SURGICAL PROCEDURES IN GENERAL PRACTICE

This is one of a series of articles contributed by invitation

CONCUSSION AND COMPRESSION

BY

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In the article published last week at page 685 attention was directed to the damage caused by violence to the scalp and the skull; but while these lesions may in themselves be very important and have far-reaching consequences, stress must be laid upon the fact that one of the foremost considerations in all head injuries must be the condition of the brain itself. Our treatment must have as its object not only the preservation of the life of the patient but his protection from untoward after-effects of cerebral injury in the form of headaches, fits, impaired concentration, and other unpleasant sequelae. Increase in motor traffic has caused a corresponding increase in the number of head injuries, and the majority of these are of such a character that some damage to the brain is produced. It is important for us to visualize the nature of this damage, the way in which the brain reacts to it, and the processes involved in recovery when such takes place.

When confronted with a patient who has been rendered unconscious by a head injury we have to decide on three points: (1) whether he has merely been concussed or whether the brain has been extensively damaged; (2) whether surgical intervention is called for immediately or, if the condition is such as not to permit this at the moment, whether operation is likely to be required at a later stage; and (3) how we are to manage the case in order to prevent subsequent interference with cerebral function.

Cerebral Concussion

Professor Wilfred Trotter (1924) has defined concussion as "an essentially transient state due to head injury, which is of instantaneous onset, manifests widespread symptoms of a purely paralytic kind, does not as such comprise any evidence of structural cerebral injury, and is always followed by amnesia for the actual moment of the accident." Avoiding discussion relating to the nature of cerebral concussion, except to state that it is probably of vascular origin and in the nature of an acute and unsustained compressive anaemia, one need only say that this definition accurately describes the condition, which is of everyday occurrence, and that it is well supported by clinical experience. From the definition it will be apparent that the subject of a head injury who is unconscious for half an hour or longer is not suffering from concussion. In such cases we must postulate cerebral damage such as contusion or laceration. It also follows that the patient who can accurately relate the details of the accident and remembers the precise impact which caused his head injury has not been concussed, and the medico-legal significance of this will be obvious.

It would appear that there is a latent interval in the registration of cerebral impressions, and that a sudden interference with the registering mechanism, such as is conceivably produced by a severe head injury, cuts off impressions which have been streaming into the brain for some time beforehand. Thus the cyclist who is concussed may remember riding along a road and seeing a motor-car coming round a corner, and then all is cut off

until he finds himself being attended to on the roadside. This period of amnesia appears to be a kindly provision on the part of Nature, since severe psychic shock might ensue if the precise details of the accident were to remain clear in the mind of the person who was injured. The period of amnesia, which is characteristic of both concussion and gross damage to the brain, must be regarded as a great saving factor.

Contusion and Laceration

The next group of cases we have to consider consists of those patients who, following a head injury, are unconscious for a variable interval; the unconsciousness may last for minutes, hours, or days, or it may even be of weeks' duration. In these cases there is gross cerebral damage, the brain being extensively bruised and possibly lacerated as well. Bleeding into the cerebrospinal fluid may be shown by its intimate admixture with blood when obtained by lumbar puncture. From the very first, also, there may be evidence of local damage to the brain in the form of focal signs such as a monoplegia, hemiplegia, or ophthalmoplegia.

It is important to examine the nervous system as soon after the accident as possible, because focal signs then present have been produced by primary injury of the brain. These signs have a different significance from those which appear later while the case is under treatment, when they may arise through compression of the brain by oedema or by blood-clot and may call for relief by decompression. There is much to be said for the American practice of having medical officers on ambulances, because, apart altogether from the importance of first-aid treatment, particularly in cases of haemorrhage and in spinal and other injuries, an early expert examination in a case of head injury is of value in determining the subsequent management of the case.

Surgical Intervention

Indications for Immediate Operation.—Compound fractures of the vault of the skull, like compound fractures elsewhere in the body, require urgent surgery to protect the patient from infection (see article in last week's issue). With this great exception very few cases of head injury require immediate operation. At this early stage operation is practically never required to deal with intracranial haemorrhage—not because haemorrhages do not occur in these early phases of head injury, but because, when they occur, they are rapid, widespread, are associated with extensive damage to the brain, and are so quickly fatal as to be beyond the art of surgery even if this be at once available.

Indications for Delayed Operation.—In simple depressed fractures elevation should be undertaken when the patient's condition permits, and only after adequate preparation. The indications for operation during the course of treatment are discussed later.

Management of Cases of Brain Injury

After examining the patient to ascertain whether there is a scalp wound, depressed fracture, or clinical evidence of a fracture of the base of the skull (see previous article), we should note whether there are any signs of focal damage to the brain such as facial or limb palsies or ophthalmo-

plegia. The significance of such signs at this stage has already been discussed. They must be regarded as the result of local damage produced by the injury, and constitute as it were the primary or basal clinical picture, for changes in which we must be on the alert.

The patient is put to bed in quiet and darkened surroundings, with the head elevated to reduce intracranial venous congestion, and fluids by the mouth are allowed in only very limited amounts or are withheld altogether. He is given six ounces of magnesium sulphate solution (30 per cent.) by the rectal route. The solution must be slowly and gently introduced, and its administration continued regularly at six-hourly intervals. The reason for this dehydration (by withholding fluids by the mouth and giving rectal magnesium sulphate) is gradually but persistently to reduce the quantity of cerebrospinal fluid within the skull and so give the bruised and torn brain space in which to expand as it reacts to the injury. The watery fluid is incompressible, and so only by its withdrawal or by the opening of the skull can the necessary room for this expansion be obtained. If the damaged brain is allowed to swell and expand the chances of its recovery are very much greater. We obtain evidence of this when we compare the reaction to injury shown by organs such as the kidney and the testis. If the kidney is bruised it swells (its capsule being readily distensible), and although there may be haematuria for a time the organ gradually returns to its normal size and function. The testis, on the other hand, is prevented from swelling because of the inelastic tunica albuginea, which in this sense resembles the cranium; and following a blow such as a kick there is subsequent shrinkage and withering up of the organ, the parenchyma of which undergoes a pressure atrophy because of the restraining and strangling influence of the inexpandable capsule. So it is with the brain: if we carry out persistent dehydration we both check the inevitable reactionary oedema and permit the injured brain to swell within the skull and make such a recovery of function that the patient is free from unpleasant after-effects. This dehydration treatment must be continued for at least ten days, and longer in severe cases (for example, in a case where the patient was unconscious for three weeks it was maintained for over a month), and may then be gradually replaced by giving, by the mouth each morning, doses of from one to four drachms of sodium sulphate while continuing to limit fluid intake.

Restlessness.—Traumatic delirium or cerebral irritation is sometimes a distressing feature of head injuries, and is probably the result of frontal polar contusion and widespread oedema. Since instituting routine dehydration, however, I have seen it less often and in a less intense form. The best drugs for the restlessness are the bromides, chloral and its allies such as chloralamide (20 grains, twice daily), and hyoscine (1/100 grain) given hypodermically. In most cases of brain injury morphine is better avoided because of its depressive effect upon respiration. The gently continued dehydration must be persevered with even if the restlessness of the patient makes it difficult.

Lumbar Puncture.—As a routine this is neither necessary nor advisable. It is occasionally useful in indicating the state of the intracranial pressure and in showing whether bleeding into the subarachnoid spaces has occurred. As a means of withdrawing fluid from the skull and reducing the intracranial tension it is too drastic, and the sudden pressure readjustments made upon the damaged brain are probably harmful. It should therefore not replace the gradual and continued dehydration treatment already advocated. As a means of ridding the cerebrospinal fluid

pathways of blood which tends to set up reactions in the leptomeninges it may be practised with advantage every two to three days in those cases in which the cerebrospinal fluid is intimately mixed with blood. Not more than 15 c.cm. should be removed at a time, and this only slowly—that is, with the patient's head low, and through a needle of small bore.

Operation during Treatment Course

If dehydration has been steadily carried out reactionary oedema, should it occur at all, will be minimal in amount and in its effects, and if focal signs arise during the course of the dehydration treatment we must infer that these are due to clot compression—that is, a steadily accumulating intracranial haemorrhage. It is therefore very important to obtain a neurological clinical picture at the earliest possible moment, and, as previously pointed out, this forms a basal clinical pattern. A monoplegia which is present immediately after a head injury must be regarded as due to damage in the motor area of the cortex or along the course of the pyramidal fibres, caused by the initial tearing or bruising of the brain. On the other hand, a monoplegia arising during the course of dehydration treatment is due to accumulating haemorrhage and usually gradually increases in degree and extent, so that before long it becomes a hemiplegia. Such a focal sign of compression appearing in these circumstances is an indication for craniotomy, to evacuate the clot and relieve the pressure. These signs of local compression which arise subsequently to the primary injury have been aptly termed epiphenomena by Geoffrey Jefferson (1933).

Traumatic Intracranial Haemorrhage

Intracranial haemorrhage following head injury may occur either within or outside the meninges, and very occasionally takes place within the substance of the brain itself. The clinical features are those of focal or general compression of the brain—for example, the presence of paralytic phenomena (a monoplegia becoming a hemiplegia), slowing of the pulse rate, or the onset of stupor which may deepen into coma. Respiratory failure may occur, particularly if the haemorrhage is in the posterior fossa, and the heart may continue beating after respiration has ceased. In such circumstances artificial respiration and an emergency posterior fossa decompression are indicated (Rogers, 1933).

Operation in intracranial haemorrhage is done to relieve the brain of the ill-effects of the steady accumulation of blood-clot. When signs of compression appear craniotomy must not be delayed, and efforts to determine the site of the clot compression must be made. The site of compression is frequently revealed by the presence of contralateral weakness or paralysis of an arm or leg or of both limbs, or by a paralysed pupil on the same side as the clot. Progressive drowsiness in such cases is a warning that craniotomy is needed, because if the patient is allowed to sink into coma from clot compression the results of operation are not nearly so good as if this is performed when the patient is still conscious or merely drowsy. The classical signs of middle meningeal haemorrhage—namely, unconsciousness following a blow, a period of return of consciousness (the lucid interval), drowsiness and loss of consciousness again—are by no means always present, but this form of intracranial haemorrhage will not be overlooked if, when managing the case along the lines here laid down, we are on the alert for signs of compression. We must be on the watch for epiphenomena.

Treatment of Intracranial Haemorrhage

Details of operative treatment are beyond the scope of this article. Blood-clot may be removed through burr or trephine openings or through an osteoplastic flap cut for the purpose. If burr openings are used several may be necessary, and small scoops for removing clot and a stream of hot saline are useful accessories.

Illustrative Cases

The following cases illustrate the application of the principles of treatment which have been considered.

Case 1.—A farm labourer aged 33 was admitted to hospital semiconscious and restless. In a brawl he had received a blow over the left temple from a part of a steel plough. While he was undergoing dehydration treatment cerebral irritation was a prominent feature. Three days after admission, while still semiconscious, he developed a right monoplegia, which soon progressed to a hemiplegia. Intracranial haemorrhage was diagnosed and a left osteoplastic craniotomy carried out. A large amount of extradural blood-clot (83 grammes) from a ruptured middle meningeal artery was removed and the bone-flap replaced. He made a complete recovery.

Case 2.—A schoolboy aged 12, when playing at school one morning, was struck over the left parietal bone by a small stone which had been thrown by another boy. He was only slightly dazed and did not lose consciousness. That night he had a headache, but was otherwise well. During the following week his parents noticed that he was becoming more and more drowsy, and five days after he had been struck he was semicomatose. He was seen by his doctor, who sent him to hospital. Craniotomy was performed at the site of the blow, and outside the dura mater a large mass of blood-clot from a torn middle meningeal artery was found and removed. He regained consciousness shortly afterwards and recovered completely.

Case 3.—A contractor aged 32 received a blow on the head during a fight. He walked to his home. The next morning he suffered from headache and vomited. A week later, because of persistent headache, he was sent to hospital by his doctor. A lumbar puncture revealed lemon-coloured cerebrospinal fluid, the pressure of which was normal. He was kept in bed and placed on dehydration treatment, but was inclined to be anxious and irritable. Fifteen days after his admission he became drowsy and developed left hemiplegia, and examination showed choking of both optic disks. During the day he steadily became more drowsy. Intracranial haemorrhage, probably subdural, was diagnosed, and operation was performed forthwith. The dura was tense and bluish, and when opened a large collection of old blood-clot was revealed. This was removed and the subdural space washed out with saline. He made a complete recovery.

Case 4.—A consulting physician aged 53, who had sustained a head injury in a motor accident, was admitted to hospital comatose and cyanosed, with stertorous breathing and a right monoplegia. He remained unconscious for three weeks. His cerebrospinal fluid was intimately mixed with blood, and a diagnosis was made of extensive cerebral contusion and laceration. X-ray examination showed a fracture through the squamous temporal bone of the right side running into the base. He was given the dehydration treatment to which reference has been made, and was kept quietly in bed. Although in consultation craniotomy was urged by colleagues because of the focal sign (monoplegia) it was not carried out, since it was concluded that as the sign had been present from the first it was caused by the primary contusion. He remained unconscious for three weeks, but made a complete recovery and is now back in consulting practice. There were no indications of an accumulating intracranial haemorrhage in this case. Dehydration was all that was necessary.

REFERENCES

- Jefferson, G. (1933). *British Medical Journal*, 2, 807.
Rogers, Lambert (1933). *Ibid.*, 2, 100.
Trotter, W. (1924). *Ibid.*, 1, 816.

INTERNATIONAL CONGRESS ON RHEUMATISM AND HYDROLOGY

OPENING OF OXFORD MEETING

The International Congress on Rheumatism and Hydrology was held at Oxford from March 28 to 30, and is being followed by the International Congress on Rheumatic Diseases at Bath from March 31 to April 3. The Oxford meeting, which was the fifteenth anniversary of the International Society of Medical Hydrology and the tenth of the International League against Rheumatism, was attended by about 200 representatives. Only one of the several delegates expected from Austria was present, and none from Czechoslovakia and other countries in Central Europe, and it was stated that an announced delegate from Soviet Russia was a political prisoner. But German and French were heard as frequently as English in the discussions. The first words uttered, indeed, were in German, when, at the ceremonial opening at the Sheldonian Theatre, Professor H. Vogt of Berlin proposed the election of Sir Farquhar Buzzard as President.

President's Address

SIR FARQUHAR BUZZARD devoted much of his brief address to describing the part played by Oxford men in promoting the study of hydrology. His first example was Dr. Walter Bailey of New College, Regius Professor of Medicine, who in 1587 described the medical properties of the springs of Worcestershire. After congratulating the editorial committee responsible for the volume entitled *A Survey of Chronic Rheumatic Diseases*, published in commemoration of the bicentenary of the Bath Royal National Hospital,¹ which, he said, was a challenge to the medical profession to spend yet more of its time and energy in attempting to overcome some of the most distressing and disabling scourges of all civilized races, Sir Farquhar Buzzard continued:

"The problem is one—and there are several others in medicine—in which the clinician, the pathologist, the bacteriologist, and the biochemist need all the help and information they can obtain from the student of preventive and industrial medicine, from the statistician, and from the social worker. Some help from all these sources is already available, but insufficient attempts have as yet been made to correlate the contributions and control the results with scientific accuracy. Is it too much to hope that sufficient money may be forthcoming from Government or other sources to establish centres for these studies where the social worker, the statistician, the clinician, and the pathologist may work together under the guidance of experts in preventive medicine and social hygiene?"

He also touched on the timely significance of the fact that experts were present from Germany, France, Belgium, and other countries, conferring in an atmosphere of entire friendliness, seeking only truth, and assured of good will and understanding. "Would that matters of far less importance than health could be discussed in a similar spirit."

Climate in the Causation of Disease

The first discussion, held at the University Museum, was on "Wet and dry climates and weather in the causation of disease." Dr. E. P. POULTON of Guy's Hospital, in an opening paper, said that he had found that to plot absolute humidity against temperature provided a measure of climate which was easier to assess than the more complicated "relative humidity." On this basis he had plotted average monthly values for humidity against temperature, and he showed curves for Calcutta and other places in India and for Kew and various meteorological stations in Great Britain. Each station had its characteristic curve, and could be classified into types according to geographical influences. A hot summer day in England

¹ Oxford University Press. Pp. 346, 31 illustrations. 18s.