BRITISH MEDICAL JOURNAL

LONDON SATURDAY APRIL 10 1937

HEAD INJURIES IN GENERAL PRACTICE *

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In a single surgical charge in the Edinburgh Royal Infirmary over the period of a year seventy cases of head injuries were admitted, 5.5 per cent. of the total admissions, and the mortality was 14.2 per cent. I have quoted these figures to show what is already known—that this type of injury is relatively common, increasingly so, and that it is associated with a considerable risk to life.

Significance of the History

It is important to have some record of the history of the accident if certain pitfalls of diagnosis are to be avoided, for there are many conditions apart from head injury which are associated with unconsciousness. The position is all the more confusing when there are no witnesses of the occurrence, the only evidence available being the fact that the individual was found lying unconscious, perhaps bleeding from a scalp wound.

Some months ago a woman in the late sixties was admitted to my ward. She was deeply unconscious; there was a scalp wound in the right temporal region, and a flaccid paralysis of the left arm and leg. She had been found lying at the bottom of a common stair, and it was the impression of those who assisted her that she must have fallen and injured her head.

In actual fact she was the victim of apoplexy, and in this instance the head injury—if the scalp wound may be described as such—was *post hoc* to her unconsciousness, though we might have been excused if our appreciation of the position had pictured the reverse. There are similar possibilities of error in relation to the unconsciousness of drunkenness, the coma of renal failure and diabetes, the collapse of syncope, and even the intoxication of drugs.

We must remember, too, that injury to the skull and its contents may arise through indirect violence.

The roof of one of the large halls in Edinburgh was being redecorated, and a rope was passed from the upper scaffolding to the floor. A painter descended by it, and unfortunately his hands were slippery with oil, so that, instead of dropping gradually, he came down at a run and arrived at the floor of the hall in a sitting position. He apparently had some degree of concussion, but the jeers of his fellow workmen brought him to his senses, and he walked away.

Forty-eight hours later he was admitted to hospital with cerebral compression. He died, and necropsy revealed a fracture at the base of the skull encompassing the foramen magnum and passing into the posterior fossa.

These cases illustrate the danger of misconception; in one instance of assuming a head injury when none exists,

in another of overlooking cranial or cerebral injury because the *modus operandi* has been atypical.

It may be difficult to obtain an accurate account of the sequence of events immediately subsequent to the injury, but it is of considerable practical importance. Following a head injury the sequelae may follow one or other of three possibilities. There may be the temporary disturbance of a slight concussion, a brief loss of consciousness followed by a rapid return to complete normality; or there may be the deep unconsciousness of a severe concussion, the state developing in such close relationship with the accident that it appears to arise coincident with the blow; or yet again consciousness is retained for some time after the accident, but presently there is a lapse and a state of stupor develops. The first and second of these possibilities require no elaboration, their meaning is apparent, but when the third development is recorded it is apt to have a somewhat sinister significance, for it may be that it records the historical sequence of a case of cerebral compression. If the individual retains consciousness for an appreciable period of time after the accident and then passes into a stuporous condition the chances are that a lesion was initiated, the effects of which are now being manifested.

Shock and Other Injuries

There are three questions which our preliminary examination should attempt to answer:

1. Is shock present, and if so to what degree?
2. Are other injuries present apart from damage to the skull and its contents?
3. What type or types of head injury exist?

The first and second of these considerations are incidental matters, and yet they have a real importance. Shock is the sequel of severe injuries whatever their character, but it is apt to be manifest in head injuries to a disturbing degree. It is the inevitable accompaniment of concussion if the latter is in any way severe, and in actual fact there is no clear line of distinction between the two. We recognize the existence of shock, and we estimate its degree by the appearance of the patient and by the fall in blood pressure, and, having recognized it, we are entitled to draw these conclusions:

1. That in the absence of other injuries the degree of shock is a reasonably reliable indication of the severity of the head injuries.
2. That for the moment the occurrence is not without advantage, for, in so far as it is associated with a lowering of intracranial pressure through reduction of the general blood

*Abridged version of a British Medical Association Lecture given to the Dundee Branch on December 5, 1936.

pressure, it affords a temporary safeguard against bleeding immediately after the accident.

We should treat the shock, but, appreciating that it has a certain salutary influence, we should not employ the intensive stimulating methods that are used in ordinary shock, lest by so doing the blood pressure is unduly raised and intracranial bleeding ensues.

Injuries of other parts must be examined in order that their exact nature may be estimated and the urgency of their treatment decided. The injuries of motor accidents are often multiple, and I have known a man sustain a perforation of his intestine in addition to severe injury to the skull. The preliminary examination should review the general situation and allow of a decision on the relative urgency of the individual lesions.

Possible Head Injuries

Estimating the damage which may have been received by the skull and its contents is, however, the most important part of the examination. Individually or in any combination there may be the following:

1. A simple scalp wound	4. Concussion.
2. A fracture of the skull vault.	5. Cerebral compression.
3. A fracture of the skull base.	6. Cerebral laceration.

The basis upon which this classification is made lacks uniformity—concussion and compression are clinical manifestations, while scalp wounds and fractures are local pathological effects—but I know of no better presentation, and for practical purposes it fulfils our requirements.

When the patient is unconscious we begin the investigation by attempting to estimate the depth of unconsciousness. Such an estimate is a reliable index of the degree of intracranial damage, and provides a standard by which we can contrast further improvement or deterioration. Four tests are applied in the following order.

1. A simple question, such as "What is your name?" spoken in the ordinary tone of voice.
2. An imperative question spoken loudly close to the ear.
3. The induction of a painful stimulus by pressing the finger-nail against the supra-orbital notch.
4. Testing the corneal reflex.

I need not elaborate the details beyond saying that response to the first indicates a comparatively slight concussion, while failure to react to the last shows the deepest degree of unconsciousness compatible with life—cerebral activity is restricted to the automatic action of the vital centres.

Pulse, Respiration, Temperature, and Blood Pressure

It is an almost universal rule that these observations are recorded at two-hourly intervals for the first twenty-four hours and thereafter at four-hourly periods. The pulse readings are particularly important. A quickened pulse of small volume is the usual accompaniment of concussion, but in the absence of complications there is an early readjustment to the normal. A slowing pulse rate accompanied by an increase in volume is a hint that compression is developing, or at least that the vasomotor centres are being stimulated in some way. A quickened pulse rate maintained at high speed and yet showing increased volume is an indication that some degree of cerebral laceration without localized compression exists. These are generalizations, but they are sufficiently constant to justify their recognition as indications of certain pathological changes.

Changes in respiration are less constant, but they too are important. In concussion respiration is slow and

shallow. Irregularity of the respiratory rhythm, and particularly anything suggestive of a Cheyne-Stokes change, is ominous, because it is the response of an increased intracranial pressure. A much-quickened respiration points to serious cerebral laceration, generally of a diffuse character, and most medical men are familiar with the respiratory changes of advanced cerebral compression—the irregular stertorous breathing, which in its origin indicates an increasing bulbar compression and a failure of the vital nuclei in the medulla.

Temperature readings have a significance which is not fully appreciated. Assuming that extracerebral causes have been excluded, a rising temperature means that some degree of cerebral damage has been sustained. A pure uncomplicated concussion does not exhibit pyrexia, and if the sign is present something more local has occurred—a subdural ecchymosis, a cerebral laceration, or a localized compression. I know of few individual features more suggestive than a pyrexial reaction in a head injury, and in general it may be said that the earlier the manifestation and the more intense the reaction the more serious is the cerebral damage.

Blood pressure readings should always be made and the findings recorded at two-hourly intervals for twenty-four hours. In the period which immediately succeeds a head injury of any severity the blood pressure falls; as recovery advances the pressure rises. If it does so gradually and, after rising a few points above the normal readjusts itself to the average figure, the prognosis is good, and the evidences are those of a simple concussion. But if after the preliminary fall it rises to a high level and is maintained at, it may be, 180 to 200 mm. Hg there is ground for anxiety, for this is a reaction which indicates an increasing intracranial pressure. On the other hand, in injuries of extreme severity, particularly such as are associated with extensive compound fractures of the skull vault, the blood pressure may remain at a low level throughout the entire period of the illness.

Bony Injuries

If a scalp wound is present its position and characters are noted, and any haemorrhage should be arrested. By gentle retraction of the scalp wound the condition of the underlying skull can be seen and search made for a fracture, remembering always the risk of mistaking an oozing suture for a fissured fracture of the vault.

The skull vault is then palpated methodically, firm pressure being made with the finger-tips, and any painful reaction is noted. If pain is elicited in the absence of a haematoma it suggests the existence of a fracture. It is possible that the outline of a depressed fracture may be detected, but it is difficult to distinguish between a localized circular bone depression and the softened centre of a haematoma. The mouth, the nose, and the auditory meatus are examined, and, if bleeding is encountered, an attempt is made to distinguish the haemorrhage of local injury from that which arises from damage to the skull base. If blood is escaping from the external auditory meatus its characters are noted—if its consistency is more watery than normal, and if coagulation is delayed, the probability is that cerebro-spinal fluid is mixed with it.

The maxillae and the mandible are examined, and particular attention is paid to the investigation of the temporo-mandibular joints. Fractures of the middle fossa of the skull base may result from a blow upon the lower jaw; in such an instance the force is transmitted through the temporo-mandibular joints, and it is well, therefore, to make this examination a routine procedure.

Pupil Reactions and Tendon Reflexes

Our attention is now directed to the pupils. Two practical considerations are:

1. As a general rule the pupils in the average case of head injury are moderately contracted and react sluggishly to light.
2. If one pupil is widely dilated and fails to react to light, the other presenting a normal appearance and retaining the light reaction, and if the change is noted comparatively suddenly in the progress of the case, one or other of two explanations is likely—a pontine oedema or haemorrhage which is interfering with the activity of the third nucleus, or a cerebral compression developing on the same side as the dilated pupil. The latter is of significance in the recognition of the compression syndrome.

It is sometimes stated that the fundus oculi should be examined by an ophthalmoscope to determine whether a papilloedema is developing. It is true that in the later stages of compression papilloedema may develop, and it is first manifest on the same side as the compression, but, unless the examiner is trained in ophthalmoscopy, he is unlikely to find this method of investigation of practical value.

Examination of the extremities is now proceeded with. Any evidence of twitching is noted, and the limb is moved into various attitudes in order to ascertain whether there is flaccidity, or spasticity. If there is compression in the cortical motor area changes will be manifest in the musculature of the limbs on the contralateral side, at first spasticity and later flaccidity. The tendon, plantar, and abdominal reflexes are examined, but their evidences are not particularly trustworthy in head injuries, for they sometimes appear to be disorganized by severe concussion, and in the absence of any local cerebral lesion. It is inadvisable to base any definite conclusions upon them in the period which immediately follows a head injury.

An x-ray examination of the skull concludes the preliminary examination. There are circumstances in which such an examination may be impossible, but every effort should be made to secure it. It may reveal a vault fracture which has been hitherto unsuspected, and, by outlining the extent and position of the injury, it supplies most important information.

Arriving at a Diagnosis

The practical issue in the diagnosis of a head injury may be summed up in the simple question, "Is a state of cerebral compression developing?" The compressing factor may be general, as, for example, a cerebral oedema, or it may be restricted and local, as in the case of a fragment of bone driven inwards, or an extradural haemorrhage, and yet again—and this is the most significant possibility of all—it may be local yet progressive, so that eventually its effects are manifest throughout the entire cerebral area. A middle meningeal haemorrhage is the classical example of the last possibility, and of all the sequelae of the head injuries it is the one which it is most imperative to recognize, because in such an eventuality early operation is the only means by which life may be preserved. Compression factors which from the beginning are general in distribution are less urgent questions, and there are certain of them which yield to non-operative measures.

In a local sense, if the preliminary investigation has been reasonably complete, one ought to be able to decide whether there is a fracture of the vault or of the base of the skull; whether there is damage to the brain to the extent of concussion, or whether compression, general or local, is occurring.

Fracture of the Skull Vault.—This may be apparent through a scalp wound, or it may be suspected by local

pain on pressure, but the possibility is put beyond doubt by an x-ray examination—a detail of the investigation which should never be omitted.

Fractures of the Base of Skull.—The nature of the injury, the mechanism and the distribution of the force, may lead us to suspect a fracture of the base of the skull. Haemorrhage from skull apertures and cavities, or a delayed ecchymosis in the subconjunctival or orbital regions may help in diagnosis. Pain produced by firm pressure upon the mastoid processes is a reasonably reliable sign in fractures of the posterior and middle fossae. When other signs are absent the severity of the cerebral damage may indicate the possibility of fracture. X-ray examination is rarely helpful—to obtain a reliable picture would necessitate a disturbance of the patient which is not permissible; but sometimes the radiograph of the vault reveals a fracture running towards the base, and with such evidence we may draw reasonably accurate conclusions.

Concussion

The great majority of head injuries examined within a short time after the accident show evidences of concussion; compression develops later, and the time incidence of its appearance depends upon the source responsible. There is some doubt as to the nature of the process by which concussion arises, but for our purpose we may accept the view that a sudden trauma applied to the skull is conveyed to the cerebral tissues, and thus produces a temporary derangement of nuclear activity, and even perhaps a molecular disintegration of the connecting and distributing elements. The result is a depression of the entire mechanism of the central nervous system, with the qualification that its effects vary according to the situation and function of nerve elements. As might be anticipated from its exposed position, the cortex suffers most, and there is a depression of the higher centres which initiate motor and sensory activity, with loss of consciousness in varying degrees. So the process is distributed, but with this saving element, that what might be called the vital nuclei, the centres which control circulation and respiration, automatic to some extent and resistant at all times, are affected last and least.

The clinical appearance resembles that of surgical shock, with the addition, because of the locale upon which the trauma is exerted, of a disturbance of consciousness in varying degree. The state of the patient in the period immediately subsequent to the head injury may be described as follows. He may be unconscious in the first or second degree (a deeper stupor generally means that there is something more than pure concussion); the pulse rate is quickened and of reduced volume, the respirations are quickened and shallow, the blood pressure is reduced, and the temperature is subnormal; the musculature is flaccid, though occasionally a limb will take up a cataleptic attitude; the appreciation of pain is diminished, the skin is pale, and the surface temperature is lowered. This picture represents the state of concussion, or, as some prefer to term it, cerebral shock.

Onset of Compression

A variety of influences may cause cerebral compression, always serious and sometimes fatal—an indriven segment of bone, haemorrhage (subdural or extradural), the oedema which follows injury, or the oedema and congestion which develop at a comparatively late stage and are generally the result of sepsis. In each case the process at work is similar, but for purposes of description we may consider the case of a subdural haemorrhage, the result of a rupture of the middle meningeal artery.

This accident, focal in its beginnings and yet progressive in character, produces a state of affairs which affords perhaps the most illustrative and impressive features of a compression syndrome. When the haemorrhage accumulates to an appreciable extent it constitutes an addition to the already existing volume of intracranial contents, and as the intracranial space is already fully occupied accommodation is supplied by displacing a certain amount of cerebro-spinal fluid from the cerebral to the spinal distribution. The relief is a limited one, and, the compressing influence continuing to develop, the thin-walled cortical veins become affected. To some extent blood is driven out of them, and I suppose an immediate slight further degree of additional accommodation is provided, but again the benefit is short-lived, and presently the pressure on the veins leads to a congestive reaction throughout the brain substance. Compression continuing, and no further accommodation being possible, a zone of pressure anaemia appears in the immediate proximity of the compression. In depth the anaemic area is relatively small, a matter of millimetres, but in surface it will be as extensive as the area in contact with the compressing influence. Anaemia means inactivity of the cortical area concerned, the clinical evidence being a localized paralysis of the area innervated from the cortical field. Coincidentally, and beyond the zone of anaemia, the brain tissue is congested, and, as venous and sinus pressure continues to be exerted, a cerebral oedema develops. Finally, if compression continues to be exerted, the brain tends to become mobilized towards the skull base in the direction of the foramen magnum.

The significance of the change is obvious. There is medullary compression and oedema, gradual failure of the cortical nuclei which control circulation and respiration, and eventually the inevitable result—death from respiratory failure.

Early Symptoms and Signs

The late signs of compression are obvious, but under such conditions operation is generally unavailing. Early recognition is essential if good results are to be secured. If there has been a period of consciousness between the concussion and the onset of compression certain symptoms may be recorded. A severe insistent headache is complained of, there is an intolerance to bright light amounting almost to a photophobia, the mental attitude alters, and the patient becomes restless and excitable, unduly irritable, and even irrational. These are features of much significance, and, if they are noted in the post-concussion phase, they indicate a state of cerebral congestion which may well suggest the onset of compression. Ultimately the patient relapses into unconsciousness, and as insensibility deepens the general and focal signs of compression become evident. In such instances the significance of the changes are generally appreciated, for the relapse is so impressive that it demands recognition and further investigation.

Danger arises when the stupor of concussion passes almost imperceptibly into the unconsciousness of compression, and the latter state develops so gradually that its recognition is overlooked until the opportunity of affording relief is almost past. In this case the unconsciousness deepens, the face becomes flushed, there is restlessness, particularly when any stimulation is applied to the body surface, the pulse rate falls, its volume increases, and blood pressure readings are considerably increased; the respiration rate falls, but there is a coincident increase in the individual excursions; the temperature rises, the pupils are equal and contracted, the urine is retained. These

are the evidences of a general intracranial rise in pressure. If the compressing force is focal in its immediate effects certain local peripheral evidences become manifest, and these are demonstrated in most characteristic fashion when pressure is exerted upon the cortical motor area, a development which arises so typically when the compressing element is a haemorrhage from the middle meningeal artery. As a cortical anaemia develops the musculature of the opposite arm and leg and side of the face demonstrates incoordinated contractions, and with further compression a flaccid paralysis is evidenced. Lastly, if a unilateral delayed dilatation of the pupil appears it is an evidence of compression on the same side as the dilatation.

These, then, are the two aspects of compression, the one general both in distribution and in manifestations, the other focal in its peripheral effects. It is important to appreciate the distinction and to recognize the actual situation, for while the first may be treated, in its initial stages at least, by non-operative measures, the second demands surgical intervention at the earliest moment.

Diagnostic Tripod

Some further evidence is required, however, and we obtain it by lumbar puncture and the readings of a spinal manometer. The normal cerebro-spinal fluid pressure varies within considerable limits, 60 to 120 mm. of water being regarded as average readings. Where compression exists the readings are consistently higher than 120 mm., and they may even reach a height of 250 mm. This is indisputable evidence, and it has the further advantage that it affords a reasonably accurate conception of the degree of the compression. Even if a special manometer is not available lumbar puncture should still be undertaken. A normal cerebro-spinal pressure is revealed when fluid drops from the needle at the rate of one drop per second, and one can draw reasonably accurate conclusions according to the variations which are manifest. Lumbar puncture is a valuable procedure, but I suggest that it should not be practised until we have reason to suspect that compression may be appearing. To adopt it as a routine in the early stages of head injury is probably injudicious. Its evidence at this stage is not reliable, while it may be that the manoeuvre so disturbs the pressure balance that a further cranial haemorrhage is started.

To sum up the position, we are justified in diagnosing compression on three points of evidence:

1. The effects of a general increase of intracranial pressure, as revealed by the pulse, the blood pressure, the respiration, the temperature, the appearance, and general behaviour of the patient.
2. The effects of a local cortical pressure as shown by a developing peripheral paralysis.
3. The reading of the cerebro-spinal fluid pressure.

These are observations which can be made by any medical man, and in general it may be said that they are the foundation upon which the diagnosis of compression is based.

Possible Later Developments

If the concussion period has passed and compression has not developed, so that events appear to be pursuing a natural and satisfactory course, there remain two further possibilities which have to be kept in mind—a delayed compression oedema, which arises in association with cerebral laceration; and a meningo-encephalitis, the result of septic invasion extending from a base fracture which has communicated with the nose or mouth or auditory canal.

The first of these possibilities—compression oedema, a sequel to cerebral laceration—is not unduly serious, but unless we are aware of the possibility we are apt to exaggerate the significance of its development. It generally appears about the fourth or fifth day after the accident, and it is this delayed development which is apt to prove disconcerting. When all appears to be going well, and pulse and temperature have been following normal lines, the temperature rises, the pulse rate falls, and a drowsiness which may pass into complete unconsciousness affects the patient. It is a temporary change. It usually disappears spontaneously, and its progress can be arrested by one or other of the hypertonic medications which are described under treatment. The origin of the change is not fully understood, but in point of time it appears to correspond to the phase when the reactionary changes preparatory to healing of the cerebral tear are developing.

The second possibility—the development of a delayed infection—is an entirely different matter.

Last November a young man was admitted to my wards with a fracture of the middle fossa of the skull; bleeding from the left auditory meatus indicated that the fracture communicated with the inner and middle ears. His immediate recovery was all that could be desired, and sixteen days after the accident was sustained I demonstrated the case to a clinic as an illustration of a satisfactory and encouraging recovery. I alluded to the risks of delayed infection, but I felt satisfied that in this instance at least the risks were past.

I had spoken too soon; the same evening the temperature rose to 99.5° F. and the pulse rate had quickened from 80 to 110. When I saw him next morning it was evident that meningitis had developed, and in spite of our efforts he succumbed four days later to a streptococcal meningitis.

It was an experience which impressed us with the uncertainty which must attend the later progress of certain types of head injury.

Treatment of Uncomplicated Concussion

The injured man is undressed with the minimum of disturbance. He is placed in bed flat on his back, and his head is turned to one side. In this position he breathes more easily; if he vomits he is less likely to aspirate the vomited matter; and if there is bleeding into the mouth the fluid which otherwise would be swallowed will collect in the hollow of the cheek. We assume that the preliminary examination is completed and we have arrived at a provisional appreciation of the condition. At this early stage we may take it that the clinical state is one of concussion; the possibility of compression is in the future, early or remote as its genesis may decide, but at this point it need not trouble us so far as treatment is concerned. Concussion is tantamount to shock, and we treat it accordingly. The body temperature is raised by hot-water bottles or by a shock cradle. Fluids are administered, preferably by mouth, or if that is impossible by subcutaneous or rectal routes. Intravenous methods are inadvisable, for a sudden rise in blood pressure may initiate bleeding, and a too copious supply of normal saline may increase the tendency to oedema of the cerebral tissue. In a desperate case it might be permissible to administer intravenous saline by the "drip" method, but the indications are infrequent. If a scalp wound exists we take immediate steps to arrest the bleeding. I suspect that there are many head injuries in which the haemorrhage from a neglected scalp wound has been the factor that precipitated a fatal issue. A two-hourly pulse, temperature, and respiration chart is recorded, and we keep a close watch upon the state of the bladder. A distended bladder is often the cause of a restlessness on the part of the patient which the physician may ascribe to other more serious influences.

Allaying Restlessness

The patient should be kept as quiet as possible: if he is in a hospital ward he is screened off, if he is in a room by himself the room is darkened. He may become restless; many cases of head injury pass through this phase, and it may be an ominous sign. It may indicate the onset of compression, but in most cases it represents the reaction that is coincident with recovery from concussion. Restlessness presents the first problem in treatment, and means must be taken to allay it, for if it persists it is harmful. Any attempt at physical restraint is resented, and in the effort intracranial pressure rises at a time when this is least desirable. Morphine and its derivatives are contraindicated for several reasons. They are apt to increase the venous engorgement of the cerebral tissue, they depress the respiratory centre, and there is the clinical objection that the resultant hypnosis may disguise the deepening unconsciousness which otherwise would indicate the onset of compression. Experience has shown that chloral hydrate combined with potassium bromide is both efficient and safe. Fifteen to twenty grains of each drug is a suitable amount, and if necessary it may be repeated four-hourly. If the patient cannot or will not swallow, paraldehyde may be administered per rectum; it is given in a dosage ratio of 1 drachm per stone of body weight up to a total of eight drachms. In its administration there is the practical point that it is best tolerated when made up in mucilage of acacia in a proportion of two parts to one of the mucilage. There are instances in which these sedatives are insufficient, and in such an event hyoscine hydrobromide in doses of 1/200 grain hypodermically may be given, but only when less powerful sedatives have failed.

Such is the treatment adopted in the presence of an uncomplicated concussion, and in general it may be said that it is the treatment of choice in the early stages of the majority of head injuries.

Treatment of Fractures

Assume now that an x-ray examination of the skull has been carried out, and it is reported that there is a linear fracture of the skull vault. How does this affect the situation? Generally speaking the finding has no particular significance. It is a complicating feature, and implies some increase in the length of time of the convalescence, and when it is shown to occupy a situation which brings it into relation with the middle meningeal artery it may arouse in our minds the suspicion of haemorrhage from that vessel. These are special considerations, however, and individual attention is paid to them, but it is well to appreciate that the ordinary linear vault fracture is a relatively insignificant lesion, which certainly calls for no variation from the ordinary scheme of treatment.

It may be that the vault fracture is of the depressed variety. In this event are we still to adopt a conservative attitude? The answer is that under certain conditions operation is indicated, as when a scalp wound overlies the fracture, when there is radiological and clinical evidence that the fragment is in-driven, and when we have reason to suspect that the fracture is associated with a subdural or extradural haemorrhage. I do not mean to imply that all of these developments demand immediate operation. The last may be an urgent matter; the first has underlying it the anxiety to prevent intracranial infection, and therefore the sooner it is remedied the better. The decision regarding the in-driven fragment will depend on local circumstances, recognizing, of course, that the

earlier the relief the better for the patient. There is no hard-and-fast rule; but whenever possible operation should be postponed until the effects of cerebral shock have subsided.

Fracture involving the skull base I dread more than any other type of head injury, for the complications which follow in its wake. Fractures of the anterior fossa of the skull are rarely associated with immediate danger, but they are the type in which a delayed sepsis is apt to arise, because of the frequency with which they communicate with the cavity of the nose. Fractures of the middle fossa have dangers which are both immediate and remote—immediate because an extensive haemorrhage so often accompanies the break and because the line of it may involve the sella turcica and endanger the pituitary or its stalk; remote because a communication with the ear cavity may provide the avenue for infection. Fractures of the posterior fossa give us the most immediate concern because of their proximity to the vital area of the medulla, but, on the other hand, they are in large measure free from the risks of secondary infection.

Taken as a group the gravity of base fractures in contrast to fractures of the vault depends in large measure upon an anatomical fact—the relation of the dura to the bone. At the skull base the dura is closely adherent to the bone, so that when a fracture occurs a tear of the dura is almost inevitable. In the vault, on the other hand, dura separates with the greatest of ease except where sinuses exist, and the complication of dural rupture is therefore much less frequent. For the moment the knowledge that a fractured base exists need not call for any variation in treatment; we follow the lines which have been described, and we take particular care to avoid any obvious sepsis in those cavities which lie in relation to the skull base. It is for this reason that we advise the instillation of a few drops of 5 per cent. argyrol into the auditory meatus, and perhaps spray the nose and mouth with the same preparation. Restlessness is evident in many cases of this type, and if it appears should be treated at an early stage by the judicious exhibition of sedatives.

Treatment of Compression

Twenty-four hours having passed, deepening unconsciousness, alterations in pulse, blood pressure, and respiration, and a rising temperature may indicate the onset of a generalized and progressive cerebral oedema. A lumbar puncture is carried out and the cerebro-spinal fluid pressure is estimated. If the reading is less than 200 mm. of water but above the normal reading of 100 to 120 mm. the cerebro-spinal fluid should be drained until a normal pressure is reached: if on the other hand the pressure exceeds 200 mm. it is an indication for more active dehydration by the hypertonic therapy first suggested by Weed and his co-workers. If the patient is able to swallow he is given 2 drachms of magnesium sulphate dissolved in 2 ounces of water, and thereafter at intervals of two hours 1 drachm of the salt in 2 ounces of water is administered until a watery stool is evacuated. If swallowing is impossible 3 ounces of magnesium sulphate in 6 ounces of water is given in an enema. These are hypertonic solutions, and in virtue of their osmotic properties they attract and withdraw fluid from the tissues until an isotonic balance is reached. The cerebral tissues part with the fluid in common with other regions, and the oedema is thereby relieved. It is an efficient procedure; temporary no doubt, but it may prove sufficient to adjust the balance until a more stable state of affairs has been acquired.

Hypertonic solutions such as 50 per cent. glucose or 30 per cent. sodium chloride injected intravenously are powerful dehydrating agents. There must be no evidence of recent bleeding if they are to be employed, for as rapid dehydration occurs a clot may be disturbed and a serious haemorrhage ensue.

Decision to Operate

If lumbar puncture or alimentary dehydration afford relief the situation may be stabilized and symptoms of compression may not recur. If they do reappear and continue to develop, a repetition of the treatment described may be called for. But if all efforts are unavailing, and the compression persists and increases, I think we are justified in deducing that, whatever the cause of the compression, it is not a simple and general oedema. It is more likely to be a haemorrhage, which is slowly but insidiously progressive, and we are now faced with deciding whether or not to operate.

Three general considerations are, first, the operation of a simple cerebral decompression is a relatively easy one; secondly, if there is doubt it is better to operate than to delay and to regret; thirdly, if operation is to be attempted the earlier it is carried out the better. It is my impression that this means of relief is not employed as often as it might be. Too often it is delayed unduly, and, if so, we cannot expect that a local decompression will afford relief to a brain that is completely water-logged.

TECHNIQUE OF OPERATION

If the patient is deeply unconscious no anaesthetic will be required, but there are many advantages in a local anaesthetic such as 1 per cent. novocain to which adrenaline (1 in 200,000 of the solution) has been added. The infiltration is carried along a line which extends vertically upwards from the zygoma one inch in front of the external auditory meatus for a distance of three to four inches.

The superficial tissues are divided, the temporal fascia is incised, and the fibres of the temporal muscle are split and retracted so as to expose the bone of the temporal fossa. The bone is perforated with a trephine or burr at a point about two fingerbreadths above the upper border of the zygoma, and with a nibbling forceps the vault is cut away until an aperture about two and a half inches square is made. It may be that the compressing factor is found to be a haemorrhage in the extradural area, the result of a tear of the middle meningeal artery; if so, we remove the clot and ligature the vessel. Failing this finding, we open the dura in a cruciate manner, taking care to avoid the dural vessels, or, if of necessity they must be divided, picking them up with a fine round needle and stitch, and ligaturing each individually.

This is as much as the average operator should undertake. Any obvious source of haemorrhage should be controlled, but no further intervention should be practised unless the operator has special training in neurological surgery. The operation has achieved what was intended—it has relieved an increasing intracranial pressure and any haemorrhage encountered has been arrested. The dural flaps are left widely separated, the split in the temporal muscle is restored by a few interrupted catgut sutures, the line of the temporal fascia incision is closed with a continuous catgut stitch, and the skin edges are united with silkworm gut or horsehair.

Such are the steps of the operation, and it will be seen that it is a relatively easy and straightforward procedure. I anticipate that I shall be criticized for suggesting that a craniotomy should be performed by those who have no special training in general surgery and none in neurological surgery. I appreciate the criticism, but there are conditions in which the assistance of a specialist cannot be obtained, and in that event it is the man on the spot who has the responsibility and, shall I say, the privilege, of

carrying through a procedure which may be the only means by which life can be saved.

In many instances the subtemporal decompression affords relief; if it fails to do so, and if there is no response to a repetition of the alimentary dehydration, we may consider the advisability of repeating a subtemporal decompression on the opposite side, and if this fails we can but await events, with the satisfaction, however, of knowing that we have done all that it is in our power to avert disaster.

After-treatment

The after-treatment of head injuries may be summed up in the words "Go slow." Lasting harm may be done by an attempt to shorten the convalescence or to agree to an unduly early return to the stress of everyday life. The period of confinement to bed will depend upon the nature and extent of the injury; three to four weeks is a common average. A low diet with abundance of fluid is allowed, and half an ounce of magnesium sulphate each morning is a safe and efficient purgative. It is a good rule to give 10 grains of potassium bromide daily; it lessens the irritability of the central nervous system, and its employment at this early stage may prevent unpleasant sequelae in the future. For insomnia we use luminal (2 grains) or medinal ($7\frac{1}{2}$ grains) or soneryl (5 grains).

Sooner or later there comes a time when the question of resuming ordinary activity has to be considered. This is an anxious decision, and if we are to do the best for the patient it will be made the subject of experiment. A testing time when a graduated and provisional resumption of activity is permitted will be instituted. If there are sequelae in the form of giddiness and headache, mental fatigue, and inability to concentrate, we must recognize these as warnings that the time is not yet opportune, and the patient must be advised to extend his convalescence. This, the "aftermath" of head injuries, is in some respects the most difficult problem of all, but it is scarcely within our sphere for the moment, and I have mentioned it for one purpose only—that our consideration of the subject may be rounded off and reasonably complete.

W. Titkemeyer (*Med. Welt*, February 13, 1937, p. 209) draws attention to recent investigations on tobacco smoke, which consists of tars, nicotine, pyridine bases, the lower aliphatic acids, carbon monoxide, carbon dioxide, and ammonia. Nicotine is still believed to be its most poisonous constituent. Four drops are lethal in one-half to one minute; 40 mg. produce disturbances lasting twenty-four hours; five ordinary cigarettes contain a lethal amount of nicotine. The amount of nicotine absorbed by the body depends on many factors. It is detoxicated in the liver and excreted through the skin and kidneys. The gaseous contents of tobacco smoke are now said to have no effect on the general health. Its deleterious effect on the buccal mucous membrane is believed to be due partly to the raising of mouth temperature, especially in cigar and pipe smokers, and its property of getting saturated with water from the mucous membrane. These factors are more important than the chemical constituents of tobacco in the production of "smokers' catarrh." In surveying the literature the author is struck with the discrepancies in the findings of various authors, and he suggests, in the interests of public health, that a more scientific standardization in the investigation of tobacco smoke be adopted—namely, that the length to which a cigarette is smoked be indicated; that the humidity of the tobacco be noted; that the amount of tobacco smoked per cigarette or cigar be weighed; and that the length of the interval of smoking be accurately timed.

INSULIN SHOCK TREATMENT OF SCHIZOPHRENIA

BY

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The insulin shock treatment of schizophrenia is well established on the Continent; the results, as summarized in Dr. Isabel Wilson's report for the Board of Control, must be regarded as sufficiently encouraging to warrant a trial of the method in this country. The treatment has therefore been instituted at the West Ham Mental Hospital, and the results, in an admittedly limited number of cases, are fully justifying what was initially regarded as in the nature of an experiment. In the preliminary consideration of this treatment the question first to be settled was how far it would be practicable, in view of the complicated technique, within the organization of an ordinary rate-aided mental hospital. It is the purpose of the present communication to show that with a well-disciplined and enthusiastic staff it can be undertaken in the infirmary ward of such a hospital without overtaxing the staff and without any serious interference with ward routine.

Administrative Arrangements

It was decided to use the male infirmary ward, which serves the whole of the male side (593 patients). In this ward there is accommodation for fifty-three patients. In the observation section there are nineteen beds, and two single rooms and a padded room are available. Projecting from this section is a bay of six beds; this was selected for the cases under treatment, with a view to disturbing other patients as little as possible. The male nurses work a forty-eight hour week in two shifts of four nurses each. Each shift comprises a charge nurse, a deputy, and two probationer nurses. The shifts work week about, mornings and afternoons.

Realizing the need for perfect staff co-operation, the two charge nurses had the treatment fully explained to them. The difficulties and dangers were stressed, but good care was taken to reassure them and to work up their enthusiasm. They were then given the Board of Control's Blue Book to read. Incidentally, it may be mentioned that the doctor responsible had had experience of hypoglycaemic states whilst working under W. Wilson Ingram (Sydney) during his researches into the massive dose insulin treatment of diabetes mellitus.

Treatment was begun on one patient, a case of severe catatonic dementia praecox. The whole course was carried through on this patient, no further cases being begun until the treatment had been finished, so that the nurses' interest was not divided. They thus became wholly familiar with the routine and gained confidence. It took a fortnight to work up to the shock dose (140 units). During each of the two weeks the afternoon shift which was not actually doing the treatment that week was repeatedly put through the "drill" (described later).

It was found useful for the doctor to pretend to be the patient, and to act as dramatically as possible the various crises that might be expected. By the end of the fortnight both shifts were perfect in their parts. The value of this training has since been demonstrated by the fact that no emergencies have occurred during the treatments which the nurses have not been able to deal with pending the doctor's arrival. Although a patient's appearance has