MILITARY NEUROSURGERY

PENFIELD

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CANADIAN ARMY MANUAL
OF
MILITARY NEUROSURGERY

by

WILDER PENFIELD, M.D., D.Sc., F.R.C.S., C.
Director, Montreal Neurological Institute
Professor of Neurology and Neurosurgery
McGill University

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FOREWORD

IN the present war, with its new methods of combat, no less than in other wars, the medical officer in active service must face new problems. For the solution of such problems he should have ready access to the accumulated experience of army surgeons and to the recent specialized advances of modern Medicine.

This manual provides such information in a special field in which advance has been rapid. It should help to standardize treatment, to reduce mortality and decrease the incidence of complications, early and late. But in Medicine there can be no last word, no final formulation of facts; and should the need arise a second edition will be issued.

R. M. GORSSLINE,
Director General Army Medical Services.

OTTAWA,
JULY 3RD, 1941.

WILLIAM OSLER, placed upon the title page of a notebook, which was printed for the students of his first class in clinical medicine at the Montreal General Hospital, the following quotation from Froude:—

"*The knowledge which a man can use is the only real knowledge, the only knowledge which has life and growth in it, converts itself into practical power. The rest hangs like dust about the brain or dries like raindrops off the stones.*"

PREFACE

THERE are many times when a medical officer, without benefit of consultation, must treat the wounded for conditions which lie outside the field of his special training. A manual should serve him as pocket consultant and should tend to standardize treatment in the branch of the Medical Service for which it is prepared. The necessity of periodic restandardization may well make it necessary at some future time to prepare a second and revised edition.

Sufferers from injury to head and spine, more than any others, are apt to be neglected during periods of great military activity because of the time and special preparation needed for their care. Rapid sorting and transportation of such patients back to trained teams within six to ten hours of wounding is the *sine qua non* of successful treatment.

The clinical conclusions reported are based upon the work of the pioneers in traumatic neurosurgery: Weir Mitchell, Trotter, Cushing, De Martel, Sargent, Archibald, Foerster, Tinel, Wagstaffe, Jefferson and many others. The technique of the surgical approach to the treatment of acute injuries of head and spine, that is described here, was in large measure worked out by the author's associates, Lt. Colonel William Cone and Dr. Arthur Elvidge. In all fairness the former might be considered a co-author of Chapter II, although he could hardly be held accountable for its shortcomings.

The first chapter of this Manual is devoted to basic principles; the others chiefly to treatment. The subject of acute head injuries, and the early complications of such injuries, is treated in practical detail. This is supplemented by an analysis of the late complications which, in the past, have formed a frequently unnecessary cause of pension, for many of these chronic conditions can be prevented or treated at the time of injury or during convalescence. The management and early treatment of injuries of the spine are dis-

cussed with incomplete consideration of chronic back problems.

The surgery of peripheral nerves is given only a limited discussion and that largely from the point of view of surgical technique. It is recommended that medical officers responsible for such cases should have in their possession copies of the Medical Research Council Report No. 54 on the Diagnosis and Treatment of Peripheral Nerve Injuries*. That excellent pamphlet describes syndromes, anatomy and general care of peripheral nerve injuries.

Numerous alterations and improvements have been made in the text as the result of the constructive criticism of Dr. Kenneth G. McKenzie of Toronto, Dr. Gilbert Horrax of Boston, Professor Geoffrey Jefferson of Manchester and Colonel Edward Archibald of Montreal. Thanks are due to Miss Anne Dawson for the preparation of the index.

Montreal, *W. P.*
July 17, 1941.

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Chapter I

HEAD INJURIES—DIAGNOSIS AND GENERAL PRINCIPLES

TODAY the treatment of head injuries has become a challenge to the medical profession. In the darkness that now envelops warring countries at nightfall, a formidable toll of lives is taken from the occupants of unlit vehicles. The falling walls and hurtling bodies of bomb explosions, in total warfare, have added a further quota of such casualties both civilian and military. In the trench warfare of 25 years ago penetrating and traversing wounds of the head were proportionally more numerous. But now all types must be anticipated. More effective standardization of surgical treatment is urgently needed, and better results should be expected than those of a quarter century ago.

This chapter includes a brief discussion of the principles which a surgeon must understand in order to handle cases of head injury effectively. Practical rules for treatment will be found in subsequent chapters. Sets of rules may outline routine management, but they cannot provide for emergencies nor decide matters of expediency. Only common sense and surgical judgement can guide a medical officer in quick decisions. Surgical judgment must be founded on understanding, and this may serve as an excuse for the inclusion of a discussion of general principles in a practical manual of treatment.

The brain is protected against trauma by the cranial vault, but once the damage has penetrated to the brain itself the very presence of that unyielding bony covering is a complication that makes treatment difficult and death more frequent.

It is essential that among cases of severe head injury the diagnosis of the nature of the injury should be made early. The practice of leaving a patient in bed without taking ener-

getic steps to analyse his case is a frequently fatal form of neglect. Radical interference with those patients who would recover equally well if left alone is to be carefully avoided, as well as operations upon those who are already irrevocably lost. But failure to operate upon a man who could be saved from death or from preventable complications is a more reprehensible surgical sin, though less likely of recognition.

1. CEREBRAL CONCUSSION

Concussion is a violent jar or shock, but the term is also applied to the immediate condition of the brain which results from it. The effect of the blow is an instantaneous arrest of function in the brain. Ordinarily there is loss of consciousness with paralysis so that the patient falls and lies in a completely relaxed state. The pulse is apt to be weak, and respiration may cease for a short time.

The sudden jar or impact upon the skull produces paralysis of nerve cells by a mechanism which has been called molecular. This molecular effect may be explained as due to sudden stretching of nerves as suggested by Symonds (1940), or the jar may somehow alter cell membranes. Trotter (1923) conceived the cause to be the momentary cerebral anaemia produced by the impact of the blow upon the skull wall. Whatever the mechanism may be, the effect sems to be too instantaneous to be the simple result of interference with cerebral circulation (Miller, 1927). Nevertheless, temporary arrest of general circulation is often the most important associated phenomenon. Such an arrest injures the brain more rapidly than it does other tissues.

Recovery of the vital cerebral centres, if it occurs at all, may be complete. On the other hand it may be partial and in that case respiratory movements may return and then disappear in a few hours. There is little treatment that can be carried out in this early period except to keep the patient's head down and possibly to administer a stimulant, such as caffeine, adrenalin or coramine. This initial period is usually too brief to make possible the organization of artificial respiration and oxygen administration which would otherwise be indicated.

Stages of *recovery from concussion.* As a rule, movement returns while consciousness lags behind. In the most severe concussion there is complete paralysis, even of breathing. (1) In the first stage of recovery the patient breathes and his heart beats, but there is no other movement or reaction. (2) In the second stage he moves all his extremities and may respond to painful stimuli by withdrawal; pupillary reactions return. (3) This is frequently followed by a third stage in which the patient responds in a fragmentary sort of way when he is spoken to or hurt, and he may show signs of resentment, may even talk when roused. Obviously, at such times there is no more than a glimmer of his usual capacity for insight and understanding and this condition may continue for days.

In the recovery from severe concussion there are, therefore, three stages which lie between complete inactivity and the normal state. In stage one respiration returns. In stage two there is a return of somatic movements and certain pupillary and other automatic reactions. In stage three there is return of a modified form of consciousness, but with no insight and no memory.

Under rare conditions *partial concussion* may occur, due to regional paralysis of some area of the brain. For example, a boxer who is struck upon the chin may fall to the floor without losing consciousness because the major impact is conducted from the chin along the jaw to the base of the skull and the brain-stem, where the standing mechanism is temporarily paralysed. In other cases, a blow upon the head may produce a state of *automatism*, in which the individual gets to his feet and may execute some habitual activity but without being able to make decisions for himself or to carry out new instructions. In this state his bodily control is automatic or habitual but there is a paralysing disturbance within those parts of the brain, the activity of which is essential to conscious control.

2. CEREBRAL CONTUSION

Contusion of the brain most often occurs at the point of maximum impact but it may occur on the opposite side of

the skull by "contrecoup." It is obvious that when the skull is struck there may be momentary depression of the vault without necessarily any fracture, and that this depression brings about a change in the shape of the cranial cavity due to the elasticity of the cranial walls. When a person is thrown through the air and the head meets a resistant object, the momentum of the brain within the skull is also an obvious factor in the conduction of local impact.

When a man falls and receives a blow in the occipital region there is apt to be contusion or destruction of a small area at the tip of each temporal lobe, or on the anterior and under surface of each frontal lobe, *contrecoup injuries.* A similar blow upon the right parietal region may produce, by contrecoup, an area of contusion in the opposite parietal, frontal or temporal region.

When the head is not in motion and is struck by a small object of high velocity there may result only local damage with little or no general concussion and no contrecoup injury. Very small metallic missiles from blast may penetrate skull and brain with little generalized injury and the missile may well be sterile because of heat generated in its flight.

3. CEREBRAL LACERATION

Laceration occurs when there has been depression of the skull sufficient to break the dura and impinge upon the brain tissue. This often results from depressed fracture of the skull, and the local softening which occurs, together with the rapid ingrowth of newly-formed connective tissue, produces a scar which is a most frequent cause of posttraumatic epileptic seizures. Diagnosis of laceration can be made only by demonstrating that the dura has been torn or that fragments of bone or other foreign bodies have been driven into the brain.

4. CEREBRAL COMPRESSION

Cerebral compression may be produced by (1) bleeding from an intracranial artery or (2) vein outside the brain, or by (3) bleeding from an intracerebral artery or, in fact, by (4) edema of the brain itself. The development of compression occurs more rapidly as the result of arterial bleeding, less rapidly when it

is produced by a leaking subdural vein or by oedema of the brain. In all of these conditions, however, the compression may be considered acute as compared with the chronic compression that results from an expanding neoplasm or inflammatory lesion.

Signs of compression. Acute cerebral compression may arrest function in that portion of the brain which is directly pressed upon by the expanding blood clot, thus producing weakness or paralysis of the extremities of one side, aphasia, hemianopsia, or progressive decrease of mental alertness.

However, the most delicate indicators of generalized cerebral compression are autonomic rather than somatic. Alteration of the pulse is perhaps the most reliable sign of com-

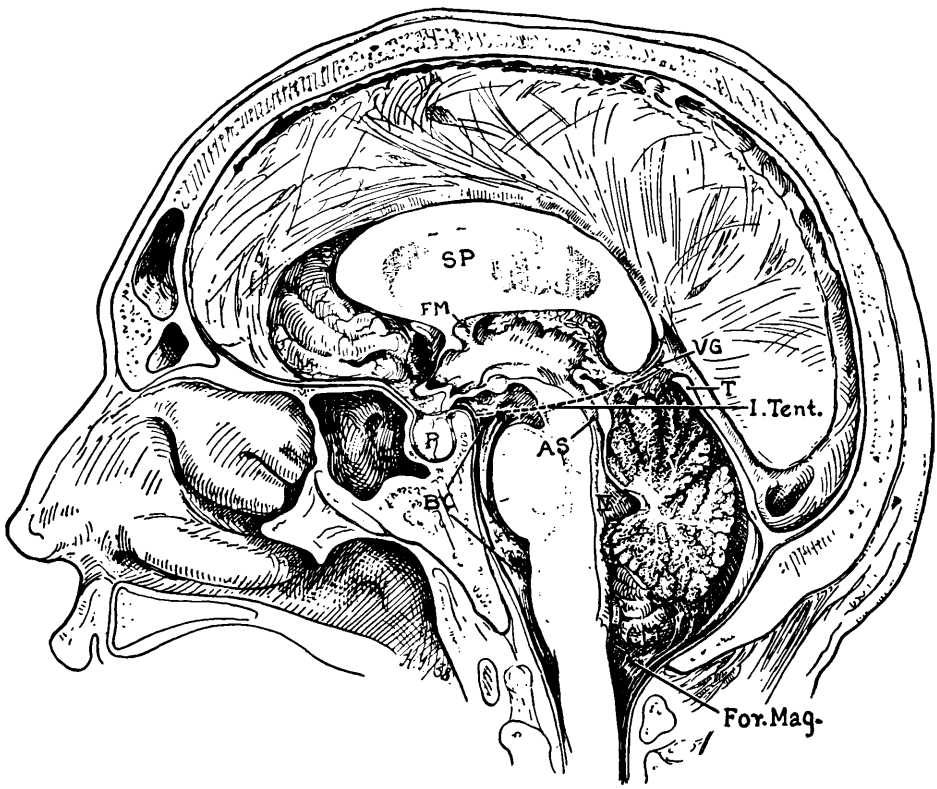


FIGURE 1

Section through skull to show structures in incisura of tentorium (1. Tent.) and foramen magnum (For. Mag.). Expansile pressure above the tentorium tends to shut off aqueduct of Sylvius (A.S.), thus producing hydrocephalus. Pressure arising either above or below tentorium may cause cerebellum to herniate into the foramen magnum and compress the medulla resulting in slowing pulse, Cheyne-Stokes breathing, etc. B.C. = basal cisternae in which cerebrospinal fluid passes forward to be absorbed in subarachnoid space.

pression. *Slowing of the pulse* rate down to 60 or 50 per minute, together with a pounding quality of the pulsation, almost invariably serves as a warning of increased pressure. This change in the quality of the pulse is due to the widening of pulse pressure. The systolic pressure is apt to rise and the diastolic pressure to fall. Later signs of dangerously advancing cerebral compression are *decreasing alertness*, *advancing paralysis*, *pupillary dilatation* on the side of the origin of the compression, and sometimes external *strabismus* of the eye on that side, and *Cheyne-Stokes breathing*.

In order to understand the mechanism of the production of the signs of *increased intracranial pressure*, it should be remembered that there are two major outlets for the contents of the cranial cavity, (a) the incisura of the tentorium, and (b) the foramen magnum (Fig. 1). It is obvious that any rapidly expanding object above the tentorium may cause the cerebral structures to herniate downward through the incisura and that there may likewise be herniation downward of the contents of the posterior fossa into the foramen magnum.

(a) *Herniation through the incisura.* This occurs whenever the supratentorial pressure becomes great enough and is associated with violent headache. The adjacent edge of the temporal lobe tends to be squeezed into the space between the incisura and the midbrain (Fig. 2), (Jefferson, 1938). This produces ischaemia of the midbrain which in turn results in *coma*. It results also in paralysis of the third cranial nerve by direct pressure at that level as shown in Figure 2. The third nerve may be compressed against the edge of the incisura (Reid and Cone, 1939) or against the clivus of the sphenoid bone (McKenzie, 1938). The oculomotor nerve innervates the pupil and produces inward rotation of the eye. Consequently the result of the paralysis of this nerve is *ipsilateral dilatation of the pupil* and, later when the nerve palsy is complete, *external strabismus* of that eye. Thus the expansile pressure may be considered greatest in the right cranial chamber if the right pupil dilates. Very rarely the dilatation is found on the side opposite to a haemorrhage. This is a sign of the utmost urgency.

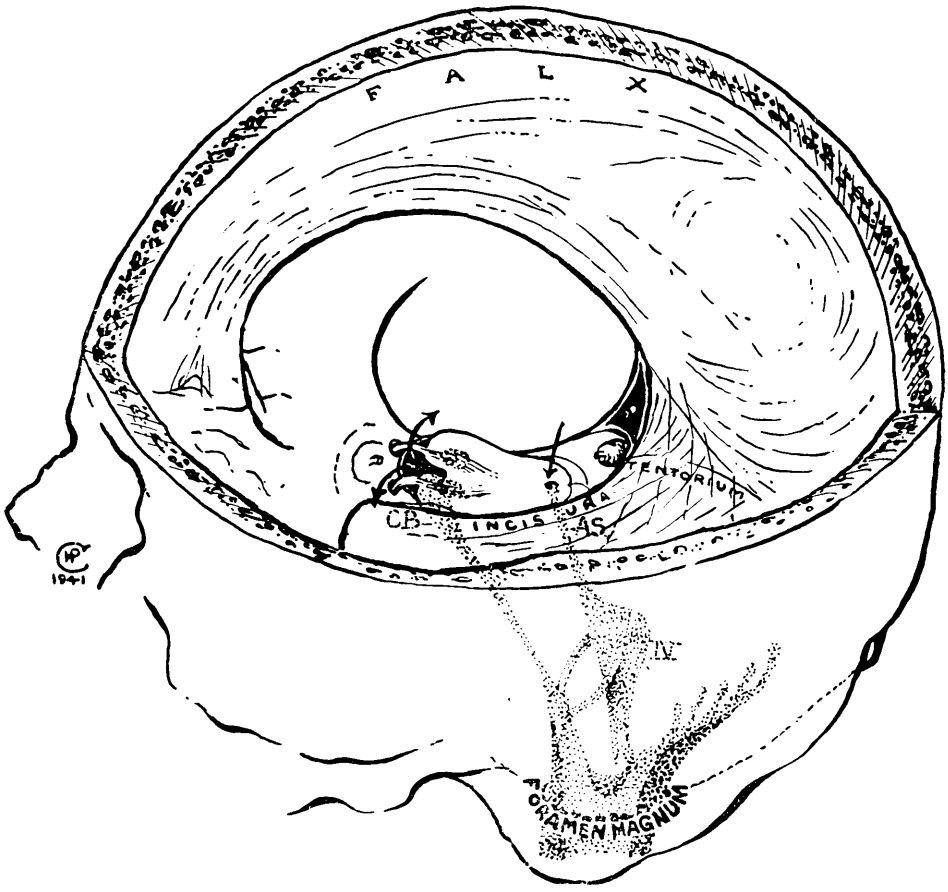


FIGURE 2

Open cranial cavity to show relationship of midbrain to incisura of tentorium; third cranial nerves seen leaving midbrain. Spinal fluid escapes normally down the aqueduct of Sylvius to the fourth ventricle (IV) and returns through the basal cisternae (C.B.) to pass into the subarachnoid spaces over each hemisphere. An expanding lesion such as a blood clot over one hemisphere tends to produce compression at incisura closing cerebrospinal fluid pathways and causing midbrain pressure with dilatation of pupils.

Furthermore the ischaemia of the midbrain may be sufficient to produce another change, i.e., the assumption by a patient of the attitude of *decerebrate rigidity*. In this attitude the trunk, legs and arms are straight and stiff and the hands pronated. These patients are apt to show recurring tonic seizures characterized by marked increase in this type of standing rigidity. Such tonic postural seizures may present elements of *opisthotonos* in which case the head is retracted and the back arched. Decerebrate rigidity is seen in cases

of very severe head injury especially when there has been bleeding within the brain-stem. Surgical interference is almost always useless in such cases.

(b) *Herniation into the foramen magnum,* or production of a *cerebellar pressure cone* also results in an easily recognized chain of final warning phenomena. The adjacent edges of the cerebellum are apt to be squeezed downward into the exit of the posterior fossa and thus the medulla oblongata is compressed (Fig. 1). This results in the *slowing pulse* and *rising blood pressure* which would seem to be due, at least in part, to vagal stimulation resulting from inadequate circulation in the medulla. If the ischaemia of the medulla becomes more severe, *Cheyne-Stokes breathing* appears. This is characterized by recurring periods of slowing or even temporary arrest of respiration. General alertness and spontaneous movement may disappear during each period of apnoea. The return of respiration is apt to bring with it, in each cycle, a return of some general bodily activity; the patient may stir and even speak. The appearance of Cheyne-Stokes breathing is the final warning that death approaches, but the patient may still be saved if he has an intracranial blood clot to be removed.

Another troublesome result of bulbar compression is *mucous obstruction* and difficulty in swallowing. The mucous secreted from the bronchi seems to become thick. It accumulates in the pharynx so that, unless it is removed or unless the patient is placed in a face-down position to allow it to run out, there may result interference with respiration and even *pulmonary collapse* because of bronchial plugging.

5. FRACTURE OF THE SKULL

Fracture of the skull is usually important, not of itself, but because of the amount of cerebral damage that may have been associated with a blow sufficient to crack the skull, and also because of the possibility of associated intracranial haemorrhage, and finally, because of the danger of its opening a pathway to the leptomeningeal spaces that may result in meningitis. Recognition of a fracture of the skull should be

by direct inspection, if there is laceration of the scalp. In any case roentgenographic examination must be made and should be carried out as early as feasible.

Fractures of the skull may be divided into four groups: (a) simple fracture, (b) depressed fracture, (c) fracture compounded by scalp laceration, (d) fracture compounded by injury of paranasal sinuses or mastoid air cells.

A simple fracture is apt to cause trouble only if it has crossed the course of the middle meningeal artery, which may run in or just beneath the bone, and which may therefore be torn by the fracture. A depressed fracture of the skull makes laceration of the brain likely; it therefore calls for the earliest possible surgical intervention that the general situation permits.

If a fracture is compounded by a laceration of the scalp or by an opening of the paranasal sinuses or mastoid cells, the danger of meningitis obviously calls for immediate attention. The danger of *posttraumatic epilepsy* is slight in the case of uncomplicated simple fracture of the skull, but the danger of such a sequel is very great in the case of depressed fracture.

6. EXTRADURAL HAEMORRHAGE

Rupture of the middle meningeal artery usually produces a haemorrhage and increasing blood clot between skull and dura, because the artery runs either within the skull or in the outer layers of the dura. Such a haemorrhage usually gives evidence of its presence within the first six hours and may well produce death within the first twelve hours after injury. Any patient who has had an initial loss of consciousness due to concussion followed by a lucid interval of return of consciousness, succeeded in turn by a second loss of consciousness, should be considered to have an extradural haemorrhage until it is proven otherwise, see page 56.

The increasing blood clot rapidly produces signs of *cerebral compression*, such as have been described above, and usually results in an advancing hemiplegia and decreasing mental alertness followed by Cheyne-Stokes breathing and death.

This may be with or without the usual terminal dilatation of the ipsilateral pupil.

7. SUBDURAL HAEMORRHAGE

This is apt to produce evidence of *cerebral compression* which comes on more slowly, making its appearance some time during the first few days. The pressure in the veins, which cross the subdural space from brain to sinus, is low and the bleeding therefore probably takes place only at intervals when the venous pressure is raised or the general intracranial pressure is lowered. It is because of the danger of this form of bleeding, that the patient's head should be kept up rather than down during the early stages of his convalescence. As such bleeding continues it gradually pushes the brain away from the dura and this displacement may rupture one subdural vein after another. A subdural clot is less apt to produce focal paralysis. Increasing generalized intracranial pressure may give the only evidence of its presence.

8. CHRONIC SUBDURAL HAEMATOMA

This condition may result from a serious or from a trifling head injury. The mechanism of formation would seem to be as follows: Enough bleeding occurs beneath the dura at the time of what may have been a trifling injury to create a blood clot there. The bleeding, however, does not continue. On the other hand, spinal fluid seems to pass through the arachnoid membrane into the fluid haematoma under the influence of osmotic tension. This results in a gradually increasing volume and there may, as well, occur successive haemorrhagic increases due to rupture of subdural veins by the expanding haematoma. No evidence of all this may appear until weeks or months after the original injury. Cerebral compression and increase of intracranial pressure then usually call attention to its presence.

A membrane forms about the fluid clot, an inner and an outer membrane. But, from the beginning, the clot within the subdural space is, of course, separated from the subarachnoid spaces by the arachnoidea which is adequate to prevent the passage of the subdural fluid, with its high pro-

tein content, through into the cerebrospinal fluid. Consequently there is no increase of temperature nor evidence of meningeal irritation; no aseptic meningitis. On occasion, there may be a slight increase in cells in the cerebrospinal fluid or a slight increase in protein, but often the fluid is entirely normal.

9. SUBARACHNOID BLEEDING

In most cases of fracture of the skull some blood will be found in the spinal fluid within the first few days after the injury. The presence of small amounts of blood in this space gives rise to no symptoms or signs. On the other hand, if the haemorrhage into the subarachnoid space is large there is usually evidence of meningeal irritation. If the blood accumulates in the basal cisterns the patient will have *neck rigidity* for a few days and he usually complains of *photophobia* which may continue for a considerable period.

From the point of view of prognosis and treatment, the finding of blood in the spinal fluid carries with it the same indications as the finding of a simple fracture of the skull. The initial puncture of the lumbar theca should therefore be made with care, and spinal fluid should be routinely withdrawn into three successive test tubes so that the red cells may be counted in each tube, as described on page 58.

It is important to recognize that blood in the cerebrospinal fluid normally disappears within five or six days regardless of lumbar puncture (Sprong 1934). Xanthochromatin, which gives the spinal fluid a yellow tinge, makes its gross appearance on the second or third day following subarachnoid haemorrhage and is entirely absent, or reduced to a faint trace, in about six days. On the other hand, if red cells are found in the spinal fluid after that period, they should be taken as evidence of continued bleeding. If the yellow discoloration continues for a long period it should be taken as evidence either of continued bleeding or that there is a blood clot of considerable size in contact with ventricle or cistern. In that case some elevation of temperature will be observed (*aseptic meningitis*).

10. INTRACEREBRAL HAEMORRHAGE

A large blood clot within the brain gives rise to increase of intracranial pressure as well as to paralysis. The diagnosis may have to be made after the exclusion of the presence of subdural accumulation. Haemorrhage within the brain may give rise to immediate seizures. This is particularly true of small subpial haemorrhages which are apt to result in a few focal attacks which are not particularly difficult to control by medication.

11. MENINGITIS

The diagnosis of meningitis requires little discussion here. For treatment, see page 49. Positive culture, or demonstration of the organism on immediate smear from the spinal fiuid, is required as final proof of its existence. The appearance of hyperthermia and stiffness of the neck lead to the suspicion of true meningitis. However, it should be remembered that a continuous hyperthermia, and even the presence of large amounts of polymorphonuclear leucocytes in the cerebrospinal fluid do not necessarily prove the existence of bacterial meningitis.

12. ASEPTIC MENINGITIS

This may occur when there is communication between a blood clot (or a space within the adjacent muscles) and the arachnoid cisterns. Such a false meningitis (meningeal syndrome of Jefferson, 1919) may give rise to recurring episodes of elevated temperature or may result in a low-grade hyperthermia that disappears only after weeks. In the case of recurring episodes the patient is apt to have a sudden headache with stiffness of the neck and elevation of temperature. If lumbar puncture be done at such a time a large increase in polymorphonuclear leucocytes may be found, even up as high as 1,000. This increase disappears within 24 hours. Such episodes seem to be produced by the sudden discharge of the accumulated product of blood clot digestion into the subarachnoid space.

13. CEREBRAL OEDEMA

Oedema of the brain may result from brain trauma. It may occur when there is no intracranial haemorrhage, or may be associated with haemorrhage as an additional element in the production of increased intracranial pressure. Cerebral oedema is apt to be at the maximum during the first five days after a head injury. The swelling doubtless results from the entrance of an increased amount of fluid into the cerebral tissue and it can be combated by the intravenous administration of glucose, sucrose or sodium chloride in hypertonic solution. Nevertheless, the effect of these *hypertonic solutions** is so transient as to be of rare practical value. Lumbar puncture may more effectively lower the pressure (see page 59).

Whatever the chemical processes may be, the clinician must realize that cerebral oedema is a phenomenon to be expected and dealt with, just as he accepts oedema produced in other parts of the body by trauma. The particular difficulty in regard to the brain arises from the rigid resistance of the cranial box which prevents the expansion of this viscus.

* Glucose solution 25%, 150-200 cc.; sucrose solution, same dosage, is somewhat more effective; saline solution 5%, 200 cc.; magnesium sulphate one ounce by mouth or in 50% solution, 2-3 ounces by rectum will also reduce brain oedema temporarily but is much less used recently because limitation of fluid intake will produce a similar result.

Chapter II

TREATMENT OF ACUTE HEAD INJURIES*

EARLY analysis of a case of head injury is important. It must be decided initially whether or not the prognosis is hopeless. If not, then energetic steps may have to be taken to prevent meningitis, paralysis and death. But this is not enough; the treatment must also be directed toward the prevention of the later sequelae: chronic posttraumatic headache, posttraumatic neurosis, and posttraumatic epilepsy, as well as the preservation of the patient's intellectual capacities.

1. SELECTION AND DISTRIBUTION

It should follow from the discussions in the preceding chapter that the early complications of head injury may be readily distinguished from traumatic *shock*. In general, the patient who is in shock has a low blood pressure and is apt to have a weak and rapid pulse. He may well be quite conscious and anxious. In contrast to this, after the initial period, a patient with a dangerous increase of intracranial pressure is apt to have a slow, strong pulse; he is apt to be drowsy; he does not present the clammy extremities of a patient in shock. If his extremities are cold they are usually dry and his temperature somewhat elevated, while the patient in shock is apt to have a low temperature.

Hopeless prognosis. The criteria which may be said to mark a case as hopeless are few. In general, they are the signs of terminal cerebral compression as described on page 13, or of direct lesions of the brain-stem. Coma associated with dila-

* The section on *Simple Head Injuries and Routine Care of all Cases* is placed last in this chapter, on page 55, so that discussion may precede it. However, the reader may prefer to turn to it directly.

tation and fixation of the pupils indicates a hopeless situation, especially if combined with bilateral plantar extension signs. Coma with recurring spasms of decerebrate rigidity may be taken as almost certainly fatal; Cheyne-Stokes breathing, with or without coma, is not necessarily fatal. If there is time and opportunity to do a subtemporal craniotomy for extradural haemorrhage, provided the patient has one, then even Cheyne-Stokes breathing should not necessarily be accepted as an excuse for inactivity.

Severe head injuries, even penetrating wounds of the brain, are not necessarily fatal. If the surgery is energetic and well directed these patients may often be saved for useful happy life.

Jefferson, referring to his series of 220 cases of head injury in the war of 1914-1918, pointed out that there were 54 simple scalp wounds, in many of which cases there were abnormal neurological signs, 53 compound fractures without dural penetration, and 113 with laceration of the dura. There were no deaths in the first two groups (an excellent record), and among the patients of the third group there was a mortality of 37.8 per cent. He observed that this mortality rate was higher than should have obtained among similar patients in civil practice.*

The reasons for the difference in civil and military figures were the greater depth of wound in the latter, the more plentiful bacterial contamination and the longer time interval between wounding and operation. Although improved facilities, such as chemotherapy, are now at hand, the only important element among these causes that we may hope to alter is the time factor. Immediate operation upon cases of head injury on the field of battle would be highly desirable. But such operations must not be undertaken until the patient reaches a hospital where the facilities are adequate.

Cushing's instructions to neurosurgical teams in the American Expeditionary Forces in 1918 were as follows:

"If a case is operated upon and a penetration found, the operation must be completed, with a primary closure following the special débridement applicable to these injuries... 'All or nothing' is a good rule

* For further discussion see Jefferson, 1940.

to apply to craniocerebral injuries—in short: evacuate these cases untreated to the nearest base (except for shaving and the application of a wet antiseptic dressing) rather than do incomplete operations. Patients with craniocerebral injuries stand transportation well before operation; badly during the first few days after operation."

In the Spanish Civil War such patients were at first operated upon in the most advanced stations, but later were all sent back to Spanish No. 2 Hospitals (Jolly, 1940). Obviously, therefore, it is necessary either to move well equipped surgical teams far forward to handle head cases, or, if this is not practicable, these patients must be sorted out early and transported back with the greatest speed to specially trained men in stationary hospitals.

If such steps are not taken, then the "head case" is inevitably marked for neglect and death while the attention of busy surgeons is turned to less formidable injuries. General instructions should be issued and enforced that *transportation of unconscious patients* must be carried out with the patient in a prone or semi-prone posture to avoid aspiration of mucous and vomitus. A warning should be sounded also that patients should not be left for recovery from shock until the period has passed when primary closure of a scalp or craniocerebral wound could have been carried out.

2. INITIAL TREATMENT

The initial treatment of patients following head injury must depend to some extent upon the general conditions which obtain at the Casualty Clearing Station, Field Ambulance, or First Aid Post where the injured man is first seen. The treatment of these wounds must also be in conformity with the general principles of wound treatment which have been adopted, principles which are of necessity in a state of evolution because of advances in chemotherapy and inevitable alterations in the nature of the destructive agents of warfare.

Medical officers may refer to such recent collections of opinion as Surgery of Modern Warfare, edited by Hamilton Bailey, 1940; also the War Primer on Wound Infection edited by W. H. Ogilvie, 1940. Except in minor details the

initial treatment about to be described is in agreement with that outlined in the "Memorandum of the Treatment of Head Injuries" distributed to Field Ambulances, Casualty Clearing Stations, and Stationary and Base Hospitals in 1939.*

If there is *unconsciousness*, the patient is usually better if placed on his side or in a semi-prone position, as he may otherwise aspirate vomitus or mucous secretions. Unless there is a particularly low blood pressure, as in shock, it is better to raise somewhat the head of his stretcher or bed. No *sedatives* should be given at this time unless the patient is uncontrollable, in which case sodium amytal, gr. 3, p.o. or gr. 3½, s.c., sodium luminal, gr. 2, s.c., nembutal, gr. 3, p.o., codeine, gr. 1, s.c. or p.o., chloral hydrate, gr. XX, p.o., or paraldehyde, dr. 3, p.r. may be used. Morphine should usually be avoided as it hides important signs of change in the patient's condition and sometimes has a definitely adverse influence upon him. Detailed record of a "head case" must be made at the earliest moment, as described below in the subsection on Routine Care, page 55. *Hot water bottles* should never be placed next to a comatose patient.

Wound closure. Procedures recommended for special cases are described in detail under the sections which follow on the treatment of compound fractures of the skull, page 38, and penetrating wounds of the brain, page 42. Scalp suture has been called by Cairns the "most important cranial operation of war." It should be done with great care. In general, scalp lacerations may be closed tightly, after careful cleaning and excision of devitalized tissue, within six to eight hours of head injury.† If the wound has had efficient initial treat-

* As described in a communication from Professor Hugh Cairns, this memorandum gave instruction to apply artery clamps to bleeders and pack the scalp wound with wet gauze soaked in antiseptic. The antiseptic which would be recommended now is proflavine sulphate, 1-1000 solution, (Russell and Falconer, 1940, also Cairns, 1940). Further instructions were as follows: Close clip the scalp and clean it well and shave if possible. Remove pack and clean out wound but do not touch brain. If there is skull fracture pack the wound lightly with antiseptic gauze, remove artery forceps and if bleeding recommences stitch the scalp loosely together over the gauze which should be left protruding between sutures. External dressing of the proflavine and firm bandage is to be applied and sulphonamide therapy begun and patient evacuated rapidly.

† Jefferson recommends that permanent suture of the scalp should not be carried out in Field Ambulances, and that sutures there should be temporary and only to arrest bleeding.

ment, such as is described in this section, closure may be carried out 12 hours after injury. It is possible that this time may be even longer if sulphonamide treatment, local and general, has been used with energy, but this time question can only be settled after further experience in this war. If the time is longer than just mentioned, or if devitalization and contamination is great, the wound should be closed with *drainage* (rubber tissue or gutta percha) in the wound or through a "stab" wound. If infection has already set in, wound closure is contraindicated, see page 48.

Antiseptics. Reliance on mild antiseptics is open to criticism. Lacerated tissues may well be treated by proflavine initially, but the skin should have more energetic and rapid antiseptic treatment. It is essential to clip the hair and clean the scalp and, if possible, to shave it.* The skin is then best sterilized with an alcoholic solution of iodine (2½%) provided care is taken to prevent burns of the skin. Iodine may also be rubbed on to the lacerated edges of scalp. Iodine should never be poured into a wound in any case, and if the dura is open this is especially true. Lacerated brain should not be painted with iodine, but if a little antiseptic reaches the brain it may be remembered that at the time when the wound is finally revised the superficial damaged parts will probably be excised, the lacerated brain as well as the scalp.

The open wound, even though the dura may be opened, may be treated with some hope of sterilization with proflavine, and, if it is not to be sutured at once, a moist dressing of proflavine may be placed upon it. Iodine may be removed from the peripheral parts of the painted scalp by means of a little alcohol or water to avoid a burn. If gauze, wet in proflavine, sterile saline solution, boracic solution, or Dakin's solution, be placed over the wound and the surrounding skin, no burn need be feared. Such a moist dressing gives best results. Mercuric solutions should, of course, never be used after iodine.

Sulphonamides. When the wound is initially sterilized, sulphanilamide powder or sulphathiazole powder, and prob-

* Horrax urges in the Manual of Neurosurgery in preparation for the American Forces that "it would be of the greatest possible benefit if soldiers could all go into action with closely cropped hair." From the surgical point of view, this would obviously simplify the cleaning and débridement of head wounds.

ably sulphadiazine, (not sulphapyridine) may be dusted into the depths of the laceration, especially if the dura has been penetrated and the brain is exposed. This is the time when the bacteriostatic action of these sulphonamides may be really effective and it has been shown that they produce no adverse effect upon the healing of the eventual brain scar. The presence of escaping cerebrospinal fluid should make them more effective.

Sterilization of head wounds. This method of initial sterilization of contaminated head wounds may therefore be summarized as follows:

(a) Wash, clip and (if possible) shave the scalp. Remove dirt and foreign bodies from the wound and secure haemostasis as described in the quoted instructions from the Memorandum.

(b) Sterilize the scalp* and the wound edges energetically with iodine on gauze and without excess overflow of the liquid. Remove dried iodine at periphery of sterilized field with alcohol or a sponge moistened in sterile water.†

(c) Clean out the wound and sterilize with proflavine, but do not explore a track which enters the brain. Dust sulphathiazole (or sulphanilamide) powder into wound, and cover the wound and the adjacent skin with gauze moistened in: (1) sterile water, (2) saline solution, (3) boracic acid solution, or (4) proflavine. If time and facilities permit, put vaseline on the scalp at periphery of sterile field and place over the wet gauze an impermeable covering, such as rubberized tissue, celloidin film, waxed paper, or vaselined gauze. If none of these coverings is at hand, cover the wet gauze with a dry dressing and bandage securely.

(d) Instead of using a sulphonamide powder, as described above, the open laceration may be simply packed with gauze moistened with proflavine sulphate. This is quite satisfactory if the time before secondary treatment is likely

* Preparation of the scalp for operation and the technique of postoperative dressings will be discussed below.

† Iodine will not burn the skin beneath a moist dressing. The moist dressing also promotes solution of the sulphonamide, if used, and keeps skin and wound in good condition for resterilization in case of a secondary procedure.

to be short, whereas the method described under (c) above is preferable if a longer interval before revision is probable.

3. NOTES ON SURGICAL TECHNIQUE

In this section miscellaneous matters will be referred to which do not fall under other headings. These matters are of common knowledge among neurosurgeons. Complete agreement has never been, and probably never will be, reached among the surgeons of democratic countries. But this text may serve a useful purpose to the surgeon who has not already well formed habits in neurosurgery.

Scalp closure. If the incision in the scalp has been extensive or if the dura has been opened, the scalp should be closed in two layers (Fig. 3A) using silk for the aponeurosis in order to prevent the occurrence of a fluid fistula through the incision after removal of the superficial skin sutures. If, on the other hand, the intracranial procedure is minimal in extent, one layer of sutures suffices to close the scalp (Fig. 3B), but in this case the needle should pass down into the galea aponeurotica to secure haemostasis. After simple scalp laceration one layer of sutures is usually enough and steel suture is even better for this purpose than silk.

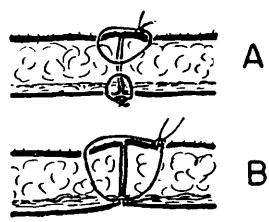


FIGURE 3

A: The scalp has been closed with a suture in the galea aponeurotica and a separate superficial suture. The aponeurotic suture provides for haemostasis and prevents escape of cerebrospinal fluid. B: Scalp closed with one suture.

Closure of galea aponeurotica. The necessity of careful closure of the scalp in two layers when the dura has been opened was repeatedly emphasized by Harvey Cushing. It is a time-consuming procedure but it prevents escape of cerebrospinal fluid which may be present beneath the scalp even after the superficial scalp sutures must be removed

(4th to 6th day). In such a case, if no closure of the galea has been carried out, the incision will open under the pressure of accumulated fluid and meningitis and hernia cerebri may result or at the least poor secondary healing of the incision. This aponeurotic suture serves also for haemostasis, see page 34. When the haemostats (Fig. 4) which were initially placed upon the galea to secure temporary hemostasis are removed, interrupted silk sutures should be passed through the galea, tied three times and cut quite short. It is a little better if the knot is placed below (as in Fig. 3A) as it has much less tendency to work to the surface later on.

Subaponeurotic aspiration. Accumulations of cerebrospinal fluid may have to be aspirated repeatedly with a small lumbar puncture needle through normal scalp. Care should be taken to sterilize the scalp well and the trocar should always be present in the needle at the time of its insertion, for an empty hollow needle is apt to carry in bits of epithelium capable of setting up infection. Aspiration is not necessary if drainage has been employed, but many surgeons prefer repeated aspiration, as Cushing did, instead of drainage, in the belief that danger of contamination is less.

Drainage of the traumatic wound should be employed in many cases, particularly if the time that has elapsed between injury and operation is over 6 to 12 hours. When to drain is discussed on page 26. Drainage is best carried out by making a stab wound, that is a separate incision at a distance from the operative incision or lacerated wound (Fig. 6), and placed over intact skull.* A rubber dam drain (Penrose tube) is placed beneath scalp and drawn out through this opening (D in Figure 13). In case cerebral débridement has been carried out, the buried end of the drain may be placed in the site of the excision through the opening in the dura. Such drainage serves to carry out excess spinal fluid and allows the escape of some blood and thus ensures initial decrease of local pressure. When the drain in question is removed, which should be within the first day or two, escape

* When the stab incision is made arterial bleeding from scalp may be bothersome, in which case the vessel should be caught in a haemostat and the electrocautery used if available. Otherwise a suture may be passed downward to the aponeurosis from the skin and tied without crossing the incision.

of fluid may be stopped because of the fact that the track leads between skull surface and scalp where it is easily compressed if desired.

Moist dressings. The surgeon, who would use drainage for cerebrospinal fluid, must understand the technique of application of *moist dressings* to scalp incisions. A small dressing, wet with sterile boracic or saline solution, should be placed so as to cover the incision and the drain hole (G in Figure 13). The sterile scalp about the periphery of the wet dressing should be covered with vaseline* (V in Figure 6 also V in Figure 13). A sheet of impermeable protective tissue should then be placed over the dressing (P in Figure 6, also P in Figure 13) so as to extend out over the vaseline around the periphery of the dressing. More sterile gauze or cotton may be placed over this and the head bandaged so as to include both ears. The inclusion of the ears, which are padded with cotton so as to avoid discomfort, will prevent dislodgement of the dressing. Instead of a circular bandage liquid adhesive may serve the purpose of preventing dislodgement.

Incision dressing. When the incision is closed it may be covered with strips of gauze about one inch wide, preferably moist when applied, so that it will cling to the skin when the rest of the dressing is removed. If 4 to 10 layers of gauze are used compression from the bandage will be more effective. Silver foil upon the incision, if available, has real advantages as it adheres even when a restless patient removes his head dressing, and it has a mild continuing antiseptic action.

Head bandage if too tight produces ischaemia and poor healing of the incision edges. It may cause enough pressure at a distance from the incision to produce a small scalp pressure sore. During convalescence, a patch on the head where hair fails to grow is a tell-tale of too tight a bandage or an evidence that the patient lay in one position with no head movement after operation. The cause of such pressure may be a combination of tight bandage and bed pressure. On the other hand, too loose a bandage fails to have the haemostatic effect which is necessary.

* The vaseline should be sterilized by heating to 200 degrees, centigrade.

When the incision has been covered either moist or dry gauze may be applied. Over this gauze it is well to put a layer of cotton wool placed posteriorly in case of cerebrospinal fluid drainage. If the dressing is the first after operation and haemostatic compression seems desirable, use firm pressure with circular bandage, but in that case it is wise to cut the bandage, at a distance from the operative field, three or four hours after its application. This will lessen the increasing tension, for scalp oedema may increase the pressure enough to result in pressure necrosis. In general, as already mentioned, it is best to include both ears to prevent dislodgement. Put vaseline on the ears and cotton wool in front of and behind each ear so as to avoid discomfort.

Anaesthesia. During any intracranial operation it is important to follow the *blood pressure* by means of a pressure cuff and stethescope with long tubes, and the pulse and respiration at regular intervals not longer than ten minutes, the result being charted.

For cranial operations the best general anaesthetic is *ether.* Care must be taken to avoid *anaesthetic explosion* if the cautery is being used. In such a case a small, sterile rubber sheet may be placed around the head so as to conduct the fumes away from the field of operation. If suction is available a suction tube may be kept near the patient's face to draw off fumes. If these methods cannot be employed then cautery should be forbidden or the ether taken away before its use.

The ether is best given by intranasal catheter or with a soft intratracheal tube. Basal anaesthesia with *avertin,* up to 100 mgm. per kilo by rectum, is most satisfactory. To this, small amounts of ether may be added. But if the patient's blood pressure is low avertin should be avoided.

Various forms of *gas anaesthesia* are unsatisfactory as they tend to raise venous pressure and cause brain swelling, and venous bleeding. Intravenous anaesthetics may be useful.

Local anaesthesia. The most satisfactory method for cranial surgery as a rule is local analgesia. In case of a large procedure the blood pressure and pulse should still be followed and charted, and this can quite well be carried out by a

specially trained nurse provided change to general anaesthesia is possible when required. Novocaine, nupercaine, procaine, etc., may be used, to which seven drops of adrenalin (1-1000) are added for each 100 cc. The author's own preference is for nupercaine. It can be used in two strengths for a procedure of considerable magnitude, nupercaine, 1-1500 solution injected into the skin and 1-4000 beneath the skin. A low limit of safety for adults is 100-125 cc. for the stronger and 100 cc. for the weaker solution. If adrenalin is placed in both, this will give complete analgesia for six hours.

For a laceration inject the local anaesthetic into the scalp in a circle about it. If a craniotomy is contemplated inject through the anaesthetized skin to periosteum as well. If it is desired to anaesthetize the temporal muscle put the stronger solution (½% novocaine or 1-1500 nupercaine) just outside the fascial covering of the muscle. After a little delay, penetrate the fascia and inject the muscle down to the bone slowly, as tension is painful. After the above injections there will be no further pain except when the middle meningeal artery is handled or the large dural sinuses and these are easily avoided or controlled. The sound of rongeuring or sawing the bone is distressing and it is wise to warn the patient each time that he will hear the noise.

Skin preparation for operation. The scalp must be shaved, carefully and well, shortly before operation; not the night before, as cuts may become infected. Wash with soap and water. In operating room clean scalp thoroughly with alcohol followed by ether. If iodine is used, which the author would recommend, at least under service conditions, paint with 5% tincture of iodine; allow it to dry and then remove it with alcohol. Be careful that no strong stain is left at periphery of field. The local anaesthetic is then injected and the incision marked out. Then repaint the actual field of operation with 2½% tincture of iodine which is not removed. Suture the towels into place. This should be carefully done as the anaesthetist, in handling the face and moving the towels, may cause puffs of air to cross the wound laden with dangerous dust. If desired, vaselined tape may be placed

under the towel edges as a further precaution against such form of contamination.

When the operation is over, a dressing which is moist (being wrung out of saline, boracic, Dakin's solution or proflavine) may be placed on the skin. This moist gauze will prevent all possible risk of an iodine burn and will stiffen and splint the scalp as it dries.

If the use of a moist skin covering seems to be too revolutionary a procedure to the surgeon, he can, of course, use a dry dressing perfectly well. There is no chance of burn in the operative field in any case because of the fluids contributed by both patient and surgeon. Reasonable care of the periphery is quite adequate to prevent burns invariably.*

Haemostasis. This is always a problem to the general surgeon when he operates upon brain or spinal cord. *Bone bleeding* is controlled by the use of Horsley's bone wax which is forced into the diploe with instrument or gauze. Any wax that may pass under the bone in the process should be removed. If no wax is available, bleeding may be stopped sometimes by crushing or compressing the bone with incomplete closure of the rongeur jaws.

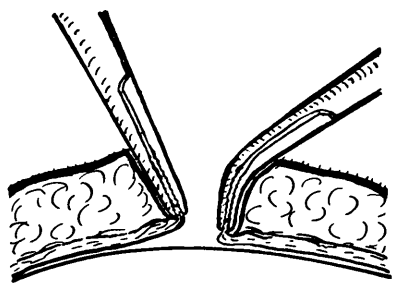


FIGURE 4

Temporary control of haemorrhage from lacerated or incised scalp by artery clamps fastened to galea aponeurotica. Straight haemostat, left, and curved haemostat, right.

Scalp bleeding is temporarily arrested by a pointed hemostat which is placed on the galea and allowed to fall outward, and either a straight or curved artery clamp may be used, as shown in Figure 4. On closure a smart "bleeder" may be cauterized if care is taken not to cauterize the skin.

* Many of the distinguished pupils of Cushing still prefer to use alcohol and bichloride for skin preparation and no one can deny that this technique, under perfectly controlled conditions, has given excellent results. Nevertheless it is not recommended at least under conditions of warfare.

For the most part, however, the bleeding must be controlled by the line of sutures in the galea (A in Figure 3) or by carrying the single suture down to the galea (B in Figure 3).

Brain bleeding is controlled by means of a Cushing silver clip or with the electric cautery. The clip must not be applied too firmly for the delicate walls of intracerebral veins or arteries are easily broken. Larger vessels may be tied with fine silk. In the case of bleeding from a vessel deep in the brain the sucker must be used. First the softened brain is removed and then the vessels which do not pass down the sucker with the cerebral tissue can easily be picked up by the sucker and a clip placed upon them or they can be cauterized.

Methods of haemostasis within the brain are of great importance. If it is necessary to choose between them, the method of suction and clip is more dependable and useful than haemostasis by means of cautery. Without any of these means the patient may well bleed to death, although hot cotton wool packs may arrest bleeding and, if absolutely necessary, the wound may be closed and the pack removed in a day or two.

With more superficial bleeding *muscle stamps* may be used. This method devised by Horsley is quite successful for bleeding veins and indeed is sometimes the only successful method when the skull has been removed over a large dural sinus which begins to bleed through its outer surface. The fresh piece of muscle is crushed out flat. This is then applied to the bleeding point and a flat piece of cotton placed on it temporarily. Hot saline or Ringer's solution is then dropped onto the cotton while cotton and muscle are compressed for a time.

Muscle bleeding may be arrested with ligatures but much time is saved if haemostats are placed on the bleeding vessels and cauterized.

Subtemporal craniotomy for ligation of middle meningeal artery. As pointed out on page 17 haemorrhage of the middle meningeal artery results in haemorrhagic accumulation of blood between the dura and skull. The clot accumulates beneath the temporal bone.

An incision about three inches long should be made in the skin of the temporal fossa one inch anterior to the external auditory meatus and slanting a little backward so as to be parallel with the fibres of the temporal muscle, as shown by the broken line in Figure 5. The temporal fascia and muscle is then incised down to the bone and reflected with a periosteal elevator to either side. To hold the muscle bundles forward and back a small automatic retractor is most useful. The bone is then opened quickly by perforator and burr or by trephine. It is best to make this initial bone opening somewhat posterior and low so as not to come down at once upon the main artery. The bone opening is then enlarged quickly with a rongeur.

If an extradural haemorrhage is present, clot will escape from the bone opening under great pressure. When the artery is exposed, as shown in Figure 5, a blunt dissector may be placed upon its bleeding point to arrest haemorrhage while enough bone is removed to expose the artery trunk below, where it may be tied or clipped.

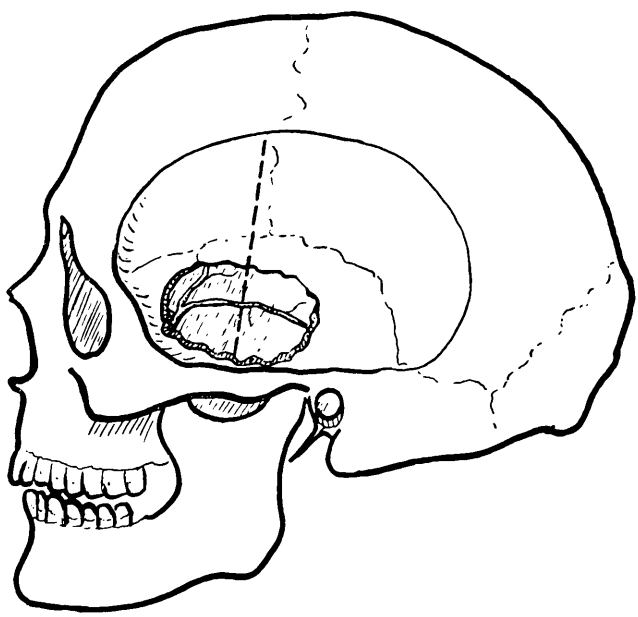


FIGURE 5

Skull opened as in subtemporal craniotomy for ligation of middle meningeal artery. The artery is seen upon the exposed dura. Broken line gives position of skin incision.

If silver clips are available, a small incision may then be made part way through the dura at the side of the artery and a small dissector passed under it. This dissector may often be passed between inner and outer layers of dura without opening the inner layer. After that a silver clip may be placed upon the artery. If a clip apparatus is not available, a small curved needle may be used to pass a silk ligature about the artery so that it may be tied.

If difficulty is encountered in either of these procedures, keep the dissector upon the bleeding point of the artery and elevate the dura with a periosteal elevator from the floor of the temporal fossa until the foramen spinosum is encountered through which the middle meningeal artery enters the skull. The foramen lies at the bottom of the curving fossa and the artery may be seen to arch inward and backward into it. The simplest way of shutting it off is then to take a small round ball of cotton about three mm. in diameter on a pointed right angled hook and wedge it downward into the foramen; or a small wooden peg may be forced into the foramen. Such a peg may be whittled out of a wooden tongue depressor.

Closure should be carried out, the muscle in layers with silk and the fascia in a separate layer, followed by galea and skin.

Subtemporal decompression. To carry out a subtemporal decompression, as described by Cushing, the approach is much the same. The bone opening should be as large as possible beneath the temporal muscle and its edges trimmed off smooth and the dura opened by radiating incisions. The muscle is then closed very carefully and the other layers as described above. The dura is opened only as high as the underlying fissure of Sylvius. This permits the temporal lobe alone to bulge against the muscular and fascial diaphragm and thus decompresses the cranial cavity.

A more effective decompression may be produced by the myoplastic method of Penfield and Cone (1936). The whole muscle fan is turned down, the bone removed and the muscle and fascia refastened by steel sutures placed through holes punched in the edge of the bone defect (Cone skull punch).

Trepanation for subdural haematoma. This is a procedure

which may be carried out quickly with little risk. It is usually best to shave the whole head and place it so that a hole can be made about one inch from the midline on either side of the head, and in at least one temporal fossa.

An incision one inch in length is made and held open by an automatic retractor. If such a retractor is not available the incision may be made a little longer, and pointed haemostats placed on the galea to check haemorrhage from the arteries adjacent to the galea, as shown in Figure 4. The bone is then opened with perforator and burr down to the dura exposing an area of dura about half an inch in diameter. Then lift the dura with a dural hook or with the point of a small curved needle held in a clamp and incise it, taking care not to wound veins which may lie beneath on the surface of the brain. If a subdural haematoma is present, blood will escape at once. A small catheter may be introduced into the subdural space and clot irrigated out from a considerable distance. A little time may well be allowed to elapse to see if the brain will come out spontaneously.

Closure may then be carried out. It is unnecessary to close the dura but great care must be given to *suturing the galea* with interrupted silk sutures as shown in Figure 3A. If bleeding should occur from the galea it will enter the subdural space and meet very little resistance until it causes fatal compression. In some cases, if a large clot is encountered, it is advisable to drain this scalp incision for 24 hours, provided the surgeon understands the technique of subaponeurotic drainage as described on page 29. Otherwise it is best to close the scalp tight. If no haematoma is encountered in either parietal region, it is usually better to make a burr hole in the temporal fossa on the suspected side.

Before operation when subdural haematoma is suspected, much help can be had by an anteroposterior *roentgenogram of the skull to show the pineal gland.* This gland is calcified in about 40% of adults, so that it may be visualized. Displacement of the pineal, even a few millimeters to one side, indicates that a clot is present. The displacement is apt to be greater when the clot is situated posteriorly rather than anteriorly in the cranial cavity.

4. TREATMENT OF COMPOUND FRACTURES OF THE SKULL

Pyogenic meningitis is likely to result from a head injury if there has been fracture of the skull compounded with laceration of the scalp. Prompt and thorough débridement and suture of the scalp will prevent infection from this source almost invariably. If a fracture of the skull is compounded with the paranasal sinuses, and particularly if there is depression and bad comminution of the walls of the sinuses, the threat of meningitis is serious. A leak of cerebrospinal fluid through the nose or one ear, in what would otherwise seem to be a simple fracture of the skull, also brings a moderate threat of meningitis and such cases should be treated routinely by mouth administration of some form of sulphonamide, as mentioned below under Routine Care.

Compound fractures of the skull constitute surgical emergencies. At operation the edges of damaged scalp should be excised and the wound opened widely.* Careful inspection

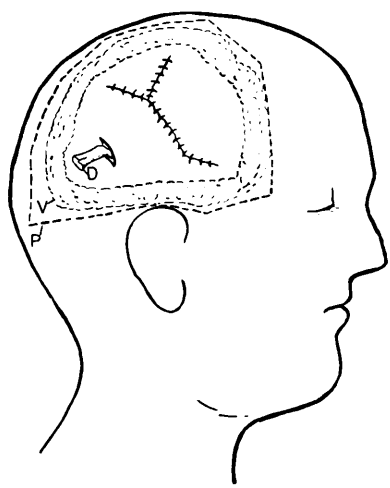


FIGURE 6

Tripod incision. A ragged laceration can sometimes best be closed in this pattern. Drain (D) emerges from incision. V = vaseline on sterile scalp. P = protective tissue over the moist gauze dressing which covers incision drain. The protective extends out to the ring of vaseline so that if cerebrospinal fluid emerges from drainage tract it will pass outward beneath the protective and into an overlying dressing with little danger of retrograde contamination from without.

* Cushing pointed out the usefulness of converting a ragged or "gutter" scalp laceration into a "tripod" or "three-legged" incision. (Fig. 6.) This often makes it easier to draw skin edges together.

will demonstrate whether a fracture is present and whether any fragments are depressed.

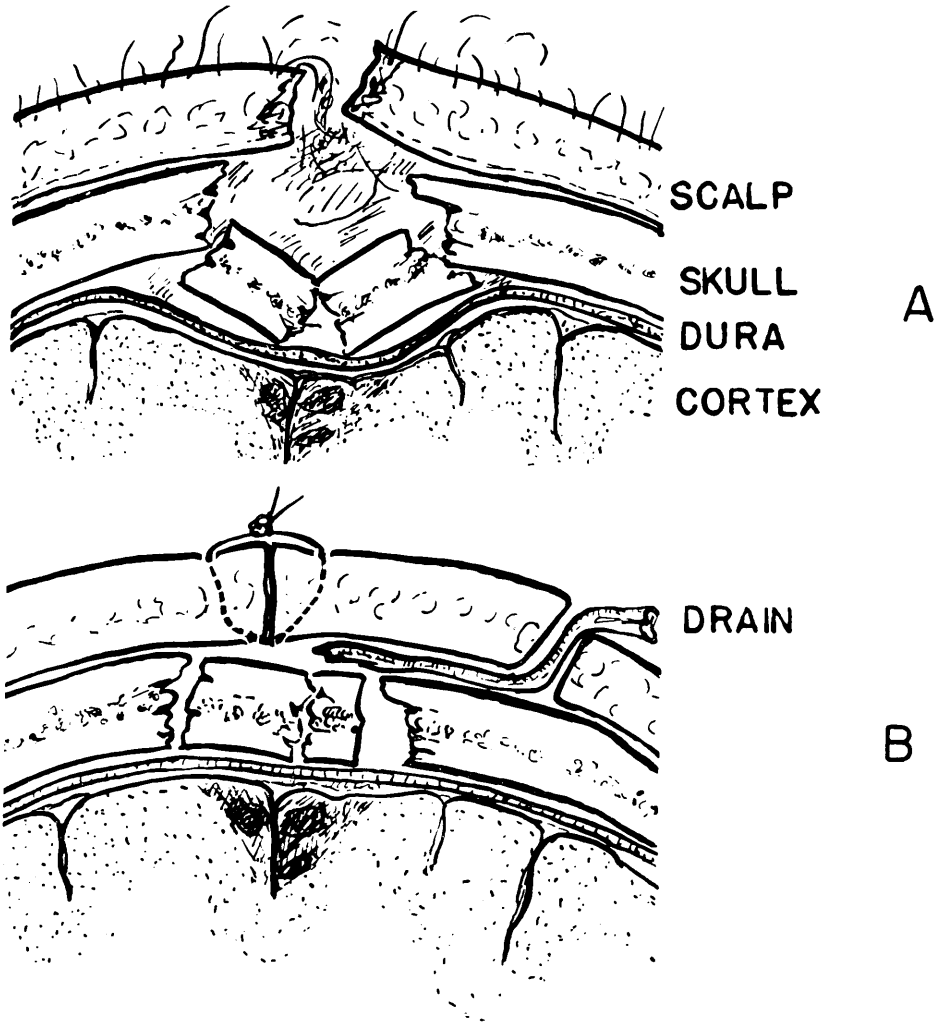


FIGURE 7

Above, depressed fracture of skull which has not torn the dura but has caused moderate contusion of the cerebral cortex. Below, bone fragments elevated; drain inserted through "stab wound"; scalp closed with one layer of silk. Bone fragments should be removed (not replaced as in the drawing) except under exceptionally favourable conditions, or when the scalp is not lacerated.

Depressed fracture. Bone fragments may be depressed and wedged in this position (Fig. 7A) without tearing the dura. Such fragments should be elevated because of the danger of eventual epilepsy. If the dura has not been opened by the injury it is usually better not to make an incision in it unless there is reason to suspect a subdural clot. If initial

treatment of the wound was unsatisfactory and if more than 6 to 12 hours have elapsed since the wounding, a scalp drain should be inserted through an incision (stab) made in the scalp at a distance from the suture line, as shown in Figure 7B. In any case the scalp must be cleaned, the edges trimmed off and sutured as early as possible, even if the elevation of the bone fragments is postponed for a time. One layer of sutures is adequate as in Figure 7B for such purpose, preferably silk or fine steel. In case the dura has been penetrated, see next subsection.

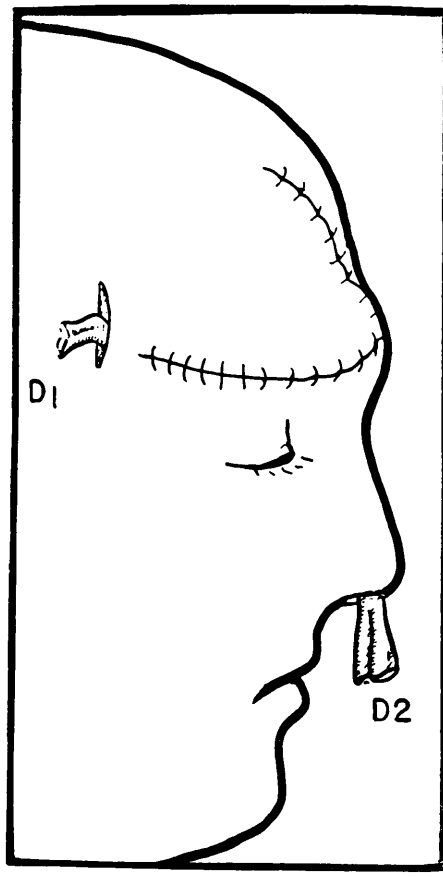
In the case of *fracture of the skull compounded with the paranasal sinuses* and with lacerations of the overlying skin, it has been pointed out by Cone that the safest and most effective procedure, after roentgenological examination, is to operate upon these *craniofacial wounds* as follows:

Carry out the usual débridement. Remove any brain tissue that may be macerated. Then close the dura carefully and completely between sinuses and brain.* Drain the subdural space by a strip of rubber tissue placed beneath the dura posteriorly and brought out through a stab wound made in the clean area of scalp (D 1 in Figure 8).

Then close the scalp laceration at least partially and turn to the sinuses. Remove whatever bits of bone there may be in the sinuses. Clean out and remove damaged mucous membrane. A rubber tissue drain (D 2 in Figure 8) should then be placed in the sinuses, bringing it downward through the nasal cavity and out the nares. The skin of the forehead may then be closed completely leaving one end of the drain in the frontal sinus to be withdrawn from the nose some days later (Fig. 9), for it may seem better to leave this drain (D 2) emerging from the incision in the forehead as well as from the nose. In case a drain is left emerging from the skin over the frontal and ethmoid sinuses and a subdural drain (D1) emerging from scalp posteriorly, the latter must be protected by the use of a moist dressing on ringed vaseline and covered with protective tissue as in Figure 6.

This type of operation, if carried out with energy and

* If dural closure is not possible the dural defect may be filled by a piece of clean periosteum removed from the skull at a distance or by fascia removed from the temporal muscle or from the patient's leg.



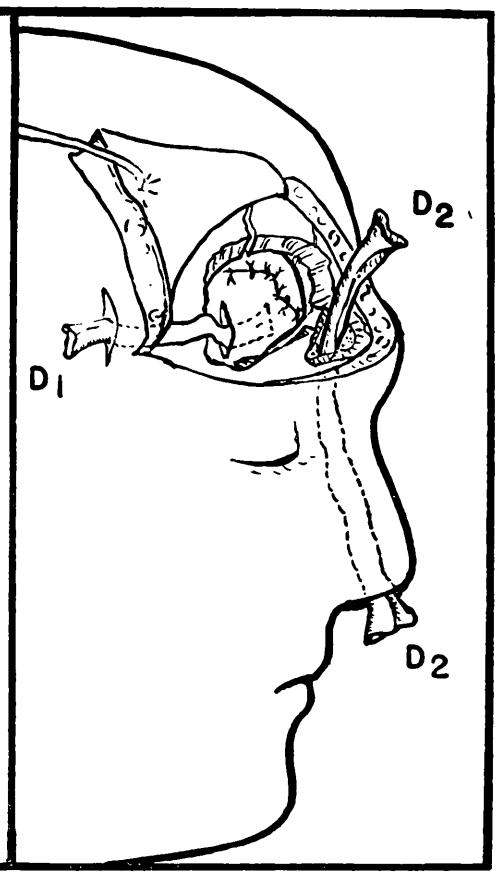


FIGURE 9 FIGURE 8

Fig. 8. Compound fracture of skull and of paranasal sinuses treated by method of Cone. Laceration in dura closed; drain (D1) inserted through incision in dura to allow cerebrospinal fluid to escape posteriorly; frontal sinuses cleaned out and drained (D2) downward into nose.

Fig. 9. Same as in preceding figure. Drainage tract (D1) to be protected against contamination by moist dressing ringed with vaseline covered with protective tissue as in Figure 6.

within the first six to eight hours, may avoid weeks and months of painful and dangerous dressings and, furthermore, avert the imminent danger of fatal meningitis. If it has been necessary to transport a patient for some distance or if other conditions have made it impossible to operate early, it is likely that this radical procedure can be carried out after a longer period, particularly if the patient has received sul-

[illegible] ges following his injury.

co-operation of a surgeon familiar with otolaryngology is of great assistance. Cosmetic repair of the forehead to fill in the excavated sinuses may be

carried out months later. In some cases, however, it is possible to replace at once certain of the fragmented bits of bone and to close the skin over a frontal sinus which has been drained downward.

Although the above treatment must be considered ideal, an alternative method of treatment of *craniofacial wounds* may be employed if the surgeon lacks experience or time. In that case the wound must be packed without closure, using gutta percha or rubber tissue next the dura or brain and sealing those parts from the sinuses as well as possible.

5. TREATMENT OF PENETRATING WOUNDS OF THE BRAIN

In the case of *depressed fracture* of the skull there should be a rule that operation is to be carried out at the earliest possible moment provided there is also laceration of the scalp. If, on the other hand, there is a depressed fracture of the skull without scalp laceration, immediate operation may be preferable, but it is permissible to elevate the bone fragments and to remove damaged brain after a delay of two or even three days.*

In general, if the dura has been penetrated during the injury, it should be assumed that there is *laceration of the brain.* The presence of softened brain, with opened dura, particularly if there are intracerebral foreign bodies such as bone fragments, means that the patient has a very good chance of developing posttraumatic epilepsy, after his immediate recovery, unless something is done within the first day or two to alter this prognostication. Prevention calls for fastidious cerebral débridement as described below.

Small metallic fragments, particularly those resulting from bomb explosions, may penetrate the skull and lodge within the brain producing only a small wound of entrance and little initial cerebral disturbance. In such cases healing often occurs without infection and surgical removal or interference may well be contra-indicated. Whether or not the incidence

* It should be remembered also that laceration of the cerebellum does not carry with it a threat of subsequent posttraumatic epilepsy, although the development of increased intracranial pressure or the threat of meningitis may force the surgeon to carry out exploration here and to remove the depressed fragments.

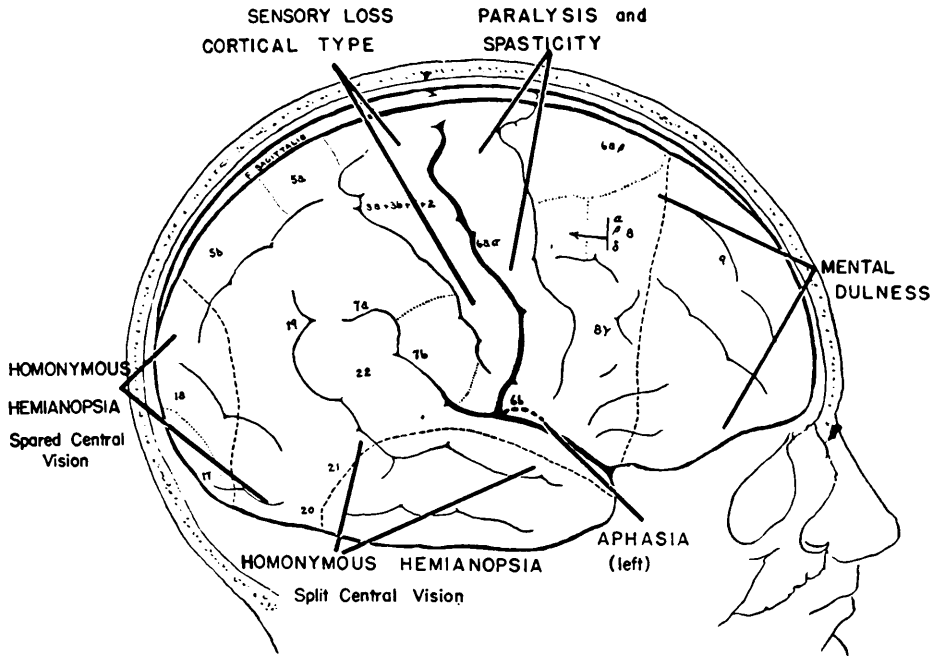


FIGURE 10

Disturbance of function resulting from lesions in different areas of the brain indicated graphically.

of posttraumatic epilepsy will be high in these cases cannot yet be foretold.

Cerebral localization must be made by neurological study. The effects of gross lesions of the cerebral cortex are indicated in Figure 10. The motor sequence in the precentral gyrus beginning above in the median longitudinal fissure and passing down to the fissure of Sylvius is as follows: toes, ankle, knee, hip, trunk, arm, hand, fingers, thumb, eyelid, vocalization, lips, jaw, tongue, swallow. Only the extremities suffer any important weakness as the result of hemispheral lesions. Lesions involving the precentral gyrus or internal capsule produce spastic paralysis.

Sensory representation in the postcentral gyrus corresponds with the motor representation. Isolated lesions here result in loss of sense of point discrimination and awkward-

of the vertex of the skull are

l sinus and often result in

marked spasticity of both lower extremities, as was pointed out by Holmes and Sargent, 1915. This follows from the fact

that the foot area is represented on each hemisphere at the median fissure.

The relationship of areas of the brain to strictly lateral projections of the skull as shown in a lateral radiogram is to be seen in Figure 11.

Cerebral débridement. Enlarge the incision in the scalp which has already resulted from laceration; excise the edges of any damaged tissue there. Especially if no scalp laceration has occurred, it may seem wiser to turn down a small osteoplastic bone flap including the skull defect. Remove the bone fragments and, in case contamination does not seem to be too great or of too long standing, they may occasionally be set aside and replaced at the close of the operation.

Enlarge the opening in the dura somewhat and also re-

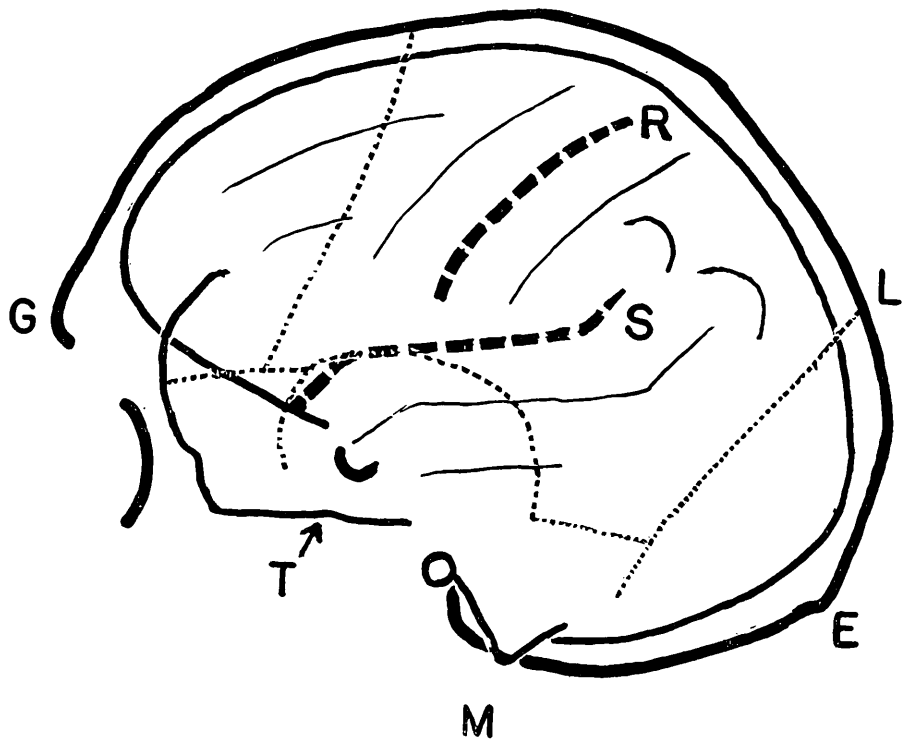


FIGURE 11

Projection of cranial and cerebral landmarks upon a lateral radiogram of the skull, the film being parallel to the sagittal plane of the head and the X-ray tube being placed at 50 centimeters' distance. G = glabellum, T = sella turcica, M = mastoid, E = external occipital protuberance, L = lambda. Broken lines indicate fissure of Rolando, (R) and fissure of Sylvius (S). Dotted lines represent sutures. Redrawn after Marie, Foix and Bertrand.

move enough additional bone so as to see clearly the amount of injury to the brain. An aspirator or a sucker should then be used without too great suction pressure. With this the softened brain can be easily removed and viable brain left behind, as for example, along the dotted line in Figure 12. The surgeon should make an effort to see that the edges of the remaining cortex have an adequate vascularization from the pial vessels. Any cerebral tissue which cannot live, or which is only half vascularized may become a future menace and is better removed. This applies particularly to the gray matter.

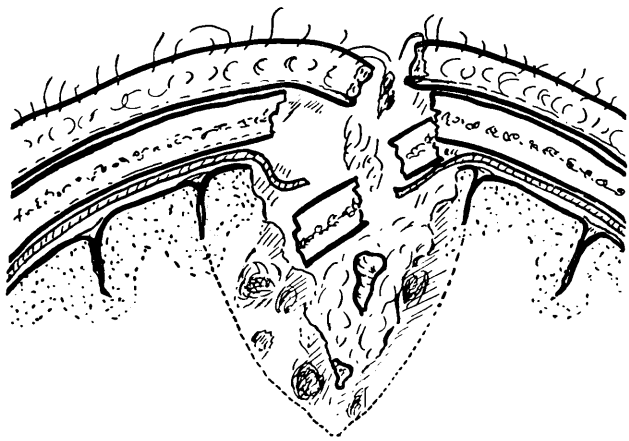


FIGURE 12

Compound fracture of skull and penetrating wound of brain.

Softened cerebral tissue can at times be washed out with a stream of Ringer's solution or may be aspirated with syringe and catheter; but a small metal sucker is a very much better instrument and it gathers up the blood vessels of the removed tissue so that they may be clipped and cut off or cauterized. The dura should then be closed leaving a fluid-filled pocket in place of the softened brain. Compare Figure 13 with Figure 12. If it is necessary to excise dura so that its closure over the defect is impossible, one may shift dura from an untraumatized area leaving the latter without brain covering, or its neighbouring intact dura may be split into two layers and reflected so as to cover the lacerated area of brain.

Foreign bodies. In the case of gunshot wounds of the brain

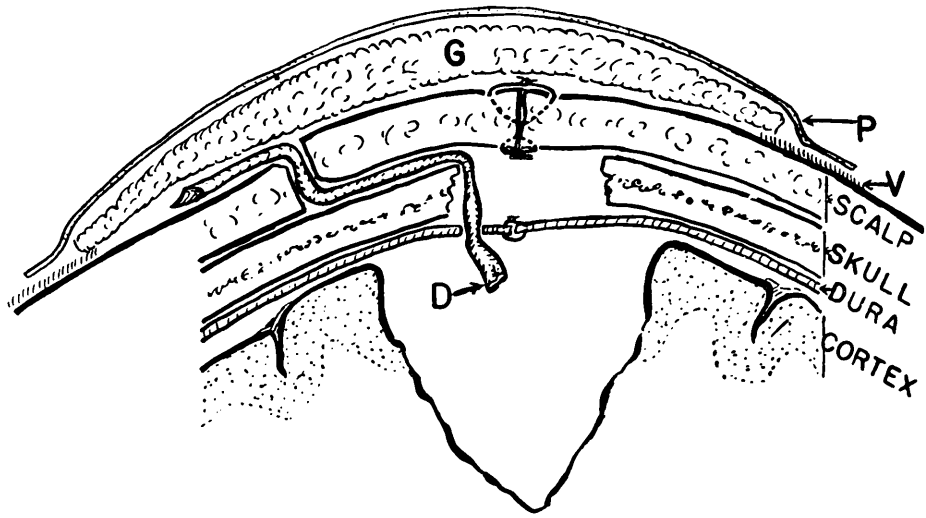


FIGURE 13

Revision and drainage of wound in the case illustrated in preceding figure. Cerebral débridement and removal of cerebral tissue by suction up to fissure lines; closure of dura; drainage (D) of cerebrospinal fluid through incision in dura; closure of scalp in two layers; application of moist dressing made up of gauze (G), ringed vaseline (V), and protective tissue (P).

where the missile has entered and may have lodged within the cerebral tissue, the use of the probe should be prohibited as it may carry micro-organisms into a sterile track. Electromagnets, if used, should be combined with visualization. Instead of this, after proper preparation and roentgenography the opening of the missile track should be exposed and a long, narrow, lighted retractor, such as that of Frazier, inserted. The surgeon should then visualize the track of the missile to its end, sucking out the softened and non-viable tissue, and clipping the bleeding vessels or cauterizing them.*

This offers the patient the best chance of survival and reduces considerably the likelihood of meningitis and post-traumatic epilepsy. During the process of sucking out the softened brain, complete removal of dirt, hair and clothing may be easily accomplished.† In some cases only gentle irrigation, aspiration with a catheter and use of long forceps to remove the foreign body may be deemed wise to avoid

* In the motor region of the cortex the surgeon should be very conservative, removing only tissue damaged beyond hope.

† Occasionally it may be advisable to leave a metallic fragment rather than to persist in its pursuit at the cost of damage to brain or danger to life. This is particularly true if the fragments are small.

haemorrhage. If such a wound clearly has destroyed the speech area in a dominant hemisphere or the internal capsule and basal ganglia, it may sometimes seem to be a kinder decision to withhold operation. Whether or not to drain such wounds is discussed on page 26.

In regard to the important subject of penetrating wounds of the brain the following may be quoted from a personal communication from Professor Geoffrey Jefferson (June 25, 1941):

"Small fragments such as commonly are thrown off by the modern aerial bomb with its terrific bursting charge, and its casing, can usually be left alone, i.e., quite different from H. E. shells. The indications for removing a metallic foreign body are, not that it is there, but how deep is it and what harm is it doing. It has been astonishing to see how often small splinters have traversed the brain and not even made the patient unconscious. It is the same as though a sudden thrust had been made with an oversize ventricular needle, i.e., usually not much harm comes of it. These fragments do not seem to carry in bacteria; they are sterile because of the velocity of their travel and pick up little from the small surface area of skin that they encounter.

"The wounds most liable to infection are gutter wounds or those with complicated scalp wounds. It is my opinion that the surface area open to infection is a dominant factor. The infection does not, in my view, come from the missile but (a) from the patient's own skin, (b) from his exploring finger, (c) from those who dress his wounds, (d) more rarely from the surgeon."

Finally the author is quite in agreement with Jefferson when he emphasizes "the essential importance of a first-rate surface operation down to the dura, but not doing much or anything inside it *unless the surgeon has made a thorough study of technique long beforehand and preferably had some training at it.*"

It may be of interest here to quote from Horrax's (1940) description of the method used by Cushing to treat "gutter wounds" and "penetrating wounds" proper. In case mechanical suction and cautery are available Horrax would also agree that more radical treatment as outlined above is probably preferable.

"The treatment of these wounds, as developed especially by Dr. Cushing, was as follows. The dirty scalp wound, large or small, was excised down to the bone, after which radiating incisions were made

from the wound edges so that scalp flaps could be retracted, giving good exposure of the skull fracture and an area around it. Burr openings were then made around the depressed or penetrated bone and this whole area excised en bloc by connecting burr holes with cutting forceps. The dural part of the wound was not excised, since it was felt inadvisable to reopen the subarachnoid space, which as a rule had already become sealed off around the torn edge of the dura. The patients were operated upon under novocain anaesthesia, and at this point they were asked to cough or strain. This caused considerable material—disorganized brain, clots, and occasionally small bone fragments—to be extruded from along the track out through the dural opening.

"The next step was to introduce into the track a soft rubber catheter attached to a syringe having a rubber bulb at its end. By means of compressing the bulb, soft, disorganized brain, clots and other débris were sucked out from the track a little at a time, the catheter being introduced farther and farther as progress was made. Fragments of bone could be palpated and located by the catheter along the track and these fragments removed by a delicate forceps. If a metallic shell fragment had been lodged in the brain this was usually found at the bottom of the track and was removed either by a forceps or by the electromagnet. The latter was brought up to the end of a long, blunted nail which was introduced into the brain track down to the metallic fragment.

"By means thus described a careful, but necessarily slow and tedious, débridement of the wound in the brain was accomplished, an average of two hours being required for the serious, deeply penetrating wounds in which there were numerous bone fragments, other débris, and perhaps one or more metallic foreign bodies. If the operation could be done within ten to twelve hours from the time the wound was received, primary closure without drainage was successful in the vast majority of cases. When operations had to be done at a base hospital, usually eighteen to sixty hours after wounding, infection was so far advanced that primary closure was disastrous. Under these circumstances the wounds were left widely open directly over the area of dural penetration. A cerebral fungus of greater or lesser size would then protrude, and in most instances, after careful dressings with gutta percha placed next to the raw surface, granulation would take place and the fungus recede as epithelialization proceeded."

Sulphonamides on lacerated brain. Chemicals of the sulphonamide group (sulphanilamide and sulphathiazole) may be applied locally in cases where there has been gross contamination and laceration of the brain, provided the sterility of the compound can be guaranteed. They are absorbed locally, producing a very high local concentration of drug. No evidence of increased reactions or more severe scarring

in brain wounds was found by Hurteau (1941) in an experimental study in which these drugs were left in the area of brain laceration. On the other hand, the important issue is the correct radical treatment by surgical methods and no dependence should be placed upon chemotherapy if this dependence replaces proper surgical therapy.

6. TREATMENT OF MENINGITIS AND INFECTIONS OF SCALP, SKULL AND BRAIN

In the treatment of meningitis it is essential first to discover, and to treat properly, the primary inflammatory process, whether this be an infected wound, an area of osteomyelitis, sinusitis, mastoiditis, or brain abscess. On the other hand, in case the avenue of contamination is a spinal fluid fistula through ethmoid plate or ruptured drum-membrane without an extradural zone of infection, radical local treatment is usually inadvisable.

The advances in chemotherapy have greatly improved the prognosis of leptomeningeal infection by pyogenic organisms. Once the diagnosis* is definitely established by finding organisms in the cerebrospinal fluid, by smear or by positive culture of the fluid, a satisfactory blood level concentration of the drug used must be obtained quickly and maintained until leucocytes disappear from the spinal fluid and the fluid pressure drops to normal. Suitable controls must be employed to avoid serious toxic effect from too long a period of administration.

In the later stages of treatment, continued elevation of intracranial pressure without hyperpyrexia suggests the possibility of brain abscess. Continued elevation of temperature with normal cerebrospinal fluid pressure suggests some cause for the temperature other than meningitis.

It will be remembered that both *sulphapyridine* and *sulphanilamide* pass through the blood-brain barrier and appear in the cerebrospinal fluid so that a meningitis produced by an organism which is susceptible to these drugs may be attacked in a satisfactory manner by oral administration. However, sulphathiazole, ordinarily so effective against

* See p. 20 for differentiation of aseptic meningitis from true meningitis.

staphylococcus, does not pass the normal meninges. Consequently, if the infecting organism is staphylococcus aureas, this drug is of use chiefly to control an extradural process such as an infected wound or mastoid. In some cases sulphapyridine has seemed to be of help in this type of meningitis, although its action against staphylococcus aureas may be in general weaker than that of sulphathiazole.*

Intrathecal serum. In staphylococcus meningitis, antiserum may be effectively injected by a needle inserted into the lumber theca, the cisterna magna, or one of the ventricles of the brain. 20,000 units of staphylococcus antitoxic serum (Connaught Laboratories) followed by 10,000 units daily for four to five days, all intrathecally, has been used very effectively in the cure of such meningitis. Before beginning serum therapy, the sensitivity should be tested (1/10 cc. of serum diluted 1 to 10 in saline injected intradermally and observed for 2 hours). If sensitive, the patient should be desensitized by a course of subcutaneous injections at one hour intervals of .05, .1, .2, .4, .7 and 1 cc. of the serum. Antiserum may also be used intramuscularly and, with care, intravenously, but it must be borne in mind that serum thus administered, like sulphathiazole, does not pass through the barrier into the cerebrospinal fluid.

In general, for any case of meningitis, fluids should be forced and *lumbar puncture* carried out each day or two in order to follow the leucocyte count and the pressure of the fluid. The cerebrospinal fluid pressure is practically always high (above 200 mm. water, usually about 300-350 mm.) when there is active infection within the leptomeninges. A low pressure suggests either that the meningitis is aseptic or that fluid is escaping elsewhere, as through a skull fracture or a draining wound. If the pressure is high, a large amount of fluid should be withdrawn when puncture is done until the pressure falls to about 100 mm. of water. This usually gives relief of headache and may have therapeutic value. However, continuous or forced drainage of fluid by needle or operative opening is no longer to be advised in the present era of chemotherapy.

* Sulphadiazine does pass from blood into spinal fluid, but at the time of writing this drug has not been given practical trial in such cases.

If the primary cause of meningitis be an infected wound that communicates with the brain and cerebrospinal spaces, it is essential, of course, to drain the wound and usually to allow cerebrospinal fluid to escape locally also. This escape of cerebrospinal fluid should then be promoted until it is gradually shut off by the growth of healthy granulation tissue.

Craniocerebral wounds, when infected, are to be treated according to principles that apply to other wounds. *Antiseptics* must, however, be carefully kept away from the brain.* Even Dakin's solution must be prohibited if there is any possibility of its penetration into the cerebrospinal spaces. The brain surface should be covered with rubber tissue, gutta percha or some form of protective. Gauze should not be laid on the brain unless it contains a completely non-irritating oily liquid.

Although some sulphonamides may be used in contaminated wounds of the brain they are probably less effective locally in the presence of pus (Thrower, 1940). We have, however, used sulphanilamide powder, also sulphathiazole, with apparently good results, just at the point where the draining cerebrospinal fluid makes its appearance on the wound surface. This was done in the belief that the drug is dissolved and thus renders the fluid bacteriostatic where it ebbs and flows near its point of exit from the arachnoid spaces.

The *dressing of a craniocerebral wound* through which spinal fluid is draining, or may drain, calls for special care. The scalp must be reshaved periodically and should be painted with iodine (2½%) from the wound edges outward at each dressing. Rubber dam drains may be used and the brain protected from gauze packs by rubber dam or vaseline gauze. A small moist dressing should be applied, much as for simple drainage of cerebrospinal fluid (p 30 and Fig. 6). At the periphery of the iodinized scalp a ring of sterile vaseline is spread as shown in Fig. 14. Gauze moistened with saline solution or boracic acid solution is placed over the wound and within the vaseline ring (dotted line in Fig. 14). Imper-

* Proflavine (0.1% in isotonic saline buffered to pH 6.2) is recommended as least harmful to the brain, Russell & Falconer (1940).

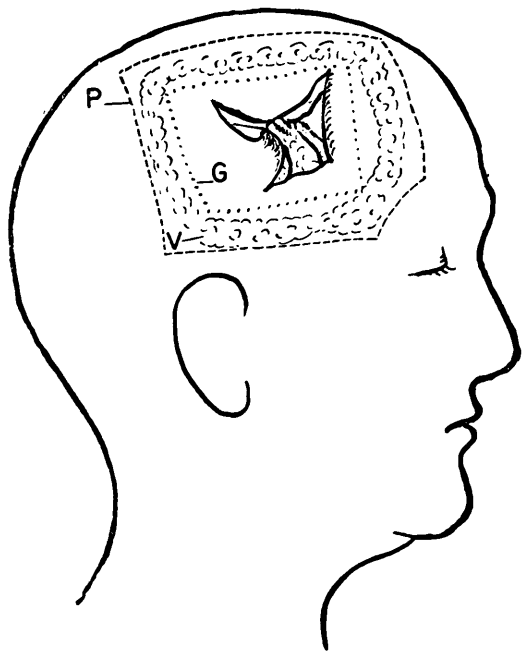


FIGURE 14

Infected craniocerebral wound covered with moist dressing. G = outline of gauze, V = vaseline ring, P = overlying protective tissue.

meable protective tissue (broken line) is laid over that and upon the vaseline ring after which a thickness or two of gauze and a Dakin's pad, or cotton wool in some form, to absorb the escaping spinal fluid. The encircling head bandage should include the ears to avoid displacement and a pencil mark added on the finished bandage so that the nursing sister can report rotation or displacement. The dressing should be changed at one to three day intervals. With such care, invasion by other organisms can be prevented for weeks during which the wound heals and the cerebrospinal fistula closes. In general, this technique applies to the dressing of a case of *brain abscess* and of *hernia cerebri*.

Hernia cerebri, or a bulging surface of exposed brain in an infected wound, calls for patience and care in treatment. The so-called *brain fungus* bulges because of increased intracranial pressure. Daily lumbar punctures will help to reduce the bulging. The dressings must be continued until the surface of the brain granulates and eventually epithelializes. After granulation, pinchgrafts of skin may be applied. The

defect may be closed by bone graft and plastic operation upon the scalp after six months to a year. If the bulging of a hernia is great, brain abscess must be suspected and an encephalogram may be necessary to settle the question of its presence. If an exposed area of dura granulates but is slow to epithelialize, the area may then be Dakinized with impunity and a secondary suture of the scalp carried out.

Brain abscess. This manual does not pretend to take up in complete detail the treatment of brain abscess. Such conditions are best referred to hospitals where specially experienced surgeons are available. However, in the case of any patient who has had a brain wound and who continues to show elevation of intracerebral pressure, brain abscess must be suspected.

If the surface of the brain is exposed in an infected wound it will bulge progressively and form a *fungus cerebri* which is most embarrassing. There are other causes for such bulging, however, among which the most frequent is internal hydrocephalus of varying degree due to subarachnoid obstruction. The obstruction is apt to be produced by inflammation or by blood left behind in the cerebrospinal passages. Consequently, a cerebral hernia should never be explored with a hollow needle until after an *encephalogram* has been carried out. If this procedure indicates a local expanding lesion, puncture and drainage should be performed forthwith.

In the case of a brain wound which has been closed or allowed to close, the appearance of increased intracranial pressure during the first few weeks after injury must likewise raise a suspicion of brain abscess. For, however energetic the administration of sulphonamides may have been, it is not a guarantee against the formation of abscess, particularly if there is a retained foreign body.

Symptomatology of brain abscess. Brain abscess does not produce an increase of temperature after the stage of local encephalitis has been followed by the walling off process. After encapsulation there is no leucocytosis in the blood. There may be some small increase in cells in the cerebrospinal fluid and there may, or may not, be an increase of protein in the fluid. It has recently been pointed out, however, by

O'Connell that hernia cerebri is frequently associated with marked rise in the cerebrospinal fluid protein in patients who have no abscess. The evidence of brain abscess is the evidence of a locally expanding intracranial lesion: headache, vomiting, papilloedema, strabismus due to abducens palsy, increased spinal fluid pressure, advancing signs of a local cerebral lesion.

Treatment of brain abscess. An abscess can occasionally be removed when extremely well walled off and chronic. An abscess may occasionally be subjected to repeated aspiration and this procedure is said to be followed sometimes by healing even when the abscess is not sterile. However, an abscess must usually be drained and the drainage maintained for a long period, following the example of MacEwen.

In approaching an abscess an opening in the skull should be made as nearly over the mass as possible. The subdural space should be walled off and if possible allowed to adhere for a day or two. An exploratory ventricular needle is then inserted through brain and capsule. When pus emerges the needle is maintained in place. A sucker is then used to aspirate a cylinder of brain about the needle and down to the capsule surface. The needle may now be withdrawn and a soft rubber tube introduced into the abscess cavity. The outer end of the tube is then split into strips which are sutured to the scalp to hold the tube in place. The largest tube possible should be used so that the abscess may turn itself inside out into it. Withdrawal of the tube must be gradual.

The treatment is simplified if the abscess is sufficiently old to have a well formed capsule. *Acute brain abscess* or purulent encephalitis occasionally threatens immediate death. Under such circumstances it has been shown by Cone that successful operation is possible. It is necessary to aspirate the infected area of brain in which multiple small abscesses may be encountered. The area is then packed with a rubber covering or a large tube is inserted and moist dressings applied by the technique described above (p 29).

Osteomyelitis of the skull can only be cured by radical treatment. If, in a case of chronic infection, bare skull is exposed at the bottom of an *infected wound of the scalp*, it is

best to use a perforator and burr to remove the bone down to the dura. The bone should then be removed with a rongeur until bone which bleeds is encountered at the periphery in all directions. If this is done, a pack of vaseline gauze* may then be laid upon the dura and the scalp replaced over the pack. The scalp may even be sutured in place temporarily. After a period of 5-10 days the pack should be withdrawn. The dura will often be found to have granulated completely, and the bone edges as well. If that is the case the scalp may be resutured and a small drain placed beneath it, to be withdrawn in a few days.

If the wound does not close it will be found that some of the skull or some of the dura has not become covered with granulation tissue. In case the bone has remained bare, then more of it must be removed. In case the dura remains bare and dead-white, the probability is that a *subdural abscess* exists. In this latter case the surgeon must eventually incise the dura but he will do so only after anxious waiting, for the *dura* is ordinarily the most important and effective barrier against the spread of infection to the leptomeninges.

7. SIMPLE HEAD INJURIES AND ROUTINE CARE OF ALL CASES

Simple head injuries, without skull penetration, and *simple fractures of the skull* are particularly numerous in the present war because of the high toll of traffic accidents during "blackout" and because of the blast of bomb explosions. Thus, among both soldiers and civilians simple skull fracture and head injury with concussion (p 10) and with different types of intracranial haemorrhage (p 17, p 18, p 18, p 19, p 20) provide clinical problems of increasing importance.

The first medical officer to see a patient who has had a severe head injury should make every effort to record the essential elements of the patient's *history.* It may be impossible to secure such a description later, and the decision in regard to therapy may depend almost completely upon this initial record. Some such forms as that outlined in Table I,

* B.I.P.P. may be used or gauze soaked in an oily preparation such as the cod liver and honey preparation called "Alphamel".

p 90, may be used to save time. Always record the nature of the accident, the existence and duration of initial loss of consciousness, and also what he was able to do immediately after regaining consciousness. If there is a history of recovery of consciousness and a secondary lapse to unconsciousness, this free interval should be looked upon as of the utmost importance—probably indicating intracranial bleeding, as discussed on p 17.

Even a hasty *physical examination* should note: (a) mental state; (b) site of wound or contusion; (c) pulse quantity and rate; (d) respiration rate; (e) bleeding from nose, ears, scalp; (f) the comparative size and the reactivity of the pupils; (g) the degree of spontaneous movement of extremities on either side. If the patient is in coma, painful stimulation should be used to arm and leg on either side in order to gain some idea as to their comparative reaction.

Roentgenographic examination should be carried out as early as possible, particularly if the injury is severe. If, however, there is dangerous shock with low blood pressure, the roentgenogram may be postponed a little while until the condition improves. Lateral stereoscopic films and antero-posterior stereoscopic films should be taken routinely unless there are special indications to the contrary.

Spinal puncture. This is advised as a routine procedure in the early stages after head injury with the understanding that consideration be given to the dangers, the indications and the benefits of the procedure as described below. If properly done, the procedure is very rarely painful, but, if the patient is in such an agitated state that it cannot be carried out quietly, it should usually be postponed, as a serious struggle may result in more harm than good. For the sake of medical officers who may be out of practice the following description is set down. See also p 77.

*Lumbar puncture technique.** Place the patient on his side at the edge of the bed, with his knees drawn up on his chest. This should flex the lower spine. If a pillow is placed between

* Lumbar puncture tray: iodine, sponges, an artery forceps to carry sponge for sterilizing skin, novocaine, ½%, hypodermic syringe and needle, lumbar puncture needle 18 and 20 gauge, three-way stopcock, spinal fluid manometer, three test tubes for collecting fluid.

the knees the posterior surface of the sacrum may be more easily placed in a vertical plane. Sterilize the skin. Choose the spinal interspace (usually the third lumbar) which is on a line from one iliac crest to the other. Inject novocaine with a hypodermic needle into the skin at that point and down to the interspinous ligament. Next replace the hypodermic needle by a lumbar puncture needle with trocar in place exactly in the midline and slanting a little cephalad. Withdraw trocar and inject novocaine. When the needle has been introduced about ¾ of its length increased resistance is felt. Withdraw about ¼ inch and inject novocaine and wait long enough to prepare stopcock and manometer. During that time the dura and periosteum become anaesthetized. Then push the needle into the dural canal. If spinal fluid does not come, explore again but do not cause unnecessary suffering by going beyond the field of analgesia. The procedure is best carried out with

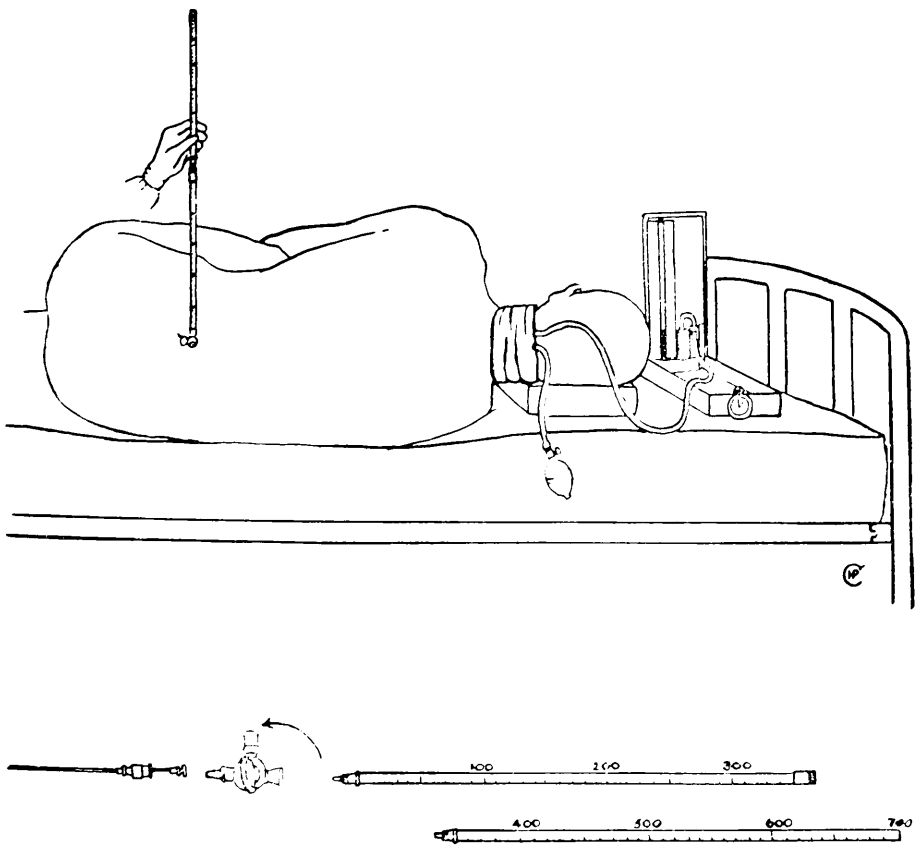


FIGURE 15

Lumbar puncture and measurement of spinal pressure with glass manometer of Ayer. Blood pressure cuff on neck for jugular compression.

gloves, but if gloves are not available the operator should mark the site for puncture with a scratch or a distinct iodine mark upon the skin before sterilizing the field. This will avoid the (time honoured ?) practice of palpating with contaminated finger the area through which the needle is to be thrust. When the first drop of fluid indicates that the canal has been entered insert the closed stopcock (Fig. 15) at once and attach the manometer so as to measure spinal fluid pressure.

The measurement of the *spinal fluid pressure* should be made with the patient horizontal and relaxed. A glass manometer, such as that described by Ayer (see Fig. 15), is best for its shows the colour of the spinal fluid at once and registers the oscillations of the fluid column. Jugular compression is of little use and may be dangerous. It should therefore be forbidden in head injury as a routine procedure, i.e., no *manometric test of Queckenstedt.* Pressure which is over 200 mm. of water may be considered as elevated. A pressure under 100 mm. is low and, if spinal fluid has not been lost in the manoeuvre of puncturing, it is suggestive that fluid is probably escaping through a fracture to be absorbed in the tissues, or that it is escaping through the external ear or nose.

Test for subarachnoid bleeding. One or two cubic centimeters of fluid should be taken in each of three test tubes. The red blood cells in each specimen may then be counted microscopically. If there has been bleeding, this gives some idea of the amount and makes it possible to rule out bleeding from the manoeuvre of puncturing. If the blood is the result of injury by the needle itself, the first tube will contain more blood and the last test tube less. If the blood is coming from the cranial cavity, the reverse will be true or the amount will be the same in each tube.

Dangers of spinal puncture. The question of whether a lumbar puncture should be carried out in cases in which there seems to be imminent danger of the development of meningitis, may be open to debate on academic grounds, but from a practical point of view we have never seen evidence that this increases the danger of invasion of the meninges by an extracranial infectious agent.

Lumbar puncture during the first few hours after head injury does not carry with it any added risks unless there is evidence of very greatly increased pressure and particularly if there are signs of bulbar compression such as Cheyne-Stokes breathing.* Under such circumstances, if lumbar puncture is done, a needle of small bore should be used and only a small amount of fluid removed. This is to avoid the possibility of promoting herniation of the cerebellum down into the foramen magnum, a danger which must be considered with very high pressure. It is a good routine practice to reduce the spinal fluid pressure by no more than half of its initial value if it is found to be high.

Indication for lumbar puncture. However, from a practical point of view lumbar puncture may, and should, almost always be carried out early in other cases of severe and mild head injury as part of the initial examination for its diagnostic value and as a prevention of later complications. Whether or not it should be repeated frequently during the period of convalescence, depends upon the individual case. If there is a good deal of brain oedema, with headache which makes the patient restless, repeated lumbar punctures may be most helpful.

It should clearly be recognized, however, that repeated punctures serve only to reduce pressure and to increase comfort, for Sprong (1934) has demonstrated that: "The spinal fluid obtained always represents a high dilution of blood. Therefore the amount of blood eliminated by lumbar drainage is always amazingly small." He calculated the amount of whole blood removed thus and found that in no case were more than ten drops of whole blood removed by an entire series of lumbar punctures in spite of the fact that some patients in his series were punctured two and three times daily.

Effect of spinal puncture. Nevertheless, although it is impossible actually to remove appreciable amounts of blood in that way, spinal puncture or cisternal puncture does serve to reduce the pressure within the ventricles temporarily, to

* Rapidly increasing hemiplegia or a dilating pupil are indications for operation and contraindications for lumbar puncture. Decerebrate rigidity, see page 15, may also be a contra-indication to lumbar puncture.

measure the intracranial pressure periodically, and to increase the comfort of the patient. Early lumbar puncture after head injury is also the best preventive of late *posttraumatic headache,* see p 68.

Ward routine. After the initial careful study of the patient, he should be placed flat in bed, or with only one pillow, and with the head of the bed elevated 5 to 10 inches above the foot. This is done in order to reduce the pressure of blood in the veins of the neck and thorax and thus the venous pressure within the cranial cavity. This lessens the danger of bleeding from intracranial veins. For the same reason, straining during enemas, at stool or from vomiting, may increase the patient's danger. In cases in which there is obvious continued venous bleeding into the subarachnoid space, as shown by repeated lumbar punctures over a period of a week or two, the bleeding may often be stopped by placing the patient in a sitting (Fowler's) position and by keeping him as quiet as possible.

A *comatose patient* requires special care. Unless he is receiving constant attention he should be placed in a semi-prone position so that the mucous which fills his nasopharynx (see p 16) may drain out and not be aspirated. When placed upon his back the mucous should be aspirated by suction. Such patients must be turned periodically or moved at regular intervals, for instance every hour when the pulse is taken. *Hot water bottles* should be prohibited. Occasionally, such a patient may be fed by small stomach tube introduced through the nose, but care must be taken to inject air first and the escaping air auscultated over the stomach.

A *case record,* which should be kept by the *nursing sister* in attendance, should include, from the beginning, a charted record of the pulse taken every half hour, the blood pressure* every hour to begin with and then every four hours, and the respiration rate and temperature every four hours. She should note carefully changes in mental alertness, in comfort, in strength of hands and feet, and in pupillary size and reaction. The changes to be expected in a fatal termination from compression are illustrated in Fig. 16.

* If, because of lack of experience or lack of time, the nurse is unable to take the blood pressure reliably, this may be omitted.

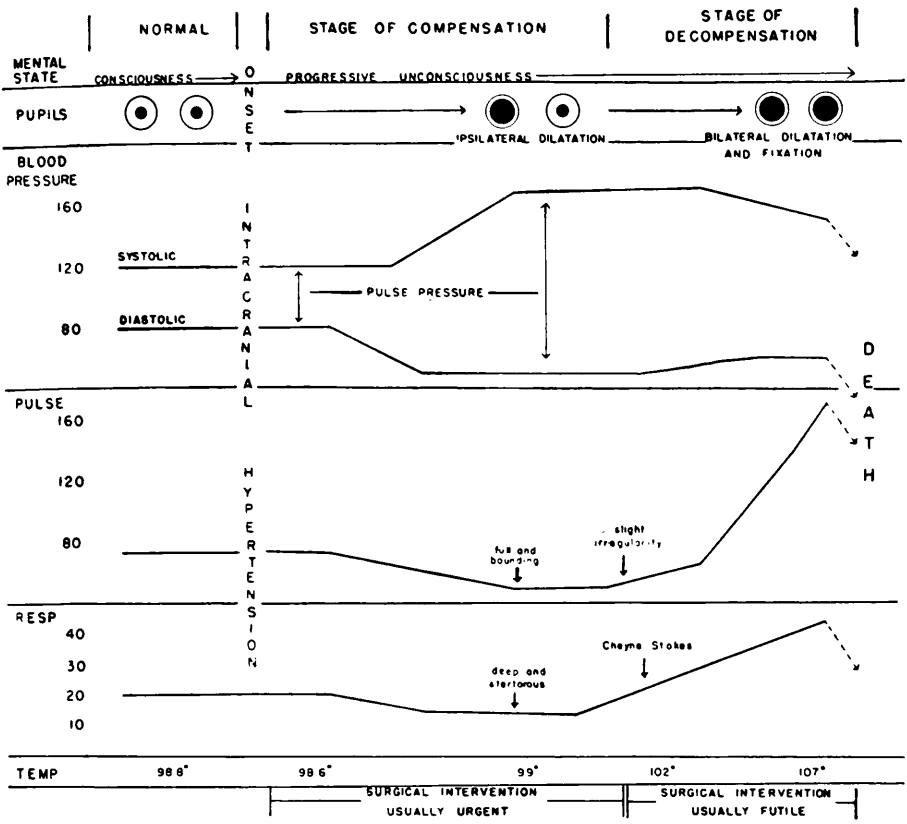


FIGURE 16

Illustrative chart showing: changes in mental state, pupils, blood pressure, pulse rate, respiration rate and temperature before and after the onset of fatal increase of intracranial pressure.

In regard to *medication*, it is better to use no sedatives if possible. At all events, morphine should be prohibited as this is apt to hide certain signs of danger. If the patient is very restless and hard to control, barbiturates may be given by mouth or by hypodermic injection, and paraldehyde, 3 dr., by rectum. If the headache is great, aspirin and phenacetin may be given by mouth, and, if necessary, codeine. Codeine serves very well in such cases and does not seem to have the bad effects of morphine.

The *unreasonable patient*, who becomes violently active and cannot be reasoned with, is better controlled by *gentle restraint* rather than by tying him down to his bed for this may increase the actual amount of struggling. It is better to have him sitting up quietly rather than supine and struggling. It is occasionally true that a patient, who has received

a severe head injury, may seem to be rational during a perio of a week or ten days when he does not heed orders and i violent and entirely uncooperative. For this period he usuall has no recollection at a later time and he must be treate with forbearance. In our experience this condition occur particularly when there are large amounts of blood in th cerebrospinal fluid, although a bloody fluid will not of itsel produce such a state.

Hyperthermia (pyrexia) in patients with severe lesions o the brain does not necessarily indicate that an infection ha set in. *Neurogenic hyperthermia* is produced by paralysis o the mechanism of heat elimination. The representation o heat control is located in the diencephalon near the posterio part of the third ventricle. Therefore a lesion above the sell turcica or in the brain-stem below this, or even in the uppe spinal cord, may disturb the regulation of temperature.

It is usually not difficult to recognize neurogenic hyper thermia. The patient's hands and feet are cold and may b mottled. His skin is dry while his temperature rises to 102 or even 106° F. and above. He does not have chills. In con trast to this a patient with a beginning infection is flushe and feels hot.

Proper treatment is to expose his body completely excep for a loin cloth. This is usually enough to bring the tempera ture down. But, if not, aspirin may be given and ice bag may be placed in the axillae and wet towels applied to th skin.

Nursing sisters should be on the lookout for this refle hyperthermia and should routinely cover any patient ver lightly following craniotomy or head injury unless his tem perature is below normal. As such a patient may have onl partial control of his temperature, the nurse can use hi rectal temperature as her guide in covering him, considerin 99°F. to 100.5° normal. It is possible to kill an unconsciou patient or one with a high spinal paralysis by covering hin too warmly.

Hyperthermia is not infrequently associated with hyper pnoea and tachycardia, also of reflex origin. This likewis points to a dangerous disturbance in the diencephalon. It i

apt to be fatal in one to two days even if the temperature is controlled. Hyperpnoea can be combated to some extent by small doses of morphia (1/12 gr. q. 1 h.).

Duration of bed rest calls for careful consideration in each case. Conservative treatment in clinics throughout the world has come to include provision for a minimum of three weeks in bed, without sitting up, for all cases of severe head injury. Any patient who has had blood in the spinal fluid, fracture of the skull or complete unconsciousness for longer than 20 minutes, should be considered to come within this group. It is during this three week period that the most serious immediate complications may make their appearance.

But when patients have been studied carefully at the outset a greater latitude of treatment is to be recommended, and if each case is considered individually, men, whose condition is otherwise good, may well be restored to normal activity much more rapidly.

In a personal communication on this point Dr. K. G. McKenzie has written: "I may say that my practice for some years now has been to allow patients up much earlier than we used to. In general, my rule now is to let them up a day after the patient feels like getting up; for instance, I would not hesitate about letting a compound depressed fracture case get up a day or two after operation if he feels like it."

It seems reasonable that if a patient has had mental disturbance continuing beyond the day of his injury, if he has a continuing increase of intracranial pressure as indicated by spinal fluid pressure measurements or severe headaches, or if he presents neurological signs of a cerebral lesion, it is much wiser to prolong the period of bed rest. On the other hand, in the case of a depressed fracture uncomplicated by prolonged loss of consciousness it may well be preferable to have the patient up as advised by McKenzie. The prevention of posttraumatic headache and dizziness probably depends not so much on the duration of therapeutic bed rest as upon early lumbar puncture (see p 68).

In case there has been *cerebrospinal otorrhoea*, the external auditory meatus on that side should be cleaned out gently and sterile cotton placed loosely in the meatus. It is better

not to manipulate the drum in any way and the exter canal should not be irrigated. If there is *cerebrospinal rhin rhoea*, as indicated by dripping of colourless or pink fl from the nose, when the patient is prone, it is best to cle the nostrils carefully and perhaps to drop into the nose oily fluid, or antiseptic such as argyrol, but there is nothi to be gained by plugging the nostrils with cotton. Irrigatic of the nasal passages should be forbidden as well as sniffi and sneezing. In either case it is best to administer sulphani mide or an allied compound by mouth until the fluid esca ceases.

If the rhinorrhoea becomes chronic it is usually due to t fact that a bit of dura is caught in a fracture line ne frontal or ethmoidal sinuses. The dura acts as a wick, a the condition should eventually be treated by cranioton and closure of the fistula from within the cranial cavity l fascial transplant.

Finally, as described on p 13 et seq., certain *danger signa* most of which are signs of advancing compression, should recognized by all in attendance on a case of acute head injur nursing sisters as well as medical officers: falling pulse to rate of 50 or 60, rising systolic blood pressure, very seve headache, increasing restlessness, decrease in voluntary mov ment of the arm or leg, progressive dilatation and fixati of one pupil, Cheyne-Stokes respiration, decreasing ment alertness and coma.

Post Traumatic Epilepsy
Penetration Dura - 18.7%
No " " - 1.6%

Chapter III

PREVENTION AND TREATMENT OF THE LATE COMPLICATIONS OF HEAD INJURIES

THE busy surgeon who is occupied with the immediate problems of head injuries is apt to see few of the late complications of such injuries. For that reason he is prone to ignore preventive therapy.

These sequelae are of great importance to *compensation boards* and constitute a considerable proportion of *pensionable disabilities*. Posttraumatic epilepsy can be prevented in some cases by radical discriminating surgery, and the same may be said of posttraumatic mental deterioration. Neurosis is to be prevented by understanding and by clinical wisdom, whatever may be the nature of the patient's injury. Furthermore, there are means for the prevention and for the cure of posttraumatic headache which have been heretofore ignored but which should be employed in the early weeks after injury.

1. PREVENTION OF POSTTRAUMATIC EPILEPSY

The likelihood of this complication is increased, not so much by the severity of the blow, as it is by penetration of the dura. This was shown by the studies of Wagstaffe (1928). He collected 377 cases of gunshot wounds of the head which had come under his personal observation between the years 1916 and 1918 and which he later followed up, finding that, when there was any form of penetration of the dura, 18.7 per cent developed epilepsy. When the skull was fractured without penetration of the dura, only two out of one hundred and twenty-four patients became epileptic, or 1.6 per cent.

Thus, in his experience, traumatic epilepsy was nearly ten times more common with penetrating wounds of the dura than with other injuries to the head.

Other studies which likewise had to do with war-time head injuries gave a higher incidence of seizures. This difference may be attributed to a difference in the therapy or in the type of case studied. Stern (quoted by Schou, 1933) found that some 20 to 50 per cent of patients with open brain injuries later developed seizures, as against 2 to 4 per cent of patients with closed injuries to the skull. Among the patients who do develop posttraumatic epilepsy, over half of them suffer their first seizure in the first year after the injury, 1/6 in the second year and 1/16 in the third year, the incidence falling progressively in the succeeding years. Attacks may begin, however, twelve and more years after injury.

It is obvious, therefore, that penetration of the dura and laceration of the brain greatly increase the likelihood of eventual posttraumatic epileptic seizures. When the dura has been crossed by a fragmented bit of bone or by a penetrating missile, the surgeon is so occupied with the immediate danger of meningitis and of haemorrhage that the ultimate danger of epilepsy tends to be forgotten.

This danger can be greatly lessened by discriminating surgical intervention. In the simpler cases in which there is a depressed fracture of the skull, the fragments should be elevated early. *Gross penetrating wounds of the brain* must be treated by débridement, done properly, as outlined on page 44. A sketch of brain laceration is shown in Fig. 12. Obviously, the surgeon should remove the bone fragments and foreign bodies in such a case. But to prevent posttraumatic epilepsy he should also, if possible, remove the cortical tissue with a sucker over to the line of cleavage formed by a fissure, as indicated by the dotted line in Fig. 12. He should thus leave as little unvascularized gray matter as possible.

The administration of anticonvulsant drugs after the patient has recovered from his initial injury is not to be considered a method of prevention and should be discouraged. If any posttraumatic advice is to be given, it might be well to advise patients who have had a cerebral laceration against

the use of alcohol and against chronic constipation. The treatment of *posttraumatic epilepsy* must include surgical therapy in some cases. Nothing is to be lost, however, if the usual conservative methods of treatment are employed for a year or two before undertaking excision of the *meningocerebral cicatrix.*

When posttraumatic seizures do develop, *cranioplasty* for skull defects should be discouraged as useless unless the surgeon is prepared at the same time to deal adequately with the underlying brain scar. The plastic closure of a skull defect is permissible, of course, to provide against future brain injury or to relieve the headache and dizziness which sometimes result from large cranial defects.

The *surgery of meningocerebral cicatrix* will not be described in this manual. It calls for preoperative study by pneumoencephalography and electroencephalography and should only be undertaken in specially equipped neurosurgical services.

2. PREVENTION AND TREATMENT OF POSTTRAUMATIC HEADACHE

Headache in general is the commonest complication of head injury. The initial headache is apt to be due to increase of intracranial pressure and this type of headache disappears with the return of normal pressure. If chronic posttraumatic headache develops, it usually begins during the period of convalescence, but it may occur only after the patient is up and about again. Failure on the part of physicians to recognize the obvious characteristics of this type of headache, together with the dizziness which usually accompanies it, results in the unfair assumption that these sufferers are either neurotic or malingering.

Posttraumatic headache has certain charactertistics which make it easy of recognition (Penfield, 1927)(i)The ache is usually at the site of the original blow, but not always. It does not move from one part of the head to another. It is apt to be present continuously day and night. It is not associated with nausea or with migrainoid visual phenomena. There are often diurnal variations in intensity which are quite regular. The patient sometimes has a favourite position

in which to place his head at night to lessen the ache. The *posttraumatic dizziness*, which is usually associated with it, is periodic, is apt to be brought on by effort or movement, is not associated with nystagmus or with tinnitus. It lasts usually from one to five minutes. The patient often says that it is a blurring or difficulty of vision rather than a true rotatory sensation.

Posttraumatic headache seems to result from a pull upon a sensitive area of the under surface of the dura by adhesions between dura and leptomeninges. At the time of the blow, two things apparently happen: firstly, the brain is shaken into a new position within the subdural space and, secondly, adhesions form to hold it there. Exudation of plasma and blood occurs upon the under surface of the dura at the site of the blow. Subdural adhesions thus form at that point and it is possible that cerebral oedema, at first, prevents the displacement and rotation of the brain back into its normal position. Thus when the patient gets up and about after the injury, the brain is prevented from returning to its ordinary position within the subdural space by the new-formed adhesions. There results, consequently, a chronic traction upon the under surface of the dura exerted by the brain, and if the area of dura in question is a sensitive one, constant headache results.

Posttraumatic headache occurs as often after minor or moderate head injury as it does after severe injury with profound loss of consciousness. Open wounds of the brain, also routine craniotomies, rarely, if ever, give rise to posttraumatic headache. It is obvious that in such cases adhesions will form between dura and arachnoid during the convalescent stages, but the brain becomes adherent in what may be called its normal position and without the displacement that might occur as the result of a closed head injury. Those patients, in whom for one reason or another an opening in the dura has been made shortly after accident, likewise rarely, if ever, develop posttraumatic headache.

Prevention of posttraumatic headache. Among patients who have been treated by early lumbar puncture and bed rest this type of headache is a rare sequel. In a series of 97 cases of

posttraumatic headache studied by Elvidge and Lehman at the Montreal Neurological Institute it was possible to get adequate information as to the original treatment of 64. Of these less than 10 per cent had had lumbar puncture within the first 24 hours, while 62 per cent had had adequate bed rest (over 2 weeks). It seems probable that the best prevention of the condition is early lumbar puncture as described above, while the duration of bed rest may be of little importance.

Spinal insufflation. At the end of the second, third or fourth week after head injury any patient who seems to have developed a chronic headache of the posttraumatic variety should be treated by a spinal insufflation of air in order to relieve the condition before it has had time to become well established. Encephalography, with filling of the ventricles, is therapeutically useless.

Therapeutic insufflation should be carried out as follows: Place the patient on his side and with his head rotated half way to the vertical, the head of the bed being somewhat elevated (Fig. 17). Lumbar puncture is done and as much

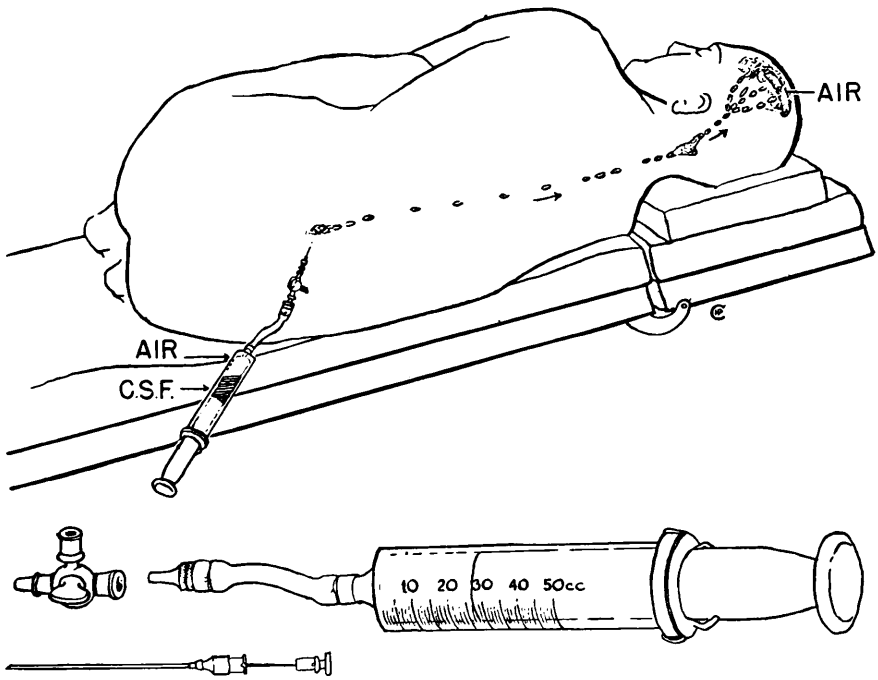


FIGURE 17

Spinal insufflation of air for treatment of posttraumatic headache.

spinal fluid withdrawn into a large syringe as possible without undue discomfort (20-60 cc.). The syringe should then be filled with air which may be filtered by drawing it through a gauze sponge. The syringe is then reinserted in the needle and air injected in 5 or 10 cc. amounts, withdrawing equal amounts of fluid, until 50 to 90 cc. of air has been injected. When injected in this way the air passes upward into the subarachnoid spaces of the basal cisterns and intergyral sulci, avoiding the ventricles, and breaks through the arachnoid membrane so that it enters the subdural space over the cerebral hemisphere. Alteration of head position every 5 minutes during the next hour will carry the air to different portions of the subdural space and thus allow the brain to pull away, breaking its adhesions. The brain thus assumes its normal resting position within the subdural space. This procedure is painful. It is well therefore to administer hyoscine, gr. 1/100 to 1/75 s.c., 90 minutes before, and morphine ¼ s.c. at the time of the puncture.

At a later stage, after the patient is up and about, spinal insufflation may also be used as treatment, but a more certain procedure, with a higher percentage of success, is the second method: direct insufflation by trepanation and lumbar puncture.

Direct insufflation. The patient is placed on his side on the operating table. A small scalp incision is made at the site of his head-pain and a hole is made in the bone with a perforator and burr. The dura is then opened. The head of the table is raised and a lumbar puncture needle is inserted and, as the spinal fluid escapes from the needle, the brain falls away from the overlying skull (see Fig. 18). The scalp is then sutured in two layers with particular attention to haemostasis because of the fact that the dura is left open. A small hollow needle may then be passed through the scalp and not more than 10 cc. more air injected into the burr hole. This further displaces brain from dura. Care must be taken at this point as tributory veins to the longitudinal sinus may be broken by the exertion of too great a pressure. To guard against this, the procedure should always be carried out under local anaesthesia so that the patient will object if too

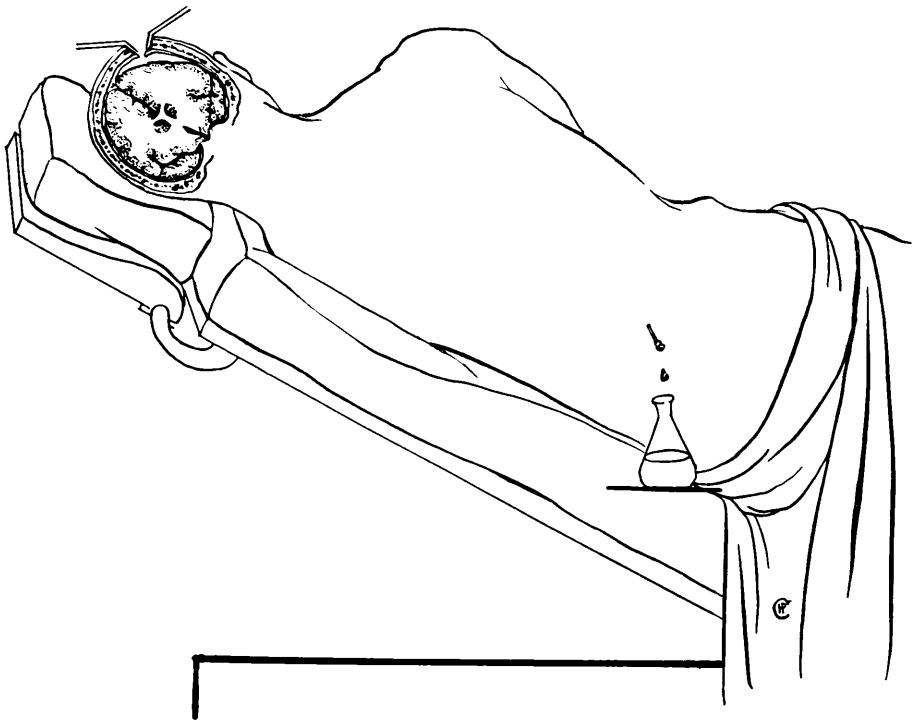


FIGURE 18

Trepanation and direct insufflation for posttraumatic headache. The spinal fluid is escaping from the lumbar needle while the ventricles collapse and air enters the subdural space.

great a traction be exerted upon such a vein. To lessen the pain morphine may be used during the period of head oscillation which should follow the procedure.

3. PREVENTION OF POSTTRAUMATIC NEUROSIS

It should be recognized that the development of a posttraumatic neurosis may sometimes be induced by faulty handling of the patient by his surgeon in the days immediately following injury. Neurosis and physical disability may coexist and the former is apt to outlast the latter. After an injury to the head a patient may well have a good deal of honest fear in regard to serious injury to his brain.

If a patient has had a minor accident, such as injury to his arm, and his physician is certain of the diagnosis and of the fact that it is not important, the patient likewise accepts that conclusion. But if he has had a head injury and if the physician is not sure of the situation, this doubt enters the

mind of the patient and may become a most important element in the establishment and continuation of a compensation neurosis. Such a doubt sets the stage for malingering, while the honest fear of brain injury promotes the development of a true neurosis.

The best method of prevention is definite early diagnosis on the part of the attending surgeon. The early *diagnosis of organic lesions*, together with recognition of true *anxiety neurosis* and of *hysteria*, without using the misleading term "shell-shock," are the important elements of prevention. In case time does not permit careful initial study under military conditions, then the patient may be labelled temporarily as: not yet diagnosed condition of the nervous system ("not yet diagnosed nervous").

There is no such thing as "*shell-shock.*" This most unhappy phrase was coined early in the war of 1914-1918 after Mott had described diffuse brain changes in a patient who had been blown up in an explosion. There grew up a belief among the laity and among the soldiers themselves during that war that proximity to exploding shells could cause an actual cerebral change. This was an error, excepting in so far as it might refer to the true concussion which we have already described. Unfortunately, the expression "shell-shock" and the conception of concussion directly producing hysteria were accepted even by some medical officers and this diagnosis was actually recorded.*

Faint-hearted individuals under the influence of fear and the discomforts of military life welcomed such a diagnosis as a satisfactory "way out," perhaps quite unconsciously. Among men in those units where the morale was high and pride in performance great, such cases were rare. In other units they were common. The practice of invaliding men back to pleasant nursing homes to convalesce from the

* The following paragraph may be quoted from Russel's (1939) experience in the war of 1914-1918: "One might recall that we saw various evidences of reaction to the exigencies of war. There were some who under very prolonged and heartbreaking strain and responsibility developed an anxiety neurosis; these were in my experience always officers, and not numerous. While they naturally excited one's sincere sympathy their problem is not what I wish to discuss at this time. Then there was the man who showed the straight reaction to fear—anxiety, trembling and jumpiness, or in some cases some mental confusion was added or even almost a stupor; but these types with common sense treatment of rest, food and an understanding appreciation, returned to duty in a few days. Besides these, a large class, which became larger the farther one got away from the front, exhibited all the evidences of *conversion hysteria*—the so-called "shell-shock."

imagined injury called "shell-shock" was the one thing best calculated to perpetuate and to create neurosis.

4. PREVENTION OF MENTAL DETERIORATION

Decreased mental capacity is a complication of head injury too often recognized only long after the injury and too rarely foreseen in the early days following the trauma. Patients complain most often of *defective memory* and of easy fatigue as the result of concentrated mental effort. Or there may be a lack of initiative and of quick insight which is only evident to those who live close to the patient, defects of which the patient himself is apt to be oblivious. The condition occurs most often in those cases in which there has been a long period of loss of consciousness or a long period of restless confusion.

Cerebral compression may be a very important element in the development of this impairment of mental activity for, during the first week or two after injury when brain oedema and varying degrees of subarachnoid haemorrhages exist, the swelling of the brain may be sufficient to close the subarachnoid spaces through which the spinal fluid from the ventricles must pass in order to be absorbed. This produces a temporary obstructive *internal hydrocephalus* and results in the permanent enlargement of the ventricles. The direct pressure upon the cortex at such times must also cause permanent cortical damage.

Consequently, it is obvious that in order to reduce brain injury intracranial pressure should be reduced as much as possible during the first weeks. This may sometimes be effected by repeated *lumbar punctures*. In other cases a large *subtemporal decompression* should be carried out.* We have not infrequently seen a drowsy patient, suffering from very intense headache during the first or second week after injury, completely relieved by subtemporal decompression, preferably of the myoplastic type. His mental processes brighten, his spinal fluid pressure drops, and a vicious cycle seems to be broken. Such preventive operations are especially urgent in the case of patients of high intelligence.

* It must be admitted that the patients who call for decompression on those grounds are rare.

Chapter IV

SPINAL INJURIES

In the treatment of this subject only those facts will be presented which are essential to intelligent handling of cases of spinal injury by the medical officers who see them early. The special procedures described are those which it is reasonable to expect that men trained as general surgeons should learn to carry out. Such procedures as laminectomy, fusion of the spine, and head traction are of great importance in a few selected cases but candidates for such treatment must be handed on to specialized neurosurgical teams.

However, it is in the initial period that any medical officer on general duty may be called upon to make a prognostic estimate and to see in perspective the whole problem presented by a case of spinal injury. Some patients can never be returned to a satisfactory existence. Others can well be preserved for the useful life which is their right by prompt and energetic action. "The skill of the surgeon is of prime importance in the surgery of war, and nowhere is it of such great importance as in the first treatment of the wounded. . . . The surgeon must show judgment in selecting for extensive operation those who have a fair chance of recovery, staying his hand from those that must die."*

1. TRANSPORTATION

Traumatic paraplegia. Before any study can be made the immediate problem is one of safe *transportation* with maintenance of a position which will not produce further injury to the spinal cord. Most fracture-dislocations occur when the subject's spine is flexed sharply. This produces a compression fracture of one or more vertebral bodies together with forward dislocation of an upper upon a lower vertebra.

* Quoted from the 1940 Lecture of the Royal College of Surgeons of Canada, W. E. Gallie (1941).

Thus the cord may be compressed on its anterior surface over the edge of the lower vertebral body. To relax and release the cord the spinal column must therefore be extended.*

Any injured man who has a paraplegia or who gives a history of bilateral paralysis of the limbs requires special care during transportation and bearers should be given routine instructions in regard to it. If a medical officer is present when the patient is first picked up, it is well first to straighten him out while traction is being exerted upon head and feet. In any case, he should not be lifted from the ground by shoulders and feet unless he is lying prone. The best routine is to turn the paraplegic over so that he lies face down upon a blanket spread upon the ground. He can then be lifted by means of the blanket onto a stretcher, and a coat or other object placed under his head to increase the extension. He may then be transported in that posture and when he is lifted off the stretcher onto a table or bed the blanket is still to be used as a lifting sling. If the injury is in the cervical region, steady traction should be exerted upon the head during any change in the patient's position so as to avoid angulation or bending.

Application of a *plaster cast* to immobilize the neck and trunk should be carried out as early as feasible, except that it is usually not wise to apply plaster to a completely anaesthetic patient. After that, transportation is possible, even to considerable distances, without danger of increasing any existing damage which the spinal cord may have received. However, the patient should usually be turned periodically during long transport, even in plaster, so that the body weight may not cause pressure necrosis. Finally, in the case of spinal fracture without penetrating wound, it is important that the patient be hurried back to a base hospital in one way or another.

When a cast is to be applied, the spine should usually be placed in extension, not in flexion. The patient may sometimes be supported in a sitting posture with the aid of a

* For an excellent recent discussion of war injuries of the spine, further reference may be made to Knight, 1940.

chin-occiput supporting halter. It is usually better, however, to place him prone upon a hammock (Fig. 19) of heavy cloth swung between two tables, or a portion of blanket may be sacrificed for this purpose. This provides extension of the spine. The hammock must be included in the plaster and then cut off when the procedure is finished and the plaster is sufficiently dry.

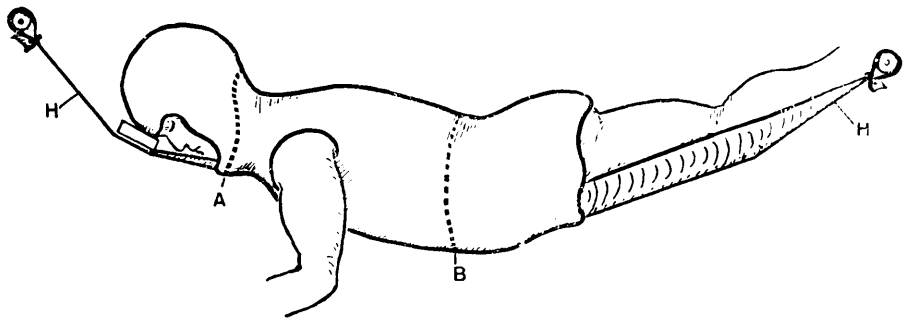


FIGURE 19

Application of plaster of Paris cast for vertebral dislocation. Patient supported in hammock of cloth (H) which may be cut off above and below plaster when the procedure is finished. For cervical dislocation the cast need not extend below broken line B. For thoraco-lumbar dislocation it need not extend above broken line A.

Casts for immobilization of the spine should be put on after protecting the skin with a thin layer of cotton wadding followed by a layer of piano felt. If insufficient felt is at hand, the bony prominences at least should be padded and pieces of flat sponge-rubber serve this purpose well. For a low fracture-dislocation the cast should include pelvis and sacrum. For a high lesion it must include the neck, head and forehead, as in Fig. 19, leaving enough room under the jaw for its movement. It is usually best to cut out a window over the abdomen as soon as the plaster is dry to provide room for respiratory movements and for the abdominal distension which may become troublesome.

2. STUDY OF A CASE OF SPINAL INJURY

At the earliest opportunity neurological examination should be made and anteroposterior and lateral stereoscopic *roentgenograms* should be taken, preferably after removal of the cast. Lumbar puncture should be carried out with the

aid of a spinal fluid manometer so that manometric determinations may be made as follows:

Spinal manometric test (Queckenstedt). The patient is placed horizontally on his side on a bed or table.* A *lumbar puncture* needle is then inserted in the third or fourth lumbar interspace and a "three-way" stop-cock attached to the needle. Into the stop-cock an upright glass manometer is inserted, as shown in Fig. 15. A mercury manometer may be used but is less satisfactory. A blood pressure cuff (Cone and Grant 1934) is then wrapped about the patient's neck (or the jugular veins may be compressed digitally). If the cuff is used, it is inflated in turn to pressures of 10, 20, 40, and 60 mm. of mercury, and the rise and fall of the cerebrospinal fluid recorded each time as in Table II on page 95 of the Appendix. If there is no block to the passage of fluid up or down the spinal canal, the fluid in the manometer will rise readily when the jugular veins are compressed.

Normally, spinal fluid pressure varies from 100 to 200 mm. of water. Jugular compression should cause it to rise to double or treble that height within 10 to 15 seconds and it should fall just as rapidly if the end of the needle is kept clear. If there is no response to jugular compression, it may be assumed that a *spinal block* exists somewhere between the foramen magnum and the lumbar puncture needle. Such a block is produced by an intraspinal expanding or inflammatory lesion or a direct pressure upon the spinal sac. It must be borne in mind that coughing or straining may still raise the pressure in the manometer even though a block be present. This is due to the fact that increasing the pressure in thoracic or abdominal veins causes the veins within the spinal canal, with which they connect, to become engorged and thus raises intraspinal pressure to that extent. Coughing and straining should be used to show that the needle is clear and that the manometer is recording. Conversely, care should be taken that the patient does not strain when the jugular veins are compressed.

Neurological examination should be carried out and

* In case of fracture-dislocation of the spine, the body should not be flexed as for an ordinary lumbar puncture but the position should be maintained as during transportation.

recorded. The degree of paralysis or impaired strength must be determined carefully. Loss of appreciation of pain and temperature sensation, loss of tactile sensation, loss of muscle-tendon sensation (i.e., sense of joint position) and of vibratory sensation (appreciation of the vibration of a tuning fork), presence or absence of control of bladder and rectal sphincter and, if this control is lost, of urethral sensation; all should be recorded. The examination may be quickly and graphically recorded on a body outline such as is shown in the appendix, Table III, page 96.

The deep reflexes (biceps, triceps, radials, ulnars, patella and achilles reflexes) may be absent for a considerable time after a severe spinal injury (days and weeks). This state has been called spinal shock. These reflexes return later and become hyperactive even though function does not return. Abdominal reflexes disappear if the injury is above the 9th thoracic spinal segment and the cremasteric reflexes as well. The plantar responses are apt to be flexor (downgoing great toe on plantar stimulation) or this reflex may be absent for a period of days or weeks after severe spinal injury, later becoming extensor. Slight spinal injury is apt to give a positive Babinski response (extensor plantar response) from the beginning.

Spinal Concussion

This is a term applied to the state of paralysis produced in the spinal cord by a sudden jar transmitted to the cord by a blow upon the neighbouring skeleton or by the impact exerted upon it from the passage of a high velocity missile through adjacent tissue. Under such conditions paralysis is of short duration, being limited to minutes or hours. When paralysis continues for a longer period the condition should be considered one of contusion of the cord.

Spinal Contusion

This may produce local softening of the cord associated with increasing oedema of the medullary substance. If laminectomy is carried out within a day or two after severe contusion, the cord will be found swollen and the pia under very great tension. This enlargement, however, is not suffi-

cient, in our experience, to produce spinal block by itself. If the surgeon incises the pia at that time, he will be horrified to see the softened portion of the spinal cord extrude itself through the hole in the pia like paste from a tube when it is compressed. If, instead, such a cord is explored at a later period, a flattened, soft segment is seen at the site of contusion. In either case the man's paralysis will probably be permanent and usually remains complete.

The experimental work of Allen (1918) suggested that if the pia was incised within the first 12 hours of contusion the decompression thus produced would save the nerve cells and tracts at that point from destruction. This seems reasonable but clinical verification is lacking, and, even in the piping days of peace, it has not proved practicable to carry out such operations. Consequently, in military surgery early incision of the cord is not to be recommended.

The most frequent cause of contusion is the blow which the cord receives when two vertebrae slip over each other at the time of fracture-dislocation. This occurs most frequently in the lower cervical and the upper lumbar regions because of the relatively greater mobility of those parts of the spine. Oedema and *haematomyelia* often follow and play an important role in the prevention of return of function of the injured cord.

Spinal Compression

A moderate degree of compression may interrupt function in the cord partially or completely and yet on its removal restoration of function may occur. The pressure produces local anaemia and it may arrest nerve impulse mechanically. It is in such cases that care of transportation is most important and relief of the pressure by manipulation or by operation is of the greatest importance. When such cases are studied, partial or complete paralysis may be found together with spinal fluid block.

Spinal Laceration

If the spinal cord receives a crushing blow or if it is severed by a missile, laceration occurs which is irremediable and this fragile organ may be found completely severed except for

wisps of tissue. When there is a direct blow upon the back, spicules of bone from the laminal arch sometimes produce a partial laceration of the cord. In such cases spinal block or x-ray study may suggest the possibility of such a lesion and operation then offers hope of recovery.

Regeneration of destroyed neurones or axones never occurs in the central nervous system although Cajal showed that there are abortive attempts on the part of axones to grow downward again. Even the posterior and anterior roots of the cauda equina do not seem to regenerate successfully after suture. Nevertheless, one may still suggest that if severed anterior roots present themselves they might well be sutured by a surgeon endowed with patience and optimism and who is not pressed for time. From the spinal ganglion outward through the whole body, nerves will regenerate actively if not prevented by separation, by scar or by bungling surgical suture.

3. TREATMENT OF FRACTURE-DISLOCATION OF THE SPINE

After roentgenographic, manometric and neurological study has been carried out, decision as to treatment and disposal of the case can be reached. If a spinal block exists, it should be considered that there may be a continuing pressure upon the spinal cord or the cauda equina. Such pressure may be exerted by a dislocated vertebral body or by a depressed laminal arch or a splinter of bone from lamina or pedicle. Pressure may also rarely result from the presence of a foreign body or from extradural oedema and haemorrhage. If the spinal fluid contains blood, cord trauma is probable, but the fluid should be removed in successive test tubes to be sure that the presence of the blood is not due to bleeding caused by the entrance of the needle, as described above on page 58.

In the case of *penetrating wounds of the spine*, operation must always be undertaken and it must be early and the excision of damaged muscle must be radical, as well as removal of bits of vertebral bone. Sulphonamides should probably be placed in the wound generously.

The following general rules may be laid down to assist in decision as to the handling of "closed" spinal injuries.

Partial paralysis with block. If the paraplegia is only partial, or if the paralysis is complete and yet some sensation below the level of spinal injury is preserved, then there is much more reason to hope for return of function, especially if evidence of a spinal block exists. In such a case operation should be considered as an immediate possibility and decision made quickly. Under these circumstances it is advisable to transport the patient to a special hospital for spinal operation or to arrange for immediate operation on the spot if an experienced neurosurgeon is available.

If interference is determined upon, the operation of choice under these conditions is *laminectomy*, and removal of the bone fragments which will often be found impinging upon the cord. At the same operation *spinal fusion* may be carried out by curettement of the articular facets and by the lateral insertion of bone fragments after the method of Hibbs; or fusion and immobilization of the vertebrae may be obtained by means of lateral bone transplants using tibia, rib, or dried beef bone (os purum). *Skeletal traction* applied by skull tongs or skull wires (Turner and Cone 1938) may be of great assistance in lesions of the cervical spine. After such radical therapy, immobilization by plaster for months will also be necessary but the patient can be gotten up much faster than when fusion is omitted.

Manual reduction. If marked displacement or angulation of the cervical vertebral column is shown by x-ray, an attempt at replacement may be made by strong, continued traction upon the head for 5 to 30 minutes by means of a chin-head halter and a general anaesthetic may be given to overcome muscular spasm. The deformity may thus be improved or corrected, after which plaster is applied. This will succeed only occasionally.

Complete paralysis without block. If there is complete absence of motor and sensory function below the level of injury and if there is no block, improvement of the patient's chances of recovery by means of operation is extremely unlikely. Nothing should be done in this case except immo-

bilization and general care. The expenditure of time and labour beyond this may well prove to be fruitless.

In case of *partial paralysis without block*, it is usually sufficient to immobilize the patient by application of a cast in hyperextension, although careful consultation is usually indicated. The same may be said of the case in which there is *complete paralysis with spinal block.*

4. TREATMENT FOR BLADDER PARALYSIS

Paralysis of the urinary bladder due to injury of the spinal cord or nerves presents a difficult practical problem. It is best not to allow the bladder to become too greatly distended at first for this reduces the bladder's resistance to the development of cystitis. Consequently, catheterization* should be carried out at regular intervals so spaced as to prevent the bladder from filling up to a point beyond 500 to 800 cc. Usually every eight hours will suffice. During the period of catheterization the patient should be given urotropin from 7½ to 15 grs. t.i.d. a.c. and, if necessary, acid sodium phosphate in amounts necessary to give the urine an acid reaction. In place of this, sulfanilamide therapy may be an effective preventive.

If spontaneous micturition does not return within a few days, an *indwelling catheter* may be inserted and this is then best attached to a *tidal drainage* system like that of Monro (Bellis 1940). This simple apparatus, which is explained in Fig. 20 and in its legend, gradually adds, to the urine in the bladder, sterile saturated solution of boracic acid under a low pressure. In case of frank cystitis, silver nitrate 1:5000, potassium permanganate 1:4000, or other similar solution may be used at a low intravesical pressure.

When the pressure rises, due to fluid accumulation or to a tendency of the bladder to contract, siphonage in the system is precipitated and this completely empties the bladder. It then fills gradually again with urine and with irrigation fluid until the whole process repeats itself. The rhythmic activity of the bladder makes possible restoration of its normal

* If the paralysis is obviously complete from the beginning, an indwelling catheter may be inserted at the start and periodic irrigation used until the tidal drainage is started as described below.

activity as soon as nerve control returns, and the indwelling catheter is remarkably well tolerated, with comfort to patient

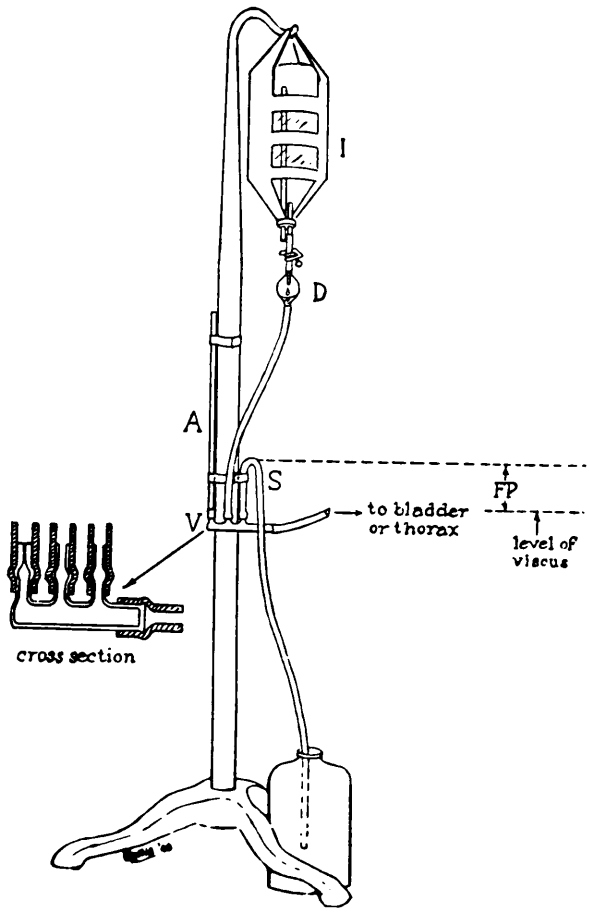


FIGURE 20

The irrigating fluid (I) is placed in a suitable flask suspended at least 75 cm. above the level of the symphysis pubis. The flow is regulated at 40–60 drops per minute, by means of a screw clamp above the drip (D). The drip should not have an air vent. The fluid then enters the horizontal arm of the modified glass Dakin's tube and passes into the bladder through the rubber tubing connected to the indwelling catheter. It also ascends into the air vent and manometer (A). The latter consists of a vertical glass tube about 6 or 7 mm. in diameter and 30–40 cm. in length. At the bottom of this air vent a valve (V) is interposed. The opening of the valve has been narrowed by fusion so that it just admits the point of a pin. The height of the siphon tube is adjusted to the desired pressure (FP) which the bladder must reach to initiate siphonage.

When siphonage is instituted the bladder is drained rapidly and the manometer slowly because of the valve. Thus the bladder is completely emptied, before the siphonage is broken. In the tidal irrigation of the atonic bladder a pressure of 2–5 cm. of water is recommended. This pressure should be increased to 10–15 cm. if the bladder detrusor is intact. When the apparatus is functioning properly, there are visible respiratory excursions in the manometer. (Bellis, 1940).

and a minimum of nursing care. In case control does not return when the patient is gotten up he may develop an automatic bladder or may be given a *suprapubic cystostomy.* Occasionally suprapubic cystostomy may be preferred from the beginning, for example, if it is desired to move the patient about.

5. SUMMARY

In conclusion, when a break has occurred in the supportive framework about the spinal canal or the vertebral column, the spinal cord or cauda equina are immediately vulnerable. Traumatic injury that has already occurred to the nervous structures cannot be repaired but function may return if no further injury occurs, and if continuing pressures are removed. The medical problem is therefore to provide transportation without increasing cord injury, to immobilize the spinal column by plaster or occasionally by operative fusion, and to remove from cord or cauda equina any continuing pressure by bone fragment or foreign body at the earliest feasible moment.

The medical officer in charge of such cases must therefore be able to carry out a spinal manometric test to detect the presence or absence of spinal block. He must be able at least to immobilize the spine in a plaster jacket. And, finally, whether or not he may relish the prospect, any medical officer may have to decide quickly who can be saved by energetic treatment.

Chapter V

PERIPHERAL NERVE INJURIES

MANY manuals and books are available which present the anatomy of the peripheral nerves and the clinical disabilities produced by lesions of these nerves such as Pollock and Davis, 1933, and Stookey, 1922. A most useful handbook of peripheral nerve anatomy for Medical Officers is the "The Clinical Forms of Nerve Lesions"* by A. Benisty. Also an excellent pamphlet, *The Diagnosis and Treatment of Peripheral Nerve Injuries, Report No. 54 of the Medical Research Council, 1920,* will serve as a practical guide in the treatment of such injuries and this should be in the hands of all Medical Officers.

No effort will be made here to duplicate the information presented in this last pamphlet. But a few additional notes will be made on the technique of nerve suture and nerve liberation.

1. NERVE ANASTOMOSIS

Immediate suture is the most successful suture. If there is no loss of substance in the nerve, the ends should be cut across cleanly so as to see the pattern of funiculi above and below. It is essential, for the best results, to approximate the corresponding funiculi. To do this the first suture should be passed, through the sheath only, on the corresponding margin of upper and lower nerve segments (A, Fig. 21). Then a second suture should be placed, on the opposite side, in the sheaths of each nerve segment. When these two initial sutures are tied the corresponding funiculi will be approximated, provided the patterns have been carefully studied. A difficulty may be encountered here also if there has been loss of nerve substance, for the pattern may have altered and thus the matching process becomes difficult. These two

* Edited by E. Farquhar Buzzard and published in English by the Univ. of London Press in 1919 as part of a series of Military Medical Manuals edited by Sir Alfred Keogh and Sir Thomas Goodwin.

sutures are then used to handle and to turn the nerve during the further stages of the procedure. Two or three more sutures are then passed through the sheath edges and tied so as to approximate the gaping sheath between the attachments, first in front and then, when the nerve is rotated, behind (B, Fig. 21).

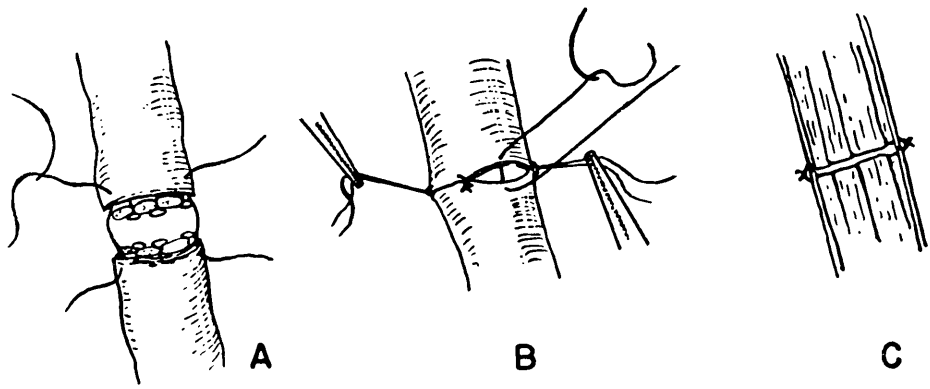


FIGURE 21

Nerve anastomosis. A = placement of primary silk sutures in nerve sheath so as to approximate corresponding funiculi; B = placement of secondary sutures using primary sutures to turn nerve; C = cross section to show corresponding funiculi opposite each other.

Suture material should be the thinnest silk on the smallest needles obtainable (see p 89). For this purpose Carrel blood-vessel sutures made of silk on straight needles, which are prepared in liquid paraffin and put up in glass capsules, are useful. It is equally satisfactory to take ordinary fine black silk and split it into its three component strands which may then be threaded into Carrel's curved arterial No. 20 needles. When carrying out the anastomosis the needles should be passed through a small amount of the sheath without touching the nerve bundles.* When the knots are tied the sheath edges should be turned outward. The ideal anastomosis leaves the funicular stumps about 1 mm. apart (C, Fig. 21). This is possible because of the elasticity of the sheath.

This fine silk has a surprising amount of tensile strength when the knots are tied. Only very rarely should a tension suture through the nerve body above and below be necessary to take up the tension present when the nerve ends are drawn

* Delicate pointed thumb forceps are essential for the handling of the nerve sheath, such as the modified jeweller's forceps shown in Fig. 22 (5).

together. The limb must be placed in a posture which will shorten the course of the nerve. It can then be maintained in that position, if necessary for weeks, by plaster or bandage and gradually straightened so as to stretch the nerve. In some cases the nerve may be transplanted to a new bed for a shorter course as described in detail in the Medical Research Council Pamphlet mentioned above.

The nerve should never be pinched. Handling of the nerves should always be carried out by means of the sheath. Fine pointed thumb forceps are therefore necessary (see appendix, page 90). The delicate needles can be manipulated best if they are held in a pointed "mosquito" haemostat.

The bed in which the freshly sutured nerve is placed should, if possible, be away from traumatized tissue so that the surrounding subsequent cicatrization may be reduced to a minimum. Envelopment of the nerve in sheets of any foreign body or membrane is to be discouraged, as a rule, for the eventual scar which results is usually denser as the result of the addition of such an envelope.

Secondary nerve suture. At a later period after the initial injury, nerve suture is more difficult and the results are, in general, less satisfactory. Nevertheless, it is never too late to hope for some recovery of function by means of nerve suture. A secondary suture constitutes an operation of election and should therefore be carried out by specially trained neurosurgeons, the patient being transferred if necessary.

When a nerve has been severed and the two ends separated, a traumatic *neuroma* forms on the end of the central segment. This is due to the fact that the naked nerve fibres of the central segment grow outward, and, when they do not encounter the peripheral segment, they grow around forming a ball, and at the same time there is a very energetic connective tissue reaction so that the bulbous neuroma which results becomes very hard and dense. When any portion of the nervous system loses its proper insulation it produces a most energetic surrounding connective tissue reaction, as though it were, in fact, a foreign body as pointed out by Trotter. Thus the closure of the sheath in ordinary nerve suture preserves the normal barrier.

In the peripheral segment the nerve fibres degenerate but the sheath of Schwann columns persists. This peripheral segment evidently exerts upon the growing fibres in the proximal segment an attraction even from a distance, so that a few nerve fibres may grow to it across a considerable gap of separation. If there is no functioning continuity of nerve the neuroma should be amputated and the ends drawn together and sutured as described above. To determine whether or not an adequate functional connection exists through an area of scar it is advisable to stimulate the upper segment electrically as it lies exposed.

Transplantation of other nerves, excised for the purpose, in order to fill a gap between upper and lower nerve segments will occasionally succeed in producing return of function, but every effort should be made first to bring the ends together by wide dissection and stretching of the segments, by posturing of the limb and by displacement of the nerve to a new bed before resort is made to transplantation.

2. NEUROLYSIS

Liberation of a nerve from scar tissue frequently results in rapid return of function without anastomosis. The nerve should be exposed and freed above and below the area of injury. If there seems to exist a considerable amount of continuity across this area the nerve should be cleaned off by sharp dissection. Palpation with finger and thumb will guide the surgeon to pare off the hard kernels of connective tissue from the nerve proper. After this paring process is finished the nerve itself feels much less dense to the palpating fingers. If only a few thin strands remain it is better to cut upper and lower segments across squarely and to proceed with nerve suture. If as much as half the normal volume of nerve remains and especially if stimulation shows that it is able to conduct impulses, it is better simply to transplant it to a fresh uninjured bed of tissue, after dissecting from it the adherent scar, without carrying out anastomosis. This procedure is called *nerve liberation* or neurolysis and is often followed by surprisingly rapid restoration of function.

APPENDIX

1. Materials and Instruments

Certain suggestions are made under this heading with no thought of laying down rules. All such equipment must, of necessity, vary with the changing needs and experience of operators.

Suture Material

Craniotomy. For use to close the dura a continuous suture of silk may be used, or interrupted, weight A, on a fine curved needle (Ferguson ½ circle No. 12). Muscle and aponeurosis may be closed by interrupted sutures, C silk on a fistula needle, heavy No. 7. The superficial layer in the scalp may be closed by a continuous lock stitch, or interrupted, using full curved No. 14 needle. The suture to be used may be dermal, fine No. 0000, or silk, or fine stainless steel No. 35 to 40.

Scalp laceration. Double layer of sutures should be employed as described on p 28, when the skull is open and cerebrospinal fluid collection is possible. For simple laceration this is also best but a single layer closure may be used (Fig. 3B). Fine steel (No. 35-40) serves very well, especially when infection is likely. *Steel suture* material, such as that prepared by George Pilling & Son of Philadelphia, is less irritating to tissues than silk and rarely acts as a foreign body, even in the presence of infection. Silk or dermal suture may also be used.

Laminectomy. The dura is closed with fine silk as above. The muscle is best closed with steel wire, No. 30–32, using a large curved or a Reverdin needle as made by Collin, Paris. The fascia is closed similarly. To fasten bone grafts in place for fusion No. 28 steel wire has been used.

Nerve suture. To anastomose the divided nerve segments, it is best to split fine silk into three strands. Mount one strand on Carrell's arterial needles, either straight or curved

(20 m/m). Artery suture may also be purchased mounted on needles and sterilized ready for use (Davis and Geck No. 6-0).

Sterilization. Horsley's bone wax requires special precaution. It may be sterilized by heating to 200° centigrade. Cool and pour into small sterile jars. The jars are then covered and wrapped and autoclaved for one hour at 250° Fahrenheit and 20 pounds pressure.

Silk for sutures is wound on cards which are then wrapped in linen and autoclaved for 20 minutes at 250° Fahrenheit and 20 pounds pressure. This does not decrease the tensile strength. The sutures may be waxed with Horsely's bone wax if care is taken not to leave gross wax on the thread. Silk can, of course, be boiled although there is danger of decreasing its strength.

Instruments and Apparatus

No pretense of making a complete list of instruments for neurosurgery is made. The following notes are incidental only, and where makers are mentioned this is for identification only. Reference may be made to Figs. 22 and 23.

Artery forceps in large supply are essential with pointed delicate tips, such as the Cushing artery clamps, straight and curved (Cairns) Lewis Bros., London (Fig. 4).

Thumb forceps with delicate pointed tips are essential for intracranial work and nerve anastomosis, such as the jeweller's forceps (Penfield) Lewis Bros., London.

Suction apparatus of some sort is essential for intracranial work, preferably run by a motor but even that attached to a water tap is helpful. The sucker of metal or glass should have a hole in its side that may be closed by the operator's thumb to vary the intensity of suction. It is well to have different sizes—J. F. Hartz & Co., Toronto.

Cushing's silver clip apparatus, for applying clips to intracranial and intraspinal vessels is absolutely essential. The large clips work best upon the dura, the small clips within the brain (McKenzie) J. F. Hartz & Co., Toronto.

Electric cautery is a great help for haemostasis in intracranial and intraspinal work. It is not necessary to use the instrument for cutting but only for coagulation.

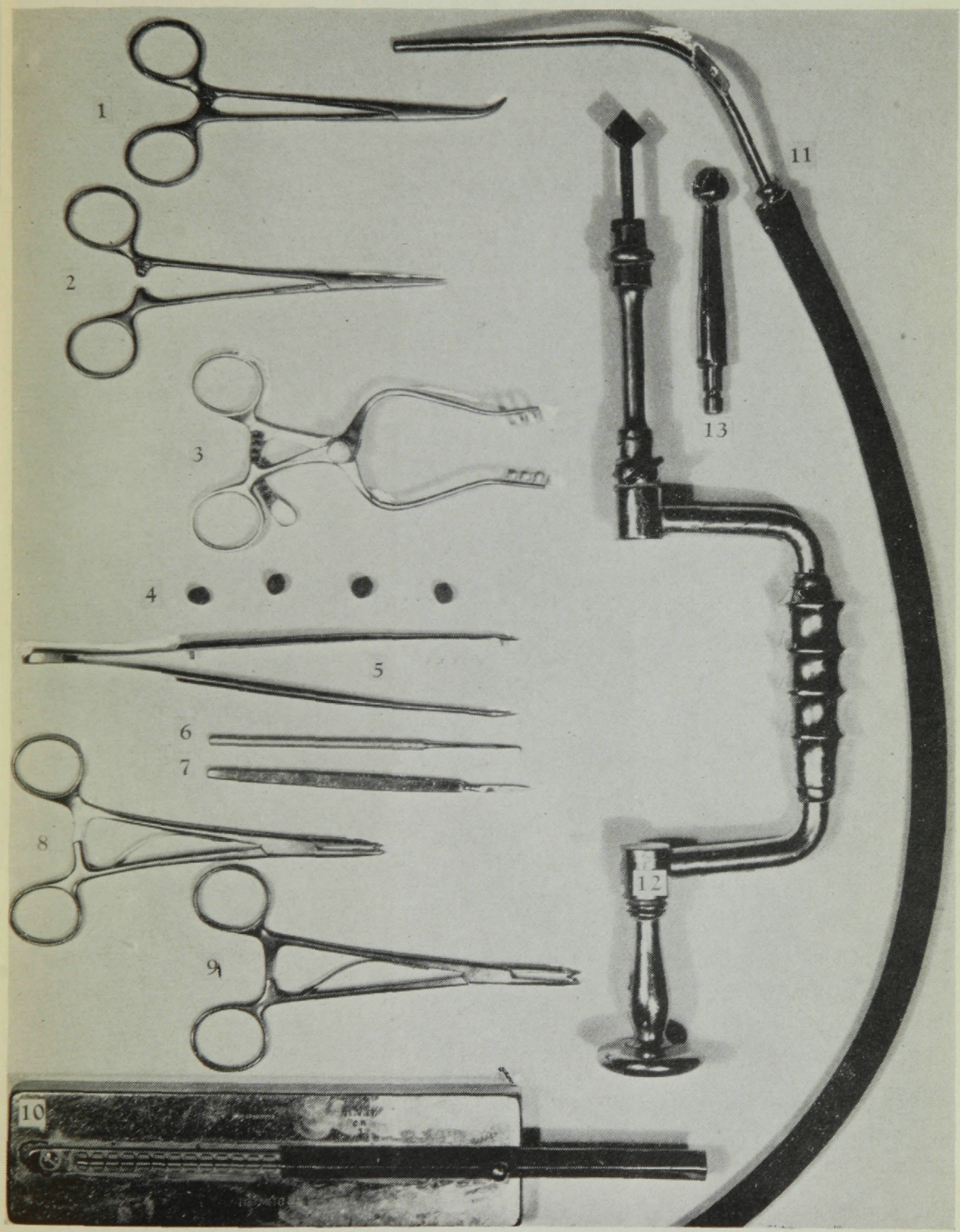


FIGURE 22

Instruments of use in simple craniotomy. 1—curved artery clamp for galea. 2—straight artery clamp for same. 3—automatic scalp retractor. 4—Horsley's bone wax. 5—jeweller's thumb forceps. 6—dural hook. 7—small scalpel to open dura. 8—Cushing holder for small silver clips. 9—same for large clips. 10—silver clip magazine. 11—sucker. 12—skull perforator. 13—burr.

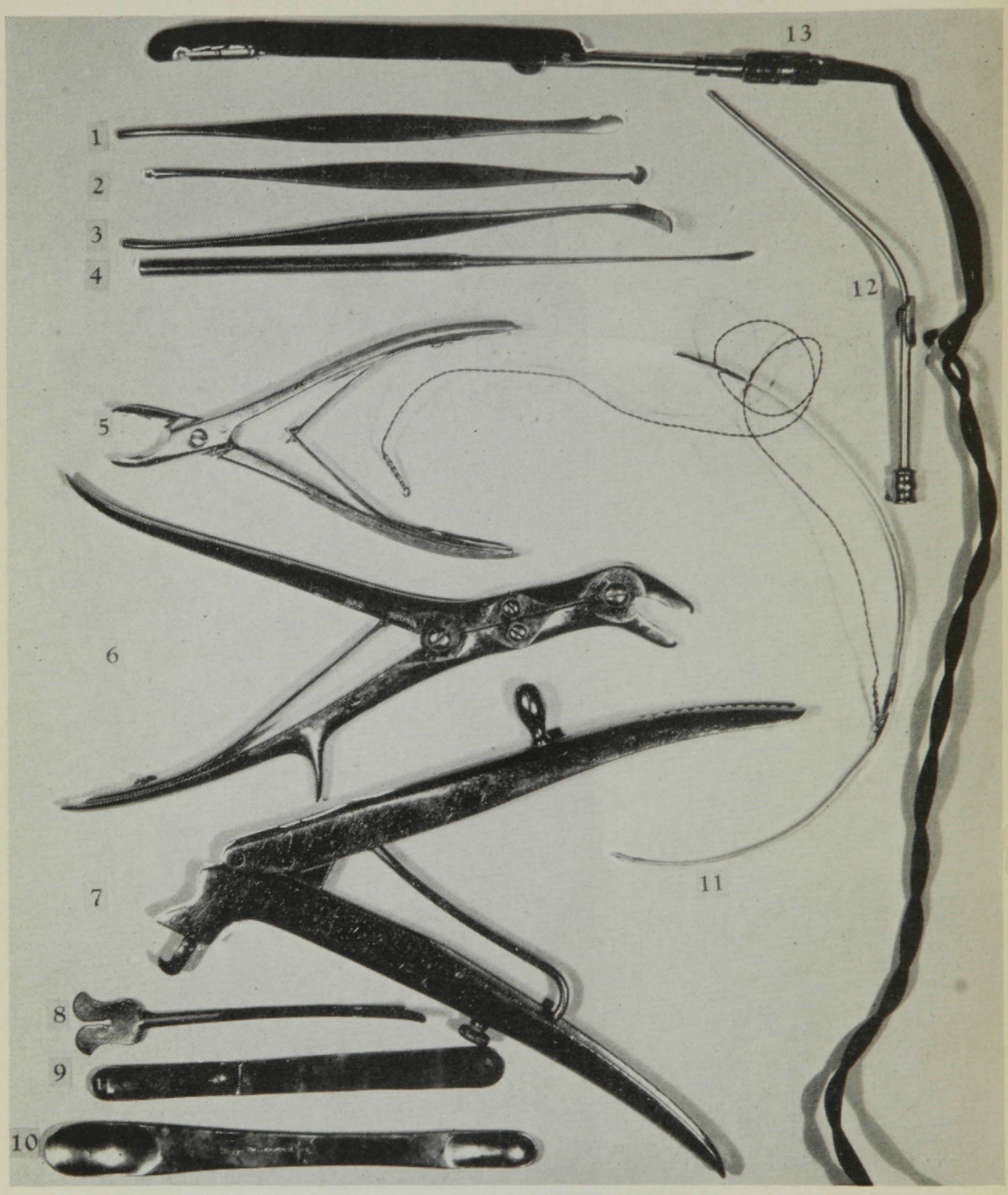


FIGURE 23

Additional instruments for larger craniotomy and cerebral débridement. 1, 2, 3 and 4—dissectors. 5—"mastoid" rongeurs. 6—Stillé rongeurs. 7—channel cutting skull forceps. 8—groove director. 9—Olivecrona dural protector. 10—Cushing brain "spoon." 11—Gigli saw guide with saw attached. 12—small sucker for removal of lacerated brain. 13—Frazier's lighted retractor.

Gigli saws are often defective. When obtainable, those from Stillé are of good quality. A satisfactory Gigli saw guide of the Stillé type is made by Lewis Brothers, London.

Burrs, trephines and rongeurs are made in great variety and of good quality. These are selected according to personal preference. The Stillé double action rongeur is satisfactory for almost all purposes.

Dissectors are most important to separate dura from bone and for innumerable purposes. A set is essential, such as 1, 2, 3 and 4 (Penfield) J. F. Hartz & Co., Toronto.

Frazier's lighted retractor, which bears a small electric bulb and is run by battery is most useful in the pursuit of foreign bodies within the brain or in cerebral débridement (Pilling & Son, Philadelphia); and for the same purpose Cushing brain spoons and the dural protector of Olivecrona made by Stillé.

Automatic scalp retractors, such as the Cone-Travers retractor made by Down Brothers, London, is most useful for the scalp. For laminectomies, the large automatic muscle retractor of Adson saves much time and trouble.

Tidal drainage tubes are modified glass distributors as used for Carrel-Dakin's irrigation, except that one of the three parallel branching tubes has a small bore (p 83). The bore may be reduced by heating.

2. EXAMINATION FORMS

In the interest of saving time and providing for standardized initial records it is suggested that forms such as are given in the following tables should be placed in the hands of all medical officers who may be responsible for examination of patients suffering from injuries of the nervous system.

INITIAL MINIMUM RECORD—CASE OF HEAD INJURY

No.....Name.....Rank.....Unit.....

Examiner.....Date.....Time of accident.....

Type of accident.....

.....Site of blow.....

Lacerations ?.....

Initial unconsciousness ?.....Duration.....

Secondary unconsciousness ?.....

Describe patient: in deep coma, in stupor with movement, drowsy, disoriented, alert, etc.....

.....

Complaints.....

Can patient walk ?.....Talk ?.....

Paralysed ?.....

Pulse rate.....Pulse Character.....

Pupils, size, R..... L..... Reaction to light.....

Respiratory rate.....Respiratory rhythm.....

Bleeding ? Nose.....Ears.....Scalp.....

Was cranial fracture seen ?.....

Notes and treatment:.....

.....

.....

.....

TABLE I.

For discussion see p. 56

LUMBAR MANOMETRIC TEST

NAME _____ DATE _____

OPERATOR _____

CUFF PRESSURE	INITIAL C. S. F. PRESSURE	AFTER 10 SEC. COMPRESSION	PRESSURE AFTER RELEASE				
			5 SEC	10 SEC	20 SEC	30 SEC.	40 SEC.
10 m.m.							
20 m.m.							
40 m.m.							
60 m.m.							
3 Coughs							
Straining							

PRESSURE AFTER WITHDRAWAL OF_____C.C._____

COLOR_____CELL COUNT_____PANDY_____

TOTAL PROTEINS_____

CONCLUSION

TABLE II

Chart to be filled in by examiner when determining presence or absence of spinal block, see page 77.

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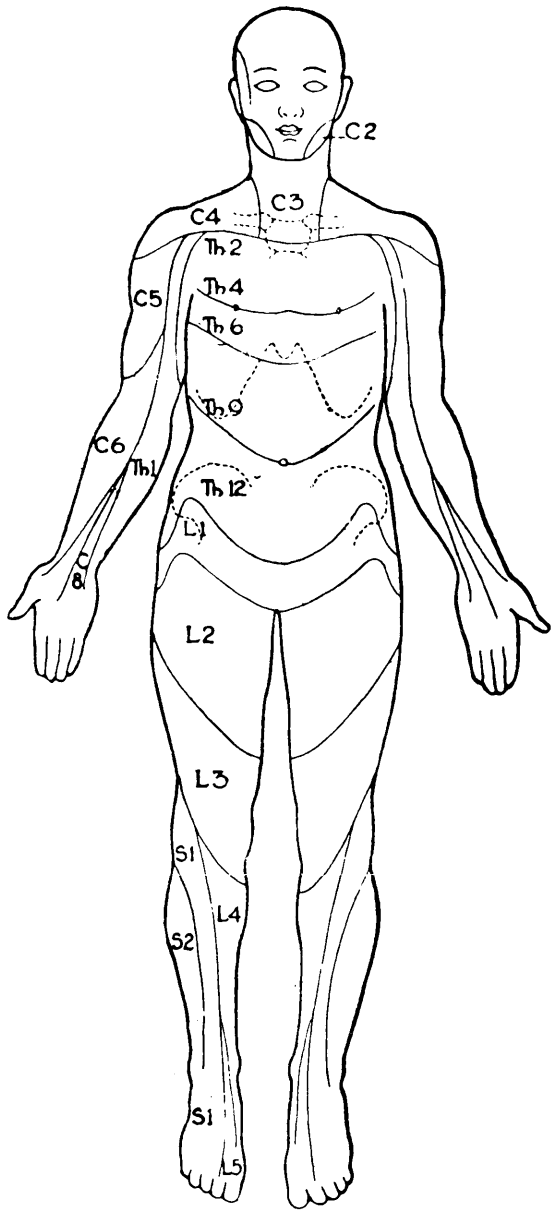
CHART - MINIMAL NEUROLOGICAL EXAMINATION

No._____ Name_____ Rank_____ Unit_____

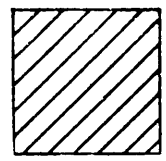
Examiner_____ Date_____

Type of sensation examined: Pain_____ Temperature_____ Touch_____

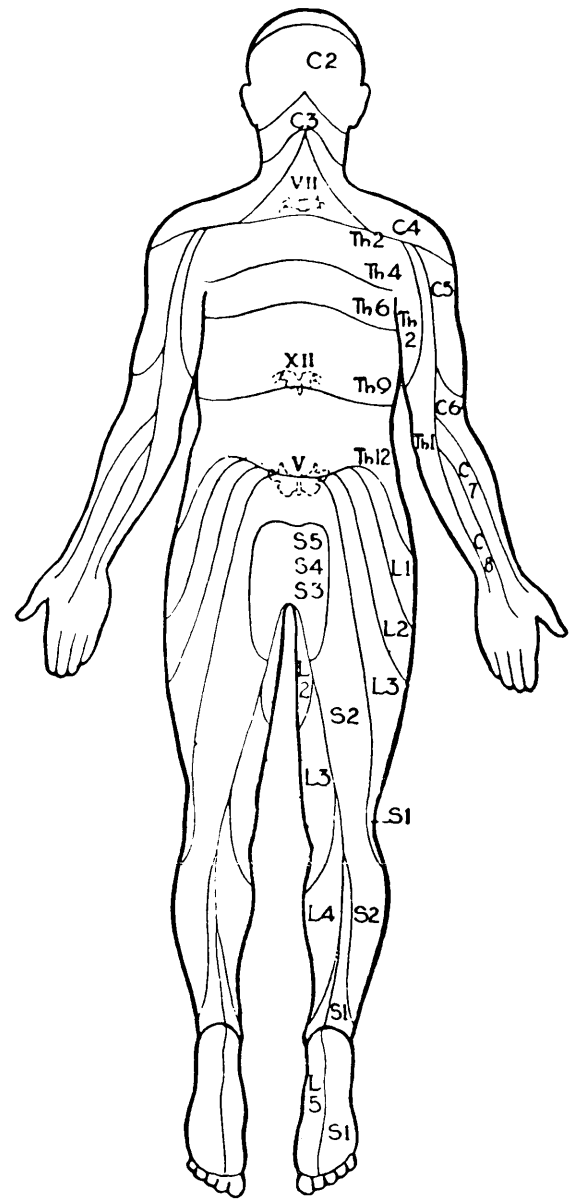
Joint-position sensation_____



To indicate decrease of sensation



To indicate loss of sensation



	Left	Right
Strength of biceps (N,W,0)		
" " triceps (N,W,0)		
" " dorsiflexion of foot (N,W,0)		
" " plantar flexion of foot (N,W,0)		
Deep reflex, biceps (0,1,2,3,4)		
" " triceps (0,1,2,3,4)		
" " patellar tendon (0,1,2,3,4)		
" " Achilles tendon (0,1,2,3,4)		
Superficial reflex, abdominal (N or 0)		
" " cremasteric (N or 0)		
" " plantar (↓flex. ↑exten.)		

Bladder control (N,0)_____

Rectal control (N,0)_____

Pupils: Size, Rt._____ Lt._____ Reaction, Rt._____ Lt._____

Indicate graphically external injuries.

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