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That the mortality rate from abscess of the brain has not been materially lowered during the past decade is evidenced by a voluminous literature on the subject.[2 3 6 10 11 14 17] As a matter of fact, there has been no reported series of brain abscess where recovery statistics compare favorably with those recorded by Macewen[16] in 1893. Dissatisfaction with our own results has led us to review the records of all our cases of brain abscess to determine, if possible, why at times we have been guilty of diagnostic error and more particularly why certain cases which at the outset appeared to offer a reasonable prognosis have frequently proven fatal. This report, therefore, is based upon data from 32 cases treated surgically and 10 cases which for one reason or another were not subjected to surgery.

Etiology.—The etiological factors in this series were essentially the same as those recorded by Evans[9] and others. The origin of the intracranial infection in 16 cases (50 per cent) was otitic disease. In the remainder the mode of entrance was from the following sources: nasal accessory sinuses, 6 cases; lung suppuration, 2 cases; cellulitis of the face, 1 case; typhoid fever, 1 case; osteomyelitis of the skull, 2 cases; penetrating wounds of the brain, 2 cases; and postoperative wound infection, 2 cases. The infecting organisms were the *Staphylococcus aureus* in 10 cases, the streptococcus in 8 cases, the pneumococcus in 6 cases, the *Staphylococcus albus* in 3 cases, and the *Bacillus typhosus* in 1 case. In 4 cases our bacteriological records are incomplete.

Pathology.—It is necessary when considering any abscess of the brain to recognize three stages of the disease, namely: the acute, subacute, and chronic.

Acute Stage.—In the acute abscess, no definite wall or capsule is found. The central necrotic mass is surrounded by hemorrhagic debris and softened brain tissue. The cerebral substance for several centimeters about the abscess cavity is edematous and infiltrated with leukocytes. Little or no fibrosis or gliosis is found in this area.

Subacute Stage.—If the acute abscess ceases to extend into and invade the surrounding cerebral tissue a reactionary wall forms about the necrotic mass. This wall consists of inflammatory tissue composed of fibroblasts, new capillary loops, astrocytes in various stages of destruction and hypertrophy, and profuse gliosis. During this intermediate stage between acute and chronic abscess, there is less edema of the surrounding cerebral substance, and symptoms of encephalitis subside.

Chronic Stage.—This stage applies to a thoroughly encapsulated abscess, the wall of which may vary from 2 to 5 mm. in thickness. The microscopic structure of the wall is essentially the same as described under the subacute stage. If an abscess of this type continues to expand, the surrounding cortical substance suffers from pressure necrosis as well as the inflammatory reaction. Eventually, the cerebrospinal fluid absorptive mechanism is encroached upon with a corresponding increase in the intracranial pressure.

Our operative group consisted of 4 acute abscesses, 16 subacute, and 12 chronic ones. The lesions were distributed in the various lobes of the brain as follows: the cerebellum, 8 cases; the temporal lobes, 10 cases; the frontal lobes, 8 cases, one of which was bilateral; the parietal lobes, 3 cases; the occipital lobes, 1 case; and 2 were subdural.

Diagnosis.—We shall not analyze the usual signs and symptoms of brain abscess. They are similar to those of any expanding lesion of the brain plus signs of infection. The value of a carefully taken history and repeated neurologic examinations in the diagnosis of brain abscess cannot be over-emphasized. In the majority of instances the presence and location are fairly easy to demonstrate, particularly in the acute and subacute stages. The primary focus is usu-

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ally obvious, though at times the pathway of infection is difficult or even impossible to prove. As an example, one case of our series developed a typhoid frontal lobe abscess two years after all symptoms of the primary disease had subsided. In 28 instances we were able to arrive at a diagnosis and determine the location of the abscess, as well as a fairly accurate understanding of its stage of development, by clinical methods alone.

We do not mean to imply that the diagnosis of brain abscess is always an easy one. Ten additional cases not included in the surgical statistics have come under our observation. None of these was operated upon. Eight of this group had unmistakable signs of diffuse meningitis on admission and a ruptured abscess was demonstrated at the postmortem examination. The other two cases had been under our observation for sufficient time to have established a diagnosis, but in neither instance did we interpret the clinical picture correctly.

We believe that this percentage of diagnostic error could be materially reduced if there was a more general appreciation of the early signs and symptoms of the disease. In analyzing the records of the 10 cases not subjected to surgery we find that there were symptoms early in the primary illness that should have indicated cerebral complication. Usually there had been headache and vomiting, nearly always lethargy, and in 4 instances there had been convulsions. In most of the cases these symptoms had been transitory and the fatal illness from meningitis was ushered in acutely after an interim of symptomatic improvement. In view of these experiences we now believe that any patient showing even transitory signs of cerebral complication from an obvious focus should be kept under the closest observation and if symptoms do not clear up completely in reasonable time, needle exploration of the brain should be carried out. If this procedure had been followed in our group certainly some of these fatal diagnostic errors might have been avoided.

In analyzing from this point of view the 12 chronic abscesses of our series we find that 8 of them showed signs of brain involvement long before they were referred to us as brain abscess suspects. Four of the patients had been subjected to operation upon the mastoids or nasal accessory sinuses and within the first few hours postoperatively they had symptoms of brain involvement, to-wit, headache, vomiting, lethargy, and convulsions. Whether or not the onset of those symptoms indicated primary development of the intracranial infection or the spread of infection from an already existent abscess is problematical.

The secondary onset of symptoms frequently seen with chronic abscesses may simulate almost any disease of the brain. Occasionally they may resemble cerebral vascular accidents. One patient of our series graphically demonstrates the difficulty of this differential diagnosis.

M. H., a woman, aged 48, eight days before admission suddenly developed a partial left hemiplegia, stiff neck, and a suggestive Kernig's sign. The only fact in the past history to suggest infection was a right mastoid operation performed three months before. The fundi were normal. Examination disclosed a partial spastic hemiplegia on the left, slight stiffness of the neck muscles, and a suggestive Kernig's sign. Spinal puncture revealed normal pressure and slightly xanthochromic fluid, containing only 6 cells per cu. mm. She was discharged with a diagnosis of cerebral hemorrhage with rupture into the subarachnoid space. We did not see the patient again, but follow-up notes from her local physician indicated that her symptoms improved for a while after she left the hospital, but began to regress, as was evidenced by increasing weakness in the left arm. One month after discharge she suddenly developed violent headache, became unconscious, and died within a short while. Autopsy revealed a large well encapsulated abscess of the right temporosphenoidal lobe which had ruptured into the ventricle. Reinvestigation of the history showed that at the time of the mastoid operation she had considerable headache and vomiting. These symptoms subsided and no great importance was attached to them. No doubt these symptoms indicated the original insult to the brain.

In 3 cases of our series in which we strongly suspected the presence of an abscess we were unable to determine its location by clinical examination alone. One of our earlier cases was subjected to several negative explorations. The lesion, however, was never located and the patient died of meningitis from rupture of the abscess into the ventricle. Since this unfortunate experience we have been confronted with a similar situation in two cases. In both of these we resorted to ventriculography and were able to locate the lesion. Ventriculography was used in another case when a brain tumor was suspected, but an abscess demonstrated. A bone flap was reflected, but fortunately before the dura was opened the brain was explored with a ventricular needle and the abscess aspirated. The patient made a complete recovery with this single aspiration of the abscess cavity. We now use ventriculography as a diagnostic aid when the location of an abscess is in question. We have, also, been able to differentiate serous arachnoiditis

and brain abscess in 6 cases by this method and have observed no harmful effects from the introduction of the air.

Treatment.—Before speaking of treatment of the abscess itself, a word might be said about the management of the primary focus. A point of view regarding the prevalent use of mallet and chisel for eradication of diseased mastoids was stressed by Hugh Cairns.[3] He called attention to the dangers of repeated percussions of the skull and suggested that many of the intracranial complications of mastoid disease might be precipitated by this operative technic. He advocated the adoption of a less traumatic method, namely, the use of burrs and rongeurs. We fully subscribe to this point of view. Furthermore, the common practice of inducing deep anesthesia for these operations, particularly when an intracranial complication is feared, appears to us likewise to be dangerous. If a diseased mastoid can be eradicated under local anesthesia with a minimum of skull trauma, then the delicate barriers to infection between the skull and the brain will be greatly protected.

It is a great temptation at times, when an abscess is suspected, to make an exploration through an already septic field such as that left by a radical mastoid operation. This practice cannot be too forcibly condemned. If a well walled off abscess is encountered and drainage adequately instituted, recovery may be uneventful. If, however, the diagnosis is incorrect, or if the abscess is not found by such an approach, a fatal meningitis or an infection in the brain along the tract of the exploratory puncture may result.

Before attempting to discuss the relative merits of the various operative procedures advocated for the cure of intracranial abscess, we should like to emphasize that selection of a method for any particular case may depend upon the stage of development of the inflammatory reaction. An acute abscess, that is, one in which the symptoms and signs of encephalitis predominate, is rarely, if ever, a surgical problem. Lesions of this type should be treated conservatively until the natural healing processes have had sufficient time to localize the infection. We advocate the use of every method at one's disposal to relieve intracranial pressure before resorting to what usually proves to be a useless exploration. The pressure can nearly always be kept under control by dehydration, and once the febrile reaction has subsided and the cerebrospinal fluid no longer contains polymorphonuclear leukocytes, then aspiration of the abscess becomes indicated. It is at this point that we consider the process to have passed into the subacute stage, and it is at this point further that surgical treatment appears to offer the best chance for cure.

It is often difficult to persuade the family and attending physician that waiting is the best policy during the acute stage. Most people cannot understand why, once the diagnosis of an abscess is made, drainage is not instituted immediately. It is our custom to make a comparison between an abscess and an ordinary boil on the surface of the body. Most people readily appreciate why it is dangerous to cut a boil during the acute stage.

There are, unfortunately, certain cases of acute brain abscess that seem never quite to localize. With a patient losing ground daily and the intracranial pressure getting out of control, one is always tempted to operate in the vain hope of changing an otherwise hopeless outlook. Four such cases have occurred in our series. All four of them died.

Most authors have considered the chronic brain abscess as offering the best prognosis to surgical treatment. Our experience does not bear out this point of view. We have had 16 subacute abscesses subjected to surgery with 12 recoveries and 4 deaths. We have had 12 chronic abscesses treated surgically with 6 recoveries and 6 deaths.

We claim no originality for any methods of treatment now in use, but have, at one time or another, used most of those advocated by other authors. In the 32 cases of our series we have followed approximately the following procedure. In most cases the abscess cavity was first aspirated as recommended by Dandy;[7] many of them were aspirated repeatedly. When it became obvious that this method was inadequate to cause healing of the lesion other more radical methods were instituted. In 17 of the cases treated by aspiration alone there were 9 recoveries and 8 deaths. However, 4 of the deaths occurred in patients with acute abscesses and were hopeless problems from the onset. The remaining 15 cases were aspirated at first, but as this method obviously failed, other procedures were instituted. In 6 of the cases Coleman's[5] small rubber catheter technic was followed, but in no instance did we feel that the catheter drained satisfactorily. However, 3 of these cases recovered,

but we question whether or not the recovery might have occurred just as satisfactorily with repeated tappings. In 9 cases where repeated tapping of the abscess cavity had failed we employed the marsupialization method as advocated by Cushing,[11] Horax,[11] and King.[15] In this group there were 5 recoveries and 4 deaths. In 1 case in which repeated tappings, catheter drainage, and marsupialization had failed we resorted to the decortication method of King[15] and were finally successful in curing the patient. We have never attempted to enucleate the abscess *in toto* as advocated by Cairns,[3] Adson[1] and others.

It is generally agreed that no one method is suitable for every case; in each case the procedure attempted should be dictated by several factors, particularly the stage of development of the abscess and the depth of the wall beneath the surface. Dowman[8] first advocated a two-stage operation, by first inducing protective adhesions between the brain cortex and the dura before establishment of drainage at a later date. Kahn[12] has modified this and made a valuable contribution to the technic of handling deeply seated chronic abscess. He advocated the creation of a large defect in the skull preliminary to drainage with opening of the dura to promote migration of the wall to the surface. In view of some of our failures with deeply seated abscesses we intend to try this method in the future as it appears to offer a two-fold advantage: (1) it promotes migration of the abscess wall to the surface; (2) it effectively seals off the subarachnoid space overlying the abscess. Even with Kahn's[12] method before resorting to wide opening of the capsule we should be inclined to attempt repeated tappings of the abscess at the second stage.

While we believe that aspiration of the abscess cavity, particularly during the subacute stage, is the method of choice, yet there are certain inherent dangers connected with it and it is not adaptable to certain cases. We have had four deaths from meningitis following the use of this operation in chronic cases. In each instance the abscess was a superficial one and contained a large amount of pus. When the aspiration had been completed the cortex fell away from the dura alarmingly and soiling of the subarachnoid space occurred promptly. Had we obliterated the subarachnoid space before aspirating the pus we believe this complication might have been avoided.

Prognosis.—In compiling our mortality statistics we have included all cases which were subjected to surgical treatment. No case has been excluded because it was an unfavorable surgical risk. The mortality rate was 44 per cent. Were we to exclude the four acute cases, which never should have been subjected to surgery in the first place, and the 2 chronic cases which had meningitis when first seen, our mortality rate would have been 25 per cent. We feel that this last figure more accurately represents the risk to which patients with chronic and subacute brain abscesses are subjected provided the diagnosis is established in a reasonable time.

In our recovered group there have been remarkably few disabling residual symptoms. Only 3 of the patients have had epileptiform seizures and one of these suffered an extensive gunshot wound of the frontal lobe in which the abscess was a secondary development.

SUMMARY AND CONCLUSIONS

The review of records from 32 cases of brain abscess treated surgically and 10 cases which for one reason or another were not operated upon disclosed certain pertinent facts which seem to justify the following conclusions:

(1) The profession as a whole is not alive to the fact that such symptoms as headache, vomiting, lethargy, or convulsions are not consistent with uncomplicated bony infection and when present indicate brain involvement. Patients with these symptoms should be considered brain abscess suspects until proven otherwise.

(2) When serious doubt exists as to the presence or location of an encapsulated abscess we favor needle exploration of the suspected area and, if necessary, ventriculography.

(3) The use of mallet and chisel and deep anesthesia in the eradication of diseased mastoids appears to incur the danger of actually inducing intracranial infection or spreading an already existent one.

(4) An acute abscess is rarely, if ever, a surgical problem.

(5) Our best results were obtained when the abscess was treated during the subacute stage. The aspiration technic of Dandy[7] appears to be ideally suited to this type of lesion except for certain large superficial abscesses.

(6) When repeated aspirations prove inade-

quate and the capsule is sufficiently tough, we favor the marsupialization technic.

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