THE PHYSIOLOGICAL PATHOLOGY OF GUNSHOT WOUNDS OF THE HEAD.

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Several valuable papers have appeared recording experiences with gunshot wounds of the head. It seems to me, however, that insufficient attention has been paid to the physiological aspects of the subject and to the importance of sepsis as a complicating factor. In analyzing the clinical histories of the present series of cases, and weighing up the causes of death and also of recovery, two facts stand out—the pre-eminent importance (1) of anatomical injury, and (2) of bacterial invasion, as rebels against the normal physiological processes of the cranial contents. In 1915 Trotter reaffirmed the principles of the intracranial circulation and the effects of the cerebral anæmia which is brought about by rises in intracranial tension. A consideration of my own cases has been carried out on parallel lines. Gunshot wounds of the head are generally fatal, not from toxæmia, but from mechanical interference with the bulbar circulation. The bulbar anæmia so produced is the result of a rise of intracranial tension above the normal, and is brought about in one of three ways: (1) By anatomical injury, including intradural hæmorrhage (subdural, subarachnoid, cerebral, and intraventricular) and traumatic œdema of brain tissue; (2) By infection, with the swelling of tissues and the exudates inseparable from it; (3) By a combination of the two foregoing entities—the common method.

The following pages are an attempt to give these various factors their relative values. The task is rendered more difficult by the absence of published experimental work upon the effects of septic processes within the skull and the methods employed by the intracranial contents in defence.

Anatomical injury, primary tissue destruction, is of paramount importance in the case of the brain, because of the physiological processes carried out by it, and on which that anatomical structure depends. To say that nowhere else in the body can death be so certainly and instantaneously produced as by interference with the vital physiological workings of the brain, is to make a generalization which needs but little modification. Microbic infection plays a double rôle; it not only upsets the general bodily functions by toxæmia, but, more important, it occasions also a rise of intracranial pressure. Owing to the subdivision of the cranial chamber into incomplete compartments, the rise of pressure is not so rapidly fatal as would otherwise be the case, as Hill and Horsley and Spencer have shown. It is a truism of pathology that all organs swell when infected, and this is as true of the brain as of any other organ. But it is to the disadvantage of the cerebrum that nowhere can increase of size be so ill tolerated as within the rigid box of the skull. The inelastic cranium admits of but slight variations in the size of its contents, and this at the expense of the cerebrospinal fluid and circulating blood. Those who have seen the swelling of arms and legs from infected war wounds will realize how fatal such swelling must be within the confined cavity of the skull.

Cerebral injuries are infected with the same organisms as wounds elsewhere. Encephalitis, ventriculitis, and meningitis have been a deadly triad in my series, and have been responsible for most of the fatalities. Which is to say that if the patient survives the initial injury, and is able to compensate for the primary upset of intracranial physiology, the outcome will depend on the severity and extent of infection.

It is on these two factors, then, of anatomical injury and of sepsis, that the expectation of life depends, and these are the fundamental grounds upon which the problem of head wounds must be attacked.

In head wounds with dural penetration we have a category in which the extent and severity of anatomical injury and the physiological disturbances consequent upon it reach a maximum. Added to this is the microbic contamination common to all gunshot wounds, converting only too frequently a comparatively simple case (that is, a fully compensated one) into one of the utmost gravity. It is obvious that the factor of anatomical injury is unavoidable. The factor of sepsis, on the other hand, we have some power to regulate or restrain, and all our efforts must be directed to its elimination, and to the prevention of the disasters which follow in its train.

One is forced to the conclusion that head wounds should be operated upon as early as possible. This series is a base hospital series. As an ideal in war no unoperated cases should be sent to the base at all; but if some must be sent, they should be those without dural penetration.

Material.—The cases on which these observations are based were treated in the head service of No. 14 General Hospital, B.E.F., between July and October, 1918. They number 220, of which 54 were simple scalp wounds, 53 fractures without dural penetration, and 113 fractures with laceration of the dura. Some of them had already been operated upon in casualty clearing stations, and these previously operated cases will be discussed separately (except the scalp wounds).

Table I.—Analysis of the 220 Cases Treated.

Cases	Scalp Wounds	Fractures of Skull, no Dural Injury	Fractures with Dural Penetration
Not previously operated upon ..	34	37	79
Operated upon in C.C.S. ..	20	16	34
Totals ..	54	53	113

Condition on Admission.—As a rule only those cases with a pulse-rate lower than 100 are chosen for distribution in base hospitals. The pulse-rate is probably the best guide for such a selection, as a slow pulse denotes a medullary circulation embarrassed but still compensated against the rise in intracranial tension caused by the wound. The difficulty is to know how far away the limits of that compensation are, and several patients arrived at the base with pulses of 100 or over. The prognosis in these last cases is unpromising, but not hopeless, as several made good recoveries against expectation. A high pulse-rate, with an unconscious, restless, and incontinent patient, is a bad sign. But, again, a certain number who were unconscious, irritable, who talked to themselves, and refused to answer questions, got better. This leads to the question, is it possible to select cases for operation? I think not. I tried to select cases at first, and bitterly regretted it afterwards. The hold these patients have on life is extraordinary, and all should be given the chance which operation affords. It is a great mistake to make an incomplete toilette because a patient's condition at first seems to be desperate. It must be all or nothing. To operate on every case brings many disappointments, but some will make recoveries little short of miraculous. No operation was performed on three cases whose condition on admission was so desperate that they died before anything could be attempted. But there were five others which, in the light of experience, should have had a more complete toilette, notwithstanding their apparent hopelessness. The most noteworthy was a patient with a through-and-through wound of the fronto-parietal area, semi-conscious, with a flaccid hemiplegia. The wound was full of smashed-up bone and brain, with gas bubbles in it. This patient lived for eight weeks instead of the few hours expected. Harvey Cushing's summing up of this subject cannot be bettered: "There is no justification in withholding operation from those in whom a prolongation of life would appear to be undesirable; no tribunal is capable of passing such judgement, and unexpected recoveries with unimpaired mental faculties sometimes follow what appear to be the most extensive cerebral injuries."

I have been able to keep in touch with nearly 80 per cent of these cases. One of my own and two of the C.C.S. cases died in England shortly after their arrival, all apparently after operations for fits. Of the remaining 74 penetrating cases (49 of my own and 25 C.C.S.), all have done well. Two cases, and perhaps a third, have a seriously impaired mentality, but it is remarkable how normal the mental condition now is of some who were most unruly and objectionable, who spent their days in hospital in disturbing others and swearing at all who came near them.

The Influence of the Age of the Wound on Operation Mortality (Dural Penetrations only).—The mortality will be found to rise step by step with the time which has elapsed between the receipt of the wound and the operation. Thus, a mortality which is nil for cases operated shortly after they were hit, rises to nearly 50 per cent when two days have elapsed without treatment. The mortality for early cases is unduly flattering, as at the base one does not get those severely wounded men who are admitted to the C.C.S. only to die shortly afterwards. Nevertheless the death-rate in cases where operation has been delayed beyond the first twenty-four or thirty-six hours is definitely higher than it is amongst those on whom an early toilette has been possible. It seems certain, therefore, that interference should be early, as it is only thus that sepsis can be prevented from obtaining a secure foothold. The curve of mortality does not rise steadily, and there is still some hope if more than three or four days have elapsed before operation can be undertaken. But to wait for meningeal adhesions to form (which probably occurs very early anyway) is unwise. It is only just to recall that the much criticized advice of Sargent and Holmes to delay operation had reference to the conditions prevailing in 1916, when special units were few, and when it was not possible to keep the cases immobilized in the C.C.S. Operated head cases do very badly when moved early.

The figures given have no reference to the day on which the patient died, but simply to the effect on mortality of the age of the wound when operated on. In nearly all cases operation was performed shortly after admission, and the varying number of days which had passed between wounding and operation depended not on the surgeon's choice but on the time occupied in transport to the base.

Table II.—Influence of Age of Wound on Operation Mortality (Dural Penetration only).

Time between Wound and Operation	Cases	Mortality
Same day as wounded ..	4	No deaths
Next day after wounding* ..	23	6 deaths (23 per cent)
Second day after wounding ..	25	12 deaths (48 per cent)
Third day after wounding ..	10	3 deaths (30 per cent)
Four and more days after wounding	9	4 deaths (44 per cent)

* 2 further cases died shortly after admission without operation.

Length of Stay in Hospital in France.—It is of vital importance that head cases should be kept at rest for a long time, as they do not stand transport well. These patients were kept under observation in France not less than four weeks, and often more; in fact the average duration of stay was thirty-four days. Some were retained as long as eight and nine weeks, the determining factor being the existence of sepsis and the accompanying unhealed wound in the form of a cerebral fungus; no case was sent across with a large fungus after July, 1918; all such cases were kept till intracranial conditions had righted themselves and the fungus was reduced and healing. The vast majority of cases were healed, and the ideal is to keep all till consolidation has occurred.

Pre-operative Investigation. — The importance of a comprehensive pre-operative examination cannot be overestimated. It should include a short history (when this can be obtained) of how the wound was caused, and its immediate effects; a rapid but complete neurological investigation; and radiographs of the head. The value of stereoscopic *x* rays

is now firmly established, and certainly no operation should be undertaken without them. Nor should the radiographer and the surgeon rest content with an examination for missiles only. Indriven bone fragments should be carefully counted, their depth measured and direction noted. A rough estimate should be arrived at as to ventricular involvement, as implication of the lateral ventricles either primarily or secondarily by extension of inflammation is of prime importance, as will be shown later. Complementary to the *x* ray is the pre-operative neurological examination. For purposes of treatment each is incomplete without the other, for the radiograph shows us what has happened to the bone, whilst the clinical investigation tells us with some degree of accuracy what has happened to the brain. Armed with information from both these sources, one is able to form a clear picture of the nature of the intracranial disturbance, and plan the operation accordingly.

Sometimes additional knowledge can be gained from the character of the injury to the steel helmet. Unfortunately few were seen, most being lost or taken away in transit. In conjunction with the site and nature of the scalp wound, the injury to the helmet often gives valuable information as to the nature of the force of impact and the direction in which it has acted. It would have been well had the steel helmets been sent down with the patients and accompanied them to England.

Types of Wounds.—As we pass through the various types of gunshot wounds, from the less to the more severe, we shall see two outstanding features: first, the anatomical extent of the injury and the accompanying neurological signs gradually increasing in severity; secondly, the havoc wrought by sepsis when it obtains access to the meningeal spaces. As regards the former, even in scalp wounds, indications of intracranial disturbance are rarely lacking; and when the bone gives way beneath the violence of impact, these evidences are more constant and unequivocal. As regards infection, we shall see that when the dura is intact or the intradural damage is very limited, no worse result usually follows than a prolongation of the time necessary for the healing of the wound; whilst in the penetrating cases sepsis leads to a train of evils.

TABLE OF RESULTS.

Group I.—Wounds of scalp with intact cranium and dura.—Cerebral concussion in 50 per cent, cerebral contusion in 43 per cent. 54 *cases, no deaths.*

Group II.—Wounds of scalp with fracture of calvarium, dura intact.—Cerebral contusion the rule. 37 *cases, no deaths.*

Group III.—Fractures of calvarium with puncture of dura: (*a*) By bone; (*b*) By missile.—Cerebral contusion inevitable, positive neurological signs. Brain substance coughed out or aspirated in 9 cases. Fungus cerebri in 2. (*a*) 9 *cases, no deaths.* (*b*) 2 *cases, no death.*

Group IV.—Fracture of calvarium with indriving of bone fragments into brain.—Severe local contusion. Fungus cerebri in 6 recoveries and 7 fatal cases. 24 *cases,* 10 *deaths* (41·4 *per cent*).

Group V.—Penetration of skull and brain by missile with indriving of bone fragments.—Severe local contusion, brain matter often presenting in wound. Fungus cerebri in 8 recoveries and 6 of the deaths. 23 *cases,* 8 *deaths* (34·8 *per cent*).

Group VI.—Penetration of ventricles by missile or bone fragments.—Fungus cerebri in 2 cases. 4 *cases,* 3 *deaths* (75 *per cent*).

Group VII.—Penetrating wounds involving face and cranial cavity. (*a*) Cranio-facial; (*b*) Petro-cranial.—Brain usually presenting in wound. Fungus cerebri in all but one. (*a*) 11 *cases,* 3 *deaths* (27·3 *per cent*). (*b*) 2 *cases,* 2 *deaths* (100 *per cent*).

Group VIII.—Through-and-through wounds of cranial chamber.—Fungus cerebri formed in all the fatal cases. 4 *cases,* 3 *deaths* (75 *per cent*).*

* It must be remarked that four of the cranio-facial wounds completely traversed the cerebral cavity; three of these recovered.

In the table of results on the previous page, Harvey Cushing's classification of these cases has been followed, with slight modification, and for the sake of clarity is appended now rather than later. No case with an intact dura died, which is to pay that membrane well-nigh as high a compliment as could be conceived. As might be expected, sepsis when superadded to severe injury takes a large toll of life; thus the mortality in *Groups IV* to *VIII* is out of all proportion to that in *Groups I* to *III.*

Summary.—Total number of cases, 170. Wounds without dural penetration, 91, no deaths. Wounds with dural penetration, 79, 29 deaths (37·6 per cent).

This result tallies very closely indeed with that obtained by Horrax in a neighbouring hospital.

Group I.—SCALP WOUNDS IN WHICH THERE WAS NO INJURY TO THE BONE.

Of scalp wounds pure and simple, I have notes on a series of 54 unselected cases. More than this number were actually admitted, but not all were recorded owing to pressure of work. In none of these was there any injury to the cranium. A bony injury *per se* is of small consequence; the important thing is the brain injury, and the frequency with which this was present will no doubt surprise many. The detailed neurological findings in these scalp cases are being published elsewhere. A few remarks, however, must be made upon them here.

Owing to the protection afforded by the steel helmet, very severe blows are frequently sustained without the bone giving way. This is particularly true of tangential wounds, in which the calvarium yields beneath the force of the impact without actual solution of continuity. This inward bending of an arc of the skull can never have been great, because the vitreous internal table remained intact in all. Nevertheless the brain sustained a definite injury in the vast majority of the cases; in fact only 5 of the whole 54 showed no neurological signs whatever. An analysis of the symptoms and signs presented shows that there are two main types of brain injury beneath an uninjured skull; the one is a general concussional upset, the other is a definite local contusion. It is very difficult to diagnose a cortical contusion when the wound overlies a silent area, so that it is probable some of the examples classed as concussional were in reality contusions of silent areas. This view is supported by a closer investigation of the wounds which overlay non-silent areas. For of such wounds (motor and sensory) there were 39, of which 17 showed signs of local contusion, or 43·5 per cent. It is only fair to assume that the same percentage holds good of the 15 wounds which overlay silent areas, although in none of these cases could positive localizing signs be detected. It is improbable that civilian scalp wounds will show so large a number of cerebral contusions, as the force producing them cannot compare with that of a projectile tearing its way through a hardened steel helmet. And in war wounds of the scalp, although the calvarium has not been actually broken, most of the men have suffered a very severe blow, sufficient to render them unconscious for a short time, and often breaking the chin-strap and tearing the helmet off the head.

Of the 17 local contusions, 11 involved the motor cortex, and 4 the visual area, whilst 2 affected both the motor area and that for general sensibility (parietal field). In 3 the nervous signs were so definite that it seemed wise to remove a bone disc, and in 2 of these an extradural hæmorrhage was found, in the other nothing. Three cases presented Jacksonian fits, which were very severe and long-continued in 2. Contralateral injury was present in 4 cases. The injuries to the visual area are a rich field for investigation of local contusion, as it is so easy here to assess the damage and follow the stages of recovery. It may be noted that the contusional damage tends to clear up rapidly, and after a week or ten days little or nothing remains of what was a definite neurological entity. Fuller particulars will be found elsewhere.

The following case is a good example of severe intracranial disturbance beneath an intact skull.

Case 1.—**Scalp wound over right motor area. Cranium intact. Extradural clot. Jacksonian epilepsy. Trephined. Recovery.**

Pte. T. P. (series No. 15). Aug. 27, 1918: Bullet wound over right parietal bone, 4 inches long, crosses lower end of Rolando. Two holes in steel helmet 6 inches apart.

Aug. 29: Admitted No. 14 General Hospital. Unconscious for a few minutes after being hit. Headache +, low frontal. Vomit −, nausea +. Palsy: Weakness left side of face, left hand-grip weak, finger movements retained but slow, subjectively hand is numb and stiff. Elbow and shoulder practically unaffected. Hand was all right when first hit, but became weaker as he walked to aid post. Leg normal. No aphasia. All tendon-jerks exaggerated. Plantar reflexes strongly flexor. Pulse 60. Mentally clear, slightly drowsy. *X*-ray, no fracture detected.

Aug. 29: *Operation*, novocain-adrenalin anæsthesia. Wound excised *en masse*, bone bared, slight bruising in diploe, no fracture. Closed with flavine cigarette drain.

Aug. 31: Neurologically, no improvement. At 6.0 p.m. next day patient had a severe Jacksonian seizure lasting 35 minutes. Whole of left side involved, but especially face and hand. *Operation*, novocain-adrenalin. Wound re-opened, 1-inch bone disc removed, extradural clot found some 6·0 mm. thick. Bone nibbled away leaving an oval defect 3 inches long. Clot sponged away. Wound closed with flavine cigarette drain. After operation patient was found to have the common post-epileptic paralysis of left arm and face (Todd's paralysis).

Sept. 2: Arm recovering, Todd's paralysis passing off. Elbow and shoulder movements normal.

Sept. 9: No further fits, hand now quite recovered, fine movements can be executed firmly and rapidly, but not yet quite normal. Subjectively, hand is still a little numb. Face recovered.

Sept. 17: Hand is now normal. Face, arm, and leg normal. No headache, giddiness, or nausea. Tendon-jerks slightly exaggerated. General condition excellent. Pulse runs 64, 72, 66, 64, to 98.

Oct. 13: Seen in England. Up and about, no headache, no giddiness, arm and face normal.

In another case in which the neurological signs were no less definite, operation revealed no extradural hæmorrhage and no splintering of the inner table. It is certain that the skull may come off scathless and yet the underlying brain suffer injury to the point of disorganization.

Group II.—CASES IN WHICH THE CALVARIUM IS FRACTURED BUT THE DURA IS NOT PENETRATED.

These cases form the link between the simple scalp wounds and those with dural penetration. They form an interesting series, exhibiting severer cerebral contusion, and, when infected, greater chronicity of healing than those in *Group I*, owing to bone infection, being in fact compound fractures with osteomyelitis of the calvarium. The series comprises 37 cases, all of which recovered. The chief findings will be discussed beneath the headings bone injury, dural injury, and cerebral injury.

The Bone Injury.—In gunshot wounds one very rarely sees the typical 'pond' depressed fracture of civilian practice. In the present series it was met with on only three occasions. The common fracture is that produced by a tangential blow in which a portion of the outer table is shot away, the inner table and diploe are broken and depressed, and this usually over a somewhat greater area than the external wound. The fragments were often piled upon one another so as to depress the dura, or a fragment might be turned edgeways and act as a strut holding the dura down (*Case* 2). A linear fracture was encountered on 5 occasions, once beneath an intact pericranium, and in 6 cases the bone injury amounted to no more than a groove or 'nick' in the bone. In 2 of these last cases, however, the pre-operative neurological findings warranted the removal of a bone disc, but in neither was anything found. From my own experience I should judge that the brittleness of the internal table has been rather overestimated. Naturally, specimens in which the internal table has given way tend to find their way into museum collections rather than those in which nothing of the kind has occurred. The casual observer is thus apt to be unduly impressed with the fragility of the internal table.

In this class of cases the important things are the degree and nature of the infection of the bone, and the extent of intracranial disturbance. The comminuted bone fragments were cultivated in 13 cases, and the results, for which I am indebted to Captain B. A. J. Peters, are tabulated below.

Table III.—Results of the Cultivation of Comminuted Bone Fragments in 13 Cases.

Case	Staphylococcus	Hæmolytic Streptococcus	Non-hæmolytic Streptococcus	B. Sporogenes	B. Welchii
1	+	0	0	0	0
2	+	0	0	+	0
3	0	0	0	+	+
4	+	0	0	0	+
5	+	0	+	0	0
6	+	+	0	0	0
7	+	0	0	0	0
8	0	0	0	0	0
9	+	0	0	0	+
10	0	0	0	0	0
11	+	+	0	0	0
12	+	0	0	0	0
13	+	0	0	+	0
13	10	2	1	3	3

The commonest organism was the staphylococcus, but it was usually in combination with the streptococcus or a pathogenic anaerobe. It will be noted that 2 of the 13 cases were sterile. One of these was clinically very clean (a piece of shell embedded and depressing the inner table, and the wound of entrance small and sealed with clot); the other became mildly septic.

It is evident that the bacteriology of head wounds is not materially different from that of wounds elsewhere, and that the picture of sepsis will be similar, though mirrored in another glass.

The Injury to the Dura.—The dura was usually bruised and dark in colour beneath the central area of the fracture. Sometimes there was a slight superficial laceration; but organisms did not penetrate in any of the cases. Rarely, a local leptomeningitis may have existed, but it never gave positive signs. A dark granular blood-clot adherent to the dura mater was frequently present, but in only four instances was there a vascular injury of moment; three times this took the shape of a laceration of the superior longitudinal sinus, and once of a branch of the middle meningeal artery. None of these gave any trouble, and were easily controlled by muscle or fascial grafts.

The Cerebral Injury.—It will be remembered that localized cerebral contusion was computed at 43 per cent of the scalp wounds, and concussional changes in almost 50 per cent more. In *Group II*, now under consideration, we find contusion in no less than 25 cases, roughly 80 per cent; and not only was it more common, but it was more severe, more unequivocal in its signs, and more protracted in its duration. This is of course due to the yielding of the bone beneath the impact of the projectile, allowing the force to exert itself freely and unhindered upon the hemisphere.

Pulping of a small area of brain tissue must occur in the majority of these cases, for the damage is in all respects more severe than in the scalp wounds.

On the clinical side, there were 11 lesions of the motor and 5 of the visual area, whilst the character of the wound and the severity of the symptoms left little doubt that contusion of a silent area had occurred in a further 8 cases. The difficulty of diagnosing a lesion of a silent area, of the frontal lobe for instance, has already been mentioned; but when the bone has been shattered and depressed and the dura extensively bruised, it seems only reasonable to conclude that the cortex immediately subjacent has not escaped hurt.

The part played by cerebral anæmia in the production of palsies was well typified by one case. It is interesting also as it is a good example of anæsthesia of cortical origin. The hand became numb immediately after receipt of the wound, but power was not impaired. At operation a piece of bone was found depressed, and the moment it was removed the patient remarked that the hand felt less numb. This immediate improve-

ment could only have been due to the relief of vascular stasis, and draws attention to the rôle of cortical anæmia in the production of disabilities of the limb. The observation is of further interest in that it could not have been made had the patient been under the influence of a general anæsthetic (for other examples of this fact, see *Cases* 3, 4, 5, 11).

Case 2.—**Gunshot wound of right parietal area, post-central, overlying approximately the sulcus parietalis horizontalis. Anæsthesia of the hand, relieved by decompression.**

Pte. A. B.—Shell wound of right parietal field, Aug. 31, 1918. Large dent in steel helmet with a hole at the bottom about 4 cm. across. Admitted to No. 14 General Hospital, Sept. 9. Never unconscious; stunned; headache generalized. No vomiting; nausea on sitting up. Palsies: Slight weakness of left side of face, hand-grip strong, arm and leg normal. Hand feels to him to be absolutely numb, and he cannot distinguish any objects placed in it; in fact he could not, to use his own expression, "tell the difference between a pen and a billiard ball." Hand is doubtfully anæsthetic to light touch, but he feels pin-prick.

Operation, Sept. 2.—Novocain-adrenalin anæsthesia. Wound excised, 1-inch disc and three large depressed fragments removed. These were piled up, and one turned edgeways held the dura down fully half an inch. On removal of this piece patient exclaimed that hand at once felt less numb. Hot saline wash, flavine. Closed with cigarette drain.

Sept. 3: Astereognosis less marked twelve hours after operation.

Sept. 6: Now easily distinguishes objects placed in his hand, but it is still subjectively numb.

Sept. 9: Sutures removed. Slight evening headache.

Sept. 17: Left hand has recovered; at times he thinks it is less sensitive than the right (presumably a case of Head's "introspective anæsthesia").

Oct. 13: Seen in England, perfectly normal. Is playing the organ.

I have notes of another case in which a similar immediate relief of symptoms occurred on removal of a piece of depressed bone, and cannot do better than insert a short account of the case here. It shows well the location of the motor centres for the foot higher on the cortex than those for the knee and hip. It shows, too, how these contusional changes clear to a certain degree within a few hours.

Case 3.—**Gunshot wound of left parietal area. Transient hemiplegia, resolving after decompression into a palsy of foot only (relief of cortical anæmia). Recovery.**

Pte. F. R.—Shell wound overlying the upper end of the left motor cortex near the middle line, Sept. 26, 1918. Operated same day in No. 33 C.C.S. Admitted No. 14 General Hospital Sept. 30. Says that when he was hit he fell immediately on his right side, which was absolutely powerless. He managed to crawl out of a shell hole, but it took him all day to go one kilometre; after twelve hours the hand had somewhat improved, but was weak. He states that the hand improved immediately when the surgeon in the C.C.S. pulled a piece of bone up. His only palsy now is of the ankle and toes; hip and knee are normal. He has right foot-drop. The knee-jerks on the right side are greatly exaggerated and there is true ankle-clonus. Plantar reflex flexor. Eighteen days later there was only slight improvement in the foot, but his general condition was excellent.

Injuries of the Visual Area. — The results of injuries to the area striata have been fully worked out by Colonels Gordon Holmes and Lister, and the present series has nothing to add to their description. One point may be noted, as it is one which has not, so far as I am aware, been hitherto recorded, and that is pain behind the eyes on manipulation of the dura mater covering the occipital lobes. What the significance of this is is not clear, but the fact remains.

The two following cases are examples of referred pain from the occipital dura; a third will be found amongst the penetrating wounds (*Case* 11).

Case 4.—C.-S.-M. W. D.—Shell wound immediately above inion, transversely across middle line. Central vision hazy, right field of vision contracted. At operation a depressed fracture was found; the dura was intact. Both dura-clad occipital lobes were exposed, and when these were sponged with hot saline the patient complained of pain in the eyes, particularly the left. Seven days later vision had improved, but was still defective.

Case 5 (For this case I am indebted to my colleague, Captain H. R. Unwin, R.A.M.C.).—Lieut. A. B.—Gunshot wound of occiput. Decompressed under novocain. On touching the dura with forceps or on sponging it, the patient complained of pain in the eyes.

Lachrymation has also been noted in some of the occipital wounds, and on occasion the eyeballs were distinctly tender on pressure.

Groups III to VIII.—**CASES IN WHICH THE DURA MATER WAS PENETRATED.**

We now arrive at the third and most important series of cases, in which the dura mater has been torn, the meningeal spaces opened up to infection, and the brain lacerated in various degrees. These are the cases which are the greatest trial to the surgeon, and the toll of life which they take is considerable. Of a total of 79 cases belonging to this category which came down to the base unoperated, 50 recovered and 29 died, a mortality of 36·7 per cent.

Anatomical Injury and Infection.—It is a well-established fact that infratentorial are more serious than supratentorial lesions, owing to the direct action on the medulla of the injury, or the swelling which it occasions. It is probable that the mortality of the former is very high indeed, that many are killed outright, some die in the forward areas, and only a few reach the base. In the present series there were 6 infratentorial lesions, and only 2 of these died (33·3 per cent). But it must be said of the 4 which recovered that only one presented a severe anatomical injury; the remainder involved no more than the superficies of the cerebellar cortex. The above figures probably give a totally erroneous impression of the true mortality of infratentorial lesions and of their real gravity.

Supratentorial lesions, not being so immediately dangerous to life, are those forming the greater part of base-hospital head material. They have nevertheless a high mortality, for 27 patients with supratentorial wounds died out of 73 (37 per cent); 11 died very rapidly, usually at a speed commensurate with the extent of the anatomical damage to the hemispheres. But death was almost always hastened by sepsis. Some lived ten to fourteen days, and then succumbed to meningitis; whilst others prolonged the struggle for three or four weeks and even for longer periods. Roughly, one can say that the causes of death are three: (1) The early deaths are due to rise of intracranial pressure by blood effusion, to traumatic œdema, or to the primary involvement of vital centres; (2) Those occurring a little later are cases which have survived the foregoing accidents, but in which microbic infection and inflammation turn the scale and raise the intracranial pressure too high; (3) Those who die later die from meningitis, ventriculitis, or abscess, often running stormy courses, but finally succumbing to bulbar anæmia.

Experience with these cases teaches the remarkable ability which the brain displays in dealing with septic processes. After a long fight, with alternating exacerbations and tranquil periods, the cerebrospinal fluid alternately cloudy (but sterile) and clear, patients may recover, or bacteria at last win their way into the cerebrospinal fluid and death ensue.

So far we have discussed the importance of anatomical injury *per se*, and the rôle of sepsis when superimposed on an injury not in itself incompatible with life. But another factor makes injuries of the hemispheres deadly—namely, the presence of the lateral ventricles within them. Ventricular infection has proved a disastrous thing in my own cases, and is, as it were, the 'Black Hand' of hemispheral wounds. The meningeal spaces possess very considerable power of forming adhesions and so limiting the spread of infection. They are able to cope with most bacteria, having least power with the streptococcus. The ependyma of the lateral ventricles has no such adaptability, and allows microbic infection to walk right through into the cerebrospinal fluid, which meets the infection by a lavish outpouring of polymorphonuclears, probably from the choroid plexus.

A great deal of attention has been directed by the surgical anatomist to the cortical representation of the brain to the skull—to surface cerebrocranial topography. The importance of such schemes is beyond dispute. But from the point of view of the operating surgeon, the deeper structures are of more importance, and of these the ventricles stand in a class by themselves. Unfortunately but little has been done in respect of the surface anatomy of the ventricles. All standard works contain at least one diagram of some classical type of cortical topography. How few contain a diagram of the surface relationships of the ventricular system. An idea of the large extent of the ventricles will be gained from *Figs.* 256 and 257, and help one to realize how frequently they must be

involved in penetrating wounds of the head. At their anterior poles they are deeply placed, and a wound of the frontal lobe must extend almost to the middle line before the ventricular cavity is pierced. The long axes of the ventricles separate from one another as they pass backwards, owing to the basal ganglia, and they become more superficial. The descending horns, sweeping forwards round the brain-stem, are also near the cortex. This is best seen in the antero-posterior view (*Fig.* 257), which shows the anterior ends of the descending or temporal horns of the ventricles lying as low as the middle of the orbit, and much outside the anterior horns.

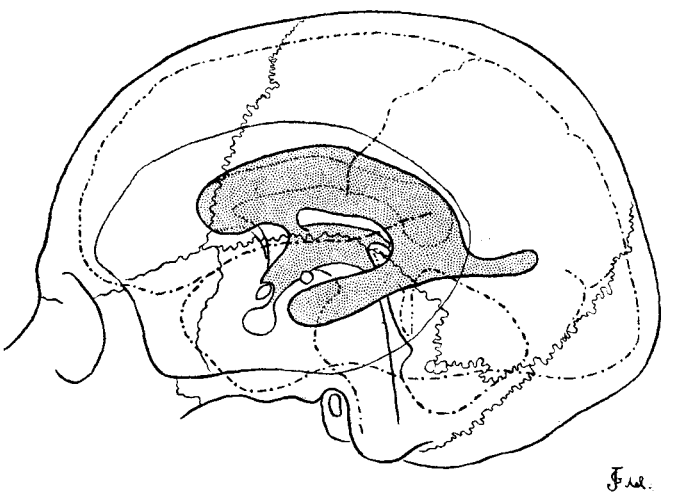

FIG. 256.—Diagram of the ventricular system, side view, modified from Symington.

Whilst ventricular penetration is a very deadly thing, it is not necessary that the ventricles should be opened primarily by the wound for a ventriculitis to be set up. In fact the ventricle has been opened in but few of these cases either by the missile itself or by bone indriven by it. It is more usual for a considerable thickness of the cortex overlying the ventricle to be severely contused and sown with bacteria. The encephalitis so originated spreads inwards, unless an efficient toilette has been performed early, until the wall of the ventricle has been involved and the cerebrospinal fluid contaminated. This has been the cause of many deaths and of many clinical storms in the convalescence of these cases. Sometimes the whole outer wall of the ventricle softens and gives way and the cerebrospinal fluid finds its way to the surface. I am convinced that the leaks of cerebrospinal fluid which one sees develop a few days after the injury do not originate from any lysis of adhesions between the brain and dura. That is to say, the source of the fluid is ventricular, it does not come from the pericerebral meningeal pool. A ventriculitis once established, the cerebrospinal fluid carries the infection by the foramina of Key and Retzius to the base of the brain. In this way the work that the meninges have done in localizing the infection is set at naught, and the whole subdural space becomes infected in this roundabout fashion. At autopsy on such cases one finds an area of encephalitis

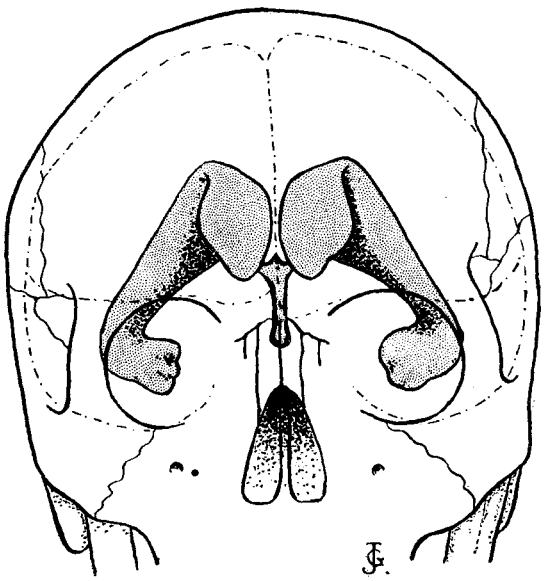

FIG. 257.—Diagram of the ventricular system, front view. Note low level of temporal horns.

up to and involving the wall of the ventricle, the ventricles themselves full of turbid fluid, the interpeduncular space and cisterna magna full of lymph-clot or purulent liquid, and the choroid plexus swollen and injected. Yet the actual cortical wound area is still firmly sequestered by meningeal adhesions. (Cf. Horrax, loc. cit., *Case* 55 and others, also Whitaker, loc. cit.)

Provided that the brain is not called upon to perform an impossible task, provided, that is, that there are no infected bone fragments left lying in the cerebral tissue, and that a delicately-handled toilette has been carried out before infective processes are too firmly established or have pushed too far afield, even ventricular involvement may be recovered from. This has occurred over and over again in the present series, both in my own and the C.C.S. cases. The patient exhibits signs of increased pressure, occipital headache, and rigidity of the neck and limbs, with rise of temperature and pulse-rate. Lumbar puncture is performed and a turbid fluid is recovered under pressure. Investigation of

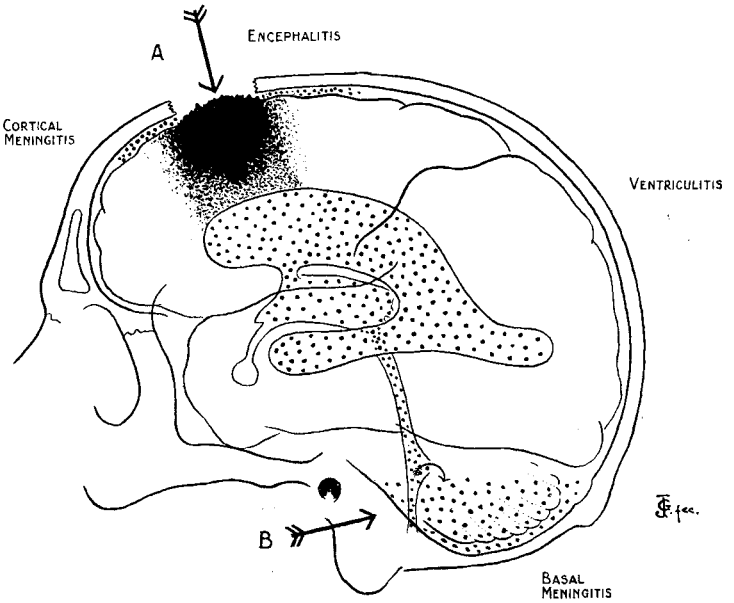

FIG. 258.—Diagram of common route of infection in head wounds. The local injury is sealed off by adhesions. (A) Local meningitis. (B) Basal meningitis, by ventricular infection escaping through the foramina of Magendie and Luschka.

this fluid proves it to be full of polymorphonuclear leucocytes ; but it is sterile on culture, and no organisms are to be found in the films. This is a puzzling finding, but has been met with often. From this point the case may take one of two courses. The patient may recover after one or more recrudescences of the same phenomena, with intervals in which he presents an absolutely or almost clear cerebrospinal fluid. Or he may go downhill, and, after an interval of two or three days, bacteria make their appearance in the puncture fluid and death shortly occurs.

The following cases illustrate this point well. The first patient had received a wound of the left temporal region a week previously, and had been operated upon in a C.C.S. The wound was extremely septic. Two days after admission meningeal symptoms appeared. Lumbar puncture was performed daily, and the varying character of the cerebrospinal fluid is set out below. It will be noted that bacteria did not appear in the fluid till the end.

Case 6.—Operated C.C.S., Oct. 13, 1918. Admitted No. 14 General Hospital, Oct. 20.

Oct. 22 : Meningeal signs declared. Pulse 60, temperature 103°. Lumbar puncture, 30 c.c. clear cerebrospinal fluid withdrawn ; yellow from old blood pigment. Headache relieved.

Oct. 23: Worse; very restless; crying out. Pulse 72, temperature 103·8°. Lumbar puncture, 45 c.c. faintly turbid fluid withdrawn: sterile to films and culture.

Oct. 24: Patient had six fits to-day, involving face only. Pulse 78, temperature 103°. Lumbar puncture, 30 c.c. cloudy cerebrospinal fluid recovered; appears to be a definite meningitic fluid; sterile to films and cultures; numerous polymorphs. Oct. 25: Better to-day. Pulse 72, temperature 100·2°. Lumbar puncture, 40 c.c. fluid withdrawn, perfectly clear. Oct. 26: Very much worse since last night. Lumbar puncture, 40 c.c. very cloudy cerebrospinal fluid removed; numerous polymorphs; on culture it grew a streptococcus. Oct. 27: Died. Pulse ran up from 64 to 112 in four hours as the medullary circulation failed.

Case 7.—Pte. J. P.—Admitted No. 14 General Hospital twelve days after operation in a C.C.S. Dirty wound left parietal area. Cerebral fungus developed five days later; patient irritable, reflexes active. Lumbar puncture, 35 c.c. clear cerebrospinal fluid withdrawn under tension. A week later meningeal signs developed; calling out with pain in head; neck rigidity. Temperature 102·8°, pulse 100. Cerebrospinal fluid leaking from fungus. Lumbar puncture three consecutive days, 40 c.c. withdrawn each time. On each occasion it was cloudy; the first day it was thick yellow; sterile on culture and films every time. Improved by puncture; fungus smaller and cleaner. Ten days later a second meningeal attack occurred, but the cerebrospinal fluid was clear. From this point on patient made a slow but uninterrupted recovery.

A sterile turbid fluid was often seen; it is not until bacteria actually enter it that the prognosis becomes serious. Only one case with a definitely infected cerebrospinal fluid recovered; in another case infection was recovered from, but the patient succumbed to a second invasion. The explanation appears to be this. The spread of infection to the wall of the ventricle results in an outpouring of polymorphonuclear leucocytes into the cerebrospinal fluid. The probable source of these is the choroid plexus, though some may come from the area of encephalitis. The leucocytes may be so numerous as to render it quite turbid. Should the encephalitis be held in check and improve, the leucocytosis passes away, leaving a clear fluid which may again become turbid. This depends on the local conditions in the brain wounds, the battle with the infection swaying this way and that with varying fortunes. When the day is finally lost, bacteria succeed in obtaining a footing in the fluid itself, and the end is not long delayed.

Table IV.—Bacteriology of Cases in which Bone and Brain were Cultivated.

Case	Bone	Brain
1	Staphylococci Streptococci	Staphylococci
2	*B. Welchii* *B. sporogenes*	Staphylococci
3	*B. Welchii* *B. sporogenes*	Staphylococci
4	Staphylococci	Staphylococci *B. Welchii* Coliform bacillus
5	Staphylococci *B. Welchii*	Staphylococci
6	Staphylococci *B. sporogenes*	Staphylococci *B. sporogenes* *B. Welchii*
7	Nil	Staphylococci

It may be urged that the absence of bacteria in the spinal theca is only local evidence, and that infection may be present within the cranium but shut off from the spinal canal. This must sometimes be true; but autopsy failed to show spinal barrier adhesions in these cases.

This brings us to another aspect of the question, namely. With what bacteria has the brain to cope, and what are its capabilities of dealing with infection?

The Nature of the Infection.—We have already referred to the bacteriology of the fractured calvarium whilst dealing with non-penetrating head wounds. It is *a priori* unlikely that the bacteriology of the infected wounds will materially differ from these, but a series of investigations was carried out with a view to establishing the exact nature of the infection. The indriven bone fragments lying in the pulverized tissue were cultivated, as well as portions of the brain substance. The results of these examinations will be found in *Table IV*.

This is a formidable toll of infection, yet all of these seven cases recovered. Both aerobes and anaerobes were almost invariably present, though the latter seem to be commoner in the bone fragments, as if the tissue juices of the brain had considerable power of dealing with them.

Indriven Bone Fragments.—These findings help one to realize the importance in prognosis of indriven portions of bone ; for, whilst one would expect that the infection in the contused brain might be cleared up, it is difficult to see what measures the cerebral tissues could take with an infected comminuted piece of bone separated from its blood-supply. Such fragments, exposed to sepsis, form veritable sequestra, and keep suppurative processes alight in the brain as elsewhere. A comparison can be drawn between those indriven into the brain and those in comminuted fractures of the femur. It is common experience how the latter prolong convalescence in wounds of the thigh. How much greater is their power for evil in the cranium ! It is conceivable that under very favourable conditions such fragments may obtain a new circulation and become organized and fixed to the cerebral tissue, particularly if very small and relatively sterile. This happy result cannot be counted on, although brief reflection will show that it must sometimes happen, for at the present date there are many people living fairly normal lives with indriven fragments organized in their brains. Fortunately I am able to confirm this by an actual observation, although the circumstances which made the experience possible were anything but fortunate. The following case first drew my attention to the importance of the subject.

Case 8.—**Gunshot wound of left frontal area, extending towards central sulcus. Gutter wound with much indriven bone. Ventriculitis, meningitis. Death.** (*Series No.* 65.)

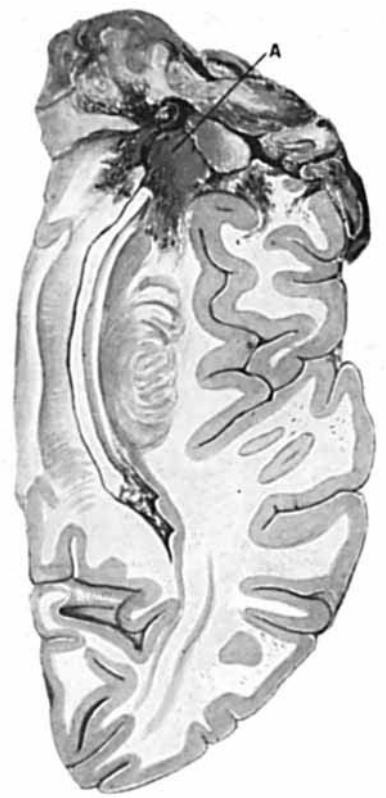

FIG. 259.—G.S.W. frontal pole (A) leading to ventriculitis of frontal horn by direct extension. The ependyma in this specimen was intact originally.

Pte. J. S.—Machine-gun bullet wound 7·5 cm. above nasion, 1 cm. to left of middle line, 10 cm. long, Aug. 10, 1918. Wound just reaches motor area. Admitted No. 14 General Hospital on Aug. 11. Patient semi-conscious ; brain fungus. Some weakness of left hand. Operation same day : decompressed by a visiting surgeon ; incomplete toilette.

Aug. 18 : Cerebral fungus has developed.

Aug. 20 : Patient complains constantly of headache ; looks worse ; meningeal symptoms ; neck and limb rigidity. Temperature 103°. Aug. 21 : Lumbar puncture, turbid white cerebrospinal fluid under pressure. Aug. 22 : Hernia larger. Report on puncture fluid, numerous polymorphonuclears and endothelial cells. Culture, free growth of streptococci.

Aug. 30 : Improved, less headache and rigidity. Lumbar puncture, slightly turbid fluid. On examination, increase of globulin, polymorphonuclears 20 per cent, lymphocytes 80 per cent. Culture, no growth. No bacteria in films.

Sept. 12 : Following last puncture, temperature fell to normal and has remained down with slight elevations only. Hernia receded and clean. Patient seems to have recovered from a streptococcal meningitis.

Sept. 18 : Meningeal signs returning, headache, neck retraction, Kernig +. Cerebral fungus developing again. Lumbar puncture, opalescent fluid, a few polymorphs and lymphocytes. Culture, no growth.

Sept. 23 : Patient is now very ill. Lumbar puncture, opalescent fluid, marked increase of globulin, many polymorphs, and a few lymphocytes. Culture, no growth. Sept. 24 : Patient still worse. Lumbar puncture, fluid cloudy. Cerebrospinal fluid grew staphylococcus and short-chained streptococcus. Sept. 25 : Died, 45 days after operation.

Autopsy.—Severe basal meningitis, not extending vertically, firm adhesions round cerebral fungus, no spread from here. On section the ventricles were found distended with turbid fluid. Examination of the softened wound area and fungus revealed eleven pieces of indriven bone, eight of which were white, dead, and free, and three adherent to the brain tissue and pink with granulations. The lateral ventricle was not penetrated by bone fragments ; infection had occurred by extension of encephalitis.

In this case, after a brave fight against odds, in the course of which one generalized attack of streptococcal meningitis had been weathered, the patient finally succumbed. The indriven fragments formed a constant source of infection; the cerebral tissue had been able to deal with only three, whilst eight more remained as veritable sequestra deep in the brain. Had these been removed at the operation the result might have been different. This case illustrates very clearly the remarks previously made about the changes in the character of the cerebrospinal fluid; how it may be turbid but sterile, and bacteria only make their appearance in it later.

Since this case was seen, no effort has been spared in effecting the removal of indriven fragments, each one being regarded as a potential source of continued infection. It is highly probable that many of the late outbreaks of infection in superficially-healed head wounds which one sometimes hears of, have their origin in such fragments. When a 'flare-up' occurs in an old wound in other regions of the body, one immediately suspects the presence of a foreign body, a piece of dead bone, of metal, or of clothing. The case of the head is not materially different. In the work of removing all indriven bone I have been fortunate in having the sympathetic support of Captain Dale, R.A.M.C., radiographer to No. 14 General Hospital, and Captain Peters, the pathologist. We have tried to form some

Table V.—Enumeration and Bacteriology of Indriven Bone Fragments.

	Case	Nature of Injury. Number of Fragments shown by *X* rays	Number of Fragments removed at Operation	Bacteriology of Fragments
Recovered	1	Lodging wound left parietal region; 8 or 9 indriven bone fragments	Missile and 10 bone fragments removed	Staphylococcus *B. Welchii* *B. sporogenes*
	2	Large wound left occipital area; at least 10 indriven fragments	15 bone fragments removed (very foul)	Staphylococcus *B. Welchii* *B. sporogenes*
	3	Lodging wound left Rolandic area; 7 bone fragments clearly distinguished, possibly more	Missile and 8 indriven fragments recovered	Staphylococcus
	4	Gutter wound left precentral area; 4 indriven fragments detected	6 bone fragments removed from brain	Staphylococcus
	5	Gutter wound left parietal area; 7 indriven fragments counted, 1 edgeways	11 fragments recovered	*B. sporogenes*
	6	Lodging wound left parietal occipital area; 3 large, 1 medium, and 1 small indriven bone fragments	Missile and 5 indriven fragments removed	Staphylococcus *B. sporogenes* *B. tertius*
Died	7	Lodging wound left parietal area; 3 or 4 indriven fragments	4 indriven fragments removed	Staphylococcus Short streptococcus
	8	Gutter wound right occiput; at least 10 bone fragments indriven	12 bone fragments recovered from brain	*B. sporogenes* *B. tertius*
	9	Gutter wound right frontal area; at least 12 bone fragments indriven	16 bone fragments removed (very foul)	Staphylococcus *B. sporogenes* *B. Welchii*
	10	Gutter wound left parietal area; 5 bone fragments detected	6 bone fragments removed from brain	Staphylococcus Hæmolytic streptococcus
	11	Lodging wound right frontal area; 13 bone fragments counted	13 bone fragments removed	Staphylococcus *B. Welchii*
	12	Gutter wound left parietal area; many indriven fragments, uncountable, hazy in wound area	10 fragments recovered	Staphylococcus *B. Welchii*

estimate as to the number of bone fragments present before operation. In actual practice it is usual to find more pieces than are shown by the *x*-ray, for many consist of inner table only and are very thin, throwing no shadow on the plate unless they happen to be edgeways to the ray. The table of 12 cases on page 275 gives an idea of the results obtained. The first 6 recovered, the second 6 died.

These figures show the extreme importance of indriven bone fragments, both as regards number and infection. It is noteworthy that the radiograph usually underestimates the number of pieces of bone, and one should not be satisfied until at least the number forecasted have been recovered.

The bacteriology of penetrating wounds may be summed up in the following table, in which the results of the bacteriological examination of 43 cases are summarized.

Table VI.—Bacteriology of: (*A*) 25 Cases which Recovered; (*B*) 18 Cases which Died.

	AEROBES			*ANAEROBES*		
	Staphylococcus	Streptococcus	Hæmolytic streptococcus	*B. Welchii*	*B. sporogenes*	*B. tertius*
A.	20	3	1	11	10	2
B.	14	4	2	9	7	1

In two cases the bone was sterile on culture. In one of these the brain grew staphylococci, so no doubt the bone would have been infected very shortly. The second sterile case was one of ventricular penetration, the only one which recovered; the inference is obvious.

It is a considerable tribute to the powers of the cerebral tissue and meninges that such infections were often successfully dealt with. It will be noted that the staphylococcus is the organism most constantly present, and the *B. Welchii* and *B. sporogenes* the next most common. The streptococcus was not often present, but it is worth noting that whilst it was found in 16 per cent of the recoveries, it occurred in 33·3 per cent of the deaths. This is very much in keeping with Whitaker's findings. Certainly the streptococcus is a most unpleasant organism, owing to its great powers of penetration and permeation. As to the anaerobes, the amount of tissue œdema they produce renders them deadly within, quite apart from any question of toxæmia.

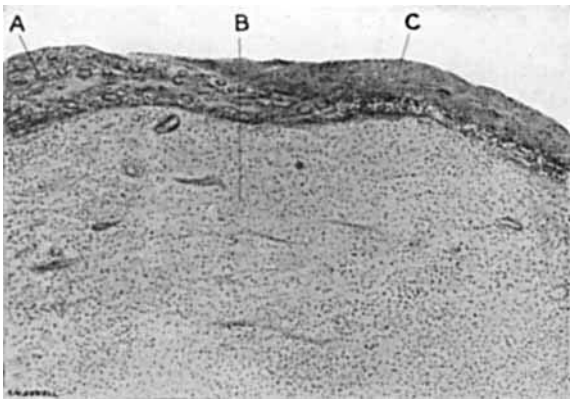

Fig. 260.—Cerebral hernia with dural adhesion. Section passes through adherent edge of dura, which is bound down by leptomeningeal adhesions. (× 12.) (A) Fungus cerebri. (B) Brain. (C) Dura mater.

Cerebral Hernia and Cerebral Fungus.*—The post-operative course varied considerably. In many the wounds healed right up; some discharged for a few days through the drainage opening and then healed; others showed a bulge which soon receded. These were cases where the anatomical injury was not very extensive and a thorough toilette had been possible. A true cerebral fungus developed in a number of cases, and was

* The term cerebral hernia will be applied to those cases which show a bulge through a defect beneath the intact skin, cerebral fungus to a granulation-covered tumour which has burst through the scalp wound.

invariably a sign of sepsis. Fungi appeared in every conceivable region, not only on the cranial vault, but also bulging through the floor of the anterior cranial fossa into the orbit. Naturally, both hernia and fungus are retarded by pressure; hence they do not develop so readily through subtemporal defects, particularly if a muscle-splitting operation has been performed. It is very remarkable to see how a cerebral hernia will force its way through an almost healed wound. The key to the whole question of cerebral fungus formation is sepsis: the aseptic cases do not produce them. Their physiological basis is twofold: a local swelling from infection, and also a general increase of intracranial pressure. These two forces combined cause a progressive bulging, the extent and duration of which are dependent on the inflammatory processes going on in the wound. If the general intracranial pressure is lowered by lumbar puncture, the hernia will recede, and the extent of the recession varies directly with the extent to which the presence of the fungus depends on increased intracranial pressure. If the local tissue œdema is great, a hernia cannot be at once withdrawn by this method. But I am a firm believer, from experience, in the rôle which lumbar puncture can be made to play in the treatment of fungus cerebri. It is difficult at first to tell how much the bulge is due to increased pressure, that is to say, to what extent it is a meningocele. This question can be answered only by lumbar puncture. I cannot agree with Leriche that fungus is the result of insufficient decompression and strangulation, though the latter may happen. A fungus is certainly a sign of failure in so far as it means that primary healing is not going to be obtained. But fungi have developed through large as well as small trepanation defects. It is true that with a large defect the prognosis is usually better; but this is because it means that a complete operation has been performed and the possibilities for drainage are good. When, on the other hand, a large defect means a very severe anatomical injury, the prognosis is bad. In my own cases the largest fungi occurred in those with the most extensive defects. There was only one case of strangulated fungus. The size of the fungus is to a certain extent an index as to the course which the case is running, an increase being usually associated with an exaggeration in the severity of the symptoms. As the meningeal signs varied, so did the hernia, the explanation being that both depend primarily on intracranial tension. It must be remarked that the classical signs of basal meningitis—suboccipital headache, head retraction, neck and limb rigidity—depend very largely on increased pressure, and in my experience were no index whatever as to the presence or otherwise of bacteria in the cerebrospinal fluid. It is true that this often shows a leucocytosis under these circumstances, but it has been found perfectly clear when all the clinical signs pointed to a declared meningitis.

A very serious complication of cerebral fungus is a leak of cerebrospinal fluid occurring through it. The mechanism of the production of these leaks has already been described—namely, the spread of encephalitis to the wall of the ventricle, and finally a rupture through the softened area, with the inevitable leak. It sometimes happens that the lateral ventricle becomes itself part of the deeper portion of the fungus. This is usually ascribed to the ventricle being drawn into the defect. It is much more likely that it is a pulsion diverticulum into a softened area.

Examination of the records of the penetrating head cases shows that fungus developed

Table VII.—Cases with Cerebral Fungus and Cerebrospinal Fluid Leakage,

Cases	Cerebral Fungus	Leakage of Cerebrospinal Fluid
No. 14 *General Hospital*		
Recovered, 50	21	1
Died, 29	17	6
C.C.S.		
Recovered, 27	8	2
Died, 7	5	2

in about 50 per cent of the cases, and that leakage of cerebrospinal fluid occurred in 11. In 3 instances the leakage stopped and the patients recovered.

It is difficult to estimate the frequency of hernia (as distinct from fungus); it must be very common as an early transient phenomenon; of course, all the fungi were technically herniæ before they broke through the scalp wound.

As to treatment, lumbar puncture stands alone as the most efficient treatment. If done early and repeated, it will control the protrusion as nothing else will. Tight bandaging, the application of corrosive and hardening liquids, and the excision of cerebral fungi, are all methods of treatment which neglect the physiological processes of the evolution of the protrusion. It can only be said of them that each one is more harmful than the last. Twenty-nine fungi were cured by conservative means, and autopsy showed that none of those which died could have profited by any of the more reckless methods of treatment.

Lumbar Puncture.—Lumbar puncture has been very freely employed in the treatment of these head cases. It has been a most valuable diagnostic and therapeutic measure. For the diagnosis of intradural hæmorrhage, both in concussional cases and in wounds of the face in which one cannot quite exclude an intracranial injury, it is without an equal. The following case may be cited as an instance.

Case 9.—**Shell fragment traversed nose and left maxilla. Doubtful cranial injury. Lumbar puncture, bloody fluid withdrawn. Explored. Penetrating wound of temporal fossa discovered.**

Pte. A. J. R.—Wounded Oct. 20, 1918. Shell fragment entered right side of nose, traversed nares and left maxilla, lodged beneath left zygoma. Admitted No. 14 General Hospital Oct. 21. Conscious. Very severe frontal headache. All tendon reflexes greatly exaggerated. *X*-ray: No penetration of cranium detected.

Oct. 21.—*Operation*: Toilette of facial wound, and missile extracted. Lumbar puncture, 35 c.c. cerebrospinal fluid withdrawn evenly stained with blood. The track of the missile seemed to be below the cranium proper, and as it was very difficult to see how the brain case could possibly have been opened, nothing further was done.

Oct. 25: Headache continues, all reflexes exaggerated. Lumbar puncture repeated, bloody fluid again withdrawn under tension (proved sterile on culture). A second *x*-ray shows a doubtful fracture of the extreme anterior and lower end of the temporal fossa. As the neurological signs were so definite and blood was still present in the spinal fluid, a further operation was decided on.

Second Operation.—Left temporal flap turned down under ether, disc removed, bone nibbled away down to floor of temporal fossa. At the extreme lower end a small tear in the dura was found and one indriven bone fragment 1 cm. square recovered from the tip of the temporal lobe. Closed with drainage after aspiration of brain débris.

The patient made an excellent recovery as far as the head went, but the left maxillary antrum had to be drained later.

This case illustrates clearly the information which may be gained by lumbar puncture in these difficult facial wounds. Albert has published some excellent examples. In this case I ought, of course, to have operated without delay on finding blood in the cerebrospinal fluid.

The punctures, with few exceptions, have been made under novocain-adrenalin anæsthesia, with the patient lying on his side. Occasionally when patients were very intractable, gas and oxygen was employed. The needle was invariably introduced in the middle line. Sometimes in the old-standing meningeal cases, the lower end of the spinal canal is so clogged with lymph that nothing can be withdrawn. In such cases punctures have to be tried higher and higher until fluid does run. Sometimes the cerebrospinal fluid will fail to flow even when the needle is not blocked by lymph. In such cases lifting the head and shoulders will almost always cause the fluid to appear.

Technique of Operation.—In the vast majority of cases local anæsthesia was used (omnopon $\frac{2}{3}$ gr. and scopolamine $\frac{1}{100}$ gr. being given an hour previously), chloroform and ether being reverted to only when the patients were extremely restless. But the writer is so firmly convinced of the superiority of local over general anæsthesia, that it was with great reluctance that the latter was employed. Even with restless cases the operation was attempted under novocain, and ether given only if the movements of the patient hampered the operative steps too greatly. The advantages of local anæsthesia lie in

lessened shock, absence of post-operative vomiting which may cloud the clinical picture, a minimum of bleeding, and convenience of posture. The last is a most important point, for a conscious patient can hold himself voluntarily in the position which gives the readiest access to the wound, and can make any change of posture that becomes advisable as the operation proceeds. He may even assist the surgeon by coughing and expelling cerebral detritus through the torn dura. As Cushing has pointed out, these patients resent jarring ; but knowledge of this tends to make the surgeon lighter-handed. Separation of the dura is sometimes painful, and referred orbital pain from the occipital dura has been commented upon above. Novocain-adrenalin analgesia reduces hæmorrhage to a minimum. Few surgeons realize the quantity of blood lost during operations. Bazett has recently shown that 17·7 per cent of the total blood volume was lost during an ordinary decompression of a depressed fracture, and 27 per cent during the operative steps taken upon a wound involving the middle meningeal. Added to the loss which has occurred since the time of wounding, such a depreciation in blood volume must be a serious factor.

For the decompression the hand trephine was used as a rule. In a few cases the Doyen bit and burr and Montenovesi's forceps were employed. There is one type of injury in which the block removal of the injured bone is very valuable ; that is, in wounds of the venous sinuses. There is an advantage in having the tear in the vein exposed instantaneously, giving a clear view and unobstructed field. Apart from this I am not convinced that Cushing's excellent results were the outcome of this or any other particular step in his technique. Aside from great gentleness and thoroughness in the toilette of the brain wound, the determining factor was probably the early date at which the operations were undertaken, thus eliminating to a great extent the factor of sepsis.

In no cases of my own series was an intact dura incised, and none seemed the worse for this conservatism. This is not to say that an unlacerated dura ought never to be opened at all ; but the conditions must be right. Asepsis is the one fundamentally necessary condition. Within the first twelve hours, before septic processes are thoroughly established, one might incise the dura with impunity ; within the next twelve this would probably entail a certain mortality, increasing in direct proportion with the time which has elapsed since wounding.

In cases where the dura was penetrated by the missile or by bone fragments, catheter suction was carried out on Cushing's lines. It is remarkable how often the catheter will remove quite large fragments of bone, but it is no less remarkable what large pieces it may quite fail to detect. In other words, the evidence that was obtained from the catheter was, in my hands, unreliable.* It is so important to make sure that all indriven bone fragments have been removed, that the introduction of a finger into the brain wound can hardly be avoided. I have made a practice of it, knowing full well the arguments against it, and yet believing that the advantages outweigh the points in its disfavour. It must be insisted, however, that the utmost gentleness and delicacy must be used. Those whose hands fall within Belloc's category in his lines to a child on " the massive paws of elder persons," those with spade-like fingers, should abstain from operating on penetrated heads.

The triradiate incision was always closed with a flavine cigarette drain, which was removed at the end of twenty-four to forty-eight hours.

Neurological Aspects of Head Wounds.—Full justice cannot be done here to the neurological pictures presented by these cases, and the results of the examinations of function have been largely excluded from this paper. A brief summary of the findings follows.

The signs and symptoms of intracranial disturbance presented by these cases naturally varied with the severity of the wound. They can in the main be subdivided into two

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* Ney admits in his recent paper that he has left bone fragments behind when using the catheter. In C.C.S. work these fragments are less likely to cause trouble later, as sepsis is not such a serious factor in the very early cases.

groups : (1) Signs and symptoms the result of general intracranial disturbance irrespective of the site of the wound, due in the main to disturbances of cerebral circulation ; (2) Localizing signs and symptoms due to injuries of special cortical areas.

Besides these signs of disturbed function, there are those of intracranial infection, the 'meningeal syndrome,' representing a second phase in the evolution of the wound.

As regards the general signs of headache, vomiting, etc., their relative frequency will be found in *Table VIII.* Even in those cases where the cerebral injury was never more than concussional, or at most a cortical contusion, these general signs were surprisingly common.

Table VIII.—The Relative Frequency of General Neurological Signs.

Conditions	Group I (Scalp Wounds)	Group II (Fractures)	Groups III—VIII* (Dural Penetration)
	per cent	per cent	per cent
Headache ..	90	97	98
Vomiting ..	16	31	45
Nausea ..	32	37	26
Stunned ..	60	67	—
Unconscious ..	32	41	61

* Seven of the recoveries in Groups III–VIII were incontinent, and 14 of the fatal cases (early condition). As 14 of this group were unconscious on admission, these figures do not refer to the total in the series, but the percentages when information could be obtained.

No useful purpose will be served here by detailing the signs of local injuries. Clearly defined examples of purely cortical injury were only encountered in *Groups I* and *II.* As soon as the dura is penetrated and cerebral laceration occurs, one meets at once the complication of subcortical division of axons in the white matter. For instance, most of the severe leg palsies were due to injury, not of the cortical area supplying the lower limb, which lies in a somewhat sheltered position, but to a division of the efferent fibres from it. So that one would see an arm-leg palsy due to *cortical* destruction of the arm area and *subcortical* division of the leg fibres. It may be added that the palsies in the penetrating groups were more severe and permanent than in the scalp wounds and depressed fractures, where the injury to the grey matter was concussional, contusional, or due to local anæmia from the direct pressure of bone (cf. *Case* 2).

As to the meningeal syndrome, consisting of headache, often occipital, nuchal pain and rigidity, head retraction or fixation, tenderness of the back, limb spasticity, Kernig, and fits, I can only repeat that the syndrome does not necessarily imply that one is going to find bacteria in the cerebrospinal fluid. The syndrome indicates increased intracranial pressure, but why it is present in some and not in others is not quite clear. The following is a case in point.

Case 10.—Pte. J. H. Admitted head service of No. 14 General Hospital two days after toilette and decompression of a penetrating wound in a clearing station. Sutured wound of left parietal eminence, clean.

Vomited twice after admission, incontinent, very restless and noisy at times. Occipital headache. Four days later meningeal signs were pronounced. Head retracted and neck stiff. Kernig +. Complains when head is raised. Tremor of limbs. Reflexes + +. Lumbar puncture, clear cerebrospinal fluid recovered, tension + +. Fluid proved sterile on culture. Patient made an uninterrupted recovery following the puncture, and began to take food the next day. Mental condition clearing.

The following is an example of the meningeal syndrome appearing with dread portent.

Case 11.—C.-S.-M. H. T., age 28. Wounded Sept. 13, 1918. Admitted No. 14 General Hospital two days later. Wounded by fragments of shell, 5 cm. above inion, wound running obliquely down to the right squama of os occipitalis. Smells exceedingly foul.

General Signs.—Headache +, improving, occipital ("like sunstroke"). Vomit +, twice

in first half hour; nausea now nil. Never unconscious. Stunned. Quite rational, a little drowsy.

Localizing Signs.—Palsies, nil. All tendon-jerks normal. Plantar reflex, flexor. Superficial reflexes normal. Vision: In left field, perception of light ('frosted glass vision'); absolutely unable to distinguish objects. In right field, considerable scotoma in the nasal quadrant; cannot distinguish colours for some distance to right of centre of pupil.

Sept. 15.—*Operation*: Type toilette. Twelve indriven fragments removed, closed with a drain. Headache relieved. Near the mid-line the dura exceedingly tender on forceps pressure, which gave deep pain behind eyes as if something were pressing on back of eyeballs.

Sept. 16: Seems to be much better.

Meningeal Syndrome.—

Sept. 17: Patient suddenly became noisy and restless, suboccipital headache, neck rigid.

Sept. 19: Fully declared syndrome, great head retraction, Kernig +, limbs flexed and stiff. Lumbar puncture, opalescent fluid under pressure. Film shows considerable number of polymorphs and a large bacillus. Culture, *B. Welchii*, staphylococcus. Died same day. (See autopsy report, Series No. 137. The occipital horn of the lateral ventricle became infected by continuity.)

Jacksonian fits were noted 48 times, distributed among 20 patients; 14 of these recovered and 6 died.

A. Recovered:—			
Group I (scalps)	.. 3 cases	4 fits	
Group II ..	.. 3 ,,	5 ,,	
Group III–VIII	.. 8 ,,	21 ,,	
B. Died:—			
Groups III–VIII	.. 6 ,,	18 ,,	

As a rule, the later a fit occurred, the less its significance. Most of the fits arose in the first two weeks, owing to the incidence of cortical meningitis during this period. Of the six fatal cases presenting Jacksonian fits, only one had a primary Rolandic lesion. Yet these six had eighteen fits between them, all died of meningitis, and at autopsy the cortex was found to be extensively involved, and even bathed in pus.

Case 12.—Left temporal wound. No palsy. Six fits eleven days after being wounded. Developed a right side hemiplegia not present before. Died. At autopsy, left hemisphere covered with pus.

Case 13.—Left frontal wound. No palsy. Twitchings right side of face three days after wounding. Autopsy: Extensive cortical meningitis of left hemisphere, collection of pus over central sulcus.

It is remarkable how easily fits can be controlled by conservative measures such as large doses of bromide (30 grs. every two hours) and by lumbar puncture. I believe it to be a great mistake to operate for fits alone in these healing cases. They do very well indeed with lumbar puncture.

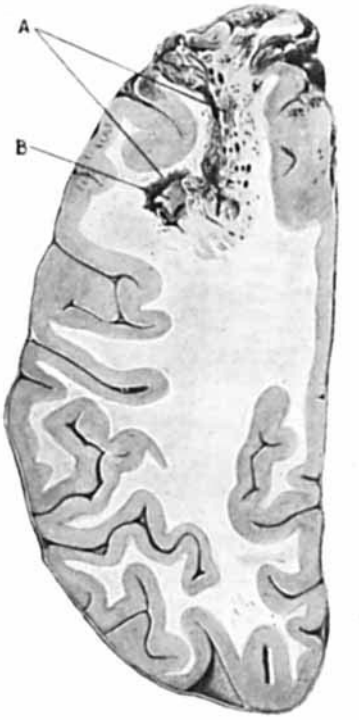

Fig. 261.—G.S.W. frontal pole; a small bone fragment (B) is lying as a sequestrum in the midst of an area of encephalitis (A).

Superior Longitudinal Sinus Syndrome.—The classical sign of this syndrome, as pointed out by Holmes and Sargent, is rigidity especially affecting the lower limbs. This they attribute to thrombosis of the superior longitudinal sinus or frontoparietal lacuna, with consequent stasis in the upper half of the motor area, the veins of which open into the lacuna named. They admit the difficulty of discriminating between the effects of direct contusion of the underlying centres and those of a blockage of the venous blood return. Cushing, in his paper (loc. cit., p. 595), attributes the whole of the symptoms to contusion of the vertex involving the mesial surfaces of both hemispheres and resulting in bilateral spastic paralysis of the lower extremities, with perhaps an accompanying palsy of one or both arms. Saviozzi inclines to the same view. In my series there were only

five mid-line vertex wounds in which the cranial dura was torn. In four the longitudinal sinus was lacerated, in one it escaped. One of each type exhibited limb spasticity. Both the cases which presented spastic paralysis were injuries coinciding with the motor cortex; when the injury did not coincide there was no palsy. It would seem, then, that cortical injury plays an exceedingly important part in the production of the symptoms of this syndrome. But it is worthy of note that cortical contusion and laceration did not result in *early* spasticity in any but these two cases. It seems probable, then, that Holmes and Sargent were right in giving this injury the honour of a special title. The cases in this series are too few to draw definite conclusions from; but, as I interpret the facts observed, they mean that spasticity superimposed on palsy denotes either a cortical hæmorrhage (as in Little's disease), or a sinus thrombosis with venous stagnation. A synopsis of the cases, two of them from the depressed fracture series (*Group II*), is given below :—

Case 14.—Vertex wound, immediately to right of mid-line, just reaches upper end of motor cortex. No palsy, but knee-jerks on left side + +, 3–4 beats of clonus left ankle. Cerebral dura not torn. Superior longitudinal sinus hæmorrhage, controlled by muscle graft. Recovered. No spasticity.

Case 15.—Vertex wound 7·5 cm. above nasion, mid-line. Cerebral dura not opened. No limb disturbances whatever. Recovered. This wound was in front of frontoparietal lacuna and motor cortex, so no limb upset would be expected.

Case 16.—Vertex wound behind coronal suture (i.e. overlying frontoparietal lacuna) but short of motor cortex. Arm reflexes + +, absent in legs. No palsy or spasticity. Dura lacerated over hemisphere. Superior longitudinal sinus almost completely divided. Fascial graft. Recovered.

Case 17.—Gutter wound of vertex, 12 cm. long, anteroposterior. Dirty. Admitted day after being wounded. Vomited yesterday. No palsy or rigidity, but leg tendon-jerks very active, arms normal. Severe 'fixed' headache, " top of head opening and shutting." Operated. Extradural hæmorrhage, with small tear in dura. Next day left arm and leg were found to be very stiff, so much so that tendon reflexes could not be elicited. Partial paralysis left leg (foot). Developed a small fungus. Evacuated early, 13 days after operation, leg still stiff, Babinski +, knee-jerks + + on left. This was either a cortical hæmorrhage or else thrombosis of a large cerebral vein. If the sinus had clotted, one would have expected the affection to be bilateral.

Case 18.—Gutter wound of vertex mainly to right of mid-line, crosses motor area. Legs in condition of spastic paralysis, adducted; left arm spastic. Operated by me twenty-four hours after wounding. Severe hæmorrhage from large rent in superior longitudinal sinus, dura torn over right hemisphere as well. This patient had a secondary hæmorrhage from the sinus seven days later, controlled by a fascial strip from the thigh. Made a good recovery, though the limb stiffness persisted for over two months.
This is the only case of secondary hæmorrhage from a venous sinus that I have met with.

It will be seen how difficult it is to distinguish between the effects of cortical contusion and those of interference with the cortical circulation. I can only repeat that *Cases* 17 and 18 were the only ones in which early spasticity was noted.

Causes of Death.—There were no deaths in this series from such extraneous causes as pneumonia, nor were there any from wounds elsewhere. So that the mortality of 37·6 per cent can be entirely attributed either to hæmorrhage and traumatic œdema, or to sepsis. A synopsis of autopsies will be found at the end of the paper; it will be seen that the average time of death was 17 days after wounding, 15 days after admission to hospital. This means that infective processes had ample time to establish themselves in the majority of cases, and were, in fact, the common cause of death. The patients who died from the primary effects of the injury, as well as one can determine, were Nos. 113, 82, 169. Of these, one died 1 day after being wounded, two 2 days after. These three were clear cases of the injury causing death unaided. The autopsies showed no signs of gross infection. One further case died of intraventricular hæmorrhage (Series No. 162); the contamination with hæmolytic streptococcus may have hastened his end. The remainder succumbed to bacterial invasion of their wounds. The clearest examples of this are Series Nos. 65, 170, 149, dying 47, 55, and 62 days after wounding. Of the remainder,

three died in the first week, five in the second week, and six in the third week, all from meningitis, either by direct spread from the cortical wound, or through ventricular involvement. The average date of death in the cases operated upon in clearing stations before admission was practically identical with that of my own series, 18 as against 17 days.

An organism was grown from the cerebrospinal fluid seven times (out of 85 punctures). Four times streptococci were found (one hæmolytic); twice *B. Welchii*; and once pure staphylococcus. I have no doubt that had more punctures been made in the terminal hours, the cultures would have been positive in every case (save the rapid deaths from hæmorrhage).

Many very severe injuries recovered against all expectation, whilst some which should have done well died incontinently.

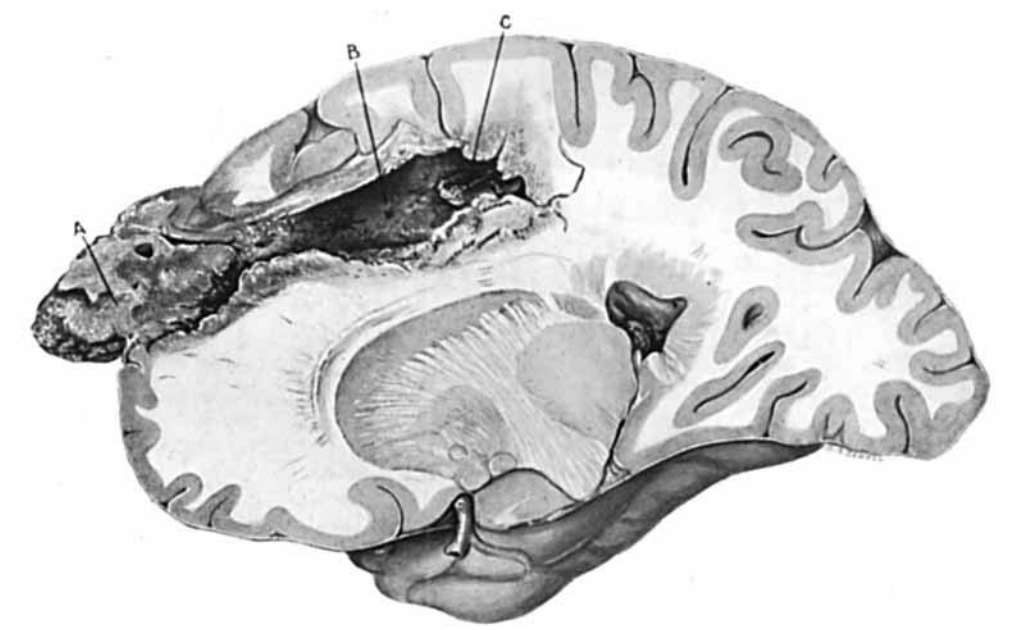

FIG. 262.—G.S.W. right frontal pole. (A) Strangulated cerebral fungus; (B) Abscess formation in the track of the lodged missile; (C) Missile. Subcortical division of the radiation from the motor area with left hemiplegia.

Reflection over the autopsy findings compels a full recognition of the part played by organisms in causing death in cerebral wounds. It compels, in many cases also, considerable wonder at the resistance which the brain and meninges can put up against infection, provided that it has room to work in—that is, if decompression is free, or repeated tappings of the cerebrospinal fluid are made.

CAUSES OF DEATH.

From effects of injury without intervention of sepsis	4
From cerebral abscess	3
From massive gas infection	2
From meningitis	19

In the deaths from meningitis, the meningeal infection clearly originated in the wound only in one case (Series No. 110). In all the others the ventricular path was of great importance. This subject has been fully discussed above. There were three deaths from abscess (Series Nos. 92, 149, 120). The most interesting was No. 92 (*Fig.* 262), where

an incomplete decompression was done, leading to a strangulated fungus cerebri holding up the discharge and converting the track of the missile into an abscess cavity.

One of the brains with massive gas infection is pictured in *Fig.* 263. It is probable that the bulk of the gas formation here is a post-mortem formation, and corresponds to the 'emphysema cerebri' of old writers. No doubt the rôle played by anaerobic bacteria in the pathogenesis of brain wounds is considerable, but actual gas-bubble formation in the cerebral tissue must be uncommon. Small bubbles in the pulped brain and bone presenting in some of the severest gutter wounds were a frequent occurrence, and not all of these cases died.

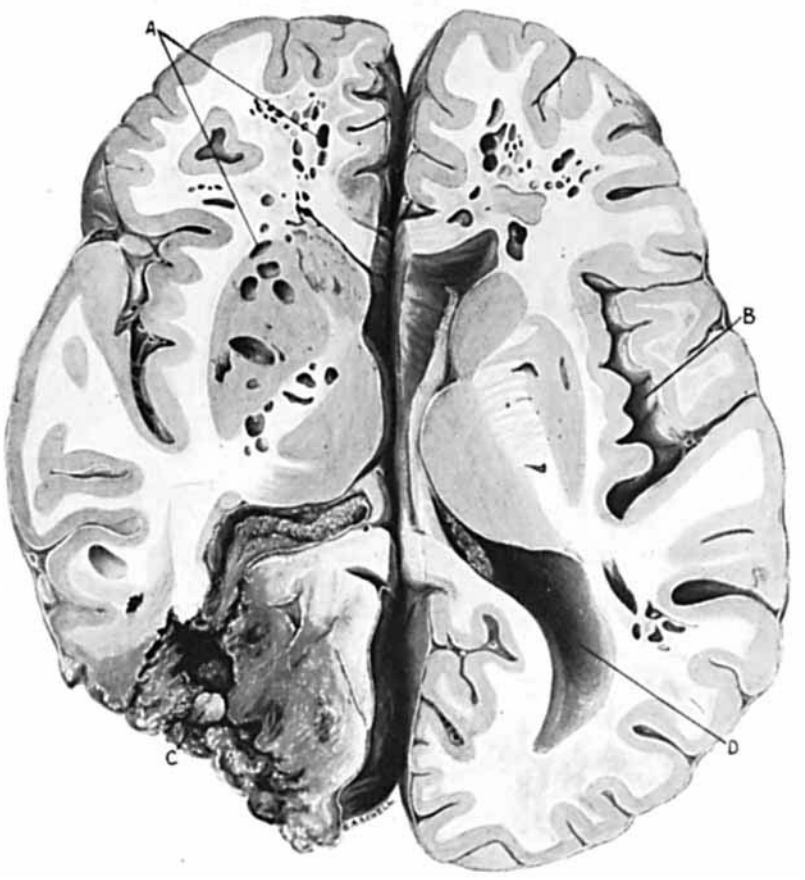

FIG. 263.—G.S.W. occipital pole, indriven bone, posterior horn of lateral ventricle directly involved. Gas infection; most of the bubbles probably post mortem, as there is no reaction around them. Hemispheres cut at slightly different levels. (A) Gas bubbles; (B) Fissure of Sylvius opened up by submeningeal gas; (C) Indriven bone fragment lying in pulped occipital pole; (D) Distended lateral ventricle.

SUMMARY.

1. In gunshot wounds of the head, as in wounds elsewhere, the prime factors are the extent of the injury, the physiological disturbance produced by it, and the incidence of sepsis.

2. Of the 150 cases in the series not previously operated upon, 29 died (19·3 per cent), all penetrating wounds of the dura of varying degrees. No case with an intact dura died. The mortality for penetrating wounds alone was 37·6 per cent.

3. With the exception of four cases which rapidly succumbed to the effects of the

injury alone, all the deaths were due to infection. The inference that head wounds should be operated upon early is obvious.

4. The common form of infection was meningitis, produced by spread either from the site of the wound, or by the medium of ventricular infection, which deposited the infection in the basal arachnoid cisterns. This latter seemed to be the commoner route, and is undoubtedly of paramount importance. Three died from cerebral abscess.

5. The nature of the infection is tabulated in the text. The bacteria present are much the same as in wounds elsewhere. The most deadly seems to be the streptococcus.

6. The importance of removing all indriven bone fragments cannot be over-emphasized. Bacteriological examination of the fragments showed them to be heavily infected with both aerobes and anaerobes. An attempt was made to enumerate all fragments and locate their depth and direction.

7. From the neurological side, three distinct aspects can be discerned: (*a*) Signs of generalized intracranial disturbance, increased tension; (*b*) Localizing signs of cortical or projection fibre injury; (*c*) The meningeal syndrome.

8. The meningeal syndrome was frequently present, often with a sterile cerebrospinal fluid, which may show a physiological leucocytosis and be quite turbid but be free from organisms.

9. Three examples of pain referred from the occipital dura to a situation behind the eyes are recorded.

APPENDICES.

I. SYNOPSIS OF AUTOPSIES: CAUSES OF DEATH.

SERIES No. 162.—Shell wound left parietal, 7 cm. vertically above porus externus. Small punctured wound, blood pumping out. Wounded Nov. 5, 1918. Admitted Nov. 7. Operated, same day.

Bacteriology: Staphylococcus, streptococcus, *B. proteus*.

Autopsy: Extensive intradural hæmorrhage, pool in cisternæ, extending over vertex and down spinal canal. All ventricles distended with ante-mortem clot. Both ventricles perforated. Origin of blood not found, ? choroidal.

Note: The only severe intracerebral hæmorrhage encountered.

SERIES No. 119.—Stinking wound right frontoparietal suture, pulped brain and bone presenting. Hemiplegic. Wounded Sept. 2, 1918. Admitted Sept. 4. No operation. Died day of admission.

Bacteriology: Staphylococcus, streptococcus, *B. Welchii*.

Autopsy, 14 hours later: Right frontal lobe pulped, massive gas infection of cerebrum. Local meningitis only.

Note: Meningeal syndrome absent. Died 8 hours after admission.

SERIES No. 115.—Bullet wound left frontal bone, very severe anatomical injury, diastasis. Wounded Sept. 2, 1918. Admitted Sept. 4. Tentative operation same day. Died Sept. 8.

Bacteriology: Staphylococcus, *B. Welchii*, *B. sporogenes*.

Autopsy: Severe lacerated wound across left frontal lobe on to right. Basal meningitis, ventriculitis, no vertical meningitis, adhesions found round wound.

SERIES No. 137.—Shell wound over right occipital, 6 cm. above inion. Left hemianopsia. Wounded Sept. 13, 1918. Admitted Sept. 15. Operated same day. Died Sept. 19.

Bacteriology: *B. sporogenes*, *B. tertius*.

Autopsy: Purulent thrombosis lateral sinus; right occipital lobe pulped; gas infection, with a few bubbles. Infection posterior horn leading to generalized ventricular infection. Severe basal meningitis extending down spinal canal; long streak of vertical meningitis running from Sylvius across left parietal field, nil on right. Cerebrospinal fluid milky, grew staphylococcus and *B. Welchii*.

SERIES No. 92.—Lodging shell wound right frontal region 7·5 cm. above vision, 2·5 cm. to right of mid-line. Wounded Aug. 28, 1918. Admitted Aug. 30. Operated same day. Died Sept. 9.

Autopsy: Strangulated cerebral fungus, large abscess cavity in track of retained missile (*see Fig.* 262). Owing to lack of adequate drainage, track of missile became converted into an abscess cavity.

SERIES No. 170.—Huge lacerated wound left frontoparietal. Flaccid right hemiplegia. Stinking wound with bone fragments and brain presenting. Wounded Sept. 3, 1918. Admitted Sept. 5. Hopeless. Tentative toilette and Carrel-Dakin Sept. 8. Died Oct. 28.

Bacteriology: Staphylococcus, streptococcus, *B. sporogenes*. Lumbar puncture seven times. Grew streptococcus, *B. sporogenes*, Sept. 25. By Oct. 15 cerebrospinal fluid was clear, but cloudy again Oct. 23, growing streptococcus and *B. Welchii*.

Autopsy: Dura firmly adherent over whole of left hemisphere. Turbid fluid in both ventricles. Thick yellow pus over base and in cisternæ, extending down spinal canal.

Note.—This case appeared so hopeless on admission that nothing was done for three days. Clinical history affords a remarkable picture of prolonged struggle with infection.

SERIES No. 149.—Craniofacial wound. Missile (large shell fragment) entered left cheek, traversed ethmoidal cells and right half of cranial chamber, and fractured vault on right side from within, without breaking the skin. Wounded Sept. 27, 1918. Admitted Sept. 29. Operated same day (face). Decompressed Oct. 9, missile removed from right hemisphere. Died Nov. 29 (62 days after).

Bacteriology: Hæmolytic streptococcus, *B. Welchii*.

Autopsy: Large abscess enclosed between ethmoid and densely adherent frontal lobes. Cribriform plate destroyed, brain glued to edges of fracture. No generalized meningitis.

Note: A remarkable case in several respects. The manner in which the missile fractured the vault from within, after traversing the cranial cavity, and, rebounding from the bone, remained in the hemisphere, is unusual. Theoretically, his abscess should have been diagnosed and evacuated. None of the classical signs of abscess were present, his pulse-rate never being below 72. He lay for over seven weeks in a disorientated mental condition, apparently about to die the whole time. The next day one would be surprised to see the 'corpse' smoking a cigarette. I was on leave when his operation was performed and never got a grip of the case. But this is making excuses!

SERIES No. 117.—Large lacerated wound left frontoparietal. Right hemiplegia. Wounded Aug. 29, 1918. Admitted next day. Operated same day. Died Sept. 6.

Bacteriology: Staphylococcus, *B. Welchii*.

Autopsy: Extensive crumbling wound left hemisphere, massive gas infection. No meningitis.

Note: This patient died without meningeal signs; no rigidity, quiet, unconscious, incontinent. Pulse rose to 130 before death.

SERIES No. 108.—Craniofacial wound. Missile entered right cheek and lay deep in skull beneath right parietal eminence. Left hemiplegia. Wounded Sept. 1, 1918. Admitted Sept. 2. Operated same day. Died Sept. 7.

Bacteriology: Not recorded.

Autopsy: Extensive superficial cortical wound, with severe infected hæmorrhage over cortex, especially on right side. No ventriculitis.

Note: Rapid death mainly from intracranial hæmorrhage, the element of sepsis being subsidiary.

SERIES No. 110.—Through-and-through wound left frontoparietal. Right hemiplegia. Brain and shattered bone presenting. Wounded Sept. 2, 1918. Admitted Sept. 4. Tentative toilette same day. Died Sept. 15.

Bacteriology: Not recorded.

Autopsy: Whole left calvarium shattered, severe hæmorrhagic encephalitis both hemispheres.

Note.—This case seemed so hopeless that operation was postponed five days, when a large loose section of frontal bone and 11 indriven fragments were removed. He lived six days more. Had the missile not decompressed him he would certainly have perished immediately from intracranial hæmorrhage.

SERIES No. 113.—Right frontal bone shattered, very severe anatomical injury. Wounded Sept. 2, 1918. Admitted Sept. 4. Tentative toilette same day. Died Sept. 10.

Bacteriology: Not recorded.

Autopsy: Large laceration anterior two-thirds of right hemisphere. Extensive cortical hæmorrhage both hemispheres. Hæmorrhages into brain to depth of 3 cm. Ventricles not involved. No discernible meningitis (too much hæmorrhage).

Note: This patient died from the pressure effects of intracranial hæmorrhage, though very likely the tension was further raised by infection.

SERIES No. 82.—Lodging wound (shell), entrance 2·5 cm. above right supra-orbital margin, missile in opposite hemisphere. Severe fissuring of vault. Wounded Aug. 26, 1918. Admitted Aug. 27. Not operated—condition desperate. Died same day.

Autopsy: Extensive injuries both frontal lobes; streaky hæmorrhage into cerebrum. No involvement of ventricles; no discernible meningitis.

Note: This patient died undoubtedly from the pressure effects of the hæmorrhage and œdema occasioned by the extensive wounds; no evidence of sepsis playing a deciding part.

SERIES No. 169.—Through-and-through wound right parietal area from above down. Wounded Sept. 1, 1918. Admitted Sept. 2. Condition hopeless; not operated. Died Sept. 3.

Bacteriology: Not recorded.

Autopsy: Through-and-through wound right hemisphere. No ventricular involvement. Punctate hæmorrhages. No meningitis.

Note: This patient was believed to have a massive gas infection. It is quite possible that he might have been saved by free decompression. No evidence of infection at autopsy.

SERIES No. 65.—Wound of left frontoparietal area. (See text, *Case* 8.) Died 47 days after wounding. Cerebrospinal fluid grew staphylococcus and streptococcus before death.

Note: The importance of complete removal of indriven bone fragments was brought home by this case.

SERIES No. 160.—Lodging wound left mastoid, missile in neck (petrocranial). Wounded Oct. 31, 1918. Admitted Nov. 2. Operated same day. Died Nov. 10. Leak of cerebrospinal fluid.

Bacteriology: *B. Welchii*, *B. sporogenes*.

Autopsy: Left temporal lobe almost completely destroyed, petrous temporal pulverized, crack in cerebellar fossa. Extensive subarachnoid hæmorrhage, with basal meningitis. Lateral ventricle involved directly. Ventriculitis.

Note: In this case sepsis was superadded to a severe injury with extensive hæmorrhage. He died only when the infection had had time fully to establish itself.

SERIES No. 95.—Lodging wound, very foul, right frontal region, 8 cm. above nasion and 3 cm. from mid-line. Wounded Aug. 28, 1918. Admitted Aug. 30. Operated next day. Died Sept. 5.

Bacteriology: Staphylococcus, *B. Welchii*, *B. sporogenes*.

Autopsy: Pulping of right frontal lobe. Missile tracked obliquely backwards and lay beneath meninges, where there was a secondary intense area of meningitis. Diffuse cortical meningitis, encephalitis involving anterior horn of lateral ventricle, generalized ventriculitis. Cerebrospinal fluid on admission milky (full of polymorphs), no growth on culture.

Note: Signs of meningitis were established on admission. Lumbar puncture on the table at the time of toilette disclosed a cloudy but sterile fluid.

SERIES No. 120.—Through-and-through wound left temporal fossa, fracture parietal and squamous bones. Wounded Sept. 2, 1918. Admitted Sept. 4. Operated same day. Died Sept. 13.

Autopsy: Local cerebral abscess due apparently to insufficient drainage. No meningitis.

Bacteriology: Staphylococcus, hæmolytic streptococcus. This patient collapsed after lumbar puncture (cerebrospinal fluid clear and sterile), and died 5 hours later.

Note: One of the three cases which died from local abscess. No clinical signs present pointing to a local collection.

SERIES No. 104.—Lodging wound right frontal bone, 8 cm. behind nasion, 4 cm. right of mid-line. Missile (shell) traversed falx cerebri. Wounded Aug. 31, 1918. Admitted and operated on next day. Died Sept. 17.

Bacteriology: Staphylococcus, *B. Welchii*.

Autopsy: Laceration both frontal lobes, severe. Encephalitis extending to frontal horn of lateral ventricle, generalized ventriculitis. Extensive cortical meningitis, cisternæ full of turbid fluid.

Note: Catheter passed at operation through the hole in the falx, but missile not recovered. Neck rigid on admission. Fungus broke through the healing scalp wound.

SERIES No. 62.—Through-and-through wound left and right occiput, exit right post-mastoid. Wounded Aug. 9, 1918. Admitted Aug. 11. Operated same day. Died Aug. 28.

Bacteriology: Staphylococcus, streptococcus (heavy infection).

Autopsy: Both occipital lobes pulped, right extends on to temporal. Massive encephalitis; ventriculitis by extension; basal meningitis. Meningeal syndrome appeared 6 days after operation.

SERIES No. 152.—Shell wound right occipitoparietal, 8 cm. above inion, 2·5 cm. to right of mid-line on to parietal eminence. Wounded Oct. 14, 1918. Admitted and operated on next day. Died Oct. 31.

Bacteriology: Staphylococcus and spore-bearing bacillus.

Autopsy: Long fissured fracture with vertical meningitis corresponding to it. Encephalitis massive, involving ventricle. Generalized ventriculitis, with extensive collection of purulent fluid in basal cisternæ.

Note.—A disappointing case. As far as the anatomical injury went he should have done well, but infection turned the scale against him.

II. THE TOPOGRAPHY OF THE VENTRICULAR SYSTEM.

It has been stated in the foregoing pages that but little work has been done on the surface relationships of the ventricular system. This is true in the main, but the remark may be thought somewhat inappreciative of the work of Retzius, Symington, Jenkins, Wakelin Barratt, Spitzka, Mme. Dejerine and Landau, and Pierre Marie, Foix, and Bertrand. The last-named have injected the ventricles with bismuth and then *x*-rayed the head, but the published prints are not of much value. Dandy's recent work in radiographing the ventricles filled with air *in vivo* is interesting corroborative evidence. In spite of the work of the above writers, the fact remains that the surface topography of the ventricles is still in an unsatisfactory condition, and there is little in it to help the surgeon. This is particularly true of the anteroposterior view of the head, and a few words explanatory of *Fig.* 257 may not be out of place. It must be said at once that no claim is made for meticulous accuracy; it is a rough working diagram. The figure was made by superposing a tracing of the end view of one of Retzius's beautiful models of the ventricles upon a tracing of the skull, both being drawn (the one enlarged, the other reduced) to the same scale. The ventricle scheme was carefully placed in correct position within the skull by reference to Symington's well-known *Cross-section Atlas*. It will be noted that the temporal (descending) horns of the lateral ventricle descend to a very low level. Reference to *Fig.* 257 makes it clear that there are few positions in which a wound can cross the mid-line of the skull without wounding a ventricle; also that an area of encephalitis does not need to be spread very deeply inwards before the ventricles are reached.

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