The treatment of cranial osteomyelitis and brain abcess / Alfred W. Adson.

THE TREATMENT OF CRANIAL OSTEOMYELITIS AND BRAIN ABSCESS

ALFRED W. ADSON, M.D.

SECTION ON NEUROLOGIC SURGERY, THE MAYO CLINIC

ROCHESTER, MINN.

SUPPURATIVE lesions of the scalp, skull, accessory sinuses, meninges and brain bear an intimate relationship to one another since the infection may spread by contiguity or travel along lymph and diploic channels, and along emissary veins which communicate with veins that pass through the dura into the substance of the brain. Appropriate surgical treatment of the localized infection often will limit its extension and prevent the more serious involvement of the meninges and brain.

The treatment of osteomyelitis of the skull does not differ from the treatment of osteomyelitis of other bones; removal of all necrotic and infected osseous tissue is required in addition to sequestrectomy. However, special consideration concerning these operative procedures is necessary, since the scalp and periosteum may have been destroyed, and removal of infected bony tissue may result in exposure of the meninges; moreover, brain abscesses are frequent sequelae of osteomyelitis of the skull.

A consideration of the anatomic arrangement of the veins in the diploe and of the emissary veins is in order, so that it may better be understood how infections may travel through communicating veins to give rise to distant areas of osteomyelitis, with or without accompanying brain abscess. Infective emboli in the arterial system are rarely responsible for osteomyelitis of the skull. Thrombosis of vessels in an extending infection of the scalp frequently spreads the osteomyelitis. The arterial supply to soft tissues and haversian canals about an osteomyelitic region serves as a barrier to limit the progress of infection by maintaining the life of the osseous tissue. Frequently, the osteomyelitic process will destroy but one table of the skull when the circulation to the opposite table has been maintained.

Gray[22] states: "The diploic spaces of the cranial bones in the adult contain a number of tortuous canals, the diploic canals (canales diploici [Breschetí]), which are surrounded by a more or less complete layer of osseous tissue. The veins they contain are large and capacious, their walls being thin and formed only of endothelium resting on a layer of elastic tissue; they present at irregular intervals pouch-like dilatations, or culs-de-sac, which serve as reservoirs for the blood.

"In adult life, so long as the cranial bones are distinct and separable, these veins are confined to the particular bones; but in old age, when the sutures are united, they communicate with one another and increase in size. They communicate, in the interior of the cranium, with the veins and sinuses of the dura, and on the exterior of the skull with the veins of the pericranium. They

consist of: (1) The frontal diploic vein (v. diploica frontalis), which opens into the supraorbital veins by an aperture in the supraorbital notch; (2) the anterior temporal diploic vein (v. diploica temporalis anterior), which is confined chiefly to the frontal bone, and opens into one of the deep temporal veins through an aperature in the greater wing of the sphenoid; (3) the posterior temporal vein (v. diploica temporalis posterior), which is situated in the parietal bone, and terminates in the lateral sinus through an aperture at the posteroinferior angle of the parietal bone or through the mastoid foramen; and (4) the occipital diploic vein (v. diploica occipitalis), the largest of the four, which is confined to the occipital bone, and opens into the lateral sinus or the torcular Herophili.

"The emissary veins (v. emissaria) are vessels which pass through apertures in the cranial wall and establish communications between the sinuses inside the skull and the diploic veins in the diploe, and the veins external to the skull. Some of these are always present, others only occasionally so. They vary much in size in different individuals. The principal emissary veins are the following: (1) A vein (v. emissarium mastoideum), almost always present, runs through the mastoid foramen and connects the lateral sinus with the posterior auricular or with the occipital vein. (2) A vein (v. emissarium parietale) which passes through the parietal foramen and connects the superior sagittal sinus with the veins of the scalp. (3) A plexus of minute veins (v. rete canalis hypoglossi) which pass through the anterior condylar (hypoglossal) foramen and connect the occipital sinus with the vertebral vein and deep veins in the neck. (4) An inconstant vein (v. emissarium condyloideum) which passes through the posterior condylar foramen and connects the lateral sinus with the deep veins of the neck. (5) A plexus of veins (v. rete foraminis ovalis) connects the cavernous sinus with the pterygoid and pharyngeal plexuses through the foramen ovale. (6) Two or three small veins run through the foramen lacerum medium and connect the cavernous sinus with the pterygoid and pharyngeal plexuses. (7) There is sometimes a small vein connecting the same parts and passing through the inconstant foramen of Vesalius, opposite the root of the pterygoid process of the sphenoid bone. (8) A plexus of veins (plexus venosus caroticus internus) traverses the carotid canal and connects the cavernous sinus with the internal jugular vein. (9) A small vein (v. emissarium occipitale) usually connects the occipital vein with the lateral sinus or the torcular Herophili and the occipital diploic vein. (10) A vein is usually transmitted through the foramen cecum and connects the superior sagittal sinus with the veins of the mucous membrane of the nose."

Osteomyelitis.—*Etiology:* Osteomyelitis results from infection of an avascularized bone or from extension of an infection into the diploic canals.[15] The most common sources for the infection are contaminated, compound, comminuted fractures of the skull, furunculosis of the scalp and extension of infections from the ear and accessory nasal sinuses, the frontal sinus being the chief offender. Diffuse osteomyelitis of the outer table is more prone to

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The denuded skull, resulting from burns by electricity and fire, from extensive lacerations, and from removal of the scalp because of tumors, ultimately, undergoes destructive processes. If the circulation to the inner table is intact, only the denuded outer table will degenerate and separate as a sequestrum.[33]

Craniotabes, gummatous osteomyelitis,[1] caused by syphilis, is a representative of the group of chronic infections. *Eberthella typhi* (*Bacillus typhosus*) at one time was a fairly common cause of osteomyelitis but today, since the introduction of vaccination, is rarely seen. An occasional case results from tuberculous involvement of the skull. Actinomycosis, too, occasionally is responsible for infective processes of the skull.

Pathology.—The process of osteomyelitis begins with the introduction of suppurative organisms into the vascular channels of the skull. If the bone is denuded of its scalp, the process will extend until adequate circulation is encountered. Frequently, accompanying cellulitis will destroy the circulation of the scalp about the denuded bony area and this further encourages extension of the osteomyelitis. The serpiginous extension along diploic canals will give rise to additional areas of necrosis beyond the original focus.[35] The infection of diploic veins results in thrombosis and the development of granulation tissue. This process may extend either to the pericranial or to the intracranial structures by extension along emissary veins. Phlegmons of the scalp develop over the necrotic bony areas and extend the infection over suture lines to new areas, through emissary and diploic veins, thus giving rise to additional osteomyelitic processes.[28] The disease may progress until the entire skull has been involved. On examination of the skull, islands of normal bone will be found between necrotic and sequestrated areas. These islands apparently have resisted the infection, owing to the fact that the circulation has been maintained and that a zone of granulation has served as a barrier in limiting the infection in the diploic canals.

The reparative process of granulation and absorption works in conjunction with the destructive process. As the bony cells die they disintegrate, are absorbed and are replaced by granulation tissue. This is readily demonstrated in a denuded area of skull. The exposed bone at first appears dry and dead-white, but sooner or later a zone, or ring, of granulation will appear at the scalp margin. In the zone of granulation there will develop a localized osteomyelitis which eventually destroys a ring of bone in the outer table of the skull. When this has taken place, the granulation tissue in the diploic spaces will actually lift from the dead-white outer table of the denuded skull (Fig. 1). Removal of the granulation tissue will reveal that the inner table is usually intact and is very vascular, owing to the fact that its circulation has been main-

tained by small arterioles from meningeal arteries. However, if the scalp has been destroyed by a severe burn, both tables of the skull will slough away spontaneously.

Symptoms.—The local symptoms are usually preceded by a history of infection. If the symptoms follow injuries of the scalp and skull, a septic temperature of low grade develops, associated with leukocytosis and with localized swelling of a soft and doughy consistency, with or without localized tenderness. If the infection results from frontal sinusitis, the swelling will appear over the frontal bone, usually on the side of the involved sinus.[7] When the infection is of hematogenous origin, as it is in syphilis, the swellings may

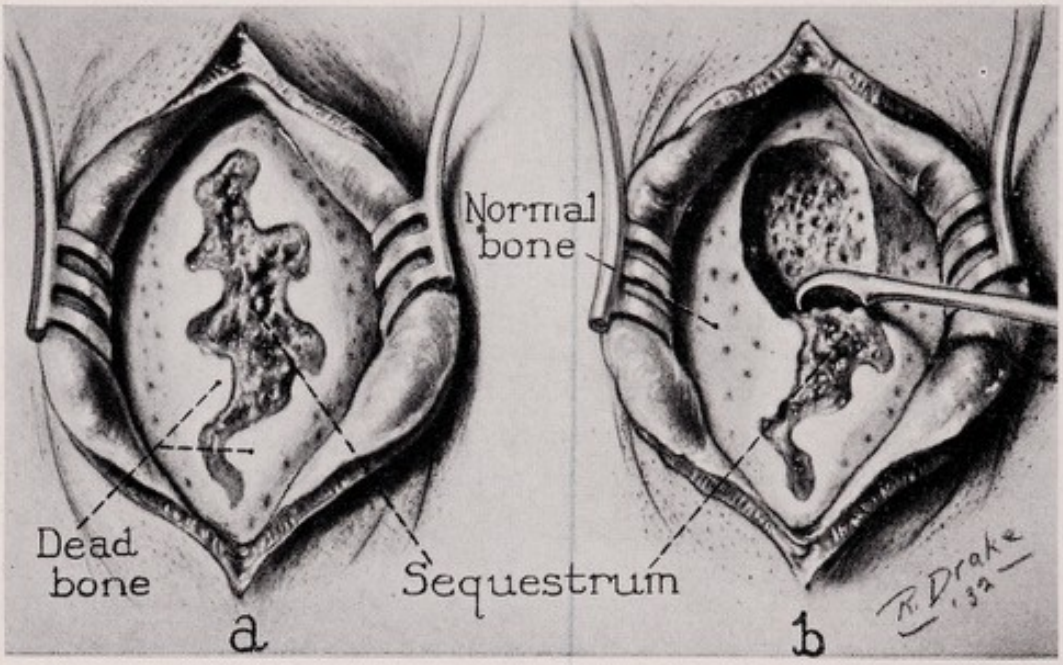

FIG. 1.—(A) Drawing showing worm-eaten erosions of osteomyelitis involving the outer table. (B) The procedure employed in removal of the sequestrum and the overhanging ledges of dead, white bone.

occur in numerous parts of the skull, but usually in those parts where the cranial tables are thickest, and where the diploic spaces are largest, as in the parasagittal portions of the frontal and parietal bones. Roentgenologic examination usually reveals motheaten erosions of one or both tables of the skull. All roentgenograms should be taken in two directions in order to demonstrate the extent of the lesions.

Surgical Treatment.—The phenomena of destruction and repair form a basis for surgical treatment, since adequate drainage of suppurative lesions of the scalp, removal of infected bony fragments and removal of dead and necrotic bone will aid in preventing or limiting the osteomyelitic process.[4] Small puncture-like incisions over fluctuating areas are of some value but are not sufficient surgical procedures to check the process. Localized osteomyelitic areas will continue to spread until the scalp has been reflected and all of the dead and sequestrated bone has been removed. Frequently the necrotic bone can be removed with a sharp curet or gouge without removing both tables of the skull (Figs. 2 and 3). However, there is less danger of the

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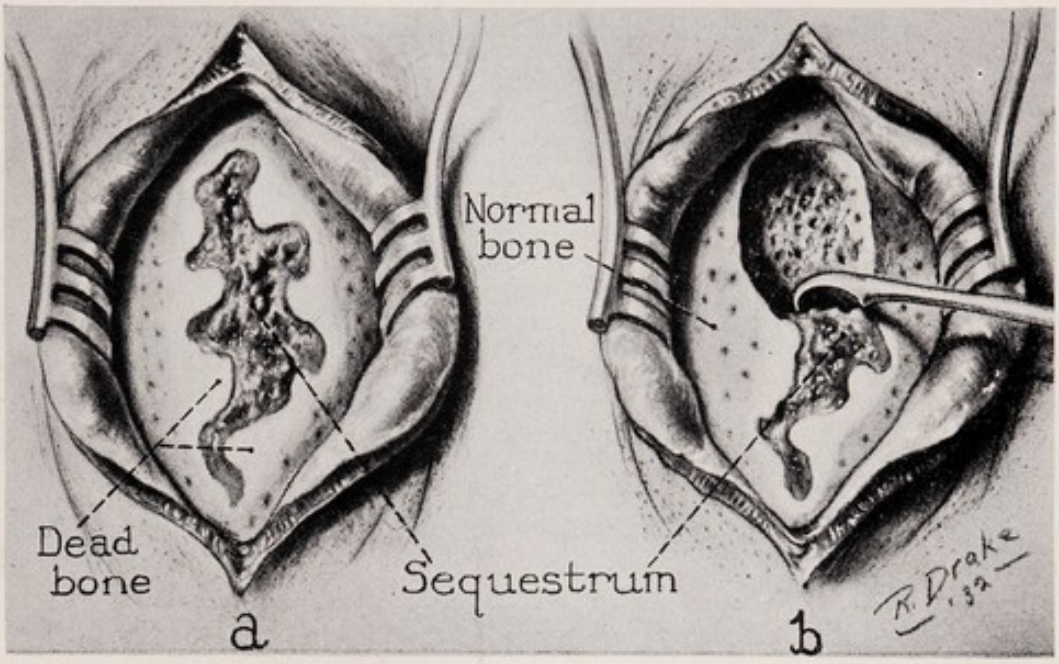

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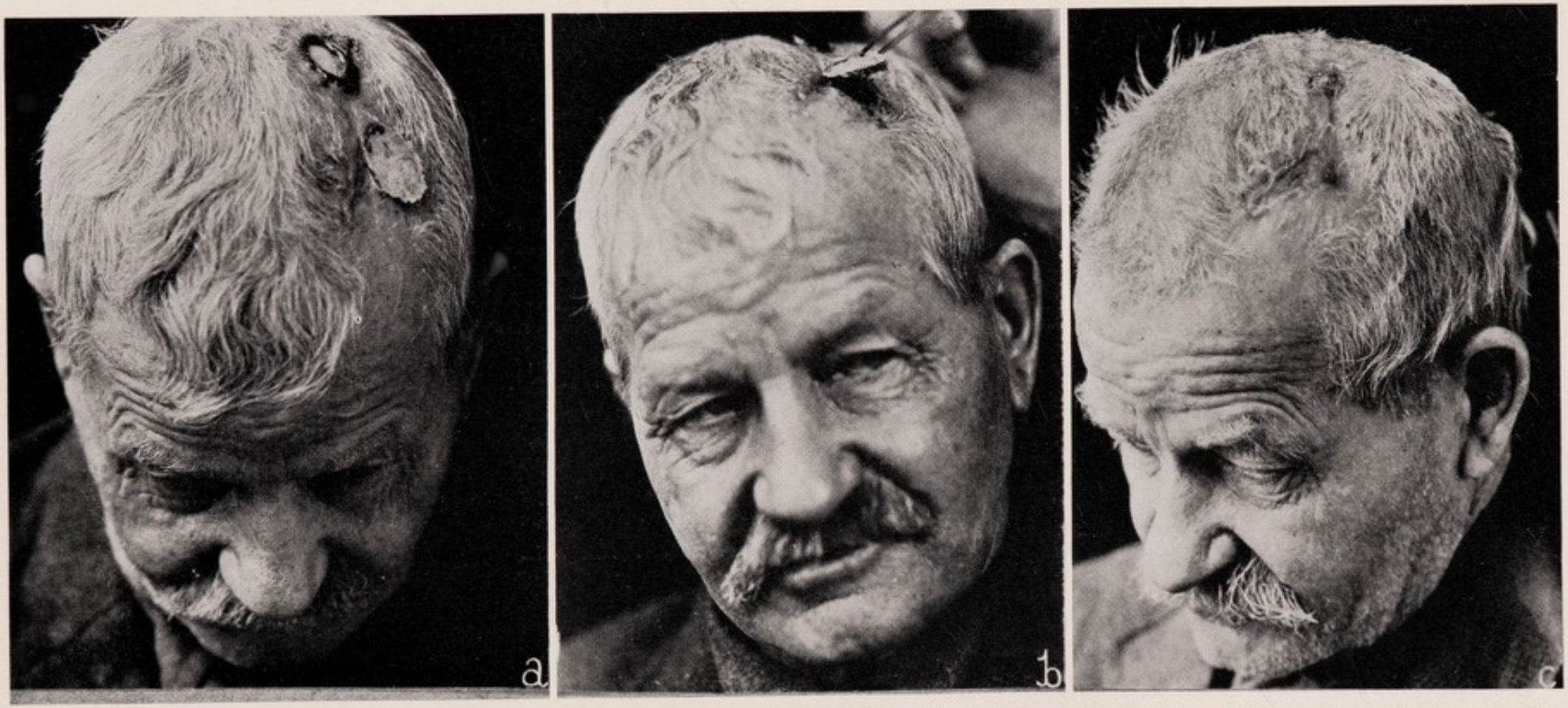

FIG. 2.—(A) Two electric burns on the left parietal area which had been present for five months without evidence of granulation. (B) Spontaneous sequestrectomy aided by the use of a forceps, which permitted granulation and healing of the wound. (C) Photograph taken six weeks following the photographs (A) and (B).

infection extending into the brain with removal of both tables of the skull, if they are necrotic, than there is when the inner table is left in place and only the outer table is removed. The dura serves as an excellent barrier to inward extension of infection provided it is not injured in the course of sequestrectomy. If extensive areas have become involved, it is reasonably safe to uncover as much as one-fourth of the skull at a time. At each stage of the operation the removal of dead and necrotic bone should be complete; however, islands of normal bone, when present, should be left in place. The periosteum likewise should be preserved and resutured after the infectious material has been removed and the field cleansed with tincture of iodine. If drainage is instituted, the drain should be removed within 48 hours, for not infrequently primary healing will be obtained. The additional areas should be treated in a similar manner at intervals of four to five days between each two operations, until all of the necrotic bone has been removed. Too often

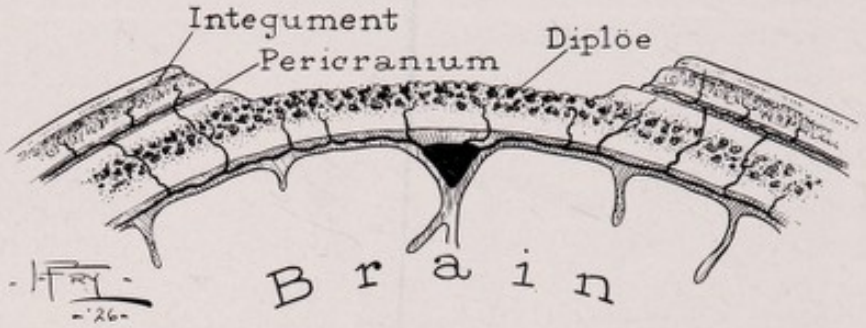

FIG. 3.—Drawing illustrating the importance of removing the necrotic outer table of the skull peripherally until normal bone is exposed, which also permits the edges of the skin to cover the fresh margins of the bone.

the surgeon is inclined to leave dead-white bone lateral to the osteomyelitic process. If this is done, the process will continue. The dead bone should be curetted away until bleeding appears from its cut edges (Figs. 2 and 3). The bleeding can be controlled with strips of gauze soaked in tincture of iodine which, if left in place, should be removed within 48 hours, together with other drainage material such as Penrose drains and rubber tubes, *etc.*

Epithelization of denuded areas of the skull can be materially hastened by removing the outer table in order to expose the diploic spaces. This will give rise to granulation tissue which serves as a bed for the epithelium (Fig. 3). Skin grafts can be employed to hasten the process of epithelization. The outer table is readily removed and the first step in the removal is to make multiple openings in the outer table with the trephine bur. After this, the ridges of bone are rongeured away or removed with a chisel, care again being taken to remove all dead bone, even though it may extend under the margin of the scalp. The uncovered inner table, with its oozing diploic veins, is protected by perforated paraffin gauze, which also encourages epithelization.

Although radical surgical treatment is the effective means of controlling the osteomyelitic process, it should be borne in mind that the virulence of the organism, the resistance, and the specific immunity of the patient to the particular organism are the combative forces which determine the activity of the

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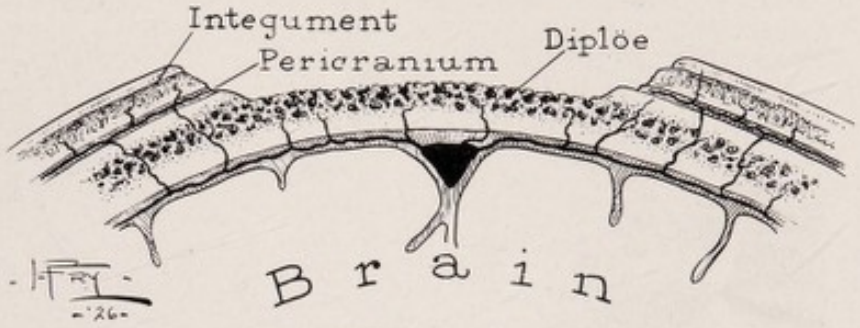

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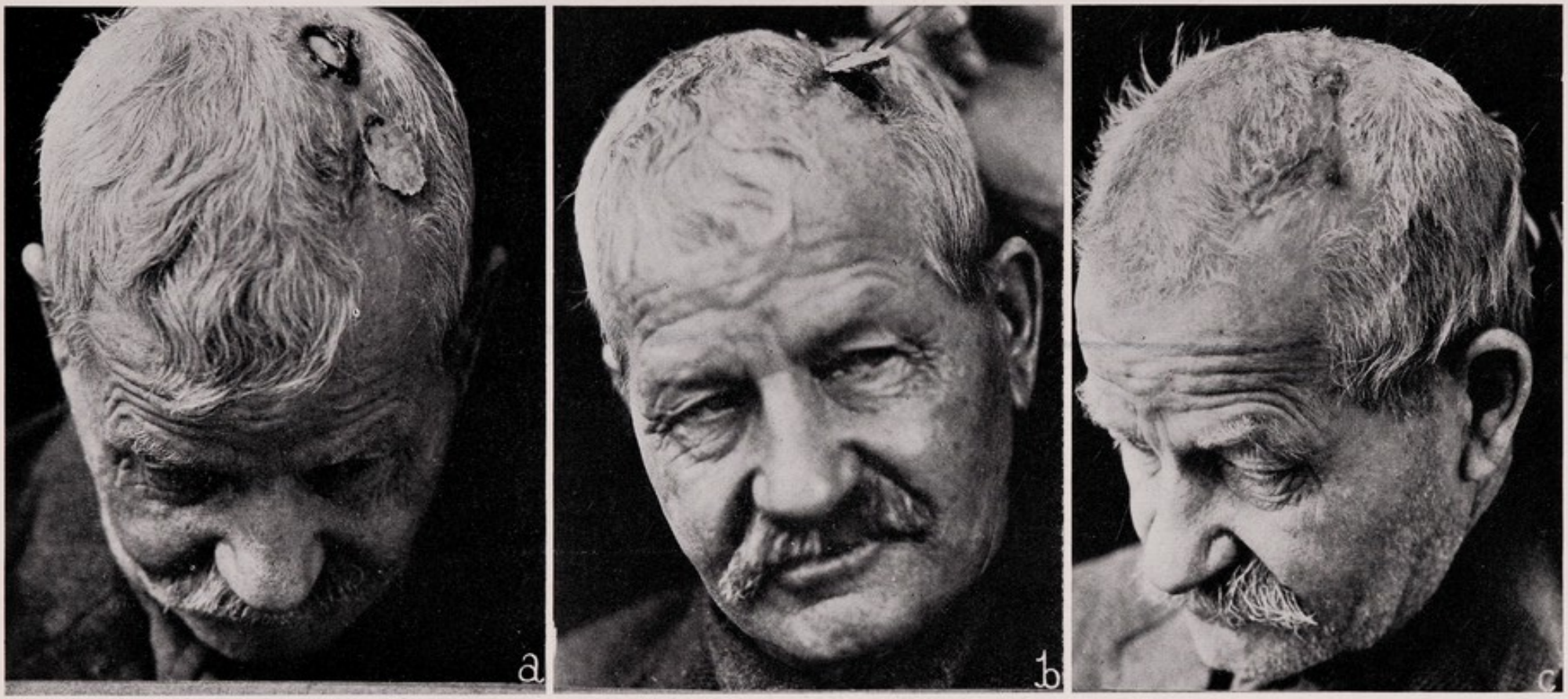

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process and the extent to which it will travel. This being true, active supportive measures should be employed. Administration of vaccines has been suggested and sulphanilamide has been employed, both of which have proved of value. High caloric diets with adequate vitamins are essential. Occasionally local application of heat has aided the circulation and hastened development in the zone of reaction, thus limiting the process.

The accompanying leptomeningitis is a serious complication and is controlled best by repeated or continuous spinal drainage and the administration of sulphanilamide. Again, a nourishing dietary regimen and excellent nursing are material aids in control of the disease.

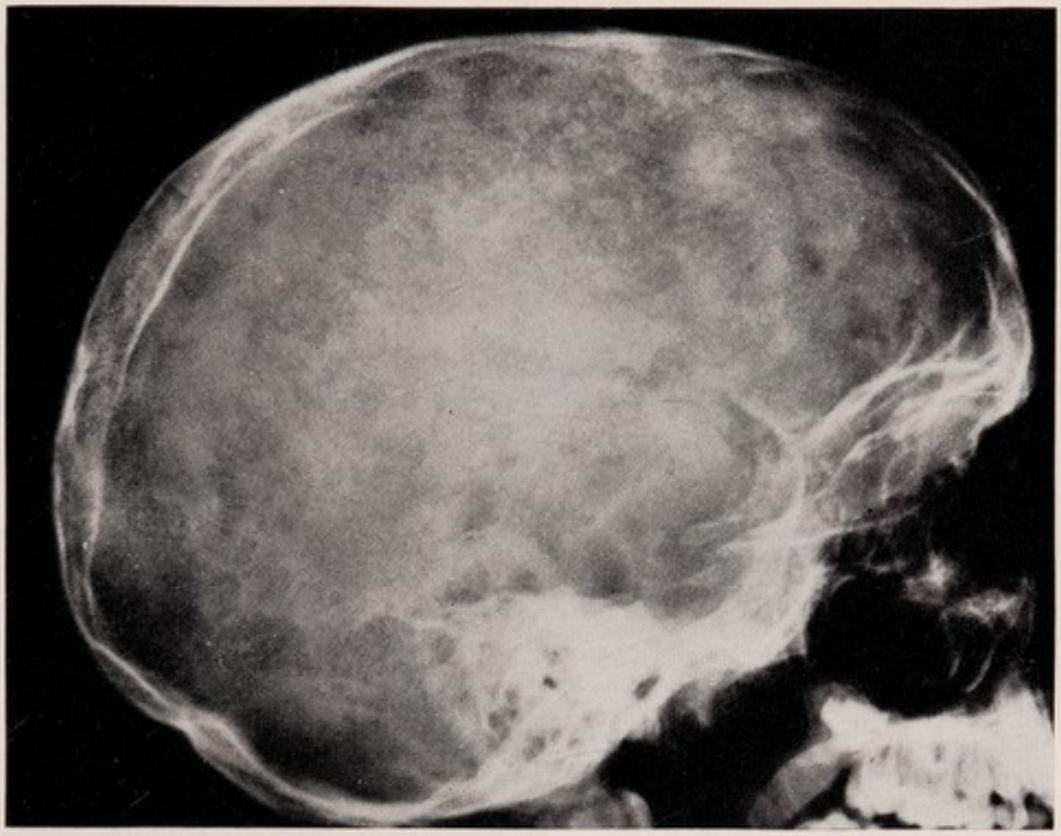


FIG. 4.—Roentgenogram, lateral view, showing extensive osteomyelitis of the left temporal, parietal, and frontal bones.

The cerebral abscess which results from osteomyelitis is one of the suspected and serious complications. When it occurs, the surgeon is confronted with the problem of deciding whether to drain the abscess before treating the osteomyelitis or to treat the osteomyelitis before draining the abscess or, indeed, to attempt to treat both at the same time. A maxim of general surgery should be invoked in the treatment of these lesions, namely: "A patient often will survive two major operations if they are performed separately but may fail to survive if both are attempted at the same time." Too much never should be attempted at one operation. Judging from my own experience, it is better to delay drainage of an abscess than to delay the operative procedure for control of the osteomyelitis. Encapsulation becomes more nearly complete if drainage is moderately delayed, whereas, the osteomyelitis process will

continue to extend and may give rise to additional brain abscesses. A number of times I have removed the necrotic bone, resutured the scalp and drained the abscess a week later through a separate incision without lighting up the osteomyelitis (Figs. 4, 5, 6 and 7). There is an occasional exception, namely: when the process has continued for several weeks; when the cerebral symptoms produced by the abscess are very marked; and when the osteomyelitic process is limited and apparently controlled. In those instances the cerebral abscess has been drained first and the osteomyelitis treated following removal of the drains. There is also the occasional case in which the osteomyelitic area is very circumscribed, and the abscess appears to be situated close to

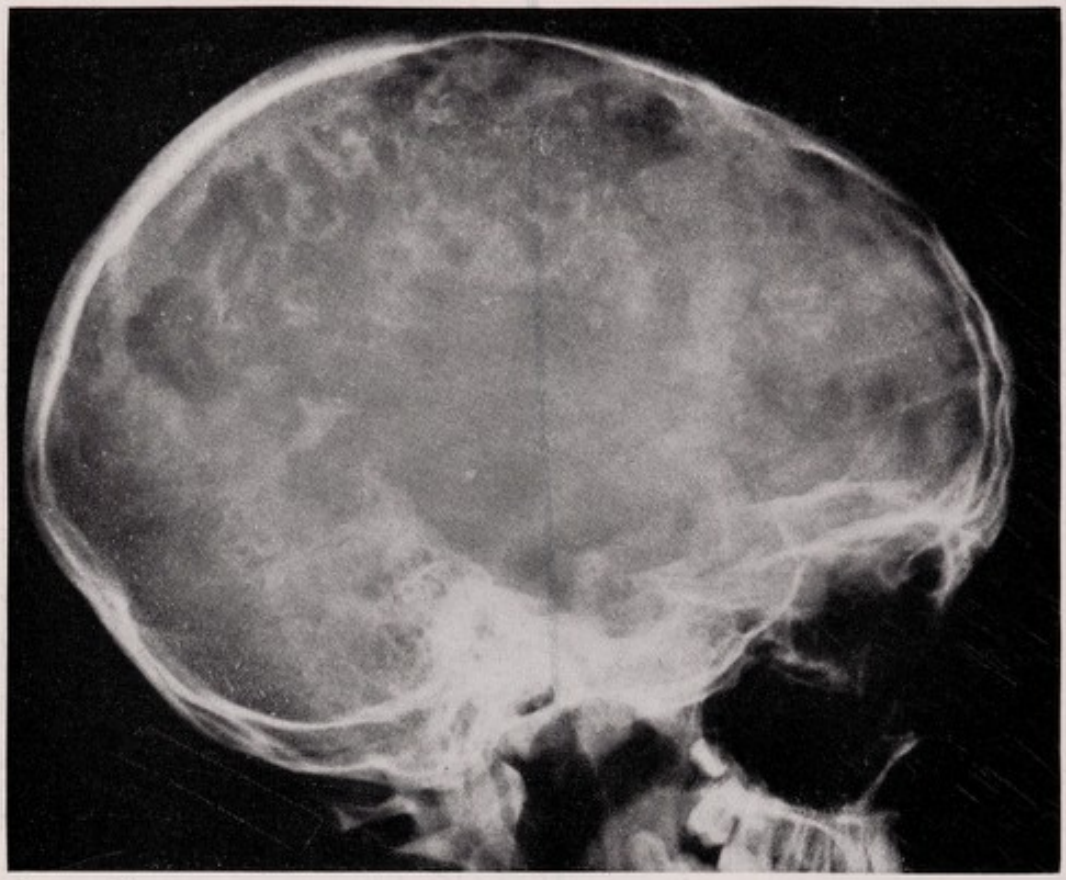
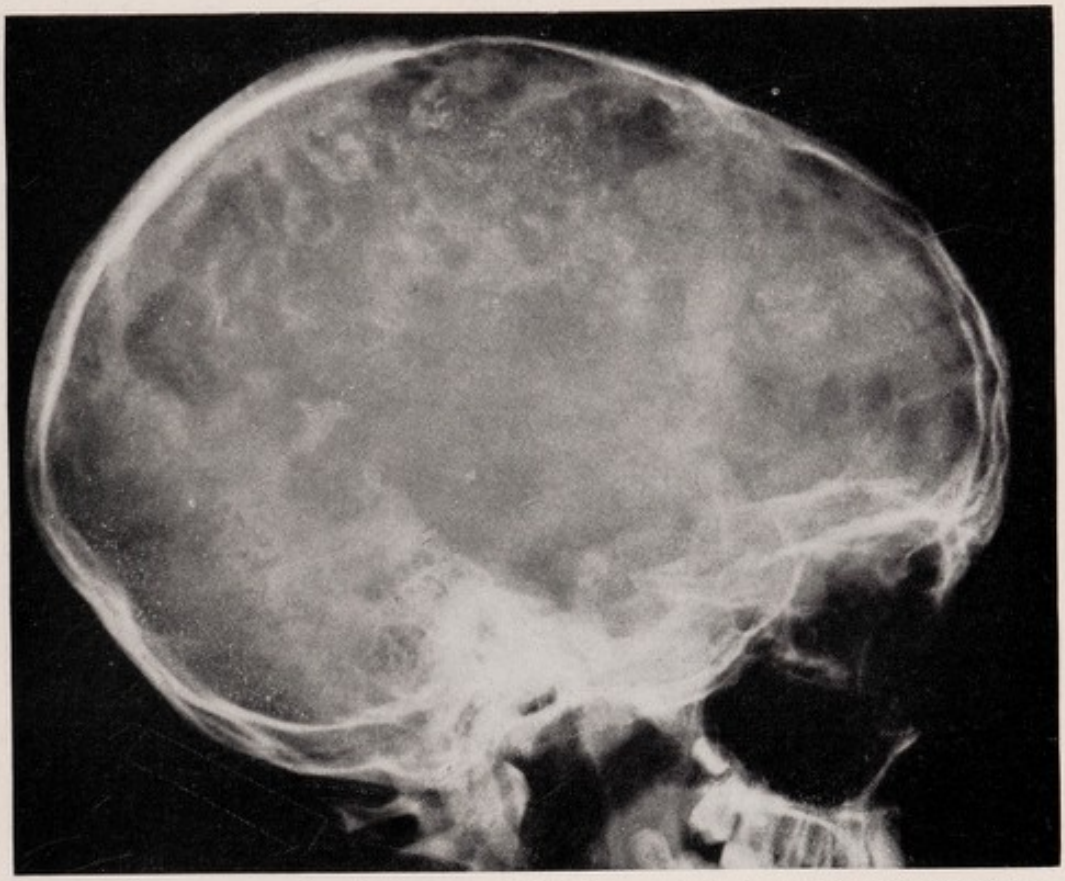


FIG. 5.—Roentgenogram, lateral view, made following removal of sequestrum and dead bone illustrated in Figure 4.

the surface. Then it is possible to drain the abscess and remove the necrotic bone at the same time through the osteomyelitic area. There is a constant danger of producing leptomeningitis in opening the dura in the presence of infected material. There is also the danger of producing an abscess by introducing a needle through infected dura into the brain when searching for a subcortical abscess.

BRAIN ABSCESS.—*Etiology:* Brain abscess results from invasion of the brain by pyogenic organisms from acute or chronic infection of the middle ear; frontal or accessory nasal sinuses; compound fractures and penetrating injuries of the skull; infections of the scalp or osteomyelitis, and from infected emboli arising from bronchiectasis, empyema, endocarditis, and general

continue to extend and may give rise to additional brain abscesses. A number of times I have removed the necrotic bone, resutured the scalp and drained the abscess a week later through a separate incision without lighting up the osteomyelitis (Figs. 4, 5, 6 and 7). There is an occasional exception, namely: when the process has continued for several weeks; when the cerebral symptoms produced by the abscess are very marked; and when the osteomyelitic process is limited and apparently controlled. In those instances the cerebral abscess has been drained first and the osteomyelitis treated following removal of the drains. There is also the occasional case in which the osteomyelitic area is very circumscribed, and the abscess appears to be situated close to



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process and the extent to which it will travel. This being true, active supportive measures should be employed. Administration of vaccines has been suggested and sulphanilamide has been employed, both of which have proved of value. High caloric diets with adequate vitamins are essential. Occasionally local application of heat has aided the circulation and hastened development in the zone of reaction, thus limiting the process.

The accompanying leptomeningitis is a serious complication and is controlled best by repeated or continuous spinal drainage and the administration of sulphanilamide. Again, a nourishing dietary regimen and excellent nursing are material aids in control of the disease.

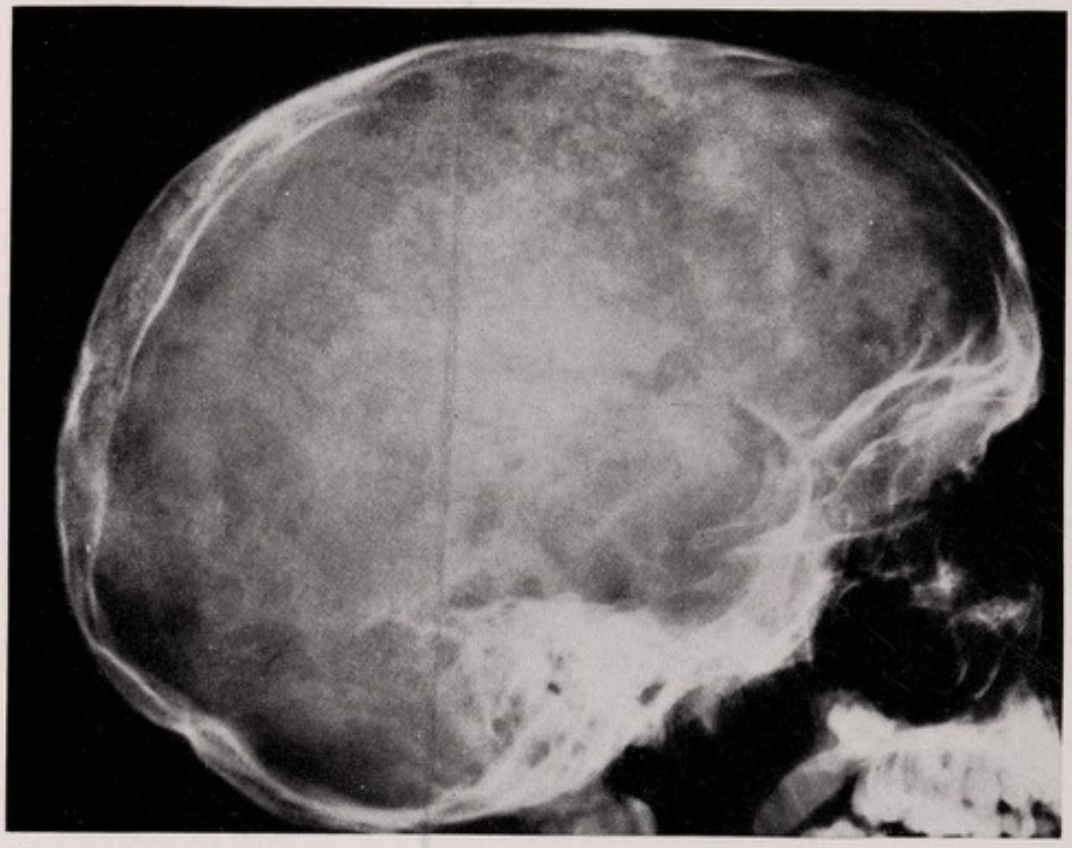

FIG. 4.—Roentgenogram, lateral view, showing extensive osteomyelitis of the left temporal, parietal, and frontal bones.

The cerebral abscess which results from osteomyelitis is one of the suspected and serious complications. When it occurs, the surgeon is confronted with the problem of deciding whether to drain the abscess before treating the osteomyelitis or to treat the osteomyelitis before draining the abscess or, indeed, to attempt to treat both at the same time. A maxim of general surgery should be invoked in the treatment of these lesions, namely: "A patient often will survive two major operations if they are performed separately but may fail to survive if both are attempted at the same time." Too much never should be attempted at one operation. Judging from my own experience, it is better to delay drainage of an abscess than to delay the operative procedure for control of the osteomyelitis. Encapsulation becomes more nearly complete if drainage is moderately delayed, whereas, the osteomyelitis process will

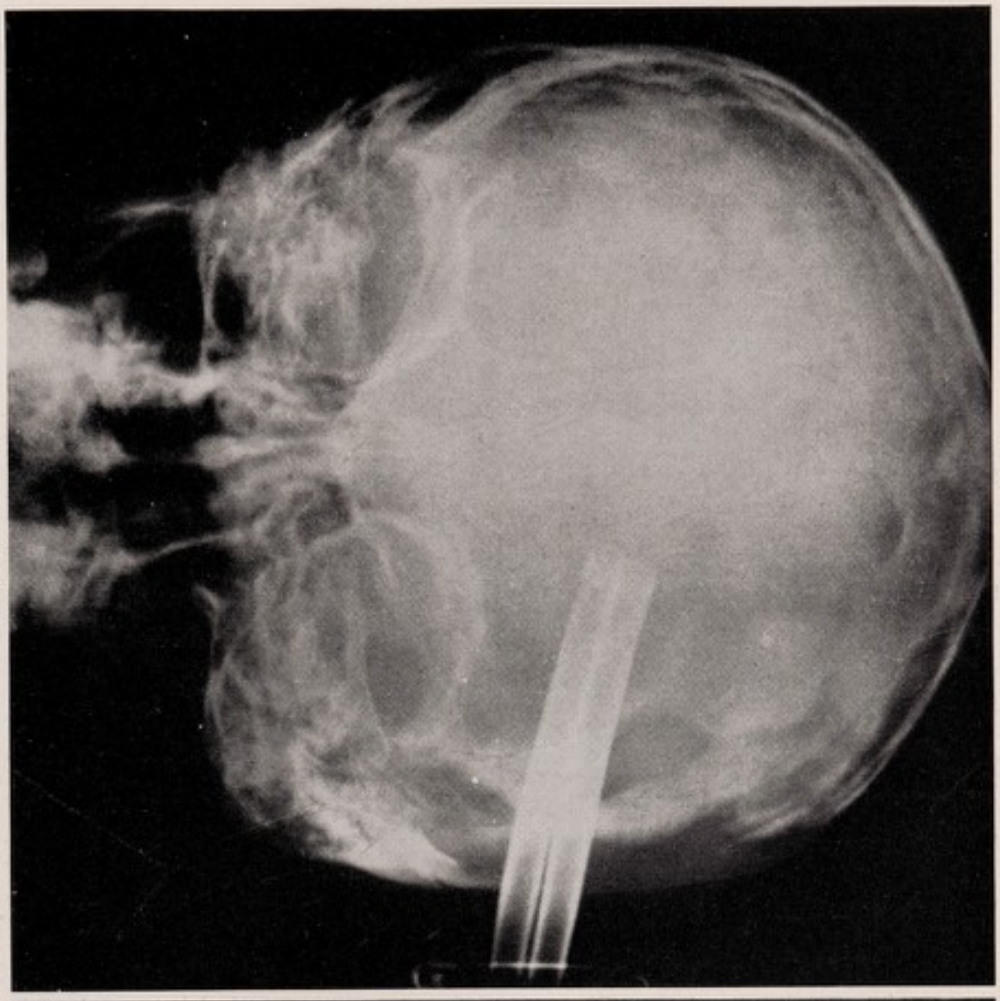

FIG. 6.—Roentgenogram, anteroposterior view, following sequestrectomy and drainage of abscess of left temporal lobe; drainage tubes in place.

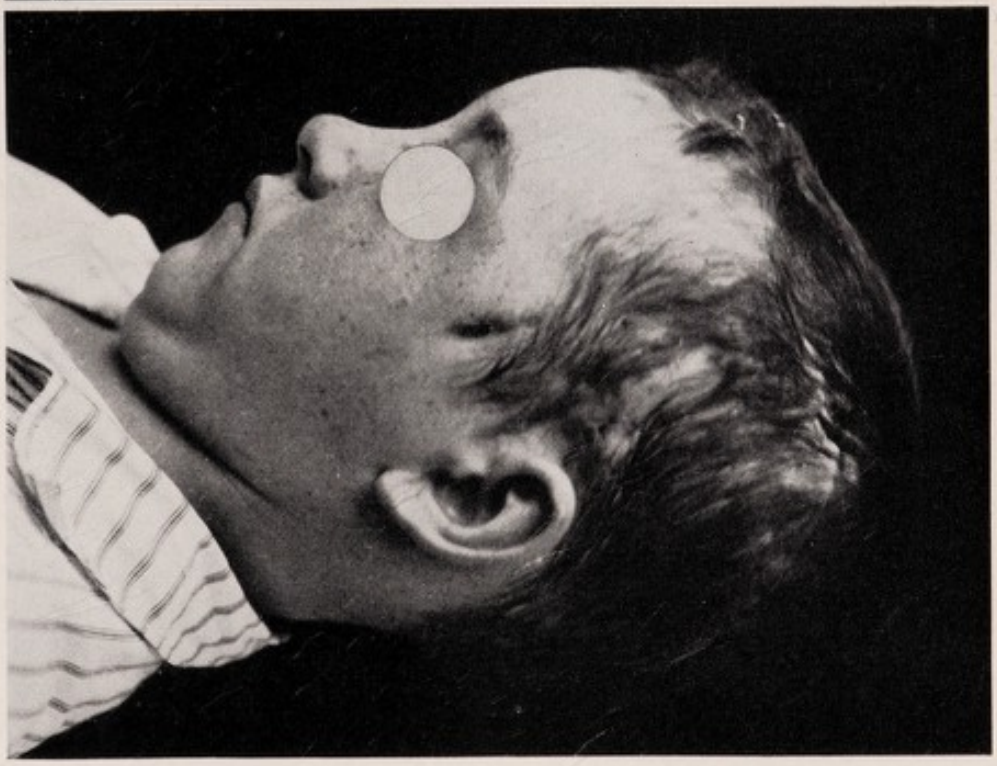

FIG. 7.—Photograph of patient following sequestrectomy and drainage of abscess of the left temporal lobe.

septicopyemia.[5, 16] Although many abscesses result from an extension of the infection by contiguity, a large number develop as a result of retrograde infection of a thrombosed vein within the brain which communicates with veins that drain infected portions of the scalp, skull or meninges.[10] Occasionally, a stalk or channel may be found to extend from the region of suppuration to the abscess but, more often than not, the cerebral abscess is situated apart from the primary focus.[32] They are situated, usually, in the white matter of the brain where the veins originate and where the circulation is less abundant.

Each abscess passes through three stages of development: The initial stage is that of localized encephalitis which might undergo recovery spontaneously, producing a syndrome of pseudo-abscess. The second stage is that of liquefaction and necrosis with encapsulation. As immunity progresses, the general symptoms of infection subside. The abscess becomes quiescent except for the symptoms of intracranial pressure and localization. The third stage represents the terminal phase of activity. Small abscesses disappear spontaneously by inspissation or resolution of the pus. Large abscesses are very likely to rupture into the ventricle or subarachnoid space and produce death unless properly drained. Therefore, appropriate treatment requires proper medical management during the initial stage and the selection of a suitable operation at a time when drainage will be most effective during the second stage. The procedure should afford adequate drainage without spreading the infection or giving cause for recurrence of the same abscess.

Macewen,[31] in 1893, was the first to recognize and localize a brain abscess but, unfortunately, he was not permitted to operate. However, he verified his diagnosis at necropsy. This experience prompted him and others to advise surgical treatment for cerebral abscesses. In reviewing Macewen's book, it is apparent that his good results were owing chiefly to the fact that most of the abscesses were encapsulated. The encapsulation suggests that the patients were brought to him for treatment after abscesses had passed through the initial stage into the second stage of development. At this time, the organisms are less virulent and many have died. The immunity, likewise, has reached its maximal efficiency.

Owing to Macewen's good results, many surgeons advise operation as soon as possible after a diagnosis of brain abscess has been made. However, they fail to recognize the fact that clinical diagnosis has improved since his time and that diagnosis of brain abscess now is frequently made in the initial stage instead of in the second stage. These early operations are responsible for the increased mortality. A suppurative process of the brain is not different from a suppurative process of any other part of the body. Therefore, similar reasoning and similar treatment should be employed. "Wait until the abscess is ripe before opening."

Infective Agent.—The organisms most frequently found in pus removed from cerebral abscesses are *Staphylococcus albus, Staphylococcus aureus,* pyogenic Staphylococci, and hemolytic Streptococci. Other varieties of bacteria

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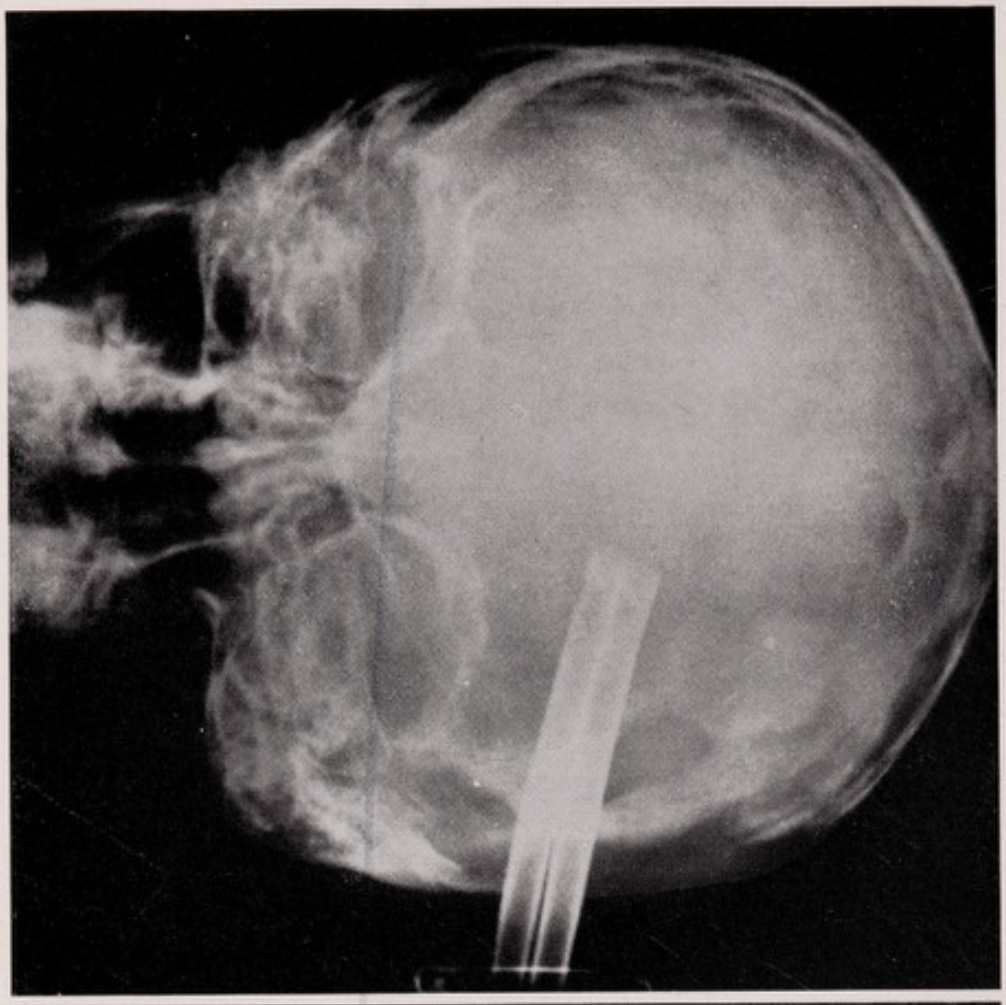

Fig. 6.—Roentgenogram, anteroposterior view, following sequestrectomy and drainage of abscess of left temporal lobe; drainage tubes in place.

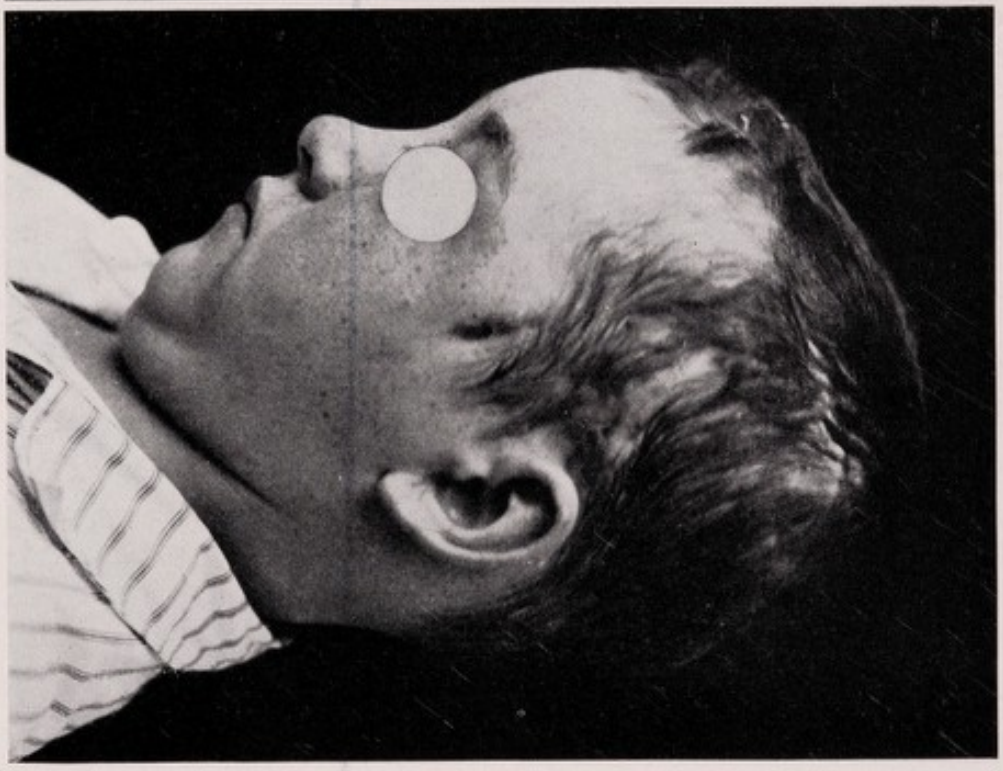

Fig. 7.—Photograph of patient following sequestrectomy and drainage of abscess of the left temporal lobe.

have been found, the nature of which depends on the source and character of the cerebral contamination.

Pathology.—Cerebral abscesses are invariably situated below the cortex.[19] Occasionally, a stalk can be seen to extend from the meninges to the abscess, but more often than not, trace of path of the invasion cannot be demonstrated. The explanation for this, I believe, is that the communicating vein, which served as a path for the organism to invade the white, poorly vascularized brain tissue, has become thrombosed. The stalk, if present, represents the zone of reaction about the infected vein. Following the inoculation of the brain, a localized region of encephalitis results. This process extends by thrombosis of more capillaries and vessels until reaction and immunity limit such extension. The center of the lesion disintegrates, liquefies, and becomes pus. Proliferation in the peripheral zone results in formation of fibrous tissue and encapsulation.

I am convinced that infections may travel through thrombosed veins and diploe for long distances, to produce abscesses without osteomyelitis. This is substantiated by the fact that abscesses do result from infections of the scalp, frontal sinus, and antrum without an accompanying osteomyelitis. The reason cerebral abscesses do not always follow osteomyelitis of the flat bones of the calvarium, frontal sinusitis, disease of the middle ear, or mastoiditis, I believe, is that venous thrombosis is limited by a zone of reaction and a collateral venous circulation. Positive blood cultures are rarely obtained. Symptoms of meningitis frequently accompany cerebral abscesses.

A single embolic abscess is relatively rare. Multiple abscesses follow pulmonary disease, particularly bronchiectasis. Also, they may represent a part of a general septicopyemic infection. The virulence of organisms producing multiple abscesses is usually so overwhelming that death results before encapsulation takes place.

Penfield[34] stated that the wall of an abscess begins to form in the first week but it is not firm enough to offer appreciable resistance to an exploratory needle until two to three weeks have passed. The course without drainage depends on the nature and virulence of the organisms. If an insufficient wall or capsule is formed, spreading encephalitis with edema of the brain may quickly terminate the patient's life.

Extension of Infection by Contiguity.—Extension of infection by contiguity is responsible for a large number of brain abscesses. The otologist often encounters an extradural abscess associated with disease of the temporal bone. Cortical abscesses of the temporal and cerebellar lobes resulting from extending infection of the ear occur but rarely. However, accompanying subcortical abscesses of both lobes are frequent, some of which have produced sufficient reaction to seal the meningeal spaces. When this occurs, it is permissible to drain the abscess at the time of mastoidectomy. If the meningeal spaces are not sealed, the introduction of a cannula from an infected mastoid wound into the brain can give rise to another abscess readily. Furunculosis of the scalp, an infected scalp wound, cellulitis, osteomyelitis, and localized

meningitis are frequent sources of cerebral abscess.[30] Abscesses resulting from infections of sinuses are owing to inward extensions through diploic channels, emissary, dural, and cerebral veins.

Relation of Frontal Sinusitis to Abscess of the Frontal Lobe.—Woodward,[38] in considering the etiology and pathology of osteomyelitis of the frontal bone, has agreed, with others, that the *Staphylococcus pyogenes aureus* is the organism most frequently responsible for the lesion, and has stated that infection of the frontal sinus occludes the ostium and places the pus under pressure. This diminishes the blood supply which, in turn, results in necrosis of the mucous membranes and thrombosis of the perforating veins. From this point, the infection spreads through the diploe, preceded by thrombosis of the diploic veins wherever death of bone has occurred. Pus and granulation tissue fill the diploe, which results in rapid destruction of the osseous elements. Furstenberg[20] has regarded the frontal sinus as eroded diploe, the outer wall corresponding to the external plates and the internal wall corresponding to the internal plates of the frontal sinus, which he believes is the chief reason for extension of the infection of the frontal sinus into the diploic spaces. Because the diploic veins communicate with emissary veins, it is apparent how the infection can reach the scalp or dura. If the infection is overwhelming and cannot be stopped by protective reaction, it reaches the cerebral veins with a resulting cerebral inoculation. Extradural abscesses over the frontal lobes are infrequent but occur often enough to warrant consideration when a diagnosis is being made. Abscesses of the frontal lobe do develop from infections of the frontal sinus without accompanying osteomyelitis, just as they develop from infection of the antrum or ethmoid and from infection of the maxilla following extraction of teeth. These infections undoubtedly follow perivascular spaces and thrombosed veins into the subcortical portions of the brain.

Symptoms.—The symptoms of cerebral abscess vary with the different stages of the disease and vary according to the systemic reaction, the degree of increased intracranial pressure, and the situation of the abscess.

Patients who have a temporosphenoidal or cerebellar abscess invariably give a history of acute or chronic otitis media with a suppurating mastoid. Abscesses in the frontal lobe are preceded by acute colds, frontal sinusitis, pansinusitis, and osteomyelitis of the frontal bone. The sudden increase of septic symptoms following a cerebral injury suggests the possibility of a developing brain abscess. Patients become apathetic and an increase in temperature occurs; the temperature remains high for the first few days but soon takes on the septic, steeple-like contour. The number of leukocytes increases to 20,000 or more. Examination of spinal fluid discloses an increase of pressure, increased concentration of protein, and an increase in the number of lymphocytes.[37] If active, suppurative meningitis develops, polymorphonuclear leukocytes appear in large numbers, causing the fluid to become cloudy. Also, the active organisms usually are identified.

Headaches and vomiting appear early. Irritability, alternating with stupor

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and with rigidity of the neck muscles, is a sign of increased intracranial pressure and meningeal irritation. The pulse is full and bounding; the rate is slower than normal. Choked disks and retinal hemorrhages likewise appear when the normal flow of cerebrospinal fluid has been disturbed.[29]

The localizing signs depend on the size and situation of the abscess. A temporosphenoidal abscess can produce homonymous defects in the visual fields, homolateral palsy of the sixth and third cranial nerves, and contralateral anesthesia, paralysis, and pathologic reflexes. An abscess in the frontal lobe may attain considerable size without producing localizing symptoms.[25] However, I have observed that the initial swelling about one eye is a good diagnostic sign of the lobe affected because the abscess is usually on the same side. Osteomyelitis of the frontal bone invariably extends to one side of the median line, suggesting the lobe involved. Although paralysis may not be present, contralateral reflexes may be exaggerated. When in doubt, it is justifiable to carry out ventricular studies and ventriculography. Cerebellar abscesses produce cerebellar symptoms, plus nystagmus[11] and reduction of homolateral reflexes. Hiccough and rigidity of the neck further indicate a lesion of the posterior fossa. Those miscellaneous abscesses that involve the brain stem and pons present bilateral pyramidal signs and bilateral palsy in the distribution of cranial nerves. Those developing from infected wounds are situated in the vicinity of the infection and those developing from nasal sinuses other than the frontal sinus are situated in the lower half of the frontal lobes.

I have seen temporosphenoidal abscesses and one cerebellar abscess produce homolateral pyramidal symptoms with partial contralateral pyramidal symptoms. The symptoms all disappeared following drainage of the abscess. The homolateral pyramidal symptoms were undoubtedly owing to partial displacement of the hemisphere by the abscess, to the opposite side, to such an extent that the crus cerebri was pressed upon and notched by the opposite margin of the tentorium at the incisura tentorii.

The pathologic process, during the initial stage of three weeks, represents a battle between destructive and reparative forces. The secondary quiescent stage, from the second to the fifth week, represents a partial victory for the forces of repair, because the abscess no longer enlarges and the pus is becoming more securely confined by a fibrous capsule. The edema disappears and circulatory disturbance, peripheral to the capsule, is repaired slowly. The recovery will continue up to five weeks, in the case of large abscesses, when apparently it comes to a standstill. During this quiescent stage the symptoms of sepsis subside and the temperature and number of leukocytes recede to normal or slightly above normal. Headaches improve, mental reactions are faster, and the number of cells in the cerebrospinal fluid returns to normal. Localizing symptoms likewise subside but, in spite of the general improvement, periodic headaches occur and choked disks and defects in the field of vision fail to disappear. Reflexes remain exaggerated and motor impairment persists. In cases of abscess of the frontal lobe, symptoms of euphoria or

depression linger. The number of leukocytes continues to rise to 12,000 to 14,000. The patient, on certain days, is apathetic, refuses to eat, and prefers to lie in bed. If it were not for the history of infection and the symptoms of the initial stage, often it would be difficult to distinguish between those symptoms caused by abscess and those produced by a cerebral neoplasm.

Symptoms may continue throughout the quiescent stage for months, until the patient is operated upon, because of an erroneous diagnosis of brain tumor, or dies from rupture of the abscess into the ventricular system or subarachnoid spaces. Small abscesses heal spontaneously with disappearance of all symptoms. Symptoms of abscesses that have been drained surgically disappear, unless important tracts and cortical centers have been destroyed by the inflammatory process. Mutilating operations likewise contribute to permanent injury of cerebral tissue. Epilepsy is a sequela to localized encephalitis and abscess, and it may appear in any case in which there has been a lesion in the frontal, temporal, or parietal lobes. The incidence of this condition may be lowered by proper and adequate drainage. Subsequent resection of the scar may offer some amelioration and relief of epileptic seizures.

Surgical Considerations.—It took many years to learn that performance of a hasty, emergency operation was futile and was accompanied by a high mortality.[21] This high mortality undoubtedly will follow if the surgeon yields to insistence that he do something as soon as a diagnosis of cerebral abscess has been made.[24] Every cerebral abscess passes through a stage of encephalitis before encapsulation occurs. It is during this stage that the infection is virulent and is disseminated most easily. Some surgeons argue that unless the necrotic tissue is removed the patient will die. This is true in the occasional case, and cases have been reported in which aspiration of necrotic material was successful; more often than not, however, the infection is disseminated by surgical intervention and the patient dies from fulminating, suppurative meningo-encephalitis. I believe the best procedure to employ during the acute stage is supportive treatment, rest in bed, high caloric diet, spinal drainage, ice bags to the head, frequent catharsis, moderate amounts of fluids, and, if the patient is comatose, occasional intravenous administration of an hypertonic solution of glucose.

Encapsulation takes place in two to four weeks. The process is an indication that immunity is being established. It is characterized by a decrease in the number of leukocytes to 12,000 to 14,000. The temperature, likewise, returns approximately to normal, 100° F. (37.8° C.), or lower. The number of cells in the cerebrospinal fluid, if increased, returns to normal. The cerebral symptoms gradually subside, but seldom disappear completely until the abscess is drained. Choking of the optic disks, if present, may continue until optic atrophy results. When encapsulation and immunity have been established, thorough and continuous drainage is necessary to effect a cure without recurrence of the abscess.

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and with rigidity of the neck muscles, is a sign of increased intracranial pressure and meningeal irritation. The pulse is full and bounding; the rate is slower than normal. Choked disks and retinal hemorrhages likewise appear when the normal flow of cerebrospinal fluid has been disturbed.[29]

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I have seen temporosphenoidal abscesses and one cerebellar abscess produce homolateral pyramidal symptoms with partial contralateral pyramidal symptoms. The symptoms all disappeared following drainage of the abscess. The homolateral pyramidal symptoms were undoubtedly owing to partial displacement of the hemisphere by the abscess, to the opposite side, to such an extent that the crus cerebri was pressed upon and notched by the opposite margin of the tentorium at the incisura tentorii.

The pathologic process, during the initial stage of three weeks, represents a battle between destructive and reparative forces. The secondary quiescent stage, from the second to the fifth week, represents a partial victory for the forces of repair, because the abscess no longer enlarges and the pus is becoming more securely confined by a fibrous capsule. The edema disappears and circulatory disturbance, peripheral to the capsule, is repaired slowly. The recovery will continue up to five weeks, in the case of large abscesses, when apparently it comes to a standstill. During this quiescent stage the symptoms of sepsis subside and the temperature and number of leukocytes recede to normal or slightly above normal. Headaches improve, mental reactions are faster, and the number of cells in the cerebrospinal fluid returns to normal. Localizing symptoms likewise subside but, in spite of the general improvement, periodic headaches occur and choked disks and defects in the field of vision fail to disappear. Reflexes remain exaggerated and motor impairment persists. In cases of abscess of the frontal lobe, symptoms of euphoria or

scess.[2, 3, 6, 8] Exceptions to this rule are when it is desirable to avoid a scar in the frontal region or when it is necessary to pass through a zone of osteomyelitis to reach the abscess. In entering the skull through a clean field, the site is chosen where the abscess is nearest the cortex or which will give the best drainage.[13] A small incision, 5 cm. in length, is made in the scalp. A craniotomy 3 cm. in diameter is usually large enough to afford ample exposure. The meninges and cortex are then sutured with interrupted stitches of catgut about the margins of the decompression to prevent separation of the cortex from the dura when intracranial pressure is relieved by draining the abscess. The meninges and cortex are further sealed and glued together by use of the electrocoagulating current. A crucial incision is made in the dura to expose an area of brain about 2 cm. in diameter. The margins of the wound are covered with wet strips of cotton in order to minimize the spread of pus between the dura and skull. The cortex overlying the abscess is frequently edematous and cyanotic in appearance.

A round-tipped brain cannula is used to locate the abscess. The resistance of the capsule of the abscess gives one the impression that the cannula is being placed against a flexible, hollow rubber ball. If the abscess has been there for a long time, the resistance may be so great that it is impossible to penetrate the capsule without incising it. Small abscesses may be overlooked, because the firm capsule is capable of deflecting the cannula unless it is directed toward the center of the abscess. As soon as the cannula enters the abscess, the trocar is removed and a Luer syringe, with an intervening rubber connector, is attached to the cannula. The pus is gently aspirated and the cavity is cleansed with small quantities of physiologic saline solution. The cavity of the abscess is explored by incising the cortex and capsule with an electrosurgical needle, using the cannula, which has been left in place, as a guide. The capsule is opened for a distance of 2 cm. in order to insert an illuminated retractor. This makes intracapsular exploration possible, permits further cleansing of the cavity of the abscess, and assures against overlooking pockets communicating with the abscess. During this procedure the capsule is retracted outward against the cortex and skull until the cavity is packed, thus preventing retraction of cerebral structures away from the skull.[18]

To assure against collapse of the cortex following drainage of an abscess, I fill the cavity, about the two inserted tubes, with loosely packed strips of iodoform gauze. This continues to keep the capsule moderately distended, instead of allowing it to crumple and give rise to loculated pockets within the capsule. The strips of gauze are shortened daily until they are removed on the tenth day. The gradual collapse and contraction of the capsule prevent recurrence of abscesses and the development of cerebral fungi.

The two tubes are sections of catheters. They are left undisturbed until after the gauze has been removed. One is shortened on alternating days until removed on the twenty-first day; the other is shortened as the sinus closes in and forces it out, which requires from four to six weeks. At operation, the tubes are fastened to the skin to prevent accidental removal during the daily

withdrawal of gauze. Following removal of stitches, the tubes are prevented from falling out by transfixing the exposed ends with safety pins and fastening these to the skin with strips of sterilized adhesive tape. Strips of vaselined gauze are placed over the margins of the wound to prevent the gauze dressings from adhering to the wound. The second tubular drain occasionally is exchanged for a smaller one after the third week but at no time is the cavity irrigated, for fear of disseminating the infection. The patient is allowed to get out of bed after the second day and to leave the hospital after two weeks. His subsequent dressings are done at the office.

Other Technics.—Aspiration by needling has a limited field of usefulness.[17] It is most useful in draining small, sterile, deeply seated abscesses. One aspiration, or two, may be sufficient to drain a sterile abscess but, when the organism still remains active, refilling continues until adequate, continuous drainage has been instituted. There is also danger of spreading the infection by repeated aspiration, as it is impossible to insert and to withdraw the needle through the same tract.

Treatment of the Capsule.—Again I find surgeons divided in their opinions as to the best treatment of the capsule. It must be accepted that if the capsule is left in place, it will result in a fibrous scar. Although this does occur, can it be avoided, and should the capsule be removed at the time of the initial operation or at a later date? Macewen,[31] Bagley,[9] Hassin,[23] Cone[14] and many others have demonstrated the pathologic changes that develop to form a capsule about the abscess. From clinical experience, it is apparent that the capsule continues to thicken for several months if the abscess is unrecognized. Therefore, it is fair to assume that the walls will collapse more readily and will be thinner if the abscess is drained properly as soon as immunity and encapsulation have taken place. To assure collapse of the capsule, King[26, 27] and Cahill[12] have suggested removal of the overlying cortex and the peripheral dome of the capsule. King and others have suggested removal of the capsule at the initial operation. This procedure hastens recovery when it is possible to remove the abscess and capsule without opening it. Bagley reported such a case and I removed such an abscess but was unaware that I had done so until the mass was opened later. The wound, in this case, healed per primam without any drainage. My experience with removal of capsules following evacuation of the pus, however, has not been satisfactory, because secondary suppurative encephalitis develops that is more troublesome to treat than the original infection. If drainage is not instituted, pus becomes inspissated and forms the caseous center of a fibrous mass. The mass will contract gradually until there remains but a small nodule of what once was an abscess, 3 to 5 cm. in diameter. The larger abscesses are more likely to rupture into the subarachnoid spaces and ventricular system than the smaller ones; consequently, it is unsafe to wait for these to disappear spontaneously. I cannot concur with those who believe that the rigid capsules will not collapse, for experience has demonstrated that all have collapsed, if properly drained. The difficulty encountered with recurrent abscesses, attributed to failure of the capsule to collapse, I believe is owing to failure to secure adequate and continuous drain-

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SUMMARY AND CONCLUSIONS

The surgeon should employ supportive measures, such as high caloric diets; also, when the infection is the result of Staphylococci or Streptococci invasion, occasional administration of vaccine and sulphanilamide are helpful.

Osteomyelitis of the skull should be treated similarly to osteomyelitis of other bones; this treatment consists of thorough sequestrectomy and removal of all dead bone. The wound should be cleansed with tincture of iodine and, if drainage is instituted, the drain should be removed within 48 hours and the scalp closed with sutures of silkworm gut.

Mortality will be lowered in the treatment of cerebral abscess if the surgeon employs some of the same principles that are employed in the treatment of suppurative lesions elsewhere in the body.[21]

In cases of suspected cerebral abscess resulting from infections about the ear, with indefinite localizing symptoms, or with localizing symptoms and signs that are conflicting, I have observed the rule of exploring the temporosphenoidal lobe before exploring the cerebellum on the side of the infected ear because of the higher ratio of incidence of temporosphenoidal abscess.

If, on study of the physical and neurologic signs, I fail to localize a suspected abscess, performance of cerebral pneumography is justifiable.

Adequate and continuous drainage should be instituted after encapsulation has taken place.

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DISCUSSION.—DR. FRANCIS C. GRANT (Philadelphia, Pa.): Doctor Adson has called attention to two very important points in the treatment of osteomyelitis: First, the retention of the pericranium, because from that pericranium, new bone will regenerate. I should like to detail a case which will substantiate Doctor Adson's opinion. I should also like to ask Doctor Adson about his treatment of the more acute osteomyelitic cases. We see, through the nose and throat service, a good many cases of acute frontal sinusitis which develop into acute osteomyelitis of the frontal bone and spread rapidly through the bone. That is the type of case of osteomyelitis which certainly, in our opinion, is very much more difficult to handle than are the chronic cases.

A female, age 12, was admitted to the hospital in 1934, following bilateral frontal sinusitis resulting from diving. She developed an acute osteomyelitis which involved both frontal bones. We had to take off all of her frontal and temporal bones on either side. This was accomplished in two stages, necessarily so, because her condition was poor, but we finally were able to get beyond the edge of the infected bone. These operations were performed in January and February of 1934. After a prolonged convalescence the wounds healed. Skin grafting was necessary.

Roentgenologic studies, in May, 1934, showed that nearly all of both frontal bones and over half of each temporal bone had been removed. But the pericranium had been preserved. Subsequent roentgenograms, in October, 1936, showed practically complete regeneration of the débrided bone. (Case of Gunshot Wound of Head and Case of Osteomyelitis of Skull. ANNALS OF SURGERY, **102**, 473–475, September, 1935.)

I should like to ask Doctor Adson one question concerning the treatment of subcortical abscess in the brain. Does he believe in cortical incision with subsequent insertion of packing into the abscess cavity or in simple tap and insertion of tube drainage?

We reviewed 31 cases that were available in the Neurosurgical Clinic, from the standpoint of morbidity, to see which group of patients had the most satisfactory final result.

We believe that the smaller the opening in the cortex to tap, or tap and drain the abscess, the more satisfactory results you will obtain, when these patients are reviewed a year or two later. We found that of 23 cases treated by tap or tap and drain, but five had hemiparesis, convulsions or other symptoms of a serious nature. In eight cases, in which a cortical incision had been made to introduce drainage, seven had a hemiparesis or a history of convulsive attacks.

I am not referring to the immediate mortality but entirely to the morbidity, and my impression is, in the treatment of abscess, if you can handle the case successfully by tap or tapping and tube drainage, the eventual results seem to be very much better.

Dr. Gilbert Horrax (Boston, Mass.): There is just one aspect of Doctor Adson's paper on which I would like to comment, and that is the treatment of the brain abscess. I feel as he does, that we have three available methods, the first of these being the simple one, which Doctor Grant has mentioned, of tapping the abscess and seeing if it is a type with which you can deal in that way. If the abscess is sterile, or in some instances if it is not sterile and can be frequently tapped, this will be sufficient, and I think undoubtedly the sequelae are less.

On the other hand, it has been my experience, as it has that of others, that there are many abscesses in which tapping is not curative. They show signs of increased brain pressure again very soon in spite of frequent tappings, and one must do something more radical for them.

In our series of 18 chronic abscesses, there were three that were handled successfully by tapping. Many of the others were tapped several times and then we had to do something more serious and more radical with them.

The second method is the open method of drainage which, as Doctor Adson says, was founded by Macewan, and if one goes back to his treatise, as we all do who are dealing with this sort of thing, one will find that his great success was not only the fact that the abscesses were long-standing and well encapsulated, but that he did use this wide open drainage, that is, a relatively wide opening, which Doctor King subsequently adopted in this country in a modified form, and with which he has been so successful.

I think the principle is the same, whether one uses Adson's, King's or Doctor Cushing's method, which consists of marsupialization of the abscess. The latter I have found more successful because many of these abscesses are near enough to the surface and the capsule is of such great strength that you can take sutures through it and sew it to the galea, and thus make a pouch of the cavity and have it entirely extracranial.

I have not seen any cases in which it was necessary to seal off the meninges. I have never seen a case where infection took place by meningitis in that way. It is always by an extension of an osteomyelitic process or by rupture into the ventricle. If the abscess is near enough to the surface, the thing to do, as Doctor King does, is to uncap that area, get down to the abscess and empty a part of its contents. The capsule will then protrude toward the surface, so that one can put in sutures and sew these to the galea, and have a perfectly outside tube, so to speak.

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DISCUSSION.—DR. FRANCIS C. GRANT (Philadelphia, Pa.): Doctor Adson has called attention to two very important points in the treatment of osteomyelitis: First, the retention of the pericranium, because from that pericranium, new bone will regenerate. I should like to detail a case which will substantiate Doctor Adson's opinion. I should also like to ask Doctor Adson about his treatment of the more acute osteomyelitic cases. We see, through the nose and throat service, a good many cases of acute frontal sinusitis which develop into acute osteomyelitis of the frontal bone and spread rapidly through the bone. That is the type of case of osteomyelitis which certainly, in our opinion, is very much more difficult to handle than are the chronic cases.

A female, age 12, was admitted to the hospital in 1934, following bilateral frontal sinusitis resulting from diving. She developed an acute osteomyelitis which involved both frontal bones. We had to take off all of her frontal and temporal bones on either side. This was accomplished in two stages, necessarily so, because her condition was poor, but we finally were able to get beyond the edge of the infected bone. These operations were performed in January and February of 1934. After a prolonged convalescence the wounds healed. Skin grafting was necessary.

Roentgenologic studies, in May, 1934, showed that nearly all of both frontal bones and over half of each temporal bone had been removed. But the pericranium had been preserved. Subsequent roentgenograms, in October, 1936, showed practically complete regeneration of the débrided bone. (Case of Gunshot Wound of Head and Case of Osteomyelitis of Skull. ANNALS OF SURGERY, **102**, 473–475, September, 1935.)

The third method is the one which Clovis Vincent has been advocating so much recently, of extirpating the abscess. We all run into these occasionally and I think that is a method which is very good at times, if the abscess is so situated that one can do it safely; but I do think if one can make use of the simpler methods, the sequelae are going to be less.

Dr. James Monroe Mason (Birmingham, Ala.): I was much interested in the remarks concerning osteomyelitis following denudations of large areas of the skull. Some years ago, it fell to my lot to care for two men who, within a few days of each other, received electric burns involving, in one instance, a large area of the scalp over the left parietal bone, and in the other, large areas over the frontal and occipital bones.

The progressive development of the osteomyelitis in the three bones followed along lines which so closely paralleled each other that it suggests what may be expected to take place whenever large areas of the bones of the vault of the skull are denuded of their coverings.

In the burns involving the frontal and occipital bones, the soft parts soon sloughed off and were trimmed away and the underlying bone was dressed antiseptically until such time as the sequestrum should become loosened. This took place within a few weeks, and the loosened bone was lifted off. We found, in each instance, that the entire outer table had become detached, but that the inner table was viable and remained in place except for a small area near the center of the wound. The entire thickness of bones was necrotic and came away with the detached outer table, leaving the dura exposed at this point. The wounds were covered with grafts and the patient recovered.

In the case involving the parietal bone, the same plan of treatment was employed, but the outcome was not so fortunate. About three weeks after the receipt of the injury, the patient suddenly developed hemiplegia and died very shortly from a large brain abscess. Autopsy revealed a similar condition in the progress of the osteomyelitis which we had observed in the other case, namely, that the entire outer table was becoming necrotic, that the greater part of the inner table was resisting the process, but that a point at the center of the involved area of bone was also necrotic. The progress of the infection went entirely through the bone at this point and a small subdural abscess had developed. This quickly extended to the brain and a large abscess was found in the parietal lobe.

Apparently, in large flat bones of the skull the inner table receives sufficient nutrition from vessels surrounding the involved area to insure its vitality when the outer table is denuded, but the circulation near the central part of the inner table may not be sufficiently active for its preservation, and necrosis follows, allowing infection to come directly in contact with the dura.

It is suggested that in large denudations, sections of bone corresponding to the center of the area be removed at once, in order that the tendency to necrosis of the inner table at this point be checked and the danger of the development of brain abscess be lessened.

Dr. Alfred W. Adson (closing): In reply to Doctor Grant's question, may I state that when the osteomyelitis is limited to the tables of the frontal sinus the lesion is treated by the otolaryngologist. If the lesion extends to the tables of the frontal bone, the operation is divided into two portions. The disease of the frontal sinus is taken care of by the otolaryngologist and the disease of the frontal bone, by the neurosurgeon.

I prefer to seal the meninges to the brain, as described, rather than to employ the technic of extirpation of the abscess, since thus there is less danger of contaminating the subarachnoid spaces. This is especially true of cerebellar abscesses.

RAPID CONTROL OF INTRACRANIAL PRESSURE

ERWIN R. SCHMIDT, M.D.

MADISON, WIS.

FROM THE DEPARTMENT OF SURGERY, UNIVERSITY OF WISCONSIN MEDICAL SCHOOL, MADISON, WIS.

THE present methods for the control of increased intracranial pressure require some time to become effective and depend upon certain physiologic principles. Even if these principles may be questioned on experimental and clinical grounds,[1, 2, 3, 4, 5, 6] from clinical evidence the mortality rate has declined under this method of treatment. Munro,[7] in 1925, reported a series of cases collected from the literature with a mortality rate of 37.8 per cent.

Fay[8] reported a series of cases in which those patients who survived six hours after admission showed a 10.4 per cent mortality, and Ochsner[9] reports a total mortality rate of 8.4 per cent. By excluding those patients who died in the first 24 hours, a mortality rate of 3.9 per cent was obtained. The method for controlling intracranial pressure we wish to present depends on mechanics alone, is rapid, and we think effective in most cases. Within the cranial cavity there are three substances that are important. Munro[10] pointed this out in 1783, and Kellie,[11] in 1824, elaborated the idea. These are the brain substance, the blood vessels with their contained blood, and the cerebrospinal fluid. A change in the volume of one means a change must occur in the volume of one or both of the other substances. The result is that the pressure in the blood vessels, especially the veins, as was pointed out by Grinker,[12] is increased and this pressure causes a rise in the systolic pressure with a slow, full pulse, with an increased pulse pressure, in the systemic circulation. Cushing called attention to the fact that our museums have many examples of fractured skulls, but there were very few examples of injured brains, and that after all the brain itself was more important than the fracture. While our clinical evidence indicates that the mortality rate is improved, little or no attention is paid to the morbidity resulting from cranial injuries.

Lowenberg, Waggoner, and Zbinden,[13] Caine,[14] Davies,[15] Hahn,[16] and McKean Downs[17] have called attention to the effect of anoxia on the brain in nitrous oxide anesthesia. Lowenberg, Waggoner, and Zbinden report three cases in which there was destruction of the cortex and basal ganglia following the use of nitrous oxide oxygen anesthesia, and one case with clinical evidence of a similar process. These authors point out that death occurs at varying times following the start of the anesthesia, varying from 20 seconds to an hour and a quarter; or it may be hours, days, or weeks before death takes place; or the case may recover with residuals present after years. Signs of anoxemia do not necessarily have to be present. Lowenberg, Waggoner, and Zbinden consider two possibilities: (1) Asphyxia; and (2) toxic effect of the gas. They find support for both ideas, but believe that the toxic effect of the gas is the more likely cause of the destruction.