POSTOPERATIVE CARE OF THE NEUROSURGICAL PATIENT

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The anaesthetist can make an important contribution to the care of surgical patients after operation, particularly in the field of neurosurgery where complications can have such a catastrophic effect. This account will discuss the period immediately after operation and also events and some rationale of care in later management when the anaesthetist will continue to play an important role.

In this Institute all major cases, including craniotomies and head injuries, are transferred to the neurosurgical intensive care unit (ICU). The anaesthetic recovery room receives spinal cases, shunts and patients who have had investigative procedures requiring general anaesthesia, but some of the latter, who are considered “high risk” neurosurgical cases or have complications in other systems, are transferred directly to the ICU. Such a unit, having efficient monitoring and highly trained personnel including the anaesthetist with his expertise in respiratory problems, can help to ensure smoother, quicker and more complete recovery of patients.

Much of the research in the “high risk” neurosurgical patient has been carried out during the operative procedure and in this issue Greenbaum (1976) and Horton (1976) have discussed the adverse effects of both techniques and anaesthetic agents on intracranial pressure (ICP) and cerebral perfusion pressure. Yet in many ways the patient is in a better situation during the operation than after operation, being adequately ventilated and oxygenated, decompressed intracranially and having, in most cases, a well-controlled arterial pressure, heart rate and rhythm. With the availability of an ICU, the combined skills of surgeon, anaesthetist and nursing staff can improve care after operation and ensure, where possible, the smooth recovery of the patient.

In broad terms, after operation the neurosurgical anaesthetist is particularly concerned with level of consciousness, intracranial pressure, respiratory problems, changes in the cardiovascular system including arterial pressure, control of temperature, fluid balance, nutrition and prevention of infection.

LEVEL OF CONSCIOUSNESS

Teasdale (1976) has described in this symposium a scale of assessment of coma. Following operation this has proved to be a simple useful guide in the day-to-day management of the patient. In his assessment the clinician must be aware of the contribution made by anaesthesia, which can be considerable. This may cause confusion of neurological assessment when, for example, a ganglion-blocking drug has been given during operation causing fixed dilated pupils for several hours. The addition of anaesthesia to depth of coma may not always be undesirable, though at one time it was the aim to keep anaesthesia as light as possible in neurosurgical procedures. Several authors have written recently about techniques of metabolic depression and barbiturate protection (Yatsu et al., 1972; Smith et al., 1974), particularly in intracranial vascular operations, which led Larson and colleagues (1974) to comment that the desire for rapid emergence from anaesthesia may become a secondary consideration. C.n.s. metabolic depression should probably be the aim in neuroanaesthesia, even if it means only a good effect on rCBF and therefore ICP. It is particularly important at this time when there is less concern about having patients as awake as possible at the end of the operation and when surgeons are not averse to the idea of ventilating patients in the period following surgery.

INTRACRANIAL PRESSURE

It has already been suggested that patients may fare better during operation than in the period immediately after surgery, and this is particularly noticeable in situations where no decompressive procedure has been carried out, for example biopsy of tumour or some investigative procedures under general anaesthesia. Leech, Barker and Fitch (1974) and Jorgensen and Misfeldt (1975) have demonstrated increases in ICP in neurosurgical operations at the termination of anaesthesia and have commented that the danger may be greater in these patients who have had no surgical decompression. A knowledge of ICP can be of great value in management after surgery of these cases and in situations where drugs have been administered and controlled ventilation instituted.
Nowadays it is common to monitor ICP, and Turner and McDowall (1976) have described various techniques in this symposium. Lundberg (1960) has emphasized the importance of immediate diagnosis and treatment which is possible with ventricular fluid pressure (VFP) monitoring and is, in some cases, lifesaving. There is, of course, the danger of infection which limits monitoring time, but G. M. Teasdale (personal communication, 1976) has recently made use of a closed system which includes an Omaya reservoir through which a small butterfly needle can be inserted at any time when VFP monitoring is required. A similar needle can be inserted into an artery, thus allowing calculation of cerebral perfusion pressure (that is, mean arterial pressure minus mean ICP).

Another useful measurement in the period following surgery is ventricular pressure response (VPR), which Miller and Garibi (1972) studied in patients by inducing small changes in volume in the lateral ventricle. They added 1 ml of saline or withdrew 1 ml of c.s.f., in some cases obtaining an early warning of the stage at which small increases in volume cause large increases in ICP. They were thus able to get a measure of brain tightness or elastance (inverse compliance). Leech and Miller (1974a) have shown that mannitol significantly reduces both VFP and VPR in primates with intracranial hypertension, the reduction of VPR being greater than that of VFP. Similarly, the reduction of VPR was more marked than VFP reduction 24 h after the start of steroid therapy. Their results suggest that both forms of therapy can reduce VFP and make the intracranial contents less susceptible to steep increases in pressure with the addition of volume to the cranium. Mannitol, in fact, having a double action in reducing brain water volume and periventricular elastance, provides a more satisfactory method than hyperventilation in protecting the brain against the dangers of increased ICP. These authors also showed that hyperventilation had less effect on elastance, although VFP is reduced with hypocapnia. Furthermore, Rowed and colleagues (1975) suggested that any beneficial effect of hypocapnia on ICP is likely to be only temporary.

**PROBLEMS OF VENTILATION**

In all patients with brain damage it is essential to have a good airway and this is particularly important in the period after operation. The patient may still be anaesthetized to a degree which will allow him to retain an endotracheal tube without respiratory depression. Blood-gases should be checked at the end of operation when spontaneous respiration has been established, and if hypoxia or hypercapnia is present then artificial ventilation may be established. The use of depressant drugs during operation can result in arterial hypoxaemia as a result of alveolar under-ventilation. These drugs can also result in the patient depending on a hypoxic drive with the danger of further respiratory depression by oxygen therapy (Leigh, 1975). Another important factor after prolonged neurosurgical operations is diffusion hypoxia, which could conceivably last for a longer period than is usually expected. If there is a need to increase the arterial tension of oxygen \( (P_{\text{aO}_2}) \), then it may be necessary to commence artificial ventilation which will allow easier control of inspired oxygen. However, every effort should be made to avoid the dangers of oxygen toxicity which may result from the use of high percentages of oxygen (Winter and Smith, 1972).

There are certain operations following which it may be desirable for the patient to retain his endotracheal tube for other reasons. This is the case with reconstructive cranio-facial operations when a tracheostomy has not been performed electively. In posterior fossa explorations, the lower cranial nerves may be traumatized, particularly in dissection of angle tumours, making it necessary to have the airway protected for a period by retention of the endotracheal tube or tracheostomy. In these cases, a nasogastric tube should also be passed in order to aspirate stomach contents during the first critical 24-h period; it can later be used for nutrition or for the administration of antacids. A useful monitor in these cases is the impedance pneumograph, which will give a warning signal if the patient stops breathing or if a ventilator ceases to function. An output can be taken from the monitor and displayed as a pattern of ventilation in spontaneously breathing patients, and this can be correlated with other measurements, for example VFP (North and Jennett, 1974).

Artificial ventilation may be necessary in patients with brain damage for other reasons, which include impairment of lung function, pulmonary oedema, multiple injuries, status epilepticus and apnoea.

*Impairment of lung function* may itself be a consequence of brain damage, and a decrease in \( P_{\text{aO}_2} \) an increase in minute volume and a reduction in \( P_{\text{aCO}_2} \) are frequent accompaniments, often despite tracheostomy and care of the airway. The patient who has suffered a head injury may have surgery for craniotomy or for other reasons and is likely to have...
special respiratory problems in the period after surgery. Denny-Brown and Russell (1941) have shown in head injuries that inhalation of secretions or vomitus occurs at the time of the injury because the larynx becomes incompetent and swallowing reflexes are interrupted. The inhalation of gastric contents can cause a severe inflammatory response in the alveolar membrane (Mendelson, 1946) and the presence of irritant material below the laryngeal inlet can result in an increase in airway resistance because of reflex bronchial narrowing. Brackett (1971) demonstrated generalized patchy areas of atelectasis which may have been the result of depressed ciliary activity. These cases can benefit from a period of artificial ventilation.

So, also, may cases of pulmonary oedema which can occur as a pathophysiological haemodynamic response to increased intracranial pressure. Ducker (1968) found that increased ICP was the only common aetiological factor in 11 young patients with pulmonary oedema. Permanent relief occurred in one patient by the reduction of ICP, which may indicate that VFP monitoring is of value in these cases as a therapeutic as well as a prognostic guide. An increase in end expiratory pressure (PEEP) will increase intra-alveolar pressure which is likely to oppose the passage of fluid from the capillaries into the alveoli and thereby improve the transfer of oxygen (Russell, Morgan and Lumley, 1971). However, a study by Esteban and colleagues (1973) showed that the effect of PEEP in the management of pulmonary oedema was variable and that it was not easy to predict which patients would benefit. There could also be an adverse effect on cardiac output.

One-third of all head injuries have multiple injuries (Lewin, 1966) and the combined head and chest injury is becoming increasingly common. Each of these carries a high risk when occurring separately. A combination of the two is particularly lethal and artificial ventilation will be urgently required. Other major injuries accompanying head injury, such as those in the upper abdomen or major limb or pelvic fractures causing immobilization, are likely to benefit from a period of artificial ventilation.

Status epilepticus can be a major complication in the period after operation and if the control of this requires the administration of depressant drugs, then artificial ventilation can sometimes be of value in the management of these cases.

Finally there is the vexed question of brain death. The clinician is not infrequently faced with a patient who has suffered severe brain damage and has become apnoeic. With the use of ventilators, such a patient may be kept alive for a period causing great distress to relatives and affecting adversely the morale of the staff of an intensive care unit. Death is generally defined as cessation of cardiovascular activity, but, in an attempt to lessen suffering to the relatives of patients, Mohandas and Chou (1971) have discussed the concept of brain death based on the integrity of the c.n.s. Having excluded depressant drugs and hypothermia as factors causing or contributing to the coma, this applies to patients who have no spontaneous respiration or motor responses above the spinal cord and in whom the pupils are fixed in the absence of evidence of 2nd or 3rd nerve lesions. Spinal reflexes may persist even when the brain stem is autolysed. Artificial ventilation and other supportive measures should then be continued for only 12 h. E.e.g. examination is not essential in the determination of brain death (Editorial, 1974). The patient is taken off mechanical ventilation for a period of 5 min commencing with a normal Paco2 and having oxygen administered by diffusion (6 litre/min by tracheal catheter). If no spontaneous respiration occurs, then it is recommended that the ventilator be switched off. However, a further period of artificial ventilation for 12 h may be commenced followed by a final test of apnoea.

In recent years there have been accounts of the use of controlled hyperventilation as a specific therapeutic measure, but this area of use is controversial. Spontaneous hyperventilation in brain damage may be the result of hypoxia, a central neurological non-chemical ventilatory drive or a secondary compensating mechanism to restore normal intracerebral pH. Hyperventilation itself can cause an increase in c.s.f. lactic acid concentration (Domonkos and Huszak, 1959), and this is likely to be associated with a low c.s.f. bicarbonate. However, Gordon and Rossanda (1968) concluded from their studies in patients with severe head injuries that low c.s.f. pH was caused, not by chronic hypocapnia, but by alterations in brain metabolism. They suggested, therefore, that this acidosis should be corrected by the use of controlled hyperventilation. C.s.f. pH was used as a guide to the degree of intracerebral acidosis, and these workers have demonstrated a reduction of acidosis, though not consistently, in both lumbar and ventricular samples in ventilated patients. While it is difficult to obtain controlled studies to assess this form of therapy, Gordon (1971) has reported clinical evidence of a mortality rate significantly lower in patients treated in this manner.
In addition to correction of intracerebral acidosis, Gordon and Rossanda (1968) claim that reduction in ICP, improvement in oxygenation and better perfusion of brain tissue are produced by the use of controlled hyperventilation. Clinicians are aware that artificial ventilation can be of use in patients with brain damage who have respiratory insufficiency, but it must also be recognized that oxygenation may be adversely affected even in patients with normal lungs. Decrease in compliance (Howell and Beckett, 1957), increase in Vd/Vr ratio (Watson, 1962) and decrease in cardiac output (Cournand et al., 1948) have been reported. Christensen (1976) found, in a study of artificial hyperventilation in cerebral apoplexy, that approximately half the patients treated in this way tolerated such a degree of hyperventilation. Clinicians are aware that perfusion of brain tissue are produced by the use of controlled hyperventilation. However, one advantage of artificial ventilation may be better control of inspired oxygen, but this should not result in PaO₂ increasing above normal values. Rossanda and Gordon (1970) advised the use of high percentages of oxygen, but this can be dangerous to the respiratory system (Winter and Smith, 1972). Patients with neurological diseases affecting respiration can be ventilated for long periods, even years, without evidence of lung impairment, but the use of high inspired oxygen can initiate respiratory failure. In a retrospective study of those patients who had developed respiratory failure, Nash, Blennerhassett and Pontoppidan (1967) concluded that oxygen was the most likely cause of this failure and not controlled ventilation.

Cerebral oxygenation may also suffer as a result of severe cerebral vasoconstriction which becomes significant when PaCO₂ reaches 20 mm Hg (Wollman et al., 1968). Below this value increases in cerebral tissue lactate/pyruvate ratio and increasing reduction of the NADH/NAD⁺ system may represent hypoxia (Granholm and Siesjo, 1969), but so long as the energy charge potential of the tissue is unaltered this hypoxia is most likely to be reversible (Granholm, 1971). Evidence that border line hypoxia exists at this degree of hypocapnia is given by the reversal of high tissue and c.s.f. lactate by the inhalation of hyperbaric oxygen (Plum, Posner and Smith, 1968). If sodium bicarbonate is infused to produce a metabolic alkalosis at PaCO₂ 20 mm Hg, rCBF will increase because of tissue hypoxia resulting from a shift to the left of the oxygen dissociation curve (Wollman et al., 1968). Similar studies, also in man, at PaCO₂ 30 mm Hg demonstrated no alterations in rCBF. Other evidence is given by the reversal of slow-wave activity in the e.e.g. which is produced by inhaling hyperbaric oxygen (Cohen, Reivich and Greenbaum, 1966). Granholm (1971) and Harp and Wollman (1973) emphasized that these observations apply to normal brain and that patients with damaged cerebral circulation resulting from arterio-sclerosis or intracranial disease of any kind cannot be expected to tolerate such a degree of hyperventilation.

The rate of delivery of oxygen to cerebral tissue is an additional factor regulating oxygenation. It has been postulated that hyperventilation will improve perfusion to ischaemic areas by an inverse steal phenomenon, but there is confusion in the literature regarding this. In areas of canine brain made ischaemic by occlusion of the middle cerebral artery, Soloway and colleagues (1968) demonstrated a reduction in the size of the infarct following the induction of hypocapnia. In a subsequent similar investigation on monkey brain (Soloway et al., 1970), the induction of hypocapnia was delayed for 1 h and they were unable to reproduce these results. Lassen and Pålvolgyi (1968) described increases in rCBF during hypocapnia in patients with apoplexy and cerebral tumours. However, Brock, Hadjidimos and Schürmann (1969) demonstrated, in a patient with ischaemic brain damage, that hyperventilation can be harmful if reactivity to carbon dioxide is still present in collateral vessels. In other studies on the effect of PaCO₂ on the brain of the squirrel monkey made ischaemic for 2 h by occlusion of the middle cerebral artery, Michenfelder and Sundt (1973) concluded that hypocapnia did not improve but rather aggravated the metabolic effects of ischaemia. There were further decreases of energy reserve measured as decreasing ATP values and increasing lactic acidosis.

Damaged brain may also have hyperaemic foci where perfusion is in excess of metabolic needs, and it has been postulated that loss of vasomotor control is a result of localized cerebral acidosis (Lassen, 1966). If this is partly a result of increased PaCO₂ then hyperventilation may minimize the damage; at the same time ICP will be decreased and this will help to maintain adequate cerebral perfusion (Knitlquist, Siesjo and Zwetnow, 1969). However, this tissue ischaemia is likely to be metabolic; tissue metabolites will have an overriding effect on CBF and hyperventilation will be of little use other than by decreasing ICP.
It seems that there is still considerable doubt about the efficacy of controlled hyperventilation as a specific therapeutic measure in brain damage. It would be interesting to know if the induction of a respiratory alkalosis to treat acute brain injury characterized by a cerebral metabolic acidosis causes a continuous c.s.f. alkalosis or if the effect is only transient. Christensen (1974) found that the effect was only transient in patients with cerebral apoplexy. C.s.f. pH adaptation occurred within 2 days. Good brain oxygenation cannot always be guaranteed with controlled hyperventilation, even under ideal conditions, nor can improved patterns of cerebral perfusion. Most clinicians would agree that artificial ventilation can be a very useful measure in controlling ICP, especially in the period soon after trauma (Crockard, Coppel and Morrow, 1973), but its value at a later stage remains in doubt. There are occasions when it is obligatory to use artificial ventilation, but in other circumstances the clinician must carefully weigh the advantages and the disadvantages of the technique before embarking on the procedure. As he watches abnormal patterns of respiration the anaesthetist, almost instinctively, has the desire to take over ventilation, but he must guard against over-enthusiasm and at all times consult with the neurosurgeon. Even if artificial ventilation is considered advisable, the situation should be reviewed regularly lest either recovery or brain death make it unnecessary. Artificial ventilation is not necessarily indicated merely because intubation or tracheostomy is performed to maintain a clear airway, nor should artificial ventilation be equated with extreme hyperventilation, which can only be harmful.

**CHANGES IN CARDIOVASCULAR SYSTEM**

It is important to avoid severe hypotension and to maintain adequate brain perfusion in the period following surgery. Tindal (1971) included drugs, hypothalamic lesions, pituitary failure, dehydration, electrolyte imbalance, haematemesis from gastrointestinal lesions and inadequate transfusion as causes of hypotension in a neurosurgical intensive care unit. Most of these are treatable, but patients who have persistent hypotension, which may be accompanied by spontaneous irreversible hypothermia, have probably sustained severe hypothalamic damage and the prognosis is very poor.

- Acute hypertension may also be dangerous by causing a “breakthrough” of autoregulation of cerebral blood flow (Skinhøj and Strandgaard, 1973), but in studies in baboons made chronically hypertensive, the mean arterial pressure was increased to a much greater value before rCBF became pressure passive (Strandgaard et al., 1975). These authors suggested that adaptative changes occur in the cerebral circulation which may help to protect the brain from further increases in arterial pressure. Hypertension can also cause an increase in brain elastance or stiffness (Leech and Miller, 1974b) and if focal oedema is already present, an increase in arterial pressure can increase its volume and extent (Klatzo et al., 1967).

It is not uncommon for the patient who has had surgery for the treatment of intracranial aneurysm to become very hypertensive in the period immediately after operation and the anaesthetist may be asked to control this. This is a confusing area because some surgeons believe that the arterial pressure should be stimulated at this time to provide a good perfusion pressure. In this unit the surgeon will often settle for “taking the top off the pressure”. Ganglion blocking drugs and phenothiazines are unpredictable in action and may be difficult to control, and because of this the author prefers to use i.v. Althesin and to institute artificial ventilation. Control of arterial pressure is very easy with this method and the condition of the patient may be improved by the reduction in cerebral metabolism and rCBF produced by drug-induced sedation.

The electrocardiograph is a useful monitor in ICU because of the association between acute cerebral damage and myocarditis (Connor, 1968). It is also well known that brain stem lesions can be associated with cardiac dysrhythmias, but Rossanda (1975) considers that, in neurosurgical intensive care, there is no special need for sophisticated instrumentation to correct these.

**CONTROL OF TEMPERATURE**

The technique of induced hypothermia for neurosurgical operations has declined over the past decade, and the tendency now is to prevent accidental decreases in temperature which may occur as a complication of prolonged operations. This helps to avoid shivering after operation and increased oxygen uptake of muscle and brain. However, there may be a limited and specialized use for hypothermia, especially in situations where main cerebral vessels are temporarily clamped. In these cases artificial ventilation should be continued into the period following surgery to ensure better control of shivering, acid–base status and cardiac dysrhythmia.

Other uses of hypothermia include the control of brain swelling and after cardiac arrest (Rosomoff,
The technique in these cases should be limited to less than 48 h as hypothermia per se can induce cerebral oedema and brain swelling if prolonged beyond this time (Bloch, 1967).

Preferential cerebral hypothermia may find a permanent place (Balcalzo and Wolfson, 1971). It has been used successfully in cardiac surgery and cold saline has been recommended for intracarotid perfusion immediately following cardiac arrest (Demian et al., 1970).

In this Institute two rooms are available in the ICU where the ambient temperature can be decreased to 10 °C. This has proved to be a better method than the use of fans to control hyperpyrexia in the neurosurgical patient. These rooms are also useful if a decision is made to induce hypothermia in a patient.

Fluid Balance

There are problems of fluid and electrolyte balance and nutrition which are peculiar to the neurosurgical patient. Periods of unconsciousness and extensive neuroradiological investigations quickly lead to dehydration, particularly in the small child. Barker (1973) showed that a patient who received multiple anaesthetics before the start of surgery was likely to develop a metabolic keto-acidosis.

As in a general ICU, it is important regularly to check weight, fluid intake and output, urinary specific gravity and electrolyte concentrations, plasma electrolytes, urea and haematocrit. In neurosurgical practice measurements of osmolality ratios are also very important in the control of fluid and electrolyte balance which may tend towards hyperosmolality or hypoosmolality. Hyperosmolality tends to occur in the early period after brain damage and may be related to a hypothalamic lesion. Over a 6-month period at this Institute, 4% of patients showed abnormalities of fluid and electrolyte balance which may tend towards hyperosmolality or hypoosmolality. Hyperosmolality tends to occur in the early period after brain damage and may be related to a hypothalamic lesion.

Nutrition

Haider and colleagues (1975) observed that patients with severe brain damage show a greater increase in metabolism than that following general trauma, and they suggested that in these cases hypercaloric nutrition in the period immediately after trauma can be of substantial benefit. The policy of fluid restriction may be difficult to combine with high caloric intake if only the parenteral route is available. Rossanda (1975) suggested that 30–50% glucose may be given with variable amounts of insulin. Single solutions for parenteral nutritional requirements have recently become available providing calories, fluids, proteins and vitamins. If the gastric route is available and absorption is good, then this is the best method of feeding these important constituents. Elemental nutrition by this route eliminates the risk of sepsis, phlebitis or thrombosis and reduces the need for constant monitoring by nursing staff.

Prevention of Infection

The anaesthetist can help to avoid infection. He should be meticulous about setting up i.v. and arterial lines in order to avoid sepsis, and particular care should be taken about the insertion of central venous lines which may be used for parenteral feeding.

Modern disposable endotracheal tubes are all adequately sterilized and should be kept so before intubation. They should not be made unsterile by placing them on benches or by applying lubrication with unsterile fingers. Throat packs are likely to cause pain after operation (Conway, Miller and Sugden, 1960), which may be followed by respiratory infection.

Renal damage is a real danger in the neurosurgical environment as in all intensive care units. Condoms and closed drainage can be used in male patients to avoid catheterization, which may lead to ascending infection. Mannitol is frequently administered during surgery, and it is a good technique to empty the bladder manually in the male patient when the muscles of the abdominal wall are relaxed. Catheterization with closed drainage may be necessary in
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females, and a bladder irrigation system using a urinary antiseptic can be used after operation.

REFERENCES


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