THE ANAESTHETIST AND THE EARLY MANAGEMENT OF ACUTE HEAD INJURIES

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SUMMARY

Brain hypoxia during the acute stage of a head injury may result from the injury itself, an inadequate airway, associated injuries and blood loss. The maintenance of a clear airway is a major responsibility of the anaesthetist and some of the difficulties involved are discussed. Artificial ventilation of the lungs will become necessary when other measures have failed to ensure proper carbon dioxide elimination and adequate oxygenation. Intermittent positive pressure ventilation, when properly performed, has no deleterious effects on the brain. The special requirements of anaesthesia for intracranial surgery are re-stated. The value of controlled ventilation and hyperventilation of the lungs as a technique in neuroanaesthesia is briefly reviewed. Reference is also made to the use of hypertonic solutions of urea and mannitol in head injuries and to the use of hypothermia in patients with brain-stem damage.

“For good results . . . following a head injury, a man or woman must have continuous and co-ordinated treatment, from the acute stages of the injury . . .” (Rowbotham, 1945).

It is estimated that 100,000 patients with head injuries are admitted to hospitals in Great Britain each year (Rowbotham, 1964). Of these, some 20 per cent are serious enough to require special treatment (Lewin, 1959a), which in many cases will involve the services of an anaesthetist. Motor vehicle accidents and falls account for the majority of head injuries, and their prevention is an international problem. Assisting in the early treatment of these injuries constitutes one of the anaesthetist’s most important duties. The purpose of this paper is to re-state relevant therapeutic principles, and to discuss the difficulties of their application during the early stages of an acute head injury.

BRAIN DAMAGE AND HYPOXIA

The harmful effects of hypoxia on the brain are summarized in figure 1. The end result of this circle of events is neuronal death.

In the early stages of a head injury, brain damage may be the result of any of the following:

The direct effect of physical trauma. Associated local sequelae, such as an extradural haematoma may, by displacing the brain, also be responsible.

Obstruction of the airway. The obstruction may be at any level of the upper or lower respiratory tract. Hypoxia, carbon dioxide retention, and venous congestion result. The disastrous effects of these on the brain are aggravated by the increased work of respiration due to airway occlusion.

Severe blood loss. Subsequently, anaemic, stagnant and hypoxic hypoxaemia occur, the last being due to an increase in the physiological respiratory deadspace (Freeman and Nunn, 1963).

Additional injuries. Thirty per cent of all patients with head injuries also have other injuries (Lewin, 1961). When severe, they greatly complicate management and worsen the prognosis.
Occasional complications of central nervous system lesions. Examples of these are rupture of the oesophagus (Maciver et al., 1956) and haemorrhage from gastro-intestinal erosions (Cushing, 1932).

These events cause a disturbance in the mechanisms responsible for oxygenating the brain. Respiratory insufficiency assumes special importance in head injuries because it is a major cause of death in those patients who survive the accident (Maciver, Frew and Matheson, 1958; Potter, 1963).

The development of pulmonary infection is an important cause of hypoxia in the patient with a head injury. It is not an immediate cause and is therefore only mentioned. Prompt and efficient management in the early stages is, however, of considerable prophylactic value against the onset of such infections.

THE AIRWAY

Factors which predispose towards respiratory obstruction in the patient unconscious from a head injury have been enumerated by Bryce-Smith (1950) as follows:

**Reflex depression.** The tongue may block the airway; aspiration of material into the lungs can occur readily when the protective cough and swallowing reflexes are obtunded or absent.

**Reduced gastric motility.** This favours vomiting and regurgitation.

**The direct results of the injury.** The airway itself may be traumatized. Blood or cerebrospinal fluid which leaks into the upper airway is liable to be inhaled.

**Pulmonary oedema.** This has been stated to be due to: an abnormally high negative intrathoracic pressure secondary to respiratory obstruction (Mushin, 1955); increased capillary permeability of central origin (Cameron, 1948), or due to hypoxia (Hayward, 1955); and reflex secretion of central origin from the tracheobronchial glands (Andrew, 1956).

**ENDOTRACHEAL INTUBATION**

The benefit of endotracheal intubation in a depressed neurosurgical patient is clearly shown in figure 2. Unnecessary intubation is, however, the potentially dangerous misuse of an excellent therapeutic procedure. Traumatic, infective and reflex complications may occur (Galley, 1959), while endotracheal tubes and their connections can become blocked by various means (Ballantine and Jackson, 1954). More simple methods of providing a clear airway, such as the adoption of the semiprone position and the use of a pharyngeal airway, are frequently effective.

![Fig. 2](image)


Endotracheal intubation may be required:

1. To provide and maintain a clear airway when this cannot be satisfactorily achieved by other means.
2. To isolate the lower respiratory tract from the digestive tract when it is not possible to safeguard the airway with the semiprone position.
3. To aspirate secretions from the lungs if the patient has a "wet" lung.
4. To control or assist pulmonary ventilation.
5. To reduce respiratory deadspace.

Tracheotomy is often performed for the same reasons. Intubation serves as a temporary tracheotomy. It may be a preliminary step immediately preceding a tracheotomy or a temporary measure which will allow the situation to be reassessed later.

Patients may be divided into three groups, according to the clinical signs present at the time of intubation. When there is deep unconsciousness
and flaccidity, intubation is simple and often urgent. In the second group there may be (a) unconsciousness with rigidity and masseteric spasm, secondary either to a hypoxic or decerebrate state, (b) unconsciousness with retention of brisk responses to stimulation, or (c) confusion giving rise to violence. Intubation can be extremely difficult in patients in this group. A mouth gag is helpful but it may be necessary to facilitate intubation by administering a drug which will reduce or abolish muscle tone. In the third group patients are conscious or near-conscious. Intubation or bronchoscopy may be necessary for such reasons as a crushed chest injury, or the inhalation of foreign material. Some form of anaesthesia may be required for patients in this group.

They frequently have full stomachs. For this reason Bryce-Smith (1950) has stated that there is "no justification" for not passing a stomach tube at "the earliest possible moment". This view must be subjected to certain modifications when endotracheal intubation is to be performed.

In the first group, reflexes are greatly depressed and the immediate considerations are to provide a clear airway and to seal off the lower respiratory tract from the digestive tract. There are no indications for passing a stomach tube until the trachea is intubated.

In the second group, there is a varying degree of reflex depression. Nevertheless, stimulation of the pharynx by laryngoscopy or the passage of a stomach tube can cause vomiting. The adoption of a head-down and lateral, or steep Trendelenburg position is an essential preliminary to either manoeuvre in order to lessen the risk of inhalation. If an effective posture cannot be achieved, the passage of a stomach tube is liable to result in the inhalation of vomitus. Alternatively the attainment of an effective posture seems to dispense with the necessity of passing a stomach tube if the trachea is to be intubated. A criticism of the steep Trendelenburg position is that it causes intense cerebral venous congestion. The writer believes that aspiration is even less desirable but that the posture should only be maintained for the shortest possible time. A lateral and head-down position is no improvement as it requires at least a 20° tilt to be effective (Elliott, 1963) and this position is also unsuitable for performing a difficult intubation.

If suxamethonium is used, the problem of regurgitation is introduced. The variety of methods (Wylie, 1963; Inkster, 1963) employed to avoid this complication illustrates how unsatisfactory they are. Many anaesthetists omit this drug altogether when there is a danger of aspiration of stomach contents. Sellick (1961) has described a method of backward pressure on the cricoid, which is a simple and convenient safeguard against regurgitation. This manoeuvre, however, is not an invariable alternative to the passage of a stomach tube, nor was it originally described as such. When there is a considerable risk of regurgitation, there is no substitute for the passage of a wide-bore oesophageal tube. Patients with a head injury fall into this category when, as is frequently the case, they have recently consumed large quantities of fluid. If Sellick's manoeuvre is to be used, it will become necessary to withdraw temporarily the oesophageal or stomach tube. Preparations for dealing with regurgitation, and the avoidance of inflation of the lungs during the period of the apnoea until intubation is performed, are precautions that must never be neglected.

Another but minor disadvantage of suxamethonium is that, in the rare event of a prolonged apnoea occurring, a valuable sign in the assessment of the patient is lost.

Chlorpromazine has also been used to facilitate intubation (Maciver et al., 1958; Matheson, Thomson and Whitby, 1959). This drug is of considerable value in the management of the decerebrate patient with hyperpyrexia (Maciver et al., 1958) and the violently confused patient (Lewin, 1958; Muskat, 1964). Advantage may be taken of its muscle relaxant properties when such patients require intubation. Chlorpromazine and similar drugs are, however, central nervous depressants. They are capable of causing arterial hypotension and tachycardia, even in small doses, and are not rapidly eliminated. The decision to use them is not, therefore, merely an anaesthetic one. It is unwise to use them for intubation purposes alone, at an early stage, such as on admission, when diagnosis may be in doubt and overall assessment difficult.

Blind nasal intubation avoids the difficulties associated with the use of these drugs. If passage of the tube through the nose is possible, intubation of the larynx is often comparatively easy.
In the third group, when intubation or bronchoscopy becomes necessary, general anaesthesia may be required. Although these patients are conscious, preliminary posturing to avoid inhalation is essential when passing a stomach tube; the respiratory insufficiency which calls for intubation will imply that coughing cannot effectively be carried out. When respiratory insufficiency is severe, further depression of respiration may be fatal if effective artificial ventilation of the lungs proves impossible. Regardless of the patient's state of consciousness, intubation should be performed without the aid of any drug capable of producing respiratory depression.

No matter how the problems of intubation in any of the three groups of patients are dealt with, efficient management demands a tipping table or trolley, upon which every head injury patient is placed on admission, and an efficient suction unit equipped with a metal pharyngeal attachment as well as catheters. Pre-oxygenation is a desirable preliminary step to intubation by whatever method is chosen.

TRACHEOTOMY

Endotracheal intubation is only a temporary measure. Mucosal inflammation and ulceration, in the area of the arytenoids and posterior ends of the vocal cords, is the price of long-term intubation. Blockage of the lumen by secretions and kinking are other hazards (Robbie and Feldman, 1963). As soon as it becomes evident that the status of the patient will not significantly alter for some considerable time, there is little point in not performing a tracheotomy as soon as it can be conveniently arranged. It is impossible to state the exact period over which an endotracheal tube can safely be left in situ. In 1950 Bryce-Smith advocated the use of tracheotomy in the management of the patient with a severe head injury. At this time tracheotomy was a relatively unfamiliar procedure. He stated that an endotracheal tube should be retained for no longer than 24 hours. A more recent view is that of Maciver and his colleagues (1958) who consider that the tube should be removed after 8 hours. Lewin's opinion of similar date (1958) is that "a decision for tracheotomy should in most instances be taken", within 24 hours. Although endotracheal tubes have been retained without harm for longer periods, 24 hours appears to be a reasonably acceptable upper limit when dealing with patients with head injuries. By this stage the patient's condition may have improved sufficiently to give rise to doubts about the necessity of doing a tracheotomy. It is wise to proceed to tracheotomy in such a patient unless the doubts warrant removal of the endotracheal tube.

Simple methods of maintaining the airway play an important part in the management of any unconscious patient. The head injury patient, who although unconscious, has no other injury, no leakage of blood or cerebrospinal fluid, and no sign of any lung involvement, may be treated conservatively (Lewin, 1959b). The test of such management occurs when the injury is severe and unconsciousness prolonged. Many patients of this type eventually require intubation and tracheotomy. Intubation of all deeply unconscious patients on admission, followed when necessary by tracheotomy, has been recommended (Maciver, Frew and Matheson, 1958). The inference is that conservative airway management is unsatisfactory in these patients and that negative clinical and radiological examinations of the chest can be misleading. It is not easy to decide whether it is better to perform intubation and tracheotomy in the absence of any indication other than the presence of a severe head injury and unconsciousness, or to wait until conservative methods of maintaining the airway fail. One régime avoids unnecessary periods of hypoxia and the other unnecessary tracheotomy.

CONTROLLED PULMONARY VENTILATION

The onset of apnoea in a patient with head injury is of sinister significance. An irreparably damaged respiratory centre is not an indication for artificial respiration. It is not always immediately apparent that irreversible brain damage is the cause of the apnoea. In such cases it is necessary to institute artificial pulmonary ventilation until the outcome is certain. In head injuries, therefore, either alone or when combined with some other injury such as a crushed chest, artificial respiration will be required if, despite earlier measures, ventilation remains or becomes inadequate. Among the earlier and concurrent measures that may be necessary in addition to maintaining a clear airway are drainage of a pneumothorax or haemopneumo-
Thorax, blood transfusion and neurosurgical intervention.

Although controlled ventilation precludes the use of a valuable sign in the assessment of the patient, there is little difficulty in deciding to use it when the need is obvious. Less obvious cases require more definite indications of ventilatory status than clinical impressions. Estimations of the mixed venous and arterial carbon dioxide tension are of considerable value in supplementing this information. These can be easily and repeatedly determined, using the rebreathing method described by Campbell and Howell (1960). Alternatively, biochemical analysis of an arterial blood sample will enable the oxygen and carbon dioxide tensions to be estimated and disturbances of acid-base balance detected. This information permits a more accurate analysis of the patient's ventilatory status to be made than if reliance is placed on the arterial carbon dioxide tension alone, and allows more accurate treatment to be instituted. Facilities for such analyses are now becoming generally available.

If controlled ventilation is to be employed, it may become necessary to paralyze the patient's muscles in order to facilitate control. The use of an opiate to depress respiration centrally is not recommended in head injury especially if severe. The administration of tubocurarine does not unduly compromise the neurological assessment of the patient if the latter is carried out when the effect of the most recent dose of tubocurarine is wearing off. In addition, if it is possible to hyperventilate the lungs, the amount of tubocurarine given can be kept at a minimum. Hyperventilation will also have the effect of reducing any rise of intracranial pressure which has resulted from hypercarbia (Ueyama and Loehning, 1963; Bozza, Maspes and Rossanda, 1961). Hyperventilation has been criticized on the grounds that the resulting cerebral vasoconstriction may lead to cerebral hypoxia (Clutton-Brock, 1957), even in the presence of an oxygen enriched atmosphere (Sugioka and Davis, 1960). This is difficult to understand, as the brain normally only extracts 20–25 per cent of the oxygen presented to it (Eckenhoff, 1963), and the brain oxygen consumption does not rise during passive hyperventilation (Kety and Schmidt, 1946). Although Sugioka and Davis (1960) measured brain oxygen tensions directly, for technical reasons their results can be questioned (Robinson, 1962). Cain (1963) has demonstrated that there is no increased production of excess lactate by the brain during hyperventilation in anaesthetized animals and therefore no evidence of cerebral hypoxia even at carbon dioxide tension values of 10 mm Hg.

Intermittent positive pressure ventilation of the lungs causes a rise in the mean airway pressure. The harmful effects on intracranial tension which may result can, theoretically, be reduced by the introduction of a negative (subatmospheric) phase. The benefits of the latter have not, however, been convincing when compared with properly performed intermittent positive pressure ventilation (Bozza, Maspes and Rossanda, 1961; Bozza Marrubini et al., 1964; Brown, 1959; Hunter, 1960; Lundberg, Kjalquist and Bien, 1959). This may be related to the fact that a rise in the arterial blood carbon dioxide tension causes an increase in the size of the vascular component of brain volume and the finding that the use of a negative phase causes a decrease in lung compliance and an associated increase in the physiological dead-space (Watson, 1962a, b). In addition a negative phase will encourage any tendency towards pulmonary oedema. In patients with cardiovascular decompensation a negative phase may be used with advantage but, again, properly performed intermittent positive pressure ventilation minimizes the likelihood of any harmful effects.

**Surgery**

It is the anaesthetist's duty to make himself acquainted with the neurological and general condition of the patient pre-operatively. Evidence of some other injury may become apparent at any stage. Deterioration in the patient's neurological state can occur rapidly and early detection is impossible unless scrupulous attention is paid to the vital signs. If abnormal signs are looked for, the anaesthetist will not be taken unawares.

The problems of intubation in the anaesthetic room are the same as in the casualty department. The most important factors in limiting cerebral oedema are complete oxygenation of the brain and the avoidance of hypercarbia. General anaesthesia, when required, needs to be at the lightest possible level but not so light as to cause coughing or straining, because of the close connection
between the mechanics of respiration and intracranial tension. Strict precautions are necessary to ensure that the patient's position on the operating table does not interfere with the patency of the airway, the venous return from the brain, or pulmonary ventilation. A decision to use controlled ventilation will be based on a number of factors. Among these are the operation proposed, the neurological condition of the patient, and the value placed upon the use of spontaneous respiration as a clinical sign. During intracranial surgery it may be necessary to take additional measures to reduce brain volume and intracranial tension in order to facilitate surgical access and exposure. If ventilation is being controlled, artificial hyperventilation may be used for this purpose (Bozza, Maspes and Rossanda, 1961; Bozza Marrubini et al., 1964; Lundberg, Kjallquist and Bien, 1959). Hyperventilation reduces only the vascular component of brain volume; the brain tissue volume remains unaltered. It could be assumed therefore that hyperventilation would be most useful in reducing any rise of intracranial pressure that has been due to hypercarbia. This is in fact true as shown experimentally in animals by Rosomoff (1963). Furthermore, Rosomoff (1963) has demonstrated that hyperventilation has a negligible effect on intracranial pressure when the brain is normotensive and in the absence of hypercarbia, because an increase in cerebrospinal fluid volume occurs to compensate for the cerebral vasoconstriction. Recently Bozza Marrubini et al. (1964) have analyzed their clinical experiences during neuro-anaesthesia for patients with space-occupying lesions. They found that hyperventilation was of considerable value in reducing a raised intracranial pressure even though hypoventilation was not in evidence preoperatively. An explanation may lie in the fact that such patients are prone to develop respiratory depression under anaesthesia. It is also possible that the normal compensatory increase in cerebrospinal fluid volume does not occur in the presence of a raised intracranial pressure (Lundberg, Kjallquist and Bien, 1959). As stated above, however, any reduction of brain volume and intracranial pressure produced by hyperventilation is necessarily limited by its lack of effect on brain tissue volume.

An effective means of reducing brain tissue volume and intracranial pressure is the intravenous administration of hypertonic solutions of urea (Stubbs and Pennybacker, 1960) or mannitol (Wise and Chater, 1962). The effect of these substances on the brain is dependent upon the creation of an osmotic gradient between the blood and the brain (Shenkin, Goluboff and Haft, 1962). This in turn depends not only on dosage but also on the rate of infusion, as both substances are excreted by the kidney and urea diffuses into the tissues. The recommended dose of 30 per cent urea is 1 to 1.5 g/kg body weight given over a period of 60 to 120 minutes (Javid, 1958; Stubbs and Pennybacker, 1960). However, a dose of 0.5 g/kg administered in 15 minutes has given satisfactory results (Shenkin, Goluboff and Haft, 1962). The larger dose of urea is frequently given more rapidly than recommended, but the sudden increase in blood volume which occurs (Coleman and Buckell, 1964) may cause circulatory collapse in older patients (Shenkin, Goluboff and Haft, 1962) and those with cardiac disease (Watkins, Stubbs and Lewin, 1961). On the basis of molecular weight the dose of mannitol (20–25 per cent) with an osmotic effect equal to 1.5 g urea per kg body weight should be 4.5 g. Urea, however, does not remain in the extracellular space and mannitol, which does, exercises a correspondingly greater osmotic effect (McQueen and Jeans, 1964). In addition, mannitol is not reabsorbed by the renal tubules and produces, therefore, a more rapid diuresis. McQueen and Jeans (1964) compared the effect on dogs' brains, of urea 1.5 g/kg administered in 90–120 minutes with mannitol 1.5 g/kg administered at the same rate. They found that mannitol produced relaxation of the brain to almost the same degree as urea and lasting twice as long, and that there was considerably less rebound rise of intracranial pressure using mannitol. During neurosurgery Wise and Chater (1962) use a dose of 2.5 to 3 g mannitol per kg body weight given over a period of 60 to 80 minutes while Shenkin, Goluboff and Haft (1962) give 1 g/kg in about 15 minutes. In the writer's experience there is a tendency in neurosurgery to administer urea or mannitol fairly rapidly in order that optimum conditions will exist when the dura is opened. The danger of giving the larger dose of urea rapidly has already been mentioned. Pulmonary oedema has occurred during neurosurgery
in a child, following mannitol 1.5 g/kg body weight administered in about 30 minutes. (Casey Smith, personal communication, 1963). This dose when administered in 10–20 minutes causes an increase in blood volume of the order of 40 per cent (Coleman and Buckell, 1964). It is therefore considered that a dose in excess of 1 g mannitol per kg should not be infused in less than 20–30 minutes without careful assessment of the neurosurgical requirements and the ability of the patient to compensate for a sudden large increase in blood volume. Both urea and mannitol are contraindicated in the presence of severe renal disease.

Thirty per cent urea is highly irritant and leakage into the soft tissues will cause necrosis and sloughing. Venous thrombosis is another hazard; a large vein should be chosen at a site well away from any traumatized area; a cut-down cannula should not be used for the infusion, as a high concentration of urea will be deposited in one part of the vein and subsequent thrombosis is almost inevitable (Watkins, Stubbs and Lewin, 1961). As far as can be ascertained mannitol is non-irritant (Wise and Chater, 1962).

Controlled ventilation of the lungs using tubocurarine, nitrous oxide and oxygen, has been introduced into neuro-anaesthesia as a preferable alternative to techniques which allow the patient to breathe spontaneously (Furness, 1957). Consistently good operating conditions and rapid postoperative recovery are major advantages of the technique. It is now widely used for many neurosurgical procedures (Ballantine, 1963; Bozza et al., 1961, 1964; Gallool, 1960; Hunter, 1960; Jones, 1960; Mortimer, 1957; Smith, 1960), an exception being posterior cranial fossa surgery (Ballantine, 1963; Bozza Marrubini et al., 1964; Hunter, 1960). In the latter instance the information obtained from the observation of spontaneous respirations is regarded by many as being invaluable. It seems fairly certain that the value of controlled ventilation in neuro-anaesthesia is mainly the result of effective carbon dioxide elimination (Bozza et al., 1964). Unlike spontaneous respiration this function of controlled ventilation is uninfluenced by the neurological condition of the patient. There are various degrees of hypoventilation. At one end of the scale the resultant hypoxia and hypercarbia cause brain damage and produce hazardous operating conditions. The treatment is artificial ventilation of the lungs. At the other end of the scale, temporary minor degrees of hypoventilation under anaesthesia with good oxygenation do not seem to harm the brain, as shown by the success over the years of techniques using spontaneous respiration. In such cases, good operating conditions may be provided by a number of methods, among them the relatively simple technique of controlled ventilation or hyperventilation. Intracranial surgery performed during the acute stage of a head injury is for the most part of a relatively minor nature and occupies little time. When general anaesthesia is required, there seems to be little justification in abolishing spontaneous respiration unless hypoventilation, because of its degree of persistence, gives cause for concern. For the less commonly performed and more major operations not involving the posterior cranial fossa, in which a substantial area of the brain is exposed either by surgery or trauma the writer generally used controlled ventilation with a muscle relaxant. Light anaesthesia can be maintained indefinitely without the risk of coughing or straining. (Topical analgesia of the larynx is considered to be unwise because of the danger of aspiration.) Post-anaesthetic recovery is rapid and an accurate assessment of the patient can be made without delay. During surgery, tracheal suction can be performed when necessary with minimal disturbance. Hyperventilation provides good operating conditions but if the brain is severely contused the more effective urea or mannitol will also be required. A correct decision to use controlled ventilation can, however, only be arrived at by considering each individual patient. To quote Bozza and her associates (1961), “the systematic and indiscriminate use of controlled respiration in all intracranial operations is definitely inadvisable”.

**FURTHER CONSIDERATIONS**

The intravenous administration of hypertonic urea or mannitol has a limited place in the management of head injury patients outside the operating theatre. Only a minority of patients benefit from such treatment (Watkins, Stubbs and Lewin, 1961). Urea or mannitol should only be used when there is clear evidence of a raised intracranial pressure and when the presence of an
intracranial clot has been excluded by burr holes or angiography.

Induced hypothermia has been used to help reduce the high mortality associated with severe head injuries involving the brainstem (Drake and Jory, 1962; Hendrick, 1959; Lazorthes and Campan, 1958; Sedzimir, 1959). Hyperpyrexia, tachycardia and a decerebrate posture are common clinical features of such injuries, but other patients may be flaccid, thermolabile and apparently moribund. Any surgically remediable condition, such as a haematoma, that may be responsible for damaging the brainstem by displacement must be treated. Hypothermia lowers the rate of brain metabolism and therefore diminishes its oxygen requirements. It also reduces cerebral oedema and therefore diminishes its oxygen requirements. It also reduces cerebral oedema and the tissue response to injury. The efficacy of hypothermia in increasing the survival rate of patients with direct brainstem damage, noted by the above workers, has been confirmed experimentally by Rosomoff (1959) who demonstrated that it was an effective means of minimizing the harmful effects of experimental brain injury in dogs. If used early and if the lesion is not too severe, it was observed to have a beneficial effect on mortality (Rosomoff et al., 1960; Rosomoff, 1959). Hypothermia may be induced with refrigerated blankets or by what Sedzimir (1959) calls "exposure therapy". Fans and a wet sheet are employed in the latter technique and ice if necessary. Chlorpromazine or promazine are used to promote vasodilatation and suppress shivering. Hypothermia is not without its disadvantages. Decreased resistance to infection is an important and lethal hazard (Drake and Jory, 1962; Sedzimir, 1959). Gastro-intestinal haemorrhage has also been reported (Drake and Jory, 1962), but this is a complication of lesions of the central nervous system (Cushing, 1932) and there seems no reason to blame hypothermia. With care and experience, and using moderate degrees of hypothermia (34°C), the masking of neurological and other clinical signs which occur is not a serious drawback (Sedzimir, 1959).

It is somewhat surprising fact that in a major neurosurgical centre in this country, hypothermia is not employed in the treatment of head injuries (Maciver et al., 1958), while in another, earlier enthusiasm for its use has waned (Sedzimir, personal communication, 1964). The writer has never seen it used during some three years at a large accident centre. Instead, normothermia, which protects the brain from the harmful effects of hyperpyrexia, is preferred. The reason is that the marked clinical improvement produced by cooling patients with brainstem damage, becomes apparent in the majority of cases when the temperature has reached normal (Sedzimir, personal communication, 1964). This statement is supported by the mortality rate for Maciver's (Maciver et al., 1958) series (26 patients) of head injuries in which normothermia was used. Although reliable comparisons cannot be made, his figure of about 40 per cent is as good as and possibly better than the combined mortality rate of three published hypothermia series (98 patients), which was around 50 per cent (Drake and Jory, 1962; Lazorthes and Campan, 1958; Sedzimir, 1959). It is worthy of note that the earlier cases of Drake and Jory (1962) were cooled to between 28°C and 33°C. Later on, however, they found that if levels between 33°C and 36°C were employed the patients improved clinically to the same extent.

The modern intensive treatment of severe head injuries has enabled patients to survive who would ordinarily have died. A proportion of these patients will, however, never again live normal lives and some will be grossly disabled mentally and physically. This depressing fact is no justification for withholding treatment since the initial severity of the injury provides no reliable indication as to which patient will preserve mental and physical faculties (and to what extent) and which will not (Sedzimir, 1959). The fact that some 50 to 70 per cent of survivors (Drake and Jory, 1962; Hendrick, 1959; Lazorthes and Campan, 1958; Maciver et al., 1958; Sedzimir, 1959) recover without mental or physical disability, underlines the value of the modern treatment of severe head injuries.

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REFERENCES


L'ANESTHESIE ET LES PREMIERS SOINS DANS LES TRAUMATISMES AIGUS DE LA TETE

SOMMAIRE

L'hypoxie cérébrale au stade aigu d'un traumatisme de la tête peut résulter du traumatisme lui-même, de l'insuffisance des voies respiratoires, de lésions associées et d'une hémorragie. Le maintien des voies respiratoires libres est une responsabilité majeure de l'anesthésiste, et on discute quelques unes des difficultés que cela comporte. La ventilation artificielle des poumons deviendra nécessaire en cas d'échec des autres mesures dans l'élimination convenable du gaz carbonique et l'oxygénation suffisante. La ventilation en pression positive intermittente, quand elle est bien réalisée, n'a pas d'effets délétères sur le cerveau. On cite de nouveau les besoins spéciaux de l'anesthésie dans la chirurgie intrac-ranienne. On fait une brève revue au sujet de la valeur de la ventilation contrôlée et de l'hyperventilation des poumons en tant que technique de neuro-anesthésie. On fait mention aussi de l'usage des solutions hypertoniques d'urée et de mannitol dans les traumatismes de la tête, et de l'usage de l'hypothermie chez les malades présentant une lésion du tronc cérébral.

ZUSAMMENFASSUNG