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BOOK TITLE } – 'THE ANAESTHETIC EMERGENCY'

11

Neurosurgical anaesthesia

ROGER TRAILL

The majority of problems presenting as acute crises during neuroanaesthesia are similar to those occurring during any type of anaesthesia. In this chapter only the problems specific to neurosurgery and neuroanaesthesia are discussed.

Monitoring montage

As with other forms of anaesthesia, the early detection of problems is important in outcome. The minimum monitoring required for a typical craniotomy is:

- *Respiratory:* Pulse oximetry (Spo_2), capnography, airway pressure, inspired oxygen concentration and an oesophageal stethoscope.
- *Cardiovascular:* Arterial pressure (invasive), central venous pressure (CVP) and ECG (lead V_5).
- *Others:* Nerve stimulator, temperature (nasopharyngeal) and hourly urine output.

SPECIFIC PRESENTING PROBLEMS

Acute increase in intracranial pressure (closed dura) or brain swelling (open dura)

Principles: Monro–Kellie doctrine

The intracranial contents are brain, CSF and blood. What we see and call the 'brain' is the cellular and interstitial components of brain tissue plus blood and CSF. The Monroe–Kellie doctrine states that when the cranium is intact the intracranial volume is fixed. Therefore increases in the volume of one or more of the compartments must be associated with a decrease in volume of one or more of the remaining compartments (compensation). There will always be a rise in intracranial pressure associated with this as some force (however small) is always needed to move substances out of the cranium. This volume–pressure relationship is classically described by the cerebral elastance curve. Initially there is a relatively flat portion and then, as the

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available compensation becomes exhausted, the curve becomes progressively steeper. On the flat part of the curve there is little change in intracranial pressure (ICP) with increases in brain volume but as ICP increases the same volume increases produce exponentially greater increases in ICP.

The effects of an increased ICP are due primarily to brain shift and not to the commonly quoted mechanism of compromised cerebral perfusion pressure (CPP) (Ryder et al, 1953). The autoregulatory curve (normally) predicts that cerebral perfusion is maintained until the CPP is reduced below 50 mmHg and that the CPP must fall to 20–30 mmHg before critically compromised flow occurs. With a non-uniform increase in ICP, which is the most common circumstance (e.g. tumours/head injuries), the downward shift of the brainstem kinks vessels, producing brainstem ischaemia (Thompson and Malina, 1959). This may begin to occur at ICPs as low as 20–30 mmHg. Cingulate gyrus herniation also produces anterior cerebral artery ischaemia by a similar mechanism.

Unfortunately during anaesthesia the usual clinical signs of brainstem ischaemia are not available until the patient is near to coning. The Cushing response occurs with marked brainstem ischaemia and is not an early sign. Electrophysiological monitors of brainstem function (brainstem and somatosensory evoked potentials) will reliably indicate when brainstem ischaemia begins to occur (before coning) (Stone et al, 1990) but is not widely used. Continuous ICP monitoring is the usual method of detection.

Most neurosurgery needs a shrunken brain but the presence of a low ICP does not guarantee a reduced brain volume (as described by the shape of the elastance curve). The presence of an elevated ICP, however, guarantees surgical conditions will be inadequate.

The causes of increased ICP and brain swelling are similar. The main difference is in the way they manifest themselves. The first we are likely to know about an increased ICP is when the surgeon tells us that the dura is tense. On the other hand, once the dura is open brain swelling is likely to be detected early by surgeons as they are often operating in confined spaces. It is only in extreme circumstances that the swelling is so marked that it causes the brain to herniate out of the craniotomy site.

The physiological consequences depend on which compartment has increased in volume, and whether this is a diffuse or localized effect. If there is associated brain shift (e.g. an acute subdural haematoma on the opposite side to the operation) there will be more problems than if there is no brain shift (e.g. swelling occurs in the operation site).

Increases in ICP (in the closed cranium) or brain swelling (when the dura is open) are caused by one or more of the following mechanisms.

Differential diagnosis

1. Anaesthetic.
   (a) Respiratory, e.g. hypoxia, hypercarbia.
   (b) Cardiovascular.
      (i) Increased cerebral venous pressure: raised CVP (overhydration, congestive cardiac failure, increased intrathoracic pressures), posture, obstruction to neck veins.
      (ii) Hypertension (see later).
   (c) Pharmacological: cerebral vasodilators, e.g. nitroprusside, nitroglycerine, volatile agents, nitrous oxide.
   (d) Hypo-osmolar fluids.
2. Surgical.
   (a) Cerebral oedema.
   (b) Haemorrhage, e.g. intracerebral, subdural, extradural, aneurysm rupture. The sudden swelling of a tumour should make one think of a bleed into the tumour.
   (c) Surgical obstruction of cerebral veins, e.g. clips (arteriovenous malformation surgery), retractors.
   (d) Surgical obstruction of CSF drainage.
   (e) Pneumocephalus, e.g. post-pneumoencephalogram, post-head injury, subdural.
3. Patient.
   (a) Progressive cerebral oedema related to reasons for surgery, e.g. tumour, head injury.
   (b) Hydrocephalus, e.g. post-subarachnoid haemorrhage, posterior fossa tumours, obstructed shunts.
   (c) General patient causes of hypotension, hypoxia, and raised CVP.

Management

1. Detect and correct any anaesthetic-related problem.
2. If anaesthesia has been stable and there are no obvious anaesthetic-related problems inform the surgeons and suggest that they look for a surgical cause for the problem.
3. Elevate head of table 10–15° (beware of the risk of air embolus).
4. Correct excessive rotation of neck.
5. Hyperventilate to a $Paco_2$ of 20–25 mmHg.
6. Reduce or cease volatile agents.
7. Cease nitrous oxide if the problem is severe.
8. Commence either a barbiturate or propofol infusion, if no EEG then as much as the blood pressure will tolerate may be given. If EEG monitoring is being used aim for the minimum dose necessary to maintain marked burst suppression, maintaining the blood pressure at baseline levels (if necessary) with a vasoconstrictor infusion.
9. If the dura is closed discuss with the surgeon whether it is safe to open the dura. It may be that rapid correction of the underlying problem is called for, e.g. draining an intracerebral haemorrhage/abscess or resection of a tumour.
10. If the dura is open and brain swelling occurs and surgical causes have been eliminated, insertion of a ventricular needle/drain or opening the cisterns may produce a marked improvement.
11. Diuretics—remember these take time to work.
    (a) Mannitol 1.5 g/kg over 20 minutes (if not already given). The

mechanism of action of mannitol is in the increase in plasma osmolality and there is no 'maximum' brain shrinkage as such. The dose limitations are related to side-effects. When brain shrinkage is needed during surgery, or when ICP increases are large and rapid reduction is needed, the volume reductions required are large. Fearnside and Adams (1980) showed that mannitol takes 15–30 minutes to act and does not have its peak effect until 60–90 minutes. We do not usually have the luxury of waiting 60 minutes to see if a smaller dose is adequate. Roberts et al (1987) have shown that the larger the dose of mannitol and the more rapidly it is given then the greater its effect. Doses ≤ 2 g/kg have been reported to be used as a means of producing cerebral protection (Little, 1978). Mannitol 1.5 g/kg is the author's usual dose. Ravussin et al (1988) showed that the increase in ICP that may occur in patients with normal ICPs are small and clinically unimportant and do not occur in patients with raised ICP.

It is suggested that smaller doses (0.25–0.5 g/kg) (Smith et al, 1986) are equally effective and that repeat doses be given only as necessary. The explanation of these apparently contradictory statements lies in the non-linear characteristics of cerebral elastance. Small volume reductions may reduce the ICP to 'normal' levels but they do not produce a markedly shrunken brain. In the intensive care unit a smaller volume of mannitol may be used as only small reductions in volume (ICP) are needed.

(b) Frusemide ≤ 1.0 mg/kg (if not already given). Frusemide 1 mg/kg reduces ICP (Cottrell and Marlin, 1981) and the combination of frusemide and mannitol is more effective than either drug alone (Millson et al, 1981). Unlike mannitol, a bolus dose of frusemide does not cause hypotension, nor can it cause intravascular fluid overload in patients with cardiac or renal impairment. There are no direct comparative studies of frusemide and mannitol but in most centres mannitol is used as first line therapy and frusemide is added. Because of the demonstrated synergy, both frusemide (0.3 mg/kg) and mannitol (1.5 g/kg) may be used, increasing the dose of frusemide up to 1.0 mg/kg if necessary (1.0 mg/kg also decreases CSF formation) (Domer, 1969).

12. Hypothermia/normothermia. Hyperthermia increases cerebral metabolic rate and should be corrected. Hypothermia has been used to treat refractory increases in ICP but is difficult to manage and has not been shown to improve patient outcome.
13. Correct hyponatraemia (see later) and normalize blood glucose.
14. Resection of swollen brain may be necessary as a last resort. If the brain is still swollen at the end of the case the dura may be left open and the bone flap not replaced.

Hypoxaemia

The problems of hypoxaemia in neurosurgery are increased due to restricted

airway access. Head injured patients may develop neurogenic pulmonary oedema, aspiration, fat embolus, pneumothoracies and lung contusions. The use of induced hypotension with nitroprusside or nitroglycerine is commonly associated with increased pulmonary shunting and the Fio_2 may need to be increased. Management of the respiratory failure should include increasing the Fio_2, hyperventilation (as for control of ICP) and correction of the underlying problem. Positive end-expiratory pressure (PEEP) should be restricted until the brain is decompressed unless 100% oxygen fails to correct the hypoxaemia. PEEP may increase cerebral venous pressure and hence ICP, potentially worsening the patient's neurological status.

Hypertension

Differential diagnosis

1. Anaesthetic, e.g. hypoxia, hypercarbia.
2. Surgical.
   (a) Surgical stimuli.
      (i) Increased level of stimuli. The critical points during craniotomies are: induction, intubation, shaving the head, cleaning the head with alcohol, Mayfield pin fixation, positioning the patient, injection of local and/or vasoconstrictor into the scalp, skin incision, periosteal elevation, sawing the bone, raising the flap and incising the dura.

      The brain substance itself does not contain pain fibres, however the dura and blood vessels do. Tension on these structures can be quite stimulating, as may manipulation of the sensory branches of the trigeminal nerve.

      (ii) Brainstem stimulation. It is not uncommon for brainstem manipulations to cause a slowing of the heart rate and an increase in blood pressure. Whether this represents brainstem ischaemia or simply distortion of cardiovascular centres is unclear. Albin et al (1976) reported a 10% incidence of transient hypertension not requiring treatment in operations around the brainstem. If the changes are prolonged (≥ 30 seconds) or marked it is sensible to warn the surgeons. ECG changes are also common: sinus bradycardia is the most common but ventricular arrhythmias may also occur and may progress to ventricular tachycardia.

      (iii) Inadequate reflex depression, i.e. failure to anticipate adequately the response to a given stimulus.

   (b) Raised ICP: Cushing's response, e.g. aneurysm rupture.
3. Patient, e.g. essential hypertension, full bladder, phaeochromocytoma.

Management

1. Correct any anaesthetic problem.
2. Pharmacological intervention.
   (a) Thiopentone 100–500 mg bolus if marked increase in blood

pressure. Thiopentone is the ideal drug in this situation, it lowers the blood pressure rapidly, vasoconstricts the cerebral vasculature, is relatively short acting, and will already be drawn up on the drug tray. Immediately administer a bolus dose (100–500 mg) depending on the size and condition of the patient and the magnitude of the rise in blood pressure.

(b) Trimetaphan (0.2% solution). This is the author's second choice. It has a slower onset than thiopentone but a similar onset time to nitroprusside and nitroglycerine and causes less cerebral vasodilatation.

(c) Isoflurane. This is author's third choice. Narcotics take too long to work and have no marked cerebral vasoconstrictor effects at the usual doses. Cerebral vasodilators such as nitroprusside should be avoided as they may increase ICP or brain swelling in hypertensive patients.

3. If it is practical ask the surgeons to stop stimulating the patient temporarily.
4. Rule out aneurysm rupture.
5. If secondary to brainstem stimulation warn the surgeon. It is not sensible to continue with operations near the brainstem if persistent hypertension occurs in the absence of other indicators of brainstem function (e.g. somatosensory or brainstem auditory evoked potentials). Even these only provide information about the pathways actually being monitored and, while they are usually affected by general brainstem ischaemia, it is possible to get isolated damage to the non-monitored parts of the brainstem.
6. Slower onset hypertension is treated with reflex depressants, e.g. narcotics, volatile agents, or non-cerebral vasodilating antihypertensives (e.g. trimetaphan, labetalol, metoprolol, esmolol, clonidine).

Hypotension

Problems specific to neuroanaesthesia include:

Differential diagnosis

1. Anaesthetic.
   (a) Mannitol. Mannitol may produce marked hypotension due to a decreased peripheral resistance which is associated with increased cardiac output (Coté et al, 1979; Domaingue and Nye, 1985). It is uncommon if ≤ 1.5 mg/kg is given over at least 20 minutes and is usually short lived if the infusion is suspended. Mannitol almost never causes an elevated blood pressure.
   (b) Dehydration.
2. Surgical.
   (a) Haemorrhage. Haemorrhage related to the brain, i.e. aneurysms, arteriovenous malformations or vascular tumours, is usually obvious during surgery (if one looks!) and with the exception of very young children it is impossible to bleed sufficiently into the closed cranium to cause hypotension from blood loss. In trauma patients, haemorrhage from extracranial sites must be considered.
   (b) Brainstem stimulation (less common than hypertension).
3. Patient.
   (a) Spinal shock. With spinal cord trauma above T6–7 the patient usually presents with varying degrees of hypotension. In the setting of a multitrauma or with an unconscious patient it may be difficult to assess neurological function. Before intubating and paralysing such a patient, if no spontaneous movement is present, check specifically to see if any movement occurs in reaction to painful stimuli. If no limbs move then one must keep in mind the possibility of spinal cord injury. The lack of obvious neurological damage does not rule out the presence of spinal fractures; iatrogenic spinal cord injury may occur if this is not considered.

   Spinal shock consists of an acute loss of sympathetic tone and leads to hypotension and bradycardia (cf. blood loss). The CVP will be low, as will the peripheral resistance, and the cardiac output is normal or slightly elevated. In the long term, blood pressure management depends on vital organ perfusion and blood pressure *per se* should not be the end-point.

   Acutely, vital organ perfusion is hard to assess and the author aims for a systolic blood pressure of about 100 mmHg. If the heart rate is slow then atropine may be used. When fluids are given the CVP tends not to rise until the venous capacity is filled and then it may increase suddenly, predisposing to pulmonary oedema. If, despite seemingly adequate fluids, the blood pressure is still low but the peripheries are warm and well perfused, a vasoconstrictor may be used. If hypotension recurs, look for other sources of hypovolaemia and consider the use of a pulmonary artery catheter (PAC).
   (b) Brainstem death. Hypotension does not occur secondary to intracranial pathology until the brainstem has effectively ceased to function. If it is in the setting of raised ICP it is the terminal phase of coning.

Management

1. Detect and correct any anaesthetic-related problem.
2. Check and correct rhythm. Look for atrial arrest with loss of atrial filling. If the heart rate is low give atropine. If the heart rate is high and the ECG shows a narrow QRS complex, assume volume depletion is the cause rather than a supraventricular tachycardia.
3. If CVP is low give fluids. Note that only iso-osmotic fluids should be used (e.g. 0.9% saline). Isotonic colloids may also be used and are more effective on a millilitre for millilitre basis than crystalloids but are more expensive. If the patient is bleeding then blood should be used to maintain the haemoglobin at about 100 g/l.

4. Check for excessive operative bleeding.
5. Check for non-intracranial bleeding.
6. If CVP is high think of pneumothorax.
7. If these do not work and the blood pressure drop is severe give metaraminol in 1 mg boluses to maintain blood pressure until the correct diagnosis is made.
8. If CVP is high and there is no pneumothorax then consider cardiac failure (e.g. infarction) or cardiac tamponade. Treat failure with inotropes (e.g. dopamine or adrenaline) and tamponade with drainage of the tamponading fluid. Cardiac tamponade in a trauma patient may indicate aortic dissection.
9. Consider unusual causes, e.g. spinal shock, drug interactions, brainstem damage, adrenal insufficiency.

Sudden patient movement/coughing

Differential diagnosis

1. Surgical stimuli (see hypertension).
2. Inadequate paralysis. The usual means of preventing patient movement is by paralysis. Monitoring of paralysis should occur at least every 10 minutes, aiming for suppression of all train-of-four twitches, repeat doses of relaxant being given when one twitch returns. An infusion may be easier and, if used, may be adjusted so that all train-of-four twitches are abolished but a post-tetanic count of at least 10 is maintained.
3. Patient conscious.

Management

1. Turn the ventilator off (this minimizes the rise in intrathoracic pressure).
2. Thiopentone 100–500 mg (see hypertension).
3. Hand ventilate the patient until the movement ceases.
4. Increase the long-term reflex depression, either centrally or by paralysis.

Unexplained bleeding—coagulopathy

Differential diagnosis

1. Disseminated intravascular coagulopathy. In severe head injuries the release of brain thromboplastins may cause disseminated intravascular coagulopathy (Astrup, 1965; Kaufman et al, 1981). This may be present by the time the patient arrives in the emergency room. Coagulation tests must be part of the initial blood testing of these patients. The failure of coagulation to return to normal is an indicator of continual brain damage. The mortality rate of patients is greatly increased in patients who have disseminated intravascular coagulopathy compared with those patients who have a similar brain injury but normal coagulation (Miner et al, 1982).
2. Dilutional coagulopathy. Patients having massive transfusions (more than half their estimated blood volume) should have regular measurements of coagulation and platelet counts (every 30–60 minutes).
3. Pre-existing coagulation defect, e.g. haemophilia.
4. Antiplatelet drugs, e.g. aspirin, sodium valproate.

Management

Replace the coagulation factors and platelets that are deficient. Fresh frozen plasma is used to replace general coagulation deficiencies but specific pre-existing coagulation disorders should be treated with specific factor replacement (e.g. haemophilia with factor VIII).

Unexpected postoperative coma

The patient must be able to be neurologically assessed within 10–15 minutes of the operation finishing so that surgical problems can be detected and treated before permanent neurological damage occurs. It is wrong to assume that after a long neurosurgical procedure the patient will take a long time to regain consciousness. One must aim for an anaesthetic technique that allows rapid recovery. There are times, however, when it is not expected that the patient will recover quickly (e.g. severe head injury with brain swelling); in this section it is assumed that the patient is expected to regain consciousness quickly.

Differential diagnosis

1. Anaesthetic.
   (a) Hypoxia, hypercarbia, hypotension, hypothermia.
   (b) Pharmacological: drug overdose, anticholinergic syndrome, drug interactions (e.g. monoamine oxidase inhibitors, lithium), residual paralysis.
2. Surgical.
   (a) Cerebral ischaemia, e.g. intraoperative ischaemia or misplaced aneurysm clips.
   (b) Increased intracranial pressure, e.g. intracranial bleeds, cerebral oedema, pneumocephalus, hydrocephalus.
   (c) Direct surgical trauma, e.g. brainstem damage, extensive frontal lobe resections.
   (d) Seizures.
3. Patient.
   (a) Hypothyroidism.
   (b) Diabetes, e.g. hypoglycaemia, hyperglycaemia (ketoacidosis, non-ketotic coma).
   (c) Metabolic disorders, e.g. hepatic and renal failure.
   (d) Electrolyte abnormalities, e.g. hyponatraemia, hypercalcaemia.

(e) Unrecognized drug ingestion, e.g. alcohol, narcotics, cocaine, phencyclidine.

Management

1. Exclude or correct physiological causes.
2. Reverse narcotic effects with naloxone 40 μg increments (≤400 μg).
3. Reverse benzodiazepine effects with flumazenil (≤0.5 mg).
4. Use physostigmine 1 mg increments (≤4 mg) if central anticholinergic syndrome suspected.
5. Check blood sugar, electrolytes, blood gases and haemoglobin.
6. Urgent computed tomography scan or angiogram depending on type of surgery; these must be done if there is any doubt about the cause of delayed awakening.
7. If all else normal, consider rare causes.

SPECIFIC CONDITIONS

Cerebral oedema

The determinants of fluid movement across cerebral capillaries is described by the Starling equation, which predicts the *net* movement of water across a capillary membrane.

$$J_v = K_f[(P_c - P_i) - \delta(\pi_c - \pi_i)]$$

J_v = net flow
K_f = filtration coefficient
P_c, P_i = hydrostatic pressures in capillary and interstitium
δ = Staverman reflection coefficient
π_c, π_i = Effective osmotic pressure in capillary and interstitium

Osmosis is the movement of solvent molecules across a semipermeable membrane down an activity gradient for the solvent. This activity gradient is created by the presence of freely moving particles (the solute) in solution (i.e. a colligative property of a solution). The more of these particles, the less the activity of the solvent and the greater the gradient between it and a pure solvent. The osmotic force is the force created by this activity gradient. The term 'semipermeable membrane' refers to a membrane that has differential permeability to solute and solvent molecules. In all animals the solvent is water. If there were no barrier to the movement of all solutes (i.e. a fully permeable membrane), then the solutes would move down their activity gradients (diffusion), come into equilibrium and no osmotic force would exist. Almost all membranes in the body (and all capillary membranes) are freely permeable to water but have differing permeabilites to solutes.

One must draw a distinction between total osmolality (normally 285 mosmol/kg in plasma) and effective osmolality. Effective osmolality relates only to the osmotic force of those solutes that cannot cross the capillary membranes in question. The Staverman reflection coefficient is the measure of the impermeability of the membrane to those solute molecules. For most capillary membranes the only solutes that do not freely cross are proteins, and therefore the effective osmotic force is only related to these proteins (the oncotic pressure). The effective osmolality of these plasma proteins is quite small (about 1.5 mosmol/kg) but results in a plasma oncotic pressure of approximately 25 mmHg. For teaching purposes we usually simplify the Starling equation by assuming that the capillary membranes are completely impermeable to proteins (the Staverman reflection coefficient is therefore 1 and disregarded). If one is only interested in the net driving force for water movement, rather than the absolute amount of the flow, the filtration coefficient (which takes into account the permeability to water and the surface area of the membrane) can also be removed and the equation restated in its usual form:

$$\Delta P = (P_c - P_i) - (\pi_c - \pi_i)$$

In addition, even if this is positive, it does not necessarily predict that the interstitial fluid compartment will increase in size because the egress from that compartment is not included. The initial movement of fluid into the interstitium will increase the interstitial pressure and impede further flow. The interstitial compartment is a gel structure with limited compliance, although this varies considerably (that of the gut is quite high). Reulen et al (1977) demonstrated the marked non-compliance of the brain interstitium and its resistance to the entry of fluid until the driving forces become quite high. This is due to its network of glial cells.

In the case of the blood–brain barrier the functional pore size is of the order of 0.7 nm compared with about 6.5 nm in non-CNS tissues), which is small enough to preclude the free passage of sodium. This means that the osmotic driving force becomes total osmolality and not oncotic pressures. Reducing the plasma sodium concentration (and its associated anion) by 0.5 mmol/l has a greater osmotic effect than halving the plasma protein concentration!

The neuronal cell membrane is essentially impermeable to these same solutes and transcellular water movement is determined purely by osmotic gradients. Hydrostatic forces have a very limited role because the cell membrane is relatively compliant and any pressure changes equilibrate by changing the cell volume.

Brain oedema has been defined as 'an abnormal accumulation of fluid within the brain parenchyma associated with a volumetric enlargement of the brain volume' (Klatzo et al. 1967). It has traditionally been divided into two types: cytotoxic and vasogenic, although it is uncommon for only one type to be present.

Vasogenic oedema occurs when an increase in the cerebrovascular permeability leads to the leakage of serum proteins into brain parenchyma. The unopposed hydrostatic forces will then move water into the interstitium, initially causing a marked rise in interstitial pressure. The hydrostatic pressure eventually overcomes this and the greater the hydrostatic pressure at the time of injury the greater the oedema formation. Fluid then

tends to spread outwards along the white matter due to the anatomical differences between white and grey matter. The ultrastructure of the grey matter resembles a tight tangle of cellular structures, whereas the white matter has a more straight and ordered structure, providing a lower path of resistance. The movement of this protein-rich fluid to areas of the brain that have a normal blood–brain barrier means that further fluid will then shift into the interstitium across these capillaries due to the increase in the interstitial oncotic pressure.

The clearance of vasogenic oedema could be by any one or a combination of three routes: CSF, blood or lymphatics (Marmarou et al, 1984). Bulk flow into the CSF, across the ventricular wall, is restricted to the early phase of oedema formation and after this the resolution of oedema is dependent on clearance of the protein. The clearance by the vascular route was considerably less than via CSF, with the lymphatics playing an insignificant role.

Vasogenic oedema occurs in tumours, trauma, infection and acute severe hypertension. Interestingly, the site of the disruption of the blood–brain barrier in arterial hypertension is the venule (Mayhan and Heistad, 1985), which is the same site as for venous hypertension and hyperosmolar disruption of the blood–brain barrier (Mayhan et al, 1986).

Hyperperfusion of the normal brain can also occur following the resection of the arteriovenous malformation. This has been called normal pressure breakthrough. When the malformation is present, the large shunt decreases the CPP in the normal brain and this leads to chronic vasodilatation and a loss of autoregulation. When the perfusion pressure increases following the clipping of the arteriovenous malformation feeding arteries, the excess blood flow may cause cerebral oedema or even intracerebral haemorrhages. Even relatively small increases in blood pressure in this setting may cause major problems.

Cytotoxic cerebral oedema is the result of swelling of the cellular components, usually due to failure of the membrane ion pumps which allows sodium and then water to enter the cell. This is commonly the result of ischaemia but can also occur with toxins (e.g. cyanide). The blood–brain barrier remains intact. A second type of cytotoxic cerebral oedema occurs when the plasma osmolality is acutely reduced, e.g. water intoxication. This reduction in osmolality causes water to enter the brain cells. Chronic changes in osmolality are much better compensated for as the brain appears capable of adjusting its intracellular osmolality to preserve brain volume. The acute normalization of chronic hypernatraemia is associated with the risk of cerebral oedema.

A third type of oedema, interstitial oedema, occurs rarely. This is characterized by an increase in sodium and water content of the periventricular white matter when overdistension of the ventricles (hydrocephalus) causes rupture of the CSF–brain barrier, permitting CSF to penetrate into the brain tissue and spread into the extracellular space of the white matter. It differs from the usual type of vasogenic cerebral oedema in that the interstitium contains almost no protein.

Ischaemic brain injuries produce both vasogenic and cytotoxic oedema. In the early part of the injury cytotoxic oedema predominates, due to failure

of the sodium–potassium ion pumps. In global complete ischaemia there is no brain oedema (as defined by Klatzo et al, 1967) as there is no overall increase in brain water, only the transfer of water from the interstitial to the intracellular compartment. In incomplete ischaemia the continual supply of water makes oedema possible. After the ischaemic insult (if it resolves) there is a period of marked hyperaemia which is associated with an opening of the blood–brain barrier and marked increases in capillary pressures. The factors determining the extent of this injury are the intensity of the preceding injury and the amplitude and rapidity of development of the reactive hyperaemia (Klatzo, 1985).

A second phase occurs after a delay, due to the ischaemic damage of the capillaries (and thus the blood–brain barrier) allowing more proteins to enter the interstitium (Wagner et al, 1983). The accumulation of a hyperosmotic mixture of proteins and necrotic cell contents further increases oedema. This spreading oedema surrounding an infarcted central zone may produce a vicious cycle of impaired microcirculation and worsening oedema, leading to an advancing zone of irreversible oedema. Maximal oedema occurs 48–72 hours after the initial insult. The resolution of an ischaemic brain injury involves the return of membrane ion pump function (if ischaemia is reversible) and the resolution of the vasogenic oedema. The later is related to the uptake of extravasated proteins by macrophagic cells and the formation of a dense mesodermal and glial scar tissue (Klatzo, 1985).

The brain swelling following brain trauma (either global, e.g. head injuries, or focal, e.g. beneath a retractor) may involve both cytotoxic and vasogenic oedema in addition to any haemorrhage that may occur. There is a cytotoxic cerebral oedema in those cells that have died or have impaired function, surrounded by a zone of altered capillary permeability leading to vasogenic oedema. These injuries may then be compounded by the physiological consequences of the neuronal dysfunction (i.e. hypotension and hypoxia), producing secondary brain injury. There is also a syndrome seen in younger head-injured patients (post-traumatic brain swelling) in which there is computed tomographic evidence of symmetrical brain swelling but without the decrease in radiolucency seen with cerebral oedema. This is thought to represent reactive hyperaemia and an increase in blood volume (Zimmerman et al, 1978).

The concepts of permeability and osmotic forces are important in fluid therapy. Zornow et al (1987) demonstrated in animals with normal brains that a reduction in colloid osmotic pressure of more than 50% did not lead to detectable changes in brain water content, whereas even small decreases in total plasma osmolality led to significant increases in intracranial pressure and brain water content. It has been demonstrated in cryogenically-injured brain that changes in plasma oncotic pressure do not cause an increase in brain water (Kaieda et al, 1989a, 1989b). They also found that the increase in brain water that occurred with reductions in total osmolality occurred only in the relatively normal brain regions. The reductions in COP that these studies used equates to a reduction in osmotic pressure of about 10 mmHg and, while this may increase interstitial fluid in other areas of the body, it is insufficient to do so in the low compliance brain interstitium.

What is not commonly realized is that one of the usual intravenous fluids (lactated Ringer's) is in fact hypo-osmolar. This fluid usually has 280 mmols/l of ions but because there is incomplete dissociation the actual osmolality is about 260 mosmol/kg. Saline 0.9%, which has 308 mmol/l of ions, has an osmolality of some 285 mosmol/kg. Tommasino et al (1988) showed that profound isovolaemia haemodilution led to increased brain water and a raised ICP, but we now realize that this was due to their use of lactated Ringer's solution for haemodilution. It is thus dangerous to use hypo-osmotic fluids in the resuscitation of neurosurgical patients.

When measuring total plasma osmolality there are uncommon circumstances (e.g. renal failure, alcohol intoxication) in which osmotically active particles (e.g. urea and alcohols) are present that are not clinically relevant because they freely cross all membranes. These increase the measured osmolality so that a 'normal' osmolality may in fact be effectively hypo-osmolar and lead to cerebral oedema.

Management

The general management of patients with raised ICP or brain swelling has been covered previously. Cerebral oedema, once the aetiological event has been treated, requires physiological support of the patient until the oedema resolves. Steroids have a role to play in patients with cerebral oedema associated with cerebral tumours but take some days to be fully effective.

The management of patients who have hyponatraemia depends on the severity of the hyponatraemia and its physiological consequences. The asymptomatic patient may be treated with water restriction and the use of saline 0.9% for replacement fluids. If a patient is symptomatic (seizures or clinical evidence of raised ICP), treatment should include the use of hypernatraemic fluids. Saline 1.8–5.0% is used with the aim of increasing the [Na] at a rate of ≤ 2 mmol/l^{-1} h^{-1}. Rates faster than this have a theoretical risk of inducing central pontine myelinolysis. This is a demyelinating syndrome, mostly seen in alcoholics, characterized by quadriplegia and bulbar and pseudobulbar signs. It has a very bad prognosis.

$$\text{Sodium deficit} = \frac{140 - [\text{Na}]}{140} * [0.6] * \text{body weight}$$

While sodium is only in the extracellular fluid one must correct total body osmolality, hence the factor of 0.6. The formula is only a guide and one needs to make hourly [Na] measurements to guide dosing. The choice of which sodium concentration to use depends on the overall water balance of the patient. If the patient is dehydrated a lower concentration may be used. Initial resuscitation, if necessary, should occur with saline 0.9%. The use of hypertonic sodium will lead to water moving into the vascular compartment and may lead to pulmonary oedema. Central venous pressure monitoring is essential if hypertonic sodium is used, and frusemide may be necessary to prevent intravascular fluid overload.

Air embolism

Pathophysiology

Fundamentally, the risk of air embolus during neurosurgery (other than as a result of accidental injection of air) relates to the development of sub-atmospheric pressures in the venous system. This occurs when the patient's head is elevated and when some portion of the venous system is further above the heart than the height of the CVP (in cm of water). Some of the veins in the head and neck (e.g. venous sinuses and diploic veins) do not collapse when subatmospheric pressures are generated, thus allowing air to enter should they be opened.

Classically this occurs in the sitting position but it can occur any time the head is elevated above the level of the heart (e.g. head-up tilt, elevation of the head by neck flexion). The greater the negative pressure the greater the amount of air that any given hole will allow in. It is possible easily to determine the risk of air embolism if one is using CVP monitoring. If one zeros the transducer at the level of the highest point of the cranium, the pressure reading will indicate the lowest possible pressure that could exist in the venous system. If it is positive there can be no risk of air entering.

An uncommon mode of air embolus is when the patient has a ventricular–atrial shunt. If the ventricular system is opened to air, the CSF may drain back into the atria and then air enters. The presence of such a shunt is either a relative contraindication to elevation of the head above the heart or an indication for the shunt to be temporarily clipped.

There have been a number of studies (Standefer et al, 1984; Matjasko et al, 1985; Young et al, 1986; Black et al, 1988) that attest to the safety and benefits of performing surgery in the sitting position, however the use of this position is becoming less frequent and in this author's institution we have not performed an operation in the sitting position since 1980 (this is despite having two neurosurgeons who had training at the Mayo Clinic). The avoidance of the sitting position does not prevent air embolus. Black et al (1988) showed that if one uses the 'horizontal' position (0–20° head elevation!), there is a lower incidence of air embolus compared with the sitting position, but it is still a clinically important occurrence (12 versus 45%).

Air embolism can be divided into two types: venous air embolus (VAE), which is quite common but may not produce morbidity or mortality; and paradoxical air embolus (PAE), in which air finds its way into the arterial system. The risk of PAE in the sitting position became recognized much later than the risk of VAE, being first reported by Gronert et al in 1979. It's exact incidence is not known, however it is probably less than 1% (Bedford et al, 1981; Black et al, 1988).

The threshold for complications with PAE is much less than with VAE. Spencer et al (1965) demonstrated that 0.4–0.5 ml kg^{-1} min^{-1} via the venous system was tolerated with minimal haemodynamic changes. In contrast to this, 0.6 ml of air/blood foam injected into the carotid artery of cats led to evidence of cerebral ischaemia (EEG slowing), cerebral swelling and an

increased ICP (Fritz and Hossmann, 1979); 0.1–0.2 ml injected into the coronary arteries of dogs causes infarction (Goldfarb and Bahnson, 1963). The other problem is that the detection of intra-arterial air is much more difficult. The surgeon's observation of air in an intracranial artery, the presence of haemodynamic/ECG changes, and changes in electrophysiological monitors are clearly much too late. The only device that allows the detection of air in the left side of the heart is the transoesophageal echocardiogram (TEE). This is exceedingly expensive and requires a dedicated person to run it; furthermore, once PAE is suspected the air cannot be removed (Cucchiara et al, 1984).

The question is: Which patients are at risk for PAE and should not have their heads elevated above the heart (even in the 'horizontal' position)? It has been presumed that the risk relates to the presence of a right-to-left vascular shunt, but transpulmonary passage of air is possible (Butler and Hills, 1979). This potentially makes everyone at risk for PAE but its low incidence, given the high incidence of VAE, suggests that it is not common.

Patients definitely at increased risk are those with known right-to-left cardiac shunts but 25–30% of the population have a probe-patent foramen ovale, which is a foramen ovale that is normally closed but may be opened by a cardiac catheter (Gronert et al, 1979). If the right-sided pressures exceed the left, then it may open and allow right to left shunting. Up to 50% of patients in the sitting position may develop a right atrial pressure in excess of the pulmonary artery wedge pressure and, presumably, in excess of the left atrial pressure (Perkins-Pearson et al, 1982). On the other hand the concept that atrial pressure gradients determine paradoxical embolism has been questioned by the work of Black et al (1989) in a pig model with an iatrogenic atrial septal defect. In their model, the relationship between left and right atrial pressures did not influence the occurrence of PAE. Using precordial two-dimensional echocardiography and the Valsalva manoeuvre (which increases right atrial pressures and decreases left atrial pressures) Guggiari et al (1988) showed that the injection of agitated saline into a peripheral vein could demonstrate a substantial proportion of patients with a probe-patent foramen ovale. It would seem reasonable to avoid the risk of VAE in these patients but Cucchiara et al (1989) showed that, despite this type of preoperative testing, PAE can still occur. Whether some assessment of the potential for PAE should be used in all patients for whom VAE is a risk is not clear.

The cardiovascular effects of VAE (in the absence of PAE) depend on the rate, and absolute magnitude, of the volume of air that enters. Usually small amounts of air precede the entry of a large volume of air. These are then carried towards the heart where there is a tendency for them to accumulate near the junction of the superior vena cava and right atrium (near the sinoatrial node), a fact that can be used to position a central venous catheter for optimal aspiration of air (Bunegin et al, 1981). The air then either accumulates in the more upright portions of the atrium or enters the ventricle. A large bolus of air (0.5–1.0 ml/kg) will fill the right ventricle and prevent ejection, leading to complete cardiovascular collapse (Durant et al, 1954; Spencer et al, 1965). The entry of smaller volumes of air will produce a

syndrome similar to thrombotic pulmonary emboli. The initial pulmonary response is a rise in pulmonary vascular resistance due to pulmonary vasoconstriction (it occurs with a volume of air too low to produce mechanical obstruction). If the rate of air entry is slow, clearance from the lungs may produce an equilibrium. At a later stage there appears to be an increase in pulmonary shunting which may produce hypoxaemia. The rise in pulmonary artery pressure will initially be tolerated but later there will be a rise in right atrial pressures, a fall in cardiac output and finally a fall in blood pressure (Adornato et al (1978)).

If the patient survives a major VAE then postoperative pulmonary dysfunction may occur.

Diagnosis

The most sensitive monitors are the precordial Doppler and the TEE. These can detect bubbles of between 0.05 and 0.2 ml and at an infusion rate of $0.02\,ml\,kg^{-1}\,min^{-1}$ and have a very low false-negative rate (approximately 3%) (Bedford et al, 1981). The TEE is slightly more sensitive than the precordial Doppler because it suffers less from interference by pulmonary disease or obesity. It can also detect PAE and give information about cardiac function. Unfortunately it is not an option for most centres.

The next most sensitive monitor is the capnograph, which will demonstrate a decrease in $P_{ET}CO_2$ at air infusion rates of $0.4\,ml\,kg^{-1}\,min^{-1}$. The specificity depends on the absence of major changes in the cardiovascular system. Any fall in cardiac output or blood pressure may be associated with a fall in $P_{ET}CO_2$. This monitor is always available in modern anaesthetics. The PAC has approximately equal sensitivity to the capnograph, the increase in pulmonary artery pressure occurring at a similar time to falls in $P_{ET}CO_2$ (Bedford et al, 1981). $P_{ET}N_2$ (provided one is not using air as part of the anaesthetic gases) is slightly less sensitive and can detect VAE at $0.6\,ml\,kg^{-1}\,min^{-1}$ (Russell et al, 1988). One advantage is that it is slightly more specific, being less effected by cardiovascular changes than the $P_{ET}CO_2$. The CVP will then increase and the cardiac output will fall prior to the blood pressure falling ($0.7\,ml\,kg^{-1}\,min^{-1}$). There is some difficulty ranking these last few changes because Gildenberg et al (1981) showed that the CVP and pulmonary artery pressure increases occurred at a similar rate of VAE (0.40 versus $0.42\,ml\,kg^{-1}\,min^{-1}$) and was not much less sensitive than the $P_{ET}CO_2$. Perhaps the practical way to resolve this argument is to point out that all techniques, other than a Doppler, detect the occurrence of VAE at air entry rates that are quite large (approximately 30 ml/min in a 70 kg person) and not much less than are needed to produce a fall in blood pressure. A Doppler detects VAE when its entry rate is, at most, a twentieth of the next most sensitive device. As an aside, the classical mill wheel murmur is only heard at an infusion rate of $1.7\,ml\,kg^{-1}\,min^{-1}$, when cardiovascular collapse occurs, and is thus useless in the detection of VAE.

The value of early monitoring devices is based on the usually valid assumption that air initially enters slowly. However, if a major venous sinus is suddenly opened, a single large bolus of air may enter, causing cardiovascular

collapse by an air lock in the right heart (the PAP will decrease instead of increasing). If the diagnosis of VAE is suspected as the cause of death it is critical that this is made known to the pathologist performing the post mortem so that the opening of the heart and pulmonary vasculature is done under water to allow air to be detected.

What monitoring should be used in cases where there is a significant risk of VAE? Firstly, one should discuss the case with the neurosurgeon and decide if the risk is justified, and this should be documented. Having decided to proceed, the only additional monitoring one needs is a Doppler device (usually a precordial Doppler). The precordial Dopplers use an ultrasound frequency of 2.0–2.5 MHz and generate a high-pitched swishing sound with blood flow, and lower pitched, more transient sounds represent myocardial and valvular movements. Air, which is an excellent acoustic reflector, produces an erratic roaring noise. The machine used should have diathermy detection so that the sound, broadcast to the whole operating room, is turned off during diathermy. Otherwise the noise will soon mean that the volume will be turned down so far that the device is useless. The Doppler probe should be positioned over the right side of the heart (to the right of the sternum between the third and sixth interspaces). It's correct placement should be confirmed by either by injecting 0.5 ml of carbon dioxide (not recommended) or the rapid injection of 10 ml of saline via the correctly placed central line, the sound of which can then be heard by all in the operating room.

The type of catheter used in patients at risk of VAE differs from the usual central line. It has been shown, in animals, that multiorifice single lumen catheters are superior to single orifice, single lumen catheters for aspiration of air, and that this improved ability to aspirate air decreases the mortality from VAE (Artru and Colley, 1985). The catheter should be located at the point at which air is mostly likely to accumulate (near the sinoatrial node). With a multiorifice catheter the proximal orifice should be located just into the superior vena cava so that the majority of the holes are located at the superior vena cava/high right atrium. Positioning can be done by a variety of techniques (radiography, pressure recordings, or ECG traces) but the most practical is the use of the ECG trace.

Commercially available adaptors (Johans ECG adaptors, ARROW Corporation) allow one easily and inexpensively to attach the chest lead of the ECG to the central line. The central line should then be filled with a sodium-containing fluid. The impedance, and interference, can be reduced by using 8.4% sodium bicarbonate (only 2–3 ml are needed). The ECG trace from a multiorifice catheter probably comes from the proximal hole (Artru and Colley, 1988), so the usually described technique for the placement of a single orifice catheter needs some modification. Normally the catheter is inserted until the P wave is equally biphasic, indicating a position in the middle of the atrium. With the multiorifice catheter it should be inserted until the P wave becomes biphasic (to indicate that one is nearing the sinoatrial node with the proximal port) and then withdrawn until the positive component has disappeared. This should result in the proximal port being some 2 cm back into the superior vena cava. The tracing should be rechecked once the arm is returned to the side as the catheter tends to move more centrally and it may need to be withdrawn slightly (Lee et al, 1984).

It is impractical to insert a PAC as well as a central venous catheter so why do I not choose the PAC instead? The PAC does provide information that the CVP does not, but none of it is more sensitive than the capnograph for diagnosis of VAE. It has previously been pointed out that the presence of a right atrial pressure greater than the pulmonary artery wedge pressure does not prevent PAE and the single non-optimally placed proximal port of the PAC is inferior to the ideally placed multiorifice catheter for the aspiration of air. In addition, the placement of a PAC entails greater risks than the placement of a peripheral central line (even when the PAC is inserted peripherally). The only unique information that the PAC provides is an assessment of cardiac output and left-sided pressures, which is not needed in the majority of cases.

Treatment of VAE

As soon as VAE is detected the surgeon should be notified and nitrous oxide (if used) should be discontinued and 100% oxygen commenced. An early warning may give the surgeons the clue they need to identify and seal a cut venous structure (sometimes dural sinuses may be the cause and bone wax is often effective). If it is not immediately obvious where air is entering it is best to flood the area with saline (considerably easier to do if the operation is being done in the horizontal position). It has been recommended that gentle compression of the jugular veins be used to increase the cerebral venous pressure, so stopping further air entry and allowing the vein to bleed, which aids in its identification. The pressure applied must not occlude the carotids and should not continue for more than 30 seconds, otherwise it may cause the brain to bulge from the wound. Unfortunately, due to drainage of blood via the vertebral venous plexus, jugular vein compression may not increase the cerebral venous pressure. Once an episode of VAE has occurred, nitrous oxide should not be used unless the entry point has been identified and closed.

If the case is being done in a 'horizontal' position then management is a lot simpler. Dropping the head of the table so that the head is below the heart level immediately prevents further VAE and allows the surgeon to search for the site of entry without any need to press on neck veins.

If the VAE has produced haemodynamic changes, one should also endeavour to aspirate as much air as possible from the central venous catheter. The use of volume and vasopressors will, hopefully, support the circulation until the air has cleared. If the cardiovascular changes are marked, the patient should be placed supine (if the sitting position is used), although this may contaminate the wound and makes any further operating impossible (another disadvantage of the sitting position).

Theoretically, placing the patient 15° head down and right side up (Durant position) will reduce the amount of air entering the heart (again this is easier to do from the 'horizontal' position). However, placing the patient fully lateral when this was not the original operating position is somewhat impractical and precludes effective external cardiac massage.

Following a large VAE, if a clearly identifiable site of entry can be found, it is unlikely that another breach will occur, the patient is haemodynamically stable and there is no evidence of cerebral ischaemic damage (brain swelling, abnormal EEG/evoked potentials), then it is reasonable to continue the operation.

The use of PEEP either in an attempt to prevent VAE or in its treatment is not advised (Lee et al, 1981; Toung et al, 1984; Grady et al, 1986) and its use may increase the risk of PAE (Perkins and Bedford, 1984).

Acute aneurysm rupture

Management

Perioperative aneurysm rupture is a potential disaster. This is especially so if it occurs prior to the surgeon being able either to clip the aneurysm itself or apply temporary clips. Batijer and Samson (1986) reviewed their experience of aneurysm ruptures. Aneurysm rupture increased the incidence of unfavourable outcome from 12 to 62%. The timing of the rupture had a major influence on outcome. Rupture which occurred before dissection had a 75% mortality, rupture during blunt dissection had a 50% unfavourable outcome rate, and if rupture occurred during sharp dissection (when either the blood pressure is low or temporary clips are used, and the surgeons are in place to treat the rupture), all patients recovered well. Rupture at clip application had a variable outcome, depending on the cause. If due to incomplete dissection it was likely to be a large and difficult-to-control tear and was associated with a 41% unfavourable outcome rate. If the bleed was due to poor clip application or some type of clip failure it was easier to control and did not necessarily lead to an unfavourable outcome.

Aneurysm rupture occurs because the wall stresses exceed the tensile capability of the aneurysm wall. Before surgical dissection the major factor determining this is the transmural aneurysm pressure, which is the difference between the intra-aneurysmal pressure (the arterial pressure at the level of the aneurysm) and the intracranial pressure. Much is made about the dangers of allowing the ICP to fall before dural opening but unless one places the patient in a steep head-up position the ICP will not be reduced below zero by any of the standard techniques used to reduce ICP (e.g. diuretics, barbiturates, hypocarbia). In the usual situation, unless the ICP starts off markedly elevated (which is uncommon), the maximum possible rise in transmural aneurysm pressure due to an ICP fall is only about 10 mmHg. This is an order of magnitude lower than the mean arterial pressure and within the variation that commonly occurs during the respiratory cycle of controlled ventilation. It is very unlikely that this would be clinically important and therefore attempting to maintain the ICP before dural opening will only result in inadequate conditions for surgery. What one needs to concentrate on is controlling the blood pressure and being meticulous in preventing it exceeding the patient's usual ward levels. One must be particularly careful during the periods of greatest stimuli (see section on hypertension).

A rupture occuring before the dural incision is a disaster. The rupture may manifest itself as a severe episode of hypertension which may be difficult to differentiate from the aetiological event. Definitive treatment requires clipping the aneurysm. The anaesthetist should cease all agents that might increase the ICP, use 100% oxygen, and change to a barbiturate infusion (methohexitone is best because of its shorter half-life but thiopentone will do). Acutely, boluses of thiopentone may be used until the blood pressure had fallen to baseline levels and then the infusion may be adjusted in order to maintain an isoelectric EEG or, if this is not in use, the blood pressure to its preoperative level. If it has not already been done, 1.5 g/kg of mannitol and 0.3 mg/kg of frusemide may be administered. The aim is to reduce the ICP to a survivable level until the surgeon can clip the aneurysm.

Using nitroprusside before the dura is opened should be avoided because cerebral vasodilatation may further increase ICP and cause coning. Once the dura is open and the surgeon is in a position to start the dissection then nitroprusside may be used to lower the mean arterial pressure to about 50 mmHg until the aneurysm is clipped or temporary clips are in place.

SUMMARY

Most emergencies that occur during neuroanaesthesia are the same as those that occur during any other anaesthetic (e.g. hypoxaemia, hypotension) and are treated in the usual way. Airway problems are a particular concern as access is usually limited. One must always consider the patient's airways, breathing and circulation before looking for specific neurosurgical problems.

We are always interested in preventing vital organ damage but in neurosurgery the potential for cerebral damage is accentuated. Once the general causes have been eliminated, increases in ICP/brain swelling are most commonly due to the underlying process (e.g. head injuries) but specific anaesthetic and surgical problems must be identified and corrected. If no correctable cause is found then head-up tilt, hypocarbia, diuretics, intravenous anaesthetics and CSF drainage are used.

Acute hypertension is dangerous because it may cause aneurysm rupture and increases in ICP/brain swelling. It is usually due to inadequate reflex depression resulting from failure to anticipate the stimuli. Acutely, it should be controlled with thiopentone and then by increasing the longer-term reflex depression either centrally (e.g. narcotics) or peripherally (e.g. labetalol). Cerebral vasodilators should be avoided.

Hypotension is usually due to blood loss or drug effects. One must not forget spinal cord damage and non-intracranial sites of bleeding as causes.

Sudden movement or coughing is treated with thiopentone and then either increasing the central reflex depression or paralysis.

Cerebral oedema is an increase in brain parenchymal volume due to excess water; it is divided into vasogenic and cytotoxic types. These are best

understood by considering the factors that determine water movement.

Air embolus may occur at any time that part of the venous system has both a subatmospheric pressure and has been breached. Air may either stay within the venous system or gain access to the arterial system. The consequences of the latter are much worse. Management is by preventing its occurrence or by early detection of its entry.

Aneurysm rupture is best dealt with by preventing it happening! If it does, and the dura is closed, one needs to control the ICP and reduce the blood pressure to its usual levels with thiopentone and then clip the aneurysm as soon as possible. Induced hypotension should only be used to reduce the bleeding once the dura is opened.

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12

Practical crisis management in the perioperative care of cardiac surgical patients

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'Better a live problem than a dead certainty!'
Contemporary Irish (cardiac) surgical proverb

This chapter is intended to deal with practical measures to correct or to manage potential catastrophes, as they present to the anaesthetist, in the perioperative care of cardiac surgical patients. Where relevant to treatment, some aspects of the pathophysiology of the problems will be explored as well. Our aim is to focus on those problems peculiar to this group of patients. The reader is referred to other relevant chapters in this monograph or more general texts for discussion of crisis management of anaesthesia or intensive care problems outside these terms of reference.

The three sections of the chapter will be devoted to problems during (1) anaesthesia, (2) cardiopulmonary bypass, and (3) immediate postoperative intensive care.

ANAESTHESIA

A. PROBLEMS AT INDUCTION OF ANAESTHESIA

Circulatory emergencies

Problem 1: Cardiac arrest

Despite optimum management, cardiac arrest can and does occur during induction of anaesthesia in cardiac surgical patients. There are clearly defined groups of patients at risk, notably those presenting with left main coronary artery disease, the patients who develop severe myocardial ischaemia in the catheter laboratory whilst undergoing angiography or angioplasty procedures (balloon, stent, atherectomy, etc.), those with tight

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