e) the patient will recover rapidly when anaesthesia has been maintained by a thiopentone infusion

ANSWERS TO MCQ QUESTIONS

1 TFTFT

The normal compensatory mechanism for any brain swelling is a reduction in the volume of intracranial CSF, with squashing of the venous sinuses. This results in a reduction of venous blood volume in addition. However arterial blood volume is not altered. It is changed by autoregulation, arterial CO_2 and O_2 tensions as well as inhalational anaesthetic agents. As a result of compensation, ICP does not rise immediately. Normal ICP is 5-13 mmHg, it is sensible to "guess" ICP as being 20 mmHg after a head injury. This encourages the clinician to think always of, or to calculate cerebral perfusion pressure as arterial pressure changes.

2 FTTTF

Hyperventilating the patient has no effect on venous blood volume as this is a passive system unlike the arterial side. Thus positioning the patient, and anything that affects venous drainage or intra-thoracic pressure will alter cerebral venous blood volume. Similarly a fall in arterial blood pressure has no effect on venous blood volume

3 FTTTF

Autoregulation is a local mechanism which alters the tension in the arterial wall and so the calibre of the artery, controlling CBF. It is a basic mechanism which maintains a constant CBF over a range of arterial blood pressures, and so prevents a fall in CBF with a fall in arterial BP until the lower limit (50 mmHg MAP) is reached when CBF falls rapidly. By causing vasoconstriction, it alters arterial blood volume, which in a decompensated situation (swollen brain) can lead to a fall in ICP as arterial pressure rises over the normal range (50-150 mmHg MAP).

4 FTFFT

When the brain is swollen and stiff the system is working at the the right hand end of the brain volume - ICP curve (fig 2 Neurophysiology). Thus anything that will increase blood volume by even a small amount will increase ICP markedly. Coughing and the head-down position will increase cerebral venous volume. A rising arterial CO_2 and falling O_2 will increase arterial blood volume. Twisting the neck can obstruct venous flow in the great veins, hence patients with high intracranial pressure should be nursed with their head in the mid-line.

5 TFTTF

Cerebral perfusion pressure (CPP) is one of the most important variables to understand and have in mind when deaing with neurosurgical patients. Cerebral perfusion pressure is calculated by SUBTRACTING ICP from MAP.

CPP = MAP - ICP

Often ICP is not known, but it is reasonable to make a sensible guess that it is approximately 20 mmHg when the brain is swollen. This situation would exist for 3-5 days followng a significant head injury. Head trauma which is sufficient to cause unconsciousness, however brief is significant. CPP will fall if arterial pressure falls, but if the patients has a high ICP, it should be treated quickly with colloid, 0.9% saline (normal) or catecholamine boluses such as ephedrine 3-6 mg. It should never be treated with a potentially hypotonic glucose solution (5% Dextrose or 4% Dextrose/0.18% Saline). These solutions are contraindicated as they will exacerbate cerebral oedema.

6 FTFTT

CBF is decreased by arterial hypocapnia because of arterial vasoconstriction. It is also reduced by a direct vasoconstrictive action of the hypnotics, thiopentone and propofol. In contrast the inhalational volatile agents dilate the cerbral vessels so increasing CBF. The opioids have no direct effect, only increasing CBF if the patient who is breathing spontaneously becomes respiratorally depressed leading to a rise in arterial CO_2 tension.

7 TTTTT

If the airway of the patient with a severe head injury becomes obstructed, intra-thoracic pressure rise so increasing cerebral venous volume and ICP. Similarly respiratory failure may be present with a raised arterial CO_2 and low saturation (hypoxia). These will induce arterial cerebral vasodilatation, increasing cerebral arterial blood and then ICP. Volatile agents will increase CBF and ICP in their own right, but if a raised arterial CO_2 is present, the increase is greater. Spontaneous breathing during anaesthesia is always associated with some

respiratory depression. This combination would be severely damaging. In this situation anaesthesia can be maintained with halothane, but the patient needs to be hyperventilated to lower arterial CO_2. Pain will also increase CBF and ICP by a direct action on cerebral arteries. Therefore it should be treated.

8 TFFFT

A multi-trauma patient with a head injury is likely to be in severe pain. Pain can increase ICP, therefore it is not only humane to treat the patient it is intracranially beneficial. However if opioids can be avoided by the use of nerve blocks this should be done. If not suitable, then small intravenous doses of an opioid can be used with the patient's neurological status closely monitored in an intensive care or high dependency environment. In patients who are being ventilated, it is quite safe to use opioids. Provided blood pressure does not fall, there is no change in ICP. If BP falls, autoregulation induces cerebral arterial vasodilatation, which, in a decompensated state, will raise ICP. A patient who is breathing spontaneously, may also have a fall in arterial saturation. This would exacerbate the effects of a rising CO_2. Supplemental O_2 should be given whenever possible to reduce the risk of hypoxia, known to occur when a patient is under the effect of an opioid falls asleep.

9 TFFFF

The increase in ICP occurs because CBF and then arterial blood volume increases. Hyperventilation reduces arterial CO_2, this induces cerebral vasoconsriction which opposes the direct dilating effect of halothane on the cerebral vasculature. Sevoflurane is less soluble than both halothane and isoflurane. Therefore both induction and recovery are rapid. Any fall in blood pressure with halothane may be significant, but is especially important when the patient is decompensated (swollen stiff brain due to oedema, trauma or other pathology). In this situation blood pressure must be kept at control levels by preventing hypovolaemia and supporting the circulation with catecholamines either as boluses or as an infusion. The small doses required in a fully saturated, hypocapnic patient are unlikely to cause arrhythmias. The patient should be carefully monitored for them.

Ether is not an ideal agent for neuroanaesthesia, but if it is the only drug available the disadvantages can be reduced by hyperventilating the patient. Spontaneous breathing should not be allowed.

10 FTFFF

Ketamine increase ICP in patients who are decompensated. Thiopentone reduces CBF by a direct effect which leads to a fall in ICP. This effect is sustained by the reduction in metabolism, also a property of propofol. Any fall in arterial pressure when CPP is critical, the situation when the patient is decompensated with a raised ICP, must be treated immediately.

The rapid recovery following induction of anaesthesia with thiopentone is due to re-distribution of the drug from the brain to other parts of the body, the fat in particular. Therefore, when it is given as an infusion there is a significant risk that it will accumulate and cause a prolonged period of unconsciousness. This is not a major problem other than making neurological assesment difficult and creating the need for an intensive care bed with ventilation postoperatively.

SHORT ANSWER QUESTION POINTS

1. A patient who has had a head injury 12h previously which caused a brief period of unconsiousness now requires an urgent general anaesthetic. Describe your management and technique with reasons.

Points to include in answers

1 Assess the patient for signs of intracranial decompensation, - Glasgow Coma Score, neurological signs: signs of other trauma, particularly the neck: concurrent clinical conditions and previous anaesthesia.

2 Note the physiology of brain swelling, the compensatory mechanism and problems of hypotension, hypoxia, hypercapnia and inhalational volatile agents.

3 Premedication: avoid, only use if the patient is very anxious and then cautious dose of sedative.

4 Describe monitoring patient, especially look for bradycardia and hypotension.

5 Describe induction, choice of agents is less important than the way they are used with an understanding of the complications, particularly

hypotension.

6 Maintenance with artifical ventilation. Again the choice of agents is less important than an adequate explanation of the properties that are useful and the side-effects and how they can be reduced.

7 Postoperative care should include: monitoring of neurological status, the signs of complications to be notes - increasing drowsiness, neurological signs, deteriorating Glasgow Coma Score: the problem off analgesia (see below)

8 Alternative techniques include nerve blocks, sciatic and femoral. They are useful but not reliable and if the situation gets out of control, the patient is worse off than if a good GA was used.

9. Peridural techniques, epidural and spinal, are potentially risky. There is the problem of coning in the presence of cerebral swelling, if a significant leak in the dural sac occurs. In addition if an epidural technique is used, injection of local anaesthetic into the epidural space will transmit a rise of pressure up to the head.

2 What are the methods of pain relief which can be used in a patient who has a fractured ankle and a recent significant head injury.

Points to include in answer

1. Use "mild" analgesics - oral or rectal. Paracetamol, and codeine phosphate are the main drugs. These preparations come as combination tablets. Alternatively, paracetamol can be given rectally and codeine by injection. Paracetamol should be given regularly initially. There is a risk of constipation with repeated doses of codeine.

2. Non-steroidal anti inflammatory drugs are very effective analgesic drugs. However, they do reduce platelet effectiveness and should be avoided while there is a risk of intra-cranial haemorrhage, perhaps for 48h following the head injury.

3. Local nerve blocks should be considered: femoral and sciatic nerve blocks. While they are unreliable for anaesthesia, they are effective for analgesia

4. Opioids can be used as the final method when others methods have been found to be insufficient. Small doses of morphine, 1-2 mg or fentanyl, 10-20mcg, are acceptable. The patient should be nursed in an area with as much experience and nursing as possible, with facilities to intubate and ventilate the patient if necessary. Neurological status should be monitored: Glasgow Coma Score, conscious level, neurological deficits, with instructions to all medical staff if there is any deterioration. Supplemental oxygen should also be given.