly to light
3. Suggested hemiparesis - left

Blood count, leucocytes 22,200
Diagnosis: Acute septicemia due to abortion. (Pathology
of brain seems to have been overlooked in diagnosis).

Post mortem: abscess cavity 2-3 cm. in diameter found in
the right cerebral hemisphere. Small rupture into
ventricle. Another small abscess located in the
pons. The left cerebellar hemisphere has been partial-
ly destroyed by an abscess which is not localized
but has been ranified through most parts of the
hemisphere.

Impression: metastatic abscess originating from septic
abortion.
Case VII. V. C. Hosp. No. 35521
A - 31 - 81

Admitted 7/2/31 History of bump on head on June 18, 1931. Ill ever since. Auto accident two years ago - trauma to head at this time. Headache and fever 6/18/31. Temp. 103 degrees.

Neurologic: head tenderness over left frontal region
Neck rigidity and Kernig
Reflexes diminished

Spinal fluid: normal pressure; 750 cells per cc. No organisms.

Temperature 7/2/31 104 7/3/31 102 7/4/31 102.6

Pulse 90 - 95
Respiration 20 - 25

Death 7/4/31

Post mortem: large left frontal lobe abscess found. Dura meningitis in region of abscess. Medullary collapse evident from coning of the tonsils.
Case VIII. F. G.  
A - 30 - 7
(Case history unavailable - Clarkson case)

Autopsy:

Entire surface of right lobe of cerebellum was covered with pus and there was slight evidence of this to the surface of the left cerebellum. The inflammatory process appeared to be localized to cerebral fossae.

Diagnosis: cerebellar abscess, evidently secondary to otitis media.

Case IX. B. S.  
A - 30 - 38

Autopsy:

Centrally located abscess in right temporal lobe.

Case X. S. V. Age 45  
A - 31 - 115

Autopsy:

Removing the dura, the leptomeninges were congested. Right temporal region was greatly softened. Central abscess found in right temporal region.

Case XI. G. Y.  
A - 29 - 40

Autopsy:

The meninges showed marked inflammation with purulent exudate scattered throughout. Section of
brain showed large frontal lobe abscess.

Diagnosis: brain abscess and meningitis.

Case XII. L. N. Age 15 A - 28 - 35

Clinical Resume;

Taken ill with severe frontal headache about March 5-6, 1928. Extensive swelling over left orbital side. Temperature and leucocyte count elevated. Operation for drainage of sinus on March 7. Patient became stuporous after operation. No signs of cerebral involvement.

Death on March 12, 1928.

Autopsy: left frontal lobe abscess.

Comment: Each of these cases reported were evidently difficult to diagnose. On the whole, the symptoms might have been regarded as atypical. Every type of abscess is present and all show a primary focus. These have all been very interesting cases and should have been gone into more thoroughly but space would not permit.
CONCLUSIONS

Brain abscesses show a wide variation as to location, but some abscess areas are far more common than others. The temporo-sphenoidal lobe is the most commonly affected, due of course to the relation to the ear. This abscess may be more readily recognized than those of other locations.

The symptom complex of brain abscess is difficult to interpret. No case seems to be absolutely typical of textbook symptoms and it seems that the experienced eye with inherent good judgment is responsible for diagnosis. These abscesses seem to simulate other intracranial complications so readily that diagnosis just cannot be made. In the future earlier signs and symptoms may be discovered which will aid in a differential diagnosis. Spinal puncture, blood counts, and temperature taking have been tremendous new aids in diagnosis.

Treatment has not been discussed here. It is a huge subject in itself, entirely a surgical measure with the outcome, as yet, far from perfection.

The exciting organism has not been discussed but well known are the members of the pus-forming groups. A few cases present an unusual organism as the exciting agent.

The cases selected from the University Hospital
have been unusually good material and have presented very difficult diagnostic problems.
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