Surgical Management of Metastatic Brain Abscess*

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Brain abscesses usually originate from one of three sources: (1) direct extension from infected paranasal and mastoid sinuses, (2) compound fractures of the skull including intracranial foreign bodies, (3) metastatic nidi from infections elsewhere in the body. It is with this third group that we are concerned in this report.

Of 50 consecutive cases of brain abscess reported by Pennybacker,15 36 per cent were metastatic. An infection anywhere in the body may be a potential source, though they most frequently originate from thoracic infections. Of Pennybacker's cases of metastatic brain abscess, 75 per cent were thoracogenic. When thoracogenic, the primary source is usually bronchiec-
tasis, lung abscess or empyema. About 50 per cent of metastatic brain abscesses are solitary and when multiple, they are either isolated or contiguous.

Until the advent of penicillin, the mortality from metastatic brain abscess was almost 100 per cent, whereas, with brain abscess of other origins, the prognosis has not been so discouraging. Grant5 reported 47 recoveries in 100 consecutive cases of brain abscess of all types treated from 1926 to 1940. In 1946, Sachs16 reported 52 deaths in 108 operated cases of brain abscess. Excluded from this series were 20 patients with metastatic brain abscesses of thoracic origin; of these 20, 19 died. Pennybacker also reported 11 deaths in 12 cases of secondary brain abscess. In his review of the literature in 1936, King33 could find only 8 cases in which metastatic brain abscess had been cured by surgery. He was impressed by the fact that all recorded cases of recovery but 1 were those in which the abscess had become encapsulated. In his article is included a report of an acute metastatic abscess which he cured. Since the use of sulfonamides and penicillin, about 8 more cases have been reported (Grant,9 Hamilton, Whitcomb and Woodhall,11 Cohen and Drooz,5 LeBeau,14 Fincher8).

Various reasons have been offered for the marked difference in prognosis between the non-metastatic and the metastatic abscesses. Some state that in the latter, the primary focus of infection has already debilitated the patient, that the infecting organisms, especially those from thoracogenic infections, are more virulent, and that often these organisms are anaerobic. Others say that a high percentage of metastatic abscesses are multiple. How-

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ever, as pointed out above, this has been proven to be true in only 50 per cent of the cases. The most significant fact is that in almost all of the fatal cases reported, death occurred within 3 weeks and usually within 2 weeks after the onset of cerebral symptoms. This fact induced King to operate on metastatic abscesses during the acute phase in an attempt to resect the infected areas of the brain. More recently the use of chemotherapy has obviated the need for surgery in this stage.

When we consider the pathology of brain abscess of hematogenous origin, we find that basically it is very similar to an abscess elsewhere in the body. It begins as an infected nidus. An area of cerebritis and, extending well beyond this, an area of cerebral edema develop comparable to the areas of cellulitis and edema about an early furuncle. The subsequent course of events depends upon the patient’s individual immunity, the virulence of the invading organisms and the size of the area of necrosis resulting from the original arterial occlusion or venous thrombosis. Undoubtedly, in some cases the processes of repair overpower those of infection and resolution occurs before real abscess formation takes place. More frequently than not, the equilibrium between the reacting elements of the brain and the invading organisms is not reached until abscess formation occurs. When it occurs, a barrier in the form of the abscess capsule is created which tends to limit the spread of the infection. This is formed by the heavy exudation of leucocytes and the proliferation of connective-tissue cells. If the infection overpowers the resistance, widespread cerebritis can occur. When an abscess becomes encapsulated, the infection may become overwhelmed by the reparative processes and heal spontaneously, as in the case of a furuncle that never drains. If it does not heal or is not drained, it may rupture from increasing intracapsular tension, resulting in another area of cerebritis and the formation of daughter abscesses.

Death, when it occurs during the acute phase, i.e., within the first 10 days, is due to the increased intracranial pressure resulting from cerebritis. When death occurs during the chronic stage, it may be due to rupture of the abscess or to increased intracranial pressure resulting from the abscess and from obstruction to the flow of ventricular fluid. If the abscess ruptures into the ventricle or the subarachnoid space it can cause fulminating meningitis.

In order to cure a brain abscess one must first carry the patient through the acute phase, i.e., the stage of cerebritis. One must then prevent rupture of the abscess and prevent death from the increased intracranial pressure of the chronic stage. As noted above, the majority of patients with metastatic brain abscesses died during the acute phase. Since the organisms in brain abscesses are usually either streptococci or staphylococci, it would seem logical to expect that chemotherapy, especially the administration of penicillin, during the acute phase might help in the struggle between the invading organisms and the reparative processes. This has proved to be the case (Hamilton, Whitcomb and Woodhall14). Many of these patients can now be carried into the chronic stage with resulting encapsulation of the abscess. When