Neurosurgical Classic—IX

ROBERT H. WILKINS, M.D.
Division of Neurosurgery, Duke University Medical Center, Durham, North Carolina

On July 21, 1876, an 11-year-old boy was admitted to the Royal Infirmary in Glasgow with the history of a head injury suffered in a fall 2 weeks previously. A small suppurating wound was present over the left eyebrow, and the boy was lethargic and febrile. Twelve days later he had a right-sided convulsion with a transient aphasia. William Macewen diagnosed a cerebral abscess, which he thought was located in the third left frontal convolution. He proposed to evacuate the abscess through a trephine opening, but the patient’s friends refused to give permission. After the patient’s death a short time later, Macewen received permission to perform the proposed operation just as he would have if the boy had been alive. Through a temporal trephine opening, the dura mater was incised and a narrow bismor was inserted into the brain for an inch and a half in the direction of the third frontal convolution. Pus welled out along the bismor, confirming the diagnosis, and a later postmortem examination revealed an abscess the size of a pigeon’s egg in the white matter of the second and third frontal convolutions.1,5,6

Macewen was convinced that this boy died unnecessarily, as did most patients with brain abscess. Over the ensuing years he attempted to reverse this hopeless situation by energetically applying to the problem his considerable skill and judgement, basing his actions on the evolving principles of the new science of bacteriology. The extent of Macewen’s success became apparent in 1893, when he published Pyogenic Infective Diseases of the Brain and Spinal Cord.6 In this work, which has remained a classic for over 70 years, Macewen presented his results in 94 patients with intracranial infections. Of these there were 5 with extradural abscesses who were operated upon, with 5 recoveries, and 19 with brain abscesses who were operated upon, with 18 recoveries. Since then these results have remained a challenge for surgeons treating intracranial abscesses.2,4,7,8

In an evaluation of Macewen’s work, Geoffrey Jefferson stated:

“The magnitude of this achievement is enhanced if we remind ourselves that although surgeons had sometimes operated upon the mastoid, the first clear description of mastoiditis as a definite entity had been given only as recently as 1877 by Frederick Bezold. Its surgery had only just been released by Lister’s work. Credit must be given to Macewen therefore as a pioneer in correct mastoid surgery, quite apart from his contributions to the complications that arise from middle ear disease. . . .

“My own views on the reasons for Macewen’s success with abscesses are that he was his own operator both on the mastoid and the abscess. . . . At the same time a considerable experience of intra-cranial abscess convinces me that Macewen could not possibly have kept his record unless he had continued to remain firm enough to reject, as he did, twenty per cent of the cases.”

Excerpts from Pyogenic Infective Diseases of the Brain and Spinal Cord are reprinted below.

References
7. Webster, J. E., and Gurdjian, E. S. The surgical management of intracranial suppuration. Methods in
CHAPTER II. PATHOLOGY OF CEREBRAL ABSCESS AND MENINGITIS.*

The pathology of abscess of the brain is so closely interwoven with that of infective meningitis, and the one so frequently accompanies the other, that the pathological aspects of both are dealt with in the same chapter.

In a given case, where a cause of infective inflammation exists on the outside of the skull, from which the interior becomes affected, the alternative, whether meningitis or brain abscess results, depends partly on the anatomical arrangement of the structures, and partly on the intensity and rapidity of the inflammatory action: which again, may be dependent upon the nature of the micro-organism and the virulence of its action. One, or more, of several intracranial conditions—pachymeningitis, lepto-meningitis, ulceration of the brain, abscess of the brain, and necrosis of the cerebral tissue, besides the involvement of the intracranial sinuses with disintegrating thrombosis—may result therefrom.

Lepto-meningitis and cerebral abscess may form independently of a visible tract of inflammation spreading inwards from the initial focus of irritation outside the cranial cavity. In such cases, the pathogenic cause has been conveyed through the vascular system by direct extension from the source of infection to the meninges or brain. This may be done by a thrombus extending through the veins into the pia or the brain; or the veins may become blocked by a localized disintegrating thrombus, portions of which, containing pathogenic micro-organisms, may be carried inwards by the reversed blood stream. If it should involve the arterial contents, a localized thrombus may result, of which, if it disintegrates, a portion may be carried from the larger vessel into the terminal capillaries in the white cerebral substance, where it would set up minute infective haemorrhagic extravasations, round which an abscess might form. . . . Inflammation may also spread through the lymphatic vessels to the brain. The perineural sheaths may likewise be the path along which the micro-organisms travel, though one very seldom finds cerebral abscess forming by perineural extension, lepto-meningitis being there the rule. . . .

In the great majority, if not in all cases, the peccant matter, which occasions both acute lepto-


meningitis and brain abscess, is organismal. Whether the various forms of intracranial disease have each a distinct specific organism which produces it, and it alone, is for the future to decide. It is, however, probable that the same organism may induce several of these intracranial lesions, the modifying circumstances being, the degree of intensity of its action, its opportunities of access to the intracranial contents afforded by the patho-logico-anatomical condition of the parts, and the degree of the restraining force of the living tissues presented by the individual.

The free communication between the naso-pharynx, the tympanum, and the mastoid antrum, is such that diseases due to micro-organisms arising in the first-named part, not infrequently affect the others, while, when the mem-brana is softened by disease and perforation ensues, organisms have free access to the tympanum directly from without. When the middle ear has become inflamed, and its mucous membrane is converted into granulation tissue and purulent exudation issues from it; then it forms a model incubating chamber for micro-organisms, which find there abundant pabulum, ever freshly poured out, and an even, temperature uniformly favourable for their development. Once they have penetr-ated into the mastoid antrum and the adjoining mastoid cells, they are so far removed from the influence of antiseptics applied to the middle through the external ear, that they rest secure and fructify uninterruptedly as long as pabulum is forthcoming. . . .

The pathogenic organisms found in suppurative lepto-meningitis and brain abscess, as tested both by the microscope and by careful plate cultivations in the author’s cases, have most frequently been the streptococcus pyogenes and the staphylococcus pyogenes aureus. . . . When pus in the intracranial suppurative lesion has been compared with that emanating in the same case from the exterior wound or otorrhoea the same organisms have frequently been found in both. . . .

The mere chronicity of an otorrhoea, without extension of serious disease, is no guarantee to the individual of future immunity. Patients so affected are at any time liable to the rapid onset of dangerous sequelae. This may arise from fresh inoculation of the discharges in the middle ear, or from the exposure of fresh areas of the membranes, brought about by progressive disintegration and the implication of these fresh areas in the pathogenic process.

Though acute attacks of infective inflammation of the skull, such as from compound fractures, frequently lead to intracranial extension of the pathogenic process, yet primary acute attacks of otitis media are rarely followed by extension of the pathogenic process to the brain or its membranes. This immunity of the cerebral structures
from implication in presence of primary though severe attacks of otitis media, is accounted for by the fact, that the bone and the mucous membrane of the middle ear are still intact, and present a barrier to infection travelling inwards. The virulence of the inflammation might, however, even under such circumstances produce thrombosis of a sinus or give rise to other intracranial complication. 

... Caries in the tympanic cavity does not usually affect the bone equally all round, but extends in certain directions. When it extends through the antrum and involves the mastoid cells the inner walls of some of which are very thin and contiguous to the sigmoid groove, the latter is apt to be involved and exposed. When it attacks the roof of the middle ear, the tegmen, though a dense plate of bone, yet is thin, and when once ulceration does secure a hold upon it, perforation of the middle fossa of the skull frequently results. When the tegmen is in this thinned brittle condition it is very apt to break on slight concussion, produced by blows on the head, or by vibration of the hammer and chisel when used in opening sclerosed mastoids. When the molecular disintegration of the middle ear proceeds inwards and forwards, the whole of the interior of the petrous bone may become, in extreme cases, hollowed out, leaving a shell externally. The labyrinth—the cochlea, and the semicircular canals—being encased in harder bone, often resist the disintegrating process, though they also are occasionally invaded. 

... The most frequent seats of perforation from middle ear disease are the tegmen over the tympanum or antrum and the sigmoid groove. When an abscess follows the former it is situated in the temporo-sphenoidal lobe, and when it follows the latter the abscess is generally located in the cerebellum. 

... When the dura mater has been exposed by osseous erosion it throws out in advance of this lesion a mass of granulation tissue from its external surface, which not only offers obstruction to the further inroads of the pathogenic process, but also assists in the absorption of the disintegrating bone. Such masses of granulation tissue, projecting into the middle ear from the dura through the erosion in the tegmen, have been mistaken for so-called “aural polypi” and have been abruptly or snared, and not infrequently serious intracranial inflammatory action has followed, apparently as the consequence. Similar granulation masses are to be found issuing from the layer of dura forming the wall of the sigmoid sinus through the sigmoid groove into the mastoid cells. These masses of granulation tissue are usually surrounded with pus, a portion of which is pent up intracranially, forming an extradural abscess, the contents of which ooze out if the neck of the granulation mass, as it issues through the erosion, be pressed gently aside. Occasionally, however, the granulation tissue may be accompanied by very little pus, the erosion taking place with all the greater insidiousness. In not a few cases there exists a small necrosis of the bone in the tegmen tympani or sigmoid groove. The extradural abscess forming on the inner side of it... 

... the softened brain tissue, in contact with the infective nidus, is prone to purulent disintegration forming a cerebral ulcer, and the pus ensuing from the ulceration being confined between the soldered membranes and the brain will probably be described as an abscess, especially when met with during an operation. The formation of a true cerebral abscess, however, occurs by a further extension from this pial accumulation and softened cerebral tissue along the paths of the cerebral vessels, which penetrate the white substance of the brain. The infective virus acting upon the vessels, and causing oedema of the brain with leucocytal exudation, often mingled with extravasation of red blood corpuscles, soon forms an area of purulent encephalitis. In the majority of fully-formed abscesses in the temporo-sphenoidal lobe, an opening into the abscess cavity exists at the base of the brain, which indicates the point of brain indentation caused by the infected conical projection in the membranes... These openings may be regarded as the area of initial invasion, and not merely as due to a secondary process of absorption of the abscess wall. Such openings in the base of the brain connected with abscess in the temporo-sphenoidal lobe are almost always located in the same place... so that, as a rule, in cases of cerebral abscess the path by which the infective processes spread from the tegmen to the brain can be traced, and the majority of abscesses develop by direct extension from the infective seat. It is therefore of great importance, in the treatment of such cases, besides dealing with the abscess, that the point of origin of the infective processes in the tissues should be sought for and their paths of extension traced, in order that they may be eradicated.

The statement that disease of the tympanum causes cerebral abscess chiefly, while disease of the mastoid cells causes cerebellar abscess, is misleading, inasmuch as the tympanum is diseased in both forms; and in the majority of cases in which the mastoid antrum and cells are affected, grave intracranial disease of the middle fossa results. The tegmen tympani is often eroded at the same time as the involvement of the sigmoid sinus occurs, and cerebral abscess in the temporal lobe is not infrequently met with, along with infective sigmoid sinus thrombosis. 

It must be admitted that there are cases in which abscess of the brain may find an exit through the erosions set up by the ulceration. 


As a rule these abscesses cannot empty themselves owing to the tortuous passages through the erosions filled with granulation masses, and on account of the resistance in their walls when encysted. Most of them end fatally when not relieved surgically. . . .

When a pathogenic embolism occurs in a cerebral artery or vein situated in the white substance of the brain, haemorrhagic extravasation ensues, accompanied or shortly followed by an exudation of leucocytes, which infiltrate the extravasation and the brain tissue in the vicinity. These leucocytes rapidly degenerate into pus, while the neighbouring nerve tissue disintegrates and liquefies. These emboli may be accompanied by anaemic or haemorrhagic necrosis, according as an artery or a vein has been blocked. This may be indicated by the appearance of the parts during operation: when they are oedematous and glistening they are anaemic, and therefore due to arteriolar thrombosis, and when they appear as reddish-brown sloughs they are haemorrhagic and due to venous thrombosis. When large arteries are blocked, necroses of extensive areas may occur which may be afterwards separated. Such large necrotic portions may also arise from very acute and infective inflammation of the brain. . . .

The tissue surrounding and forming the boundary of the acute abscess is actively inflamed. The vessels may be seen in all stages of inflammation, from hyperaemia in the outer zone to thrombosis in the inner. Surrounding the vessels are masses of exudation cells mingled with extravasated red blood corpuscles, and the glimpses of brain tissue which may be had in the less inflamed parts show it to be softened and disintegrating. When the inflammation has been great the vessels are scarcely recognisable on account of the exudation cells not only filling the surrounding parts, but also the vessel wall, and in great measure occupying its interior, the red blood corpuscles being faintly seen in some, while in others they have entirely disappeared. Irregularly distributed over the surface of the abscess are portions of minute sloughs of an oedematous greyish appearance, with pus in their periphery and in the pockets or the sinusities in the abscess wall. The surface toward the abscess is flocculent, shaggy, and irregular in outline. Occasionally little naked thrombosed vessels project from its surface into the abscess cavity at parts where the molecular disintegration has proceeded more actively in the soft brain tissue surrounding the vessel. As in processes of ulceration in other parts of the body, the small vessels become thrombosed in advance of the molecular necrosis, and so haemorrhage into the abscess is prevented. Occasionally, however, the process of disintegration is more rapid than that of thrombosis, and bleeding occurs into the abscess cavity, giving rise to symptoms of apoplectic. The zone of brain tissue surrounding the abscess is oedematous, and its vessels are hyperaemic. There is thus in the periphery of an acute abscess no other wall than a softened and disintegrated cerebral tissue filled with exudation cells and extravasated blood.

The pia mater and arachnoid tissue over an area of cerebral degeneration have often a milky and turbid appearance.

The encephalitis surrounding the abscess extends to a distance beyond the abscess itself. Were this zone of encephalitis to embrace a part of the brain whose function was known and capable of external manifestation, symptoms might arise which might lead one to localize the abscess at a distance from its actual seat.

. . . When the process of molecular disintegration has produced the abscess ceases, and the brain tissue possesses sufficient vitality to assume a formative action, the debris of disintegrated tissue, granular cells, leucocytes, blood, and inspissated pus become entangled in the meshes of fibrin, the elements for the formation of which are poured out from the living tissue. Into this mass large numbers of leucocytes penetrate. Some of these form into elongated spindle cells, bundles of which may be seen interspersed throughout the membrane. At an early period it contains no bloodvessels, and, therefore, secretion from its surface must be limited to the few leucocytes which have travelled through it from the living tissue. Later a few bloodvessels may project from the living tissue into the membrane at a point where it is abundantly penetrated by leucocytes. These vessels are, however, of the most primitive description, such as may be seen in imperfectly developed granulation tissue. They are at first so small and imperfectly developed that they can only supply nutriment to the leucocytes in their immediate neighbourhood. As they are generally to be seen on the side of the encapsulating membrane next the living tissue, they cannot provide many exudation cells for fresh pus formation.

The pus in the abscess thus becomes encapsulated by a layer of membrane of very low vitality which shuts off the living brain tissue from the dead pus within. When this capsule is complete on all sides the abscess becomes to a great extent stationary. First, because the process of molecular disintegration has ceased. Secondly, as the leucocytes which penetrate for some way into the living wall of the abscess, and which might be converted into pus were they shed on the inside of the capsule, are principally caught by the meshwork of fibrin, so that few pass into the abscess cavity; and as the lining membrane increases in thickness the difficulties of transmigration of the leucocytes increase, both on account of the greater distance they have to travel, and the increasing formation of fibrous tissue bundles, which are interspersed throughout it. Thirdly, this same membrane, although it does permit the passage of serum from
the pus cavity to the vessels in the living tissue, yet prevents the absorption of the particulate portions of the disintegrated pus. The thickness of the abscess capsule varies considerably, from one to five or more millimetres. It is generally smooth internally, and rather ragged and floeulent externally—next to living brain tissue. It is for the most part rounded or ovoid, while the boundary of the acute abscess is generally irregular in outline. The time required for the formation of the abscess capsule is indefinite, and depends on the condition of the part and the character of the inflammation. Some abscesses of at least three weeks’ duration were found to be without capsules. . . .

. . . This wall may not, however, remain permanently in the condition described. Changes take place in it from the side of the living tissue. The circumferential pressure of the brain upon the abscess may facilitate the absorption of the fluid portion of the pus. Well-formed vessels may be thrown out from the living brain tissue, and penetrating the capsule, may thus aid in the absorption of pus debris through phagocytic action. In this way it is possible for a considerable portion of a small cerebral abscess to become absorbed.

. . . On the other hand, if the capsule be replaced in part by a layer of vigorous granulation tissue, a fresh supply of pus may be formed which may augment the size of the abscess and exercise pressure from within upon the capsule which is thus apt to become thinned at parts. The abscess may then burst either into the ventricles or into the subdural space. In either case very serious symptoms ensue. . . .

. . . The tissues in the vicinity of the encapsulated abscess are not always tolerant of the foreign body in their midst. They suffer from an amount of compression, which is apt to induce degeneration and atrophy. Slight causes may then occasion in them fresh oedema and inflammation, just as encephalitis occurs round a tumour. If this encephalitis becomes purulent, a new abscess may form in the periphery of the old one, leaving the latter intact within its capsule. It is probable, however, that a leakage of the contents of the abscess into the compressed tissues in the vicinity may set free from imprisonment inflammatory elements (micro-organisms) which, meeting with fresh pabulum, regain their former vitality, and start afresh the inflammation in the tissues on the outside of the capsule. In such a case, an extramural abscess develops in the circumference of the old one, which it may set so completely free from its attachments that the encapsulated abscess may be found floating in the pus of the peripheral or secondary abscess. This was observed in one of the author’s cases, in which the primary abscess contained in its cyst-like sac was seen floating in a pool of pus.

. . . The size of a cerebral abscess cannot always be estimated by the degree of pressure produced. It is not like a solid tumour, which, to the extent of its bulk, pushes aside the living tissue in its vicinity. The molecular disintegration by which the abscess is formed hollows out an aperture in the brain which is filled by the molecular debris and pus. The abscess will, therefore, not produce the same amount of pressure on the adjacent structures as a solid tumour of the same size, so that pressure symptoms occasioned by an abscess would be less than would be produced by a tumour of corresponding bulk. On the other hand, the symptoms due to oedema and inflammatory action would probably be greater in the acute stage of abscess formation than in most tumours. . . .

There can be no doubt that the pulse is slowed in cerebral abscess owing to the pressure exerted, as after the abscess has attained a certain size its rate decreases as the abscess increases, and the moment the pressure is relieved by evacuation of the pus there is a sudden bound in the pulse rate, from for instance 40 to 120 per minute. . . . The point referred to above must here be remembered; the pus in the cerebral abscess may only occupy the area destroyed by the molecular disintegration, and, if so, little pressure can be exerted on the brain, and therefore little alteration occurs in the pulse rate. . . .

Pyaemic abscess of the brain may arise from infective matter originating in some source of infection in any part of the body. This paeanct matter entering the blood is carried by the bloodstream until it is deposited in a small vessel producing infective embolism. The diseases which have been known to give rise to this condition in the brain are, among others, disintegrating pneumonia, foetid bronchitis and empyema, foetid pericarditis, infective compound fractures, seldom acute infective periostitis, and occasionally infective ulcers of the intestines and abdominal cavity. Infective peritonitis generally ends fatally so soon after its onset that cerebral abscess has no time to form. . . .

It is well to remember that pyaemia, though generally the cause of multiple abscesses, may itself arise from intracranial suppuration, especially when the sinuses are also involved.

. . . Abscesses are rarely multiple starting when they originate in pyaemia, in which case they are in two-thirds of the cases multiple, and generally very numerous, and invade both brain and cerebellum. . . . 93 per cent of abscesses from traumatism are single: 87 per cent are single when due to otitis media. . . .

. . . After an abscess has been evacuated the brain tissue tends to fill the gap left by the removal of the pus. If it be an acute abscess, the resiliency of the brain is retained and its expansion is rapid, a few minutes to a few hours sufficing to close the gap. After evacuation of chronic encysted abscess, the expansion is much slower, taking days
before the cavity is obliterated. When the wound has been at once closed after evacuation and the dura replaced, there is a probability of the brain remaining free from adhesions, but as a rule it becomes more or less fixed to the dura. When the wound has to be healed by granulation tissue, the opening into the brain meanwhile being kept patent for pus evacuation, the pia mater adheres to the epi-
trix, soldering the brain to what becomes a rigid wall. Such anchoring of the brain is subsequently apt to produce a shock on sudden movement of the patient, as on sudden rising from a recumbent or sitting posture. This physical effect directly applied to the brain itself, though acting like a drag or pull, is akin in its physiological result to a blow applied to the cerebrum. It is apt to cause unconsciousness, generally of very brief duration. The man may fall, but is able to pick himself up in a few seconds. In some few instances the unconsciousness lasts longer. When very frequently repeated, even when the effect is so slight as to be only slightly perceptible to the patient, this frequent dragging is apt, months or years afterwards, to produce encephalitis over an extended area of the brain. This cerebral irritation might lead to epileptic fits, though the author is not aware of any undoubted instances following operation for cerebral abscess. There are, however, many such cases resulting from traumatisms, in which the brain has been soldered to the unyielding parietes. . . .

CHAPTER III. SYMPTOMS OF ABSCESS OF BRAIN AND MENINGITIS.

The elicitation of a differential cranial percussion note* as an aid to cerebral diagnosis in cer-
tain gross changes of the intracranial contents, especially in children, has been practised by the author as opportunity offered during the last ten years, and as it has been found to be useful in diagnosis the following may assist those desirous of practising it.

The percussion note is obtained by the cranial walls vibrating when struck, the note being modified by the consistency and volume of the contents and their relative position to the bone. The sound elicited depends first on the susceptibility of the skull to vibrate, and secondly on the effect which the intracranial contents exert on these vibrations. When struck, a thin cranium vibrates more easily than a thick one. A skull may be so thick that it vibrates little, if at all, to ordinary digital percussion. In the cranium of an infant, whose bones are only united by a membrane, and where they lie somewhat loosely on the brain, the percussion note elicited is so slight, dull, and flat, as scarcely to be perceptible. Should, however, the contents of such a cranium increase sufficiently to

produce tension of the whole parietes—bones and intervening membrane—then the note becomes clear, particularly as the density of the contents of such a case is low—the brain containing a consid-
erable quantity of serous fluid. In the child whose fontanelles remain open, especially in delayed closure from serous over-distension, a clear drum-
like note is elicited on percussion. . . .

. . . The author has found this clear percussion note in over forty children and young adolescents who have had distended ventricles arising from many different causes. In tumours of the cerebel-

lum it is an aid to diagnosis. When present, along with abscess, it points to involvement of the cere-
bellar fossa. . . .

CASE XXVII. Cerebral abscess in temporo-sphenoidal lobe; complicated with localized purulent meningitis of cerebellar fossa, both due primarily to chronic otitis media purulenta. Coming under observation seven days from onset of symptoms. Duration of illness sixteen days prior to evacuation of abscess. Operation. Complete recovery.

H.M., aged 9 years, admitted into the Children's Hospital, Glasgow, on 26th January, 1898. Affected with purulent meningitis, accompanied by encephalitis terminating in cerebral abscess, involving the greater portion of the right temporo-sphenoidal lobe, due pri-

marily to otitis media purulenta, an injury being prob-
ably the determining cause.

History.—Heredity.—Both parents are alive and well. Patient is one of a family of four, one of whom died when three months old from "inflammation of the brain." Other two are healthy.

Personal.—The parents believed patient to have been in good health previous to this illness. Though they had never observed discharge from his ears, yet on admis-

sion his right external ear was found to contain foetid inspissated pus.

Present Illness.—Fourteen days before admission the patient fell head first down a flight of stone stairs. He rose immediately, apparently unjured, and went about as usual. A week afterwards he complained of a severe pain in his head in and around the right ear, which compelled him to remain in bed. Since then he has had complete anorexia. Three days previous to admis-

sion, the pain extended to the frontal region. On the following day he vomited repeatedly, and a swelling was observed over the right mastoid. He has had constipa-
tion of bowels for several days, and has urinated in-

voluntarily.

Condition on Admission.—Patient was in an irri-
table state, semi-insensible and delirious, but could be occasionally roused to realize his surroundings. He fre-

quently cried out and tossed about, and was evidently suffering pain, referred to his right ear and correspond-

ing side of the head. His pupils were equal, contracted, and sluggish. The movements of the eyeballs were free, and there was occasionally a slight transient convergent

* This was later named "Macwen's sign".—R.H.W.
squin of right eyeball. There was a degree of congestion of both retinas. A fluctuating swelling was observed behind the right ear over the mastoid. The right external auditory meatus contained foul smelling pus, and the membrana tympani was perforated. With the exception of the paresis of the right external rectus, there was no observable paralysis. There were very slight erratic twitchings of groups of muscles of limbs, mostly on the left, but also on the right side of body, and equally on both sides of the face. There was an herpetic labial eruption. His temperature was high, and his pulse rapid. On admission an incision was made to relieve the sub-periosteal mastoid abscess, and vent was given to cut three drams of foetid pus.

Operation for Meningitis (28th January).—Two days after admission, the temperature having remained high, the pulse rapid, and the meningeal symptoms persisting, along with an increasing and now marked retraction of the head, delirium and other indications of brain involvement, the following operation was performed.

The mastoid antrum was opened. It was found to be enlarged, filled with cholesteatomatous masses and pus. The cerebellar fossa was then opened. The sigmoid sinus was covered with pus and a mass of granulation tissue, varying from 1/4 inch to 1/2 inch in thickness. Though its lumen was markedly encroached on, it was still patent. Granulation tissue also covered the dura behind the sinus in the cerebellar fossa, and several drams of pus were evacuated from between the skull and that membrane. The pus from the cerebellar fossa was submitted to bacterial examination, and both by direct stained specimen and culture experiments the streptococci pyogenes were obtained. The tegmen tympani was carefully scrutinized, but no erosion was discovered.

Relief to all the meningeal symptoms followed this operation. The temperature, pulse, and respiration all became normal. The muscular tremors, the crying fits, and the delirium ceased, the pupils resumed their normal mobility, and on the third day the last evidence of the retraction of the head and neck disappeared. Following this improvement, it was observed that the pulse and temperature became subnormal. On the fifth day after the operation he became drowsy, disinclined to speak, and when both eyes were exposed to a light, the right pupil was a shade larger than its neighbour and distinctly more sluggish. All these symptoms increased until the ninth day after the operation when the evidence of pressure on the brain was marked. He was then extremely drowsy and difficult to rouse. There was a passivity over the left side of the face, and the left arm when lifted up dropped in a heap. The right arm, though feeble, the patient still controlled. Both lower limbs moved actively when the toes were pinched. An operation for the relief of cerebral abscess was resolved upon. The symptoms pointed to abscess in the temporosphenoidal lobe; but as there had existed so much purulent meningitis in the cerebellar fossa which the former operation had revealed, it was deemed expedient to first subject the exposed cerebellar area to a careful scrutiny.

Operation for Cerebral Abscess (6th February, 1898).—The already exposed sinus was first inspected, and as some pus still exuded from the granulation tissue covering the dura behind the sinus, a quarter inch disc of bone was removed from the occiput covering the cerebellar fossa. The anterior half of the aperture left by this disc was covered with granulation tissue, while the posterior half exposed normal and almost transparent membranes, through which the underlying brain was just perceptible. There was no haziness or milkiness of the membranes over this area, such as would have been expected had there been an extension of the meningitis in this direction. Attention was now directed to the temporosphenoidal lobe, and as a preliminary a fresh scrutiny of the tegmen tympani and tegmen antri was made, but no visible erosion was detected.

The incision which had previously exposed the mastoid antrum was extended upwards over the squamous plate of the temporal, and from it a half inch disc of bone was removed from point half an inch above and a little behind the external auditory meatus. At this point the dura was healthy, and so transparent that a flake of plastic effusion 1/4 inch in diameter was seen either overlying or within one of the compressed pial vessels. This speck of effusion could be altered in position by probe pressure applied from the external surface of the dura mater. The surface of the brain was markedly avascular, probably due to the pial vessels being flattened against the dura and skull from intracranial pressure, but possibly from thrombosis of some of the larger trunks. When the membranes were incised the avascular brain bulged into the trephine aperture. A hollow needle was introduced into the temporosphenoidal lobe in an inward and downward direction; when it had penetrated the brain substance for about half-an-inch, there was first the escape of some gas followed quickly by a jet of pus, greenish in colour and of foul odour. A pair of forceps were afterwards introduced through the healthy brain tissue and expanded, when the pus was given free vent, and with it there came a series of cerebral sloughs, some of considerable size. The amount of pus and disintegrated cerebral tissue, though not accurately measured, was approximately about three ounces. After this matter was removed and the brain tissue held aside, a large cavity was seen to exist. Its limit in the anterior and posterior directions was not visible from the trephine opening, its depth was fully an inch, and a probe could be passed backward for about two inches and forward for about one and a half inches before coming in contact with brain matter. Considering the size of the child's head and that of the cerebral cavity, the greater portion of the temporosphenoidal lobe must have been obliterated. The brain tissue exhibited very little tendency to fill the gap, which remained as a cavity of the above dimensions at the end of the operation. The cavity was not syringed, lest infective particles might be disseminated especially into the lateral ventricles; but it was evacuated as thoroughly as possible, the head being placed in several directions to facilitate complete evacuation by gravitation. Iodoform and boracic powder were then introduced into the cavity, a short decalcified drainage tube was fixed in it by means of a stitch, and a wood-wool dressing was applied.

The pulse, which ranged from 56 to 70 before operation and was 72 before the evacuation of pus, increased in rapidity immediately after the operation, and when the patient was removed to bed was to be found to be 104, and two hours afterwards 116; subsequently it subsided to normal...
tion. At the former dressing the brain cavity no longer existed, and only a minute portion of the drainage tube lay exposed on the granulating surface, the remainder having been absorbed. The external wound healed by granulation tissue. . . .

CASE XXXIX. Case of cerebellar abscess with partial thrombosis of sigmoid sinus, arising from infective otitis media. Operation. Recovery. Coming under observation on the sixtieth day of illness. Duration of illness ninety days.

J.R., aged 38 years, was seen in consultation with Drs. Wood Smith and Barr, on 13th February, 1892, suffering from cerebellar abscess, with partial thrombosis of sigmoid sinus.

History.—Heredity.—Family history good. Personal.—Patient has had left facial paralysis and deafness since early childhood, but for many years there has been no suppuration in the left ear. Otherwise he has been in good health.

Previous Illness.—On 15th December, 1891, he got chills; felt pain in the right ear. Six hours afterwards he awoke in great agony with pain in the right ear, shooting thence through his head. Some days later the pain had further extended to the vertex and back of head and right side of neck. Along with the pain he had deafness in that ear. After a fortnight’s treatment in bed he felt stronger, but the pain and deafness persisted. He was however able to be taken to the country for a change of air, from which he benefited, but was never free from pain in the head and back of right side of neck. He returned from the country on the 11th February, and the same night and next day the pains becoming much aggravated, the writer was asked to see him on 15th February.

Condition when First Observed.—He then complained of pain over right ear, vertex, and more especially over the back of head and right side of neck, and he had pain on pressure over the course of the upper third of the jugular vein and at the apex of the posterior cervical triangle, in both of which regions the tissues were thickened and contained several enlarged deep cervical glands. There were several small superficial mastoid glands likewise enlarged and painful to touch. The pain he described as persistent and throbbing.

The motor power (with exception of left facial paralysis), sensations, and reflexes, were normal.

A slight purulent discharge emanated from the right ear, and the membrana tympani had a wash-leather appearance. There was slight papillitis in both fundi, especially the right. The pulse was 55 per minute, and the temperature was 99°F. His lungs were free from evidence of infective invasion. These symptoms pointed toward thrombosis of the sigmoid sinus with cerebellar suppuration, and immediate operation was advised.

Operation (13th Feb., 1892).—The mastoid antrum was opened and found to contain granulation tissue and pus. The sigmoid groove was then exposed. Masses of granulation tissue, sprouting from the knee of the sigmoid sinus, projected through the groove into the mastoid cells. These granulation masses being removed, the dura of the cerebellar fossa was found thinned at the inner and anterior side of the sigmoid sinus, and a drop of pus exuded by the inner side of the sinus through a minute erosion in the cerebellar dura. The dura was opened at this part, and this gave vent to about three drachms of pus, which issued from the cerebellar tissue, which was at that part softened. The sigmoid sinus, though distinctly thickened, was not opened, as the mass of granulation tissue surrounding it in all probability had acted as a protection against the entrance of infective organisms. The cavity was washed out with a weak carbolized solution, the antrum as well as the middle ear being cleansed. The ossicles and membrane were left intact.

From that time the pain previously experienced greatly diminished, and he was soon able to have refreshing sleeps and to awake feeling well. The pulse still continued slower than normal, 50 to 55, though his normal pulse and that of some other members of the family was about 60. His temperature was normal. At the termination of a week he was quite free from pain. The tumefaction of the tissues at the upper part of the neck had almost disappeared. A fortnight afterwards the enlarged glands had greatly subsided. The wound closed by granulation tissue from below upwards. He made an uninterrupted recovery and regained his former health and strength. . . .

Prognosis.—Cerebral abscess must always be regarded as a serious affection imminently dangerous to life. The great majority of cases not dealt with surgically end in death within a short period, generally a few weeks. On the other hand, there is no cerebral affection more amenable to surgical treatment, and none which offers better results. An uncomplicated cerebral abscess, whose position is clearly localized, if surgical measures are adopted for its relief at a sufficiently early period, is one of the most hopeful of all cerebral affections. It is much more so than suppurative leptomeningitis, or even than infective thrombosis of the sigmoid sinus, unless the latter can be operated on before the disintegration and dissemination of the infective contents of the vein take place. After aseptic evacuation of the abscess, not only is the patient likely to recover, but in many instances it leaves no perceptible permanent mental or bodily damage. Within a few days, sometimes hours after the evacuation, the mind becomes bright and intelligent, the paralysis passes away, and within a few months the optic neuritis has disappeared, leaving the visual function unimpaired.

Occasionally, after evacuation of the abscess, there may be some sodering of the brain to the skull, or to the almost equally rigid dura mater or fibrous covering filling up the trephine aperture, though this is much less than what occurs after the removal of tumours. When this occurs the patient may be subject to occasional faintness on sudden exposure to great heat or on sudden exertion. One man, a blacksmith to trade, says that after he had been cured of his abscess he occasionally felt on sudden effort, especially when beside the furnace, a faintness coming over him, which caused him to drop what he held in his hands and to lie down for a moment. This occurred several
times the first year, and seldom afterwards, as he carefully avoided exposure to great heat or sudden exertion. He was capable of strenuous and protracted exertion provided it was not sudden.

The prognosis without operation will in some measure depend upon the size of the abscess; the smaller ones occasionally being absorbed, the larger ones usually ending in death. The position of the abscess in the brain will also affect the prognosis. If it be situated deeply, so as to involve the more vital parts, death will speedily follow. On the other hand, a superficial abscess not operated on may be evacuated, if a sufficient vent is formed for it by pathological processes through the dura and bone. This, however, is rare, and even those so connected with the exterior of the skull generally end fatally sooner or later.

Even an abscess which has become encysted, and has remained for a considerable period without evincing any signs of its presence, may be a source of danger. The encysted abscess may burst, or it may act as a foreign body round which fresh inflammation is set up . . . and if situated in the cerebellum may cause death by oedema of the brain and serous distension of the ventricles. An encysted abscess may, however, become absorbed; but until it does so it is a menace to the patient's life, as it is still apt to have fresh suppuration induced in its vicinity whenever fresh organisms are introduced, probably gaining access by the original channel of infection. The source from which the abscess sprang remaining active is an additional element of danger, both by way of occasioning fresh suppuration round the abscess, and also by creating fresh foci of inflammatory disturbance, such as sinus thrombosis or lepto-meningitis.

The cure of cerebral abscess depends on its early detection, accurate localization, and speedy and efficient evacuation under aseptic precautions.

Course of cerebral abscess.—The time occupied by cerebral abscess in developing and pursuing its course when uninterrupted by treatment is variable. Acute abscess runs its course in from two to six weeks, whereas chronic cerebral abscess, when encapsulation takes place, may last for a very long period—months to many years. As a rule, abscess arising from infective otitis media runs a more rapid course than abscess arising from infective traumatism, without direct exposure of the brain. No doubt the nature of the specific organism introduced influences the duration of the course.

When the skull is bruised, indented, and ingrained with dirt without fracture, great care is necessary in order to remove the whole of the infective matter . . .

If fracture exists, the edges of the fractured bone ought to be inspected, and if impregnated with foreign matter, they ought to be refreshed with a chisel. . . .

. . . When the dura has been penetrated, and the inner membranes, or the brain itself, inoculated with infective products, the dura ought to be freely opened, in order to expose for inspection the inner membranes, as well as the brain; also to facilitate the removal of foreign matter, and prevent the ultimate extension of deleterious products. When, in addition to the puncture of the brain and its membranes, the cerebral substance has been bruised, it is very difficult in the midst of disintegrating brain tissue and extravasated blood to ensure the removal of all extraneous matter. When doubt on this point exists, it is preferable to leave the wound open—exposed to the influence of iodoform gauze,—first to permit of the free escape of inflammatory products, should they unfortunately form; and secondly to secure the direct application of antiseptics.

All sources of suppuration or infective matter in the orbital, nasal, buccal, oral, and pharyngeal cavities, and in the maxillary and frontal sinuses ought to be removed, or, when this is not practicable, the discharges ought to be rendered and maintained aseptic. This is sometimes difficult without extensive operation in lesions of the nasal fossa or its annexa, but as long as an infective source remains it is an element of danger and a menace to the patient. Cario-necrosis of the skull should be similarly dealt with. The frontal and maxillary sinuses, when in a state of persistent suppuration, may require trephining to afford free vent to the pus, and for the purpose of removing from the walls infected granulation tissue and eroded bone. The proximity of the frontal sinuses to the brain render infective processes in them very hazardous, especially when accompanied by erosion of their posterior wall. When the frontal sinuses are involved, both ought to be exposed. The neglect of this precaution in one case, reported to author, led to a fatal issue from supplicative lepto-meningitis, originating in the opposite frontal sinus from that which was exposed. . . .

As chronic purulent otitis media and the extension of inflammatory processes to the mastoid antrum and cells is the primary focus which leads most often to intracranial inflammatory lesions, the eradication of the otitis media must be regarded as the most potent factor in the prophylaxis of inflammatory cerebral lesions. . . .

. . . When the patient has presented symptoms of infective meningitis, extradural abscess or abscess of the brain, the mastoid antrum and the

CHAPTER V. TREATMENT.

. . . Abscess in the brain, infective thrombosis of the intracranial sinuses, and lepto-meningitis originate in primary infective foci. The first step in prophylaxis is to prevent the occurrence of such foci, the second to eradicate them when present. . . .
... By enlarging the opening in the tegmen, extradural abscess may be thoroughly evacuated. The extent of bone removed must be determined by individual circumstances, but it ought to be sufficient to prevent re-accumulation of fluid between the dura and the bone. No pockets or crevices are to be left, and all granulation tissue should be removed. It is safe to open the tegmen freely in an outward direction from the seat of the perforation. Before injecting fluid—if such be deemed necessary—between the skull and the dura ascertain the limits of the extradural space, and whether there be an opening through the dura into the inner membranes or the brain. If no such opening exist, then the extradural cavity may be washed out with safety, and dressed with iodoform and boracic acid powder and iodoform gauze. . . .

. . . When pus issues through an aperture in the dura mater from the intradural structures, after the tegmen has been opened sufficiently and the extradural surface cleansed, the dura is opened freely. If the pus be found gathered in the arachnoid or pial meshes, or if it issues from ulceration of the brain surface, it may be thoroughly dealt with from the opening in the tegmen. The inner membranes ought to be opened, and the cerebral surface washed with a stream of antiseptic solution, and dusted with antiseptic powder. No drainage tube is required if the openings through the tegmen and dura have been sufficiently free.

. . . When pus is found to issue through the dura above the tegmen from a cerebral abscess in the temporo-sphenoidal lobe, the abscess may be evacuated by enlarging the aperture in the tegmen, and also extending the aperture outwards through the squamous portion of the temporal. Such an opening into the cerebrum suffices for temporary purposes, but though it always ought to be made in order to eradicate the source of the infection, it is not safe to trust to it alone, as in many cerebral abscesses there are sloughs of brain tissue which cannot be easily removed in this way, but require a larger opening in the skull for their evacuation. If these sloughs be allowed to remain in the brain, they retain infective matter, which maintains the irritation, and may give rise to fresh abscess formation.

. . . In abscess of the temporo-sphenoidal lobe, it has been proposed to make the opening through the skull at various points. It ought to be made as near to the seat of disease as possible. In the majority of cases the abscess in the temporal lobe originates in the tissues directly above the tegmen tympani or antri, and therefore an opening in the squamous portion of the temporal above the tegmen will be the nearest point to the cerebral abscess.

When the mastoid antrum has already been opened, the incision in the soft parts made to expose the antrum is extended upwards for a couple of inches above the posterior zygomatic root. The soft tissues including the periosteum are reflected by the peristeal elevator, and a disc of bone half an inch in diameter is removed. The centre pin of the trephine is placed at a point in line with the posterior osseous wall of the external auditory meatus, and three quarters of an inch above the posterior root of the zygoma. . . .

When the dura is exposed, any purulent secretion which may exist extradurally is removed, and the colour and appearances of the dura are noted. . . . Before opening the dura, it is well to cover the exposed osseous surface and its cut edge with the iodoform and boracic acid powder, rubbing it into the cut osseous surface, in order to protect these parts from contamination by the infective pus about to be withdrawn. . . .

With the object of exploring for pus, and removing it when found, either of three instruments may be used—a cannula armed with a trocar, a hollow needle, or a pair of sinus forceps. The instrument ought to be inserted in an inward, downward, and slightly forward direction, so as to impinge, if it went far enough, against the cranial aspect of the tegmen tympani. While the instrument is being inserted, a slight to and fro lateral movement ought to be imparted to its point, with the view of ascertaining whether it has entered a cavity, inside which its extremity can move without resistance. When the sinus forceps are used the blades are several times gently expanded as they are introduced. . . .

. . . When the pus is found, its rate of flow from the cannula varies according to the amount of intracranial pressure, and when the abscess is becoming emptied the flow will be influenced by the respirations, increasing during expiration. Along with the pus, molecular debris and minute sloughs of brain tissue may be extruded through the cannula, but the larger sloughs, which are so frequently present in brain abscesses, cannot escape in this way, they require a large opening for their removal. For this purpose, a quantity of brain tissue, existing between the abscess and the surface of the brain, may require removal, or the peripheral brain tissue may be turned aside. While the cannula is retained in the abscess cavity as a guide, the cerebral tissue may be removed by means of a Volkmann's spoon, or it may preferably be turned aside by introducing a pair of dressing forceps closed and opening them in situ. Frequently, when the forceps have thus been expanded, one or more of the sloughs flow between
their blades and can at once be removed. Once the opening in the cerebral tissue has been sufficient, the sloughs are often extruded by the intracranial pressure, and are carried slowly to the surface of the brain. If the sloughs be too large to be thus extruded, they may be gently detached from the abscess wall, and assisted out by the forceps and the sharp spoon, being necessary to discriminate carefully between the slough and the healthy brain tissue. It is of importance to remove these sloughs thoroughly, if they remain they are apt to set up fresh irritation and abscess in their periphery by the infective matter which impregnates them, and which is contained in their interstices. The retention of such sloughs is one of the most fruitful sources of reproduction of abscess after evacuating the primary one, and is also a cause of delayed healing. It is true that were they rendered aseptic they could be absorbed, but this would be difficult to secure when they arise from an infected source. The leaving of cerebral sloughs is as dangerous as not eradicate the primary source of the infection or the channels by which it spreads to the brain.

... After removal of the sloughs, the cavity is washed out with a boracic or weak carbolized watery solution (1–100). In order to do this a second cannula is introduced by the side of that first inserted, the calibre of the second being at least a half greater than that of the former. A stream of antiseptic fluid is caused to flow gently through the cannula with the finer calibre, and allowed to find an exit by the larger tube. It need scarcely be added that the stream is to be gently introduced, and any resistance met with must not be overcome by forcing the fluid through, but by ascertaining that the tubes are not occluded by debris from the abscess cavity, and this applies especially to the exit tube. Fluid introduced with considerable force into the abscess cavity would burst it and disintegrate the brain tissue, and might inflict irreparable damage. The surgeon must be satisfied that the fluid is going into the abscess cavity and that it returns by the exit cannula, otherwise he must desist from injecting...

After the stream of antiseptic fluid returns clear, before the removal of the cannulae, the head of the patient ought to be turned so as to allow the fluid to gravitate out. One ought to bear in mind that the fluid introduced may cause dispersion of the pus and dissemination of the infection, unless within a firmly formed capsule. In the absence of proper flushing arrangements instead of washing out the abscess cavity the introduction of the iodoform and boracic mixture is advisable.

... Where the abscess is seated near the floor of the middle fossa or has a direct communication with it, the opening in the tegmen ought to be enlarged so as to gain access to the abscess cavity from below, if this has not already been done. The larger cannula inserted at this point serves to remove the fluid injected through the finer cannula introduced by way of the opening in the squamous, and more thorough washing of the abscess cavity is thereby secured. This aperture in the tegmen and membranes serves afterwards for drainage purposes, for which it is admirably situated, being at the lowest level of the abscess cavity when the head is raised on a pillow. When this method is adopted the Eustachian tube and the external ear are both shut off by filling the middle ear with iodoform and boracic powder. ... After the evacuation of an acute abscess, drainage tubes are (for the reasons stated) of little value, provided the whole of the infective matter has been removed. If doubt exists on this point, a drainage tube is introduced. There can be no doubt that the pulsations of the brain cause, by striking against the tube, an irritation, which is increased if the tube be rigid. In chronic abscesses the drainage tube may be required. An absorbable decailiated chicken-bone drainage tube is introduced into the abscess cavity and stitched to the skin so as to retain it in position. Its inner margin is made to project just within the outer wall of the abscess cavity.

When the abscess cavity is still foul and pus formation is likely to proceed, an india-rubber, glass, or other non-absorbable drain is preferable, but it ought to be removed as soon as possible—twenty-four to forty-eight hours. In the event of an opening having been made between the tegmen antri or tympani and the abscess cavity, then the drainage is best conducted through it, and the opening through the squamous bone is allowed to heal. ...

Where the surgeon has succeeded in rendering the abscess cavity aseptic, the dressings are left for three weeks untouched, provided the temperature is normal and there is no stain through the dressings. At the expiry of that time they are removed, and the wound is either absolutely healed or there is a superficial granulating surface, according as the wound has been stitched or left open.

When the abscess cavity has been left infected, the dressings require to be changed daily and the cavity syringed out. In this case, boracic powder alone ought to be used instead of iodoform mixture, as the constant changing and renewing of the powder might induce the toxic effect of the iodoform. A great preventive against iodoform intoxication is the use of very large scales of iodoform instead of fine powder, as the latter is much more quickly absorbed. ...

Abscess of the cerebellum, arising from otitis media, is usually secondary to suppuration in the vicinity of the sigmoid sinus, and is often accompanied by thrombosis of that vessel. It seldom
ensues from extension, by way of the internal auditory meatus, infective inflammation passing by this channel being much more frequently followed by lepto-meningitis. It is well to expose the sigmoid groove first, with the view of ascertaining the condition of the sinus. In such cases it is generally covered by granulation tissue, from which pus exudes. This ought to be removed along with the extradural pus. If there be an erosion in the dura at either side of the sigmoid, it would aid in indicating the locality of the fistula leading to the cerebellar abscess. Occasionally the pus from the cerebellum exudes by the inner side of the sinus, and it is possible, by extending the osseous incision inwards, to widen the aperture in the dura sufficiently to permit the free escape of discharge, and also to wash out the cavity. It is much better, however, to open the cerebellum from the outer and external aspect of the sinus as the abscess can always be reached in this way, while the facilities for evacuation and manipulation are greater and the subsequent drainage much more under control. The manner of evacuation, washing, and drainage is similar in detail to that given under treatment of temporo-sphenoidal abscess.

In operating upon cerebellar abscess cases, it occasionally happens that respiratory difficulties are experienced. When this occurs the operation ought to be proceeded with rapidly. Altering the position of patient’s head relatively to the trunk sometimes effects an improvement in the breathing. The anaesthesia ought to be as light as is consistent with quietness of patient. In two cases reported the respiration was suspended during the operation, and was maintained by artificial respiration in one for twenty-four hours, in the other for six, the heart meanwhile acting rhythmically. The operation was abandoned when the respiratory difficulties ensued. It would have been much better to have finished the operation while maintaining the respiration, as the evacuation of the large abscess might have saved the patient’s life by relieving the pressure on the respiratory centre.

**Abscess of the frontal lobes.**—These abscesses may be opened either from the front of the brow or from the temporal regions, the choice being determined by the precise locality of the abscess. From the brow any abscess in the frontal may be reached, though those situated in the back part of the frontal would be nearer the surface of the temporal fossa. The skull in front of the brow is thicker than in the temporal fossa, but, in the operations through the latter, the temporal muscle has to be divided and separated, and its planes of fasciae might be exposed to suppuration were they brought into contact with infective matter.
from the abscess. With proper precautions this ought to be avoided. The frontal sinus ought to be avoided in trephining from the brow, though in idiopathic frontal abscess the frontal sinus is frequently involved, and, if so, ought to be opened as a preliminary. Infection extending through the cribiform plate of the ethmoid may cause abscess near the mesial aspects of the frontal, and trephining through the frontal sinus and about the position of the middle line would suffice for the further search for pus. If the cribiform plate requires to be exposed, it is best done by removing a quarter-inch disc slightly above the glabella—remembering that the frontal lobes dip at this point to the level of the nasion. . . .

After the tongue and mouth have been cleansed, milk diet or fluid nutriment is given in small quantities, at first peptonized, as the patient's digestion and assimilation are weak. The light fluid nutriment is continued for a fortnight or three weeks, and until the temperature is normal. A week after the operation, if the bowels have not been moved, a purgative, which will not cause vomiting, and which will give an easy motion without straining, is administered. The patient is kept in bed for from four to six weeks after the operation, however well he may be. In no case is he allowed to rise before the wound is quite healed—which is generally at the end of a fortnight or three weeks. If there be secondary abscess formation, there will be further delay in healing the wound, and also in permitting the patient to rise. It is better to exceed the period of quiet rest in bed than allow accidents to occur by permitting the patient to rise too soon.

CHAPTER VI. RESULTS.

In the foregoing clinical record the cases which have been selected for publication are those which presented features of pathological or semiological interest, and they have for the most part been recorded in extenso, so as to form a contribution to the data from which conclusions may be ultimately formulated. This was felt to be especially necessary, as in consulting the scattered literature of the subject, the cases of intracranial pyogenic infective diseases recorded previously to the last few years were, for the most part, found to be fragmentary and defective. . . .

The following is a résumé of the results obtained by the author, and they present the data upon which the foregoing observations have, for the most part, been founded. In the first group of cases presented, there has been great difficulty experienced in keeping in touch with the majority of the patients after their recovery from simple ablation of the mastoid. While many had the goodness to present themselves regularly as asked, others did so or perhaps one or two occasions and then ceased to attend. The statistics of the ultimate results, to this extent, have been impaired. . . .

Of the twenty-five cases of abscess of the brain mentioned in the table, there were nineteen operated on, with eighteen recoveries. In these nineteen cases there were twenty-two abscesses evacuated, with twenty-one recoveries. One might almost conclude that in uncomplicated abscess of the brain, operated on at a fairly early period, recovery ought to be the rule.