GENERAL ANAESTHESIA FOR NEUROSURGERY

R. GREENBAUM

Anaesthesia for neurosurgery, especially intracranial operations, requires conditions which maintain the perfusion of the brain with blood. This perfusion results from the cerebral perfusion pressure: the difference between the arterial and the intracranial pressures.

Anaesthesia must also facilitate surgical access by reducing brain volume and minimizing surgical haemorrhage. Other common problems arising in the conduct of neurosurgical anaesthesia include the diagnosis of neurological damage and the treatment of haemorrhage during operation.

In the past decade, anaesthetic techniques have evolved which are designed to provide the necessary conditions for intracranial surgery, and incorporate the physiological information gained from the wealth of research on the effects of anaesthetic drugs and techniques on intracranial pressure, cerebral blood flow and cerebral metabolism. Many reviews and monographs reflecting the current interest in neuroanaesthesia have appeared in recent years (for example Hunter, 1964; Bozza Marrubini, 1965; Gilbert, Brindle and Salindo, 1966; Michenfelder, Gronert and Rehder, 1969; McComish and Bodley, 1971; Smith and Wollman, 1972; Gordon, 1975; Shapiro, 1975a, b).

The particular hazards of intracranial hypertension, and of intracranial vascular surgery, have been stressed, as these form special risk groups.

ANAESTHETIC PROBLEMS

General

Although most neurosurgical operations are performed with the patient in the supine or lateral position, it is frequently necessary to use positions which tend to cause kinking and distortion of the endotracheal tube and inaccessibility of the patient’s head.

Surgery may be prolonged and, therefore, lead to a progressive decrease in the patient’s temperature. There may be steady and sometimes heavy blood loss throughout this period. Surgical diathermy is used frequently, but must not interfere with essential monitoring of the circulation. Respiration (if spontaneous) may be influenced by the surgical procedure.

Patients often have disturbances of consciousness, may be dehydrated from vomiting, have aspiration pneumonitis and may require hormone replacement. Systemic hypertension is commonly associated with intracranial hypertension.

After operation there is a possibility of unconsciousness, or of airway problems caused by damage to the 9th, 10th or 12th cranial nerves, or of central respiratory failure. Patients who have had surgery for depressed fractures of the skull, pericortical tumours or extradural haematoma have a high incidence of convulsions following surgery.

Cerebral perfusion

Patients who have pre-existing increased intracranial pressure from a space-occupying lesion, oedema or hydrocephalus are especially at risk from any increase in intracranial pressure. Adverse effects from increases in intracranial blood volume follow from intracranial mass shifts or “coning” or local areas of inadequate perfusion pressure (McDowall, 1975).

Cerebral perfusion is influenced inter alia by the arterial carbon dioxide tension, arterial pressure, venous pressure, anaesthetic drugs and hypoxia. These factors are under the control of the anaesthetist.

Arterial carbon dioxide tension. Carbon dioxide is a most potent cerebral vasodilator, and the cerebral blood flow changes associated with it are linearly related to the cerebral blood volume (Risberg, Ancri and Ingvar, 1969; Phelps, Grubb and Ter-Pogossian, 1973). The decrease in intracranial pressure produced by hypocapnia leads to a gradual redistribution of intracranial constituents, resulting in an increase in c.s.f. volume which tends to restore intracranial pressure, but will provide good operating conditions at craniotomy (Furness, 1957; Hayes and Slocum, 1962; Bozza Marrubini, Rossanda and Tretola, 1964). Even in disease states in which there is generalized loss of autoregulation, there may be preservation of reaction to \( P_{a\text{CO}_2}\) and hypocapnia may actually restore autoregulation (Paulson, Oleson and Christenson, 1972). There is no advantage in reducing \( P_{a\text{CO}_2}\),
below 25 mm Hg and there is a risk of cerebral ischaemia with lower arterial carbon dioxide tensions (Gotoh, Meyer and Tagaki, 1965; Schettini, Cook and Owre, 1967).

Hypocapnia increases intracranial pressure as a result of increased cerebral blood flow and vascular engorgement. Autoregulation is abolished and, therefore, hypertension will cause cerebral oedema (Marshall, Jackson and Langfitt, 1969).

Hypertension. In the normal brain at normocapnia, autoregulation of cerebral blood flow prevents increases in intracranial pressure when arterial pressure increases. However, if autoregulation is impaired or abolished, intracranial pressure passively follows arterial pressure fluctuations. Many neurosurgical patients have focal or global loss of autoregulation.

In the normal brain, the upper limit of autoregulation is at a mean pressure of 125–140 mm Hg (Strandgaard et al., 1974). When this is exceeded, hypertensive breakthrough occurs and the blood–brain barrier is breached, leading to forced vasodilatation and focal plasma leakage (Johansson, Strandgaard and Lassen, 1974). It is essential, therefore, to prevent hypertensive incidents by avoiding drugs which increase systemic arterial pressure; by suppressing the hypertensive response at intubation (Alexander and Lassen, 1970; Hulme and Cooper, 1972; Shapiro, Wyte and Harris, 1972; Burney and Winn, 1975; Greenbaum et al., 1975), at skin incision and other painful manoeuvres (Shapiro et al., 1972) and, finally, at the end of anaesthesia (Leech, Barker and Fitch, 1974; Jørgensen and Misfeldt, 1975). It is rational to allow only gradual restoration of the arterial pressure after deliberate hypotension and perhaps to control the arterial pressure and maintain hypocapnia after operation in patients with disturbances of autoregulation.

Hypertensive episodes have the added hazard of increasing the risk of haemorrhage, especially in aneurysm surgery.

Hypotension. Normal autoregulation maintains constant cerebral blood flow until mean cerebral perfusion pressure decreases below 60 mm Hg (Lassen, 1959, 1964; Oleson, 1973). This lower limit is increased in hypertensive patients (Strandgaard et al., 1973).

Many patients with brain pathology have focal or global loss of autoregulation. In these patients, reductions in intracranial pressure and brain bulk will accompany decreases in arterial pressure (Mazzia, Ray and Arturio, 1956). Nevertheless, it is generally assumed that hypotension should be avoided until satisfactory decompression of the brain has been achieved (Cushing, 1902).

Halothane and sodium nitroprusside cause hypotension with cerebral vasodilatation (Griffiths et al., 1974; Stoyka and Schutz, 1975). The intracranial pressure increases until the abolition of autoregulation and hypotension produce a decrease in intracranial pressure (Turner et al., 1975). Autoregulation may take up to 2 h to recover (Keaney, McDowall et al., 1973; Keaney, Pickerodt et al., 1973). Trimetaphan causes hypotension with no change in cerebrovascular resistance and, therefore, no vasodilatation. Thus, intracranial pressure does not increase (Turner, J. M. et al., 1976 in preparation) but there is a decrease in CMRO₂ and evidence of anaerobic metabolism (Stoyka and Schutz, 1975).

Venous pressure. Increase in central venous pressure is directly transmitted to the intracranial veins and may also increase c.s.f. pressure secondarily to pressure changes in the vertebral canal (Williams, 1970). Insufficient expiratory time, high expiratory resistance, coughing or straining on the endotracheal tube will increase intracranial pressure (Hunter, 1952; Schettini, Cook and Owre, 1967). Flexion, extension or rotation of the neck all produce large increases in intracranial pressure, possibly by obstructing the cerebral venous outflow (Campkin and Turner, 1972) (fig. 1).

Cerebral venous pressure may be only slightly reduced by a negative expiratory phase (Schettini, Cook and Owre, 1967) and generally, if the patient is well relaxed, has a free expiratory pathway and an adequate expiratory time, there is little to be gained from a negative phase (Lundberg, Kjällquist and Bien, 1959).

Spontaneous ventilation during anaesthesia is always associated with increased abdominal muscle tone and expiratory muscle activity (Freund, Roos and Dodd, 1964) and, therefore, tends to increase the intracranial pressure.

Anaesthetic drugs. All currently available inhalation anaesthetic agents cause an increase of intracranial pressure (McDowall, 1975), including nitrous oxide (Hendriksen and Jørgensen, 1973; Greenbaum et al., 1975). I.v. barbiturates and Althesin lower intracranial pressure, whereas ketamine produces increases (Shapiro, Wyte and Harris, 1972; Shapiro et al., 1972).

The neuromuscular blocking agents appear to have no direct effect on intracranial pressure, except perhaps for occasional increases with tubocurarine (Tarkkanen, Laitinen and Johansson, 1974). Neuro-
leptanalgesic drugs, for example droperidol and fentanyl, tend to produce a small reduction of intracranial pressure if no hypercapnia is permitted (Fitch et al., 1969a). Diazepam has little effect (Maekawa, Sakabe and Takeshiko, 1974; Cotev and Shalit, 1975). Hyperventilation almost always prevents the increase in intracranial pressure caused by halothane (Adams et al., 1972) and nitrous oxide (Hendriksen and Jørgensen, 1973; Greenbaum et al., 1975). Although patients are occasionally encountered who show a persistent increase despite hypocapnia. Hypoxia. Arterial oxygen tensions less than 50 mm Hg cause increases in cerebral blood flow and intracranial pressure (McDowall, 1966) and this persists for some time after the incident (Loehning, Veyama and Veda, 1962; Freeman and Ingvar, 1968; Kogure et al., 1970).

ANAESTHETIC MANAGEMENT

It is possible to derive solutions to the problems discussed in the foregoing paragraphs, and embody them in the conduct of the anaesthetic.

Assessment before operation

The anaesthetist's care of the patient often commences some days before major surgery, and may be required for anaesthesia for investigations.

It is essential to recognize those patients who have increased intracranial pressure, as the adverse effects of anaesthesia are greatly magnified in them (Fitch et al., 1969b; Fitch and McDowall, 1971). The presence of papilloedema, depression of consciousness and the degree of neurological deficit must be assessed. If there has been vomiting, there may be dehydration and electrolyte abnormalities. However, no attempt should be made before operation fully to hydrate patients with intracranial hypertension. The general medical condition of the patients and their fitness for anaesthesia must also be assessed.

Many patients are receiving anticonvulsant, steroid or antihypertensive drug therapy. Patients with grossly increased intracranial pressure should have ventricular drainage or mannitol therapy before operation. If gliomas or other causes of cerebral oedema are present, steroid therapy such as betamethasone 8 mg every 6 h should be instituted.

Blood should be cross-matched, as there is often more than 1 litre of haemorrhage, and replacement with large volumes of crystalloid solutions is contraindicated. The preoperative visit also allows assessment of the suitability of either the radial artery (Allen, 1929) or the dorsalis pedis artery for cannulation.

Alert patients are naturally apprehensive before a craniotomy and, as depressant and sedative premedication is usually contraindicated, it is important that reassurance and explanation are kind and thoughtful.

Atropine 0.5 mg reduces oral secretions, while diazepam calms the patient and is a useful anticonvulsant. The latter has little effect on the intracranial pressure and, in modest doses, produces little respiratory depression (Maekawa, Sakabe and Takeshiko, 1974).

Induction of anaesthesia

With suitable care, there should be few patients who arrive in the anaesthetic room with grossly increased intracranial pressure and without further reserve for an increase in intracranial volume.

The induction technique must prevent hypoxia and hypercapnia and minimize responses to laryngoscopy and neck movements. The following sequence achieves these requirements:

(i) Atropine 1 mg followed by practolol 0.4 mg/kg body weight. The β-adrenergic receptor blockade attenuates both the arterial pressure response to intubation (Prys-Roberts et al., 1973) and the concomitant increase in intracranial pressure (Greenbaum et al., 1975).

(ii) The co-operative patient is asked to hyperventilate.

(iii) A small dose of non-depolarizing neuromuscular blocking agent, for example gallamine 20 mg is given to reduce the fasciculations and hyperkalaemia which are caused by suxamethonium (Weintraub, Heisterkamp and Cooperman, 1969).

(iv) I.v. thiopentone or Althesin, given slowly to avoid a decrease in arterial pressure, immediately followed by:

(v) Suxamethonium. This acts quickly to provide conditions for intubation and prevents the carbon dioxide retention that is almost inevitable when longer acting non-depolarizing agents are used to provide conditions for endotracheal intubation (Greenbaum et al., 1975).

(vi) Controlled hyperventilation must immediately follow loss of consciousness and, when full muscle relaxation is produced,

(vii) rapid, gentle intubation of the trachea should be performed after spraying the larynx and upper trachea with local anaesthetic. A non-kinkable
endotracheal tube must be used and fixed to the face with meticulous care after checking that it is correctly positioned. A nasogastric tube is a useful precaution in long operations which may be associated with gastric dilatation. A pharyngeal pack is necessary in patients in whom a leak of blood or c.s.f. into the nasopharynx is possible.

(viii) Hyperventilation, with no expiratory obstruction using alcuronium or pancuronium plus neuroleptanalgesic drugs i.v. and a 70% nitrous oxide/oxygen mixture. The arterial $P_{CO_2}$ is maintained at 25–30 mm Hg.

The effects of this induction sequence on intracranial pressure and arterial pressure are shown in figure 1. The effect of neck movement is clearly demonstrated.

Alternative methods which have been used to prevent hypertensive surges at intubation involve the use of 2–4 mg trimetaphan or larger doses of thio- pentone (Shapiro et al., 1972; Shapiro, 1975b).

Preparation for surgery

Monitoring and measurements.

(1) Arterial pressure and heart rate. Direct cannulation of the radial artery at the wrist is a simple and safe procedure, allowing arterial pressure and heart rate to be displayed either on dials, an oscilloscope or a simple anaeroid manometer (Zorab, 1969). Alternatively, the dorsalis pedis artery is often easy to cannulate (Johnstone and Greenhow, 1973; Spoerel, Deimling and Aitkin, 1975).

(2) Electrocardiogram.

(3) Temperature—oesophageal or rectal.

(4) Central venous pressure requirement is indicated in operations where severe bleeding is expected or where the sitting position is used.

(5) A precordial or oesophageal stethoscope is useful and is indicated especially in operations in the sitting position and for infant craniotomies (Horton, 1974).

(6) Expired carbon dioxide concentration.

(7) Intracranial pressure is no longer a purely research tool, but gives invaluable clinical information.

Other monitoring and measurements sometimes used include arterial and jugular bulb oxygen and carbon dioxide tensions, cerebral blood flow, e.g. and estimations of cerebral metabolites.

Blood and heat loss. Blood loss must be replaced, avoiding excessive overhydration or hypotonicity which might worsen cerebral oedema (Fishman, 1953). An infusion should be set up using compound sodium lactate solution and incorporating a blood warming device.

It is useful to prepare a second separate infusion for use with mannitol or hypotensive agents.

Heat loss can be minimized by the use of a “space rescue” blanket—a polyester sheet surrounded by laminates of aluminium foil. The impermeable material prevents heat loss from convection and evaporation, and the aluminium prevents radiant heat loss (Baum and Scopes, 1968; Horton, 1974). Alternatively, the patient may be placed on, and covered with, water circulation blankets.

![Fig. 1. The effects of the induction of anaesthesia on intracranial pressure and arterial pressure. The effects of head manipulation on intracranial pressure are also demonstrated. A: atropine 0.6 mg; B–C: practolol 40 mg; C–D: spontaneous hyperventilation; D–E: thiopentone 225 mg; F: suxamethonium 100 mg; G: laryngoscopy; H: endotracheal intubation; I: hyperventilation with 66% nitrous oxide in oxygen mixture; AP = arterial pressure.](image-url)
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**Iatrogenic injury.** Care must be taken to protect the eyes by covering the closed lids with waterproof non-irritant adhesive plaster. There must be no compression or stretching of peripheral nerves, and vulnerable skin pressure areas such as the forehead, ears and nose must be protected. Precautions should be taken to minimize stagnation of blood in the legs.

**Position for surgery.** The surgical position is chosen to give access and must allow good venous drainage from the site of operation by elevating the head above the heart. Care should be taken to avoid flexing or rotating the neck, which might obstruct the neck veins.

Frontal craniotomies are performed in the supine, brow-up position; temporal and posterior fossa craniotomies are satisfactory in the lateral or lateral/prone position with some rotation of the neck depending on the site of the lesion. The full prone position gives adequate exposure for most posterior fossa and cervical spine operations, but meticulous care must be taken to support the body by the iliac crests and the chest in order to leave a free, uncompressed abdomen and vena cava.

Having positioned the patient, it is kinder to shave the patients under anaesthesia rather than before operation.

Finally, the patient should be inspected and the position of the endotracheal tube and its secure fixation and that of anaesthetic tubing and other equipment checked.

**Maintenance of anaesthesia**

The basic technique is hyperventilation to an arterial carbon dioxide tension of 25–30 mm Hg with 70% nitrous oxide and oxygen. Muscle relaxation is maintained by incremental doses of non-depolarizing blocking agents given regularly to prevent any possibility of coughing or straining. An adequate depth of anaesthesia can be maintained using droperidol with fentanyl or phenoperidine.

It is arguably safer to use halothane or an alternative volatile anaesthetic supplement during hypocapnia than to allow an excessively light patient to suffer hypertensive episodes during surgical stimulation of skin and bone cutting (Shapiro et al., 1972b).

Attempts have been made to avoid the disadvantages of volatile agents, by decreasing intracranial pressure and depressing cerebral metabolism with thiopentone infusions (Hunter, 1972) and Althesin.

The problems of delayed recovery and hypotension with these techniques require further study.

Blood loss should be replaced when over 10% of the patient's estimated blood volume is lost.

**Active reduction of intracranial pressure**

When the intracranial pressure is known to be high, or when the dura is tight and the brain swollen, one or both of the following measures is indicated:

1. C.s.f. can be drained via a ventriculostomy. In patients in whom only a small increase of intracranial pressure is present and there is communicating hydrocephalus, for example aneurysm surgery, drainage may be via an indwelling lumbar spinal needle (Hart and Willatts, 1975). However, drainage should be limited to a rate of less than 5 ml/min, and limited in volume, otherwise arterial hypertension and cardiac arrhythmias are common (Barker, 1975) and a rebound increase in intracranial pressure may occur (Nornes and Magnaes, 1971).

2. Mannitol in a 20–25% solution can be given in a dose of 0.5–1.0 g/kg body weight over 10 min. In this dosage, there is unlikely to be the gross brain shrinkage which can lead to a subdural haematoma from tearing of cortical veins and dural sinuses (Marshall and Hinman, 1962). Good reduction of intracranial pressure is likely within 5 min of commencing the infusion, and the improvement of the intracranial volume/pressure relationship lasts for up to 2 h (Miller and Leech, 1975). Mannitol can also be helpful by making the brain softer and more easily retractable (Leech and Miller, 1974).

Mannitol causes transient hypervolaemia and may increase surgical bleeding, and should not be mixed with transfused blood as it causes some haemolysis.

The search for alternative osmotic agents has continued, but mannitol remains the safest for i.v. use in the operating theatre. Unfortunately, 20% glycerol produces severe haemolysis and haemoglobinuria when used i.v. (Hagnevick et al., 1974).

A catheter should be placed in the bladder when osmotic therapy is anticipated.

**Recovery from anaesthesia**

Patients who are expected to have a high risk of convulsions should be given phenytoin 100 mg i.v. during anaesthesia, to initiate anticonvulsant therapy. When cerebral oedema following surgery is anticipated, betamethasone or dexamethasone therapy should be commenced.
At the end of a neurosurgical operation there may be:

1. Depression of consciousness.
2. Damage to cranial nerves which limit the patient's ability to maintain a safe airway.
3. Respiratory depression.

After major procedures, where oedema is expected after operation, or where autoregulation is likely to be grossly impaired, it is wise to continue hyperventilation into the period after surgery.

Patients in whom spontaneous ventilation is to be preferred, should be given atropine and neostigmine to reverse residual curarization. The increased sensitivity of neurosurgical patients, to narcotics, suggests that nalorphine or naloxone should be administered routinely to reverse residual narcotic effects.

The ability of the patient to breathe and maintain his airway must be assessed and no carbon dioxide retention, hypoxia or asphyxia permitted or serious brain swelling will occur (Jergensen and Misfeldt, 1975; Leech, Barker and Fitch, 1974; Leech and Miller, 1974).

Anaesthesia for intracranial aneurysms

Seventy-five per cent of subarachnoid haemorrhages are caused by rupture of an aneurysm of a large vessel on the floor of the cranium (Leading Article, 1971). The age, and the general and neurological condition of the patient affect the prognosis, as does the site of the aneurysm and the timing of the surgery. The mortality of the initial haemorrhage is approximately 45% and there is a high incidence of second haemorrhage within the first 6 weeks. Unfortunately, there are increased risks with early surgery. At the time of haemorrhage, there is a massive increase in intracranial pressure which partially tamponades the ruptured vessel (Nornes and Magnaes, 1972). If the patient survives the initial cerebral ischaemia, intense cerebral vasoconstriction follows in a third of cases associated with high concentrations of catecholamines in the c.s.f. (Cummins and Lothian, 1973). Later increases in intracranial pressure may be caused by intracranial haematoma or the development of communicating hydrocephalus. Before operation, hypertension should be treated and epsilon amino caproic acid (Epsikapron) therapy instituted to reduce the risk of further haemorrhage.

Induction of anaesthesia as described should prevent surges of arterial pressure and, after dural exposure, c.s.f. can be withdrawn via a lumbar needle as necessary.

It is my policy to add carbon dioxide to the inspired gas mixture to maintain normocapnia, as the extremely low grey matter blood flows we have measured when hypocapnia is superimposed on aneurysmal vasoconstriction may be undesirable.

The use of the operating microscope and hypotensive anaesthesia provide excellent conditions for surgical dissection of the aneurysmal sac and its clipping. These developments have led to the gradual abandonment of hypothermia by most neurosurgical units in the United Kingdom (McDowall, 1971).

**Controlled hypotension.** Control of arterial pressure with halothane, trimetaphan infusion (0.1%) or sodium nitroprusside (0.01%) should be used to prevent hypertension and rupture of the aneurysm before clipping and to provide gradual hypotension to 50 mm Hg (mean) during dissection and clipping of the aneurysm. Care should be taken to avoid rebound hypertension after clipping.

Nitroprusside or trimetaphan should be administered by a separate, independent infusion using a microdrip set. Y-connections are dangerous as backflow can occur. The patient's sensitivity to the drugs must be assessed by starting with a slow rate of infusion and adjusting it carefully according to the therapeutic response. Both drugs have a rapid onset of action, and both can exhibit tachyphylaxis. Nitroprusside has a particularly evanescent action and does not produce pupil dilatation or histamine release.

Halothane may be used as the primary hypotensive agent or in addition to nitroprusside infusion to provide fine control of arterial pressure.

Nitroprusside tachyphylaxis has led to the administration of large doses of the drug resulting in fulminating acidemia and cyanide poisoning (McDowall, Keaney and Turner, 1974). However, if the dosage is limited to 1.5 mg/kg body weight in up to 3 h, there is no toxic hazard (Vesey, Cole and Simpson, 1976).

**Posterior fossa exploration**

There remains a controversy between those who advocate the use of the sitting position for optimal surgical access and low venous pressure and hence a dry operative field, and those who have learned to use the prone or lateral positions and claim to enjoy adequate access and satisfactory operating conditions. The argument is compounded by those surgeons who insist that knowledge of the respiratory pattern is the key to prevention of brain stem damage during surgery and that spontaneous ventilation is, therefore, mandatory. If spontaneous ventilation is to be used, the sitting position is superior to the prone or lateral.
In the sitting position, postural hypotension is common. Millar (1972) reported that 5% of patients showed marked hypotension on being placed in the sitting position and 42% of patients mechanically ventilated required vasopressor therapy. Bandaging the legs and thighs and gentle movement into position will reduce the risk of hypotension.

Air embolism occurred in 3.8% of 635 cases in the sitting position collected by Altenburg (Michenfelder, Gronert and Rehder, 1969). The severity of the embolism is determined by the rate and volume of air entrained and by the ability of the circulation to withstand the insult. Nitrous oxide anaesthesia will expand the emboli of air as the soluble nitrous oxide diffuses into the bubbles (Nunn, 1959), and 100% oxygen will reduce the hazard (Steffey, Gauger and Eger, 1974).

Monitoring with an oesophageal stethoscope will reveal a drum-like metallic resonance to the heart sounds when embolism occurs, which becomes a "mill-wheel" with larger volumes of embolus.

The other useful detection method employs a Doppler ultrasonic flowmeter (Edmunds-Seal and Maroon, 1969) but, unfortunately, most of these sensitive devices are unsatisfactory during the use of surgical diathermy. Expired carbon dioxide concentration, arterial pressure, electrocardiogram and the respiratory pattern in spontaneously breathing patients all give diagnostic information.

Treatment by surgical sealing of the open veins should be accompanied by jugular vein compression by the anaesthetist, and by increasing the intrathoracic pressure.

A G-suit can be used to increase venous pressure, and aspiration of the right heart via a catheter enables air to be directly removed. Oxygen, vasopressors, internal cardiac massage and aspiration or external massage have been used successfully (Gottlieb, Ericsson and Sweet, 1965).

It is clearly necessary to discourage neurosurgeons from the use of the sitting position and, like Michenfelder, Gronert and Rehder (1969), Shapiro (1975b), and Gordon (1975), we believe the advantages of controlled ventilation outweigh the added information obtainable by monitoring the respiratory pattern during spontaneous ventilation.

Careful continuous observation of the arterial pressure, heart rate and electrocardiogram warn of surgical interference to the brain stem or cranial nerves (Hunter, 1952, 1964; Lall and Jain, 1969). The incidence of damage to vital centres has been greatly reduced by the increasing surgical awareness of the problem and the improved surgical precision made possible by the use of the operating microscope.

**Hypophysectomy**

Removal of the pituitary gland is performed for tumours of the gland and for treatment of metastatic hormone-sensitive tumours. The gland is approached either by means of a frontal craniotomy or transnasally. Hormone replacement with cortisone should be commenced before operation and other hormone problems, such as diabetes insipidus or mellitus, may require therapy.

Intubation in patients with acromegaly can be difficult unless an extra-long laryngoscope blade and endotracheal tube introducers are available.

Patients with carcinomatosis are often anaemic, cachectic, have multiple bone metastases and may have pleural effusions and ascites. They should quickly be made as fit as possible and the anaesthetic risk accepted, as there is often a gratifying response to surgery.

After operation, a few patients develop severe hypothalamic disturbances of temperature and circulatory regulation.

**REFERENCES**


