Anaesthetic management of cerebral artery aneurysms at Tygerberg Hospital, 1980 - 1982

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Summary

Forty-seven patients were operated on for intracranial artery aneurysms at Tygerberg Hospital, Parowvallei, CP, between January 1980 and December 1982. Problems related to anaesthesia which are peculiar to this condition and the solution thereof are outlined.

The importance of meticulous anaesthetic management in the successful recovery of these patients, the majority of whom are young and in the productive phase of their lives, is stressed.

A plea is made for increased awareness, early diagnosis and surgical intervention to decrease the high mortality rate associated with this lethal condition.

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One reward of modern neuro-anaesthesia has been the improved prognosis of patients presenting for surgical repair of intracranial artery aneurysms.

A quarter of a century ago the anaesthetist occasionally merely rotated through the neurosurgical theatre, and rupture of the aneurysm before skin incision was not infrequent after a stormy induction. The effects of the anaesthetic technique and the agents used on cerebral blood flow and intracranial pressure were not generally known, and it is not surprising that the peri-operative mortality rate was in excess of 25%.

The realization that neuro-anaesthesia is a definitive subspecialty of anaesthesia, combined with the introduction of pharmacological protection of the brain, manipulation of intracranial pressure and brain volume, and hypotensive techniques, has reduced the peri-operative mortality rate to less than 10%.[1]

Pre-operative evaluation

The majority of subarachnoid haemorrhages (SAHs) occur in relatively young patients who are otherwise healthy. However, the cardiovascular system, respiratory system and metabolic and endocrine systems require special attention.

The occurrence of ECG abnormalities in patients with SAHs is well documented.[2-4] The ECG patterns associated with SAH may be large, wide T waves with normal or abnormal polarity, a prolonged rate-corrected Q-T interval, ST displacement and prominent U waves. The mechanism responsible for these abnormalities remains unclear. Abnormal serum potassium levels,[5] a raised intracranial pressure[6] and sympathetic overactivity[7] have been suggested as possible mechanisms.

These ECG changes occur in the absence of myocardial lesions,[8] and the importance of a baseline pre-operative ECG to assist in the interpretation of the intra-operative ECG is therefore obvious.

In the patient with a depressed level of consciousness immobility may lead to atelectasis, bronchial aspiration and infections. Respiratory problems must be excluded on clinical examination, and where indicated chest radiographs must be taken, arterial blood gas analysis performed and appropriate corrective measures prescribed.

Because of the close association between the circle of Willis and the hypothalamus, disturbances of hypothalamic function must be excluded. Autonomic activation with 'stress diabetes' may require insulin treatment and control of hypertension.[9] Inappropriate antidiuretic hormone secretion, fluid restriction and diuretic and steroid therapy all contribute to water and electrolyte disturbances; these must be excluded and corrected.

Anaesthetic risk determination

The standard American Society of Anesthesiologists' (ASA) classification should be combined with the Botterell grading of neurological status in order to predict outcome and plan the anaesthetic approach. Table I sets out the five grades suggested by Botterell *et al.*[10]

TABLE I. BOTTERELL GRADING OF NEUROLOGICAL STATUS[10]

I. **Asymptomatic or mild headache and neck stiffness.**
II. **Headache, neck stiffness and cranial nerve palsies.**
III. **Drowsy, confused or mild focal deficits.**
IV. **Stuporous with moderate-to-severe hemiparesis.**
V. **Deep coma or moribund with decerebrate rigidity.**

Methods

Premedication

Heavy premedication, particularly with opiates, is unnecessary and potentially dangerous.

Ventilatory depression (with its associated rise in arterial carbon dioxide tension ($Paco_2$) and intracranial pressure), nausea, vomiting and pupillary constriction are undesired effects of opiates and similar drugs. The benzodiazepine lorazepam, administered orally in a dose of 2,5 mg 90 minutes pre-operatively, is safe and effective as an anxiolytic agent in the alert and apprehensive patient. We recommend the addition of oral labetalol 100 - 200 mg to lorazepam. The α- and ß-blockades

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produced by this drug provide cardiovascular stability during induction, diminish the dose of sodium nitroprusside (SNP) required, prevent reflex tachycardia during elective hypotension, and reduce the incidence of postoperative rebound hypertension.[11] However, these tablets may irritate an empty stomach and can cause nausea and vomiting. The administration of intravenous domperidone 10 mg and an oral antacid such as magnesium trisilicate is therefore recommended.

Monitoring during anaesthesia

The accepted monitoring procedure for major surgery is mandatory in these patients.

Cardiovascular monitoring

Cardiovascular monitoring includes continuous display of the ECG, the radial artery pressure (after the performance of Allen's test) and the central venous pressure. The arterial line is inserted under local anaesthesia prior to induction. The importance of monitoring the arterial pressure at this stage cannot be overemphasized. A sudden rise in arterial pressure indicates a fresh subarachnoid haemorrhage, and immediate measures to protect the brain and reduce intracranial pressure must be instituted.[12]

The antecubital fossa approach to central venous pressure monitoring is preferred, since the positioning and draping of these patients renders fixation of the intravenous catheter difficult when the latter is placed in the neck. Correct placing of the central venous line is verified by the characteristic atrial waves on the oscilloscope.

Respiratory monitoring

Controlled hyperventilation is routinely used for neurosurgical anaesthesia. Capnography is therefore an essential monitor in these cases, provided it is kept in mind that apart from monitoring ventilation, capnography gives an indication of pulmonary blood flow. The effect of induced hypotension on capnography must be considered in this instance, and capnography must be correlated with the $Paco_2$.

Ventilator inflation pressure, tidal volume and frequency must be monitored. A change in inflation pressure in the absence of a nerve stimulator is an early indication that the effect of the muscle relaxant is wearing off.

Apart from an in-circuit oxygen analyser, the arterial oxygen tension (Pao_2) should be monitored at regular intervals. The inspired oxygen fraction does not indicate the Pao_2 because of the increased ventilation-perfusion mismatch associated with induced hypotension.

Monitoring by nerve stimulation

Bucking or straining on the endotracheal tube will have disastrous consequences on intracranial pressure in these patients. Train-of-four monitoring will give early warning that the effect of muscle relaxants is wearing off. Muscle relaxant supplements can easily be titrated on the train-of-four response, and on termination of anaesthesia muscle relaxant reversal can be verified.

Biochemical monitoring

Blood gases, acid-base values and electrolyte and glucose levels should be monitored at hourly intervals. Diuretic therapy is associated with potassium loss in the urine, and the stress response of SAH is associated with hyperglycaemia. The rational administration of electrolyte solutions, glucose and insulin is only possible with biochemical control. Increasing acidosis is an early index of SNP toxicity.

Induction and maintenance of anaesthesia

After the insertion of an intravenous line for the administration of fluids and drugs, and with all the monitors mentioned calibrated and on display, the patient is ready for induction.

Furosemide 10 mg and methylprednisolone sodium succinate 30 mg/kg are administered intravenously at the outset. Apart from its diuretic effect on the renal tubules, furosemide reduces the rate of cerebrospinal fluid production and promotes cerebrospinal fluid absorption.[13] It is our impression that the diuresis caused by the administration of furosemide 20 minutes before administration of mannitol prevents the hypervolaemia and the rise in intracranial pressure which would otherwise be produced by the latter substance. Furthermore, we have not seen any case of rebound cerebral oedema after administration of mannitol using this technique.

The administration of steroids remains controversial. The membrane-stabilizing effect possibly plays a role in repairing the damaged blood-brain barrier at the site of SAH. It is, however, generally accepted that steroids are effective in cases of focal lesions with oedema.[14] The latter can be expected at the site of operation and at the site of application of retractors. Workers in this field who claim that they have obtained poor results with steroids probably administer an inadequate dose. If results are to be expected pharmacological doses must be administered at induction. In our opinion the haemodynamic effects of steroids, i.e. reduced vascular resistance and an improved cardiac index, contribute to the prevention of cerebral ischaemia caused by vasospasm.

After the administration of the diuretic and the steroid the patient is pre-oxygenated and a 10 μg/kg bolus of fentanyl is administered. This dose of fentanyl contributes to a smooth induction and reduces cerebral blood flow and oxygen consumption.[14] We do not encounter difficulties with ventilation due to chest wall rigidity, probably because we administer pancuronium immediately after the fentanyl. We have not seen any case of nausea and vomiting after fentanyl administration, during induction or on recovery. Pancuronium 0,1 mg/kg is given intravenously after fentanyl, followed by a sleep dose of alphaxalone and alphadolone acetate (Alfathesin), or thiopentone when the alphaxalone and alphadolone is contraindicated. Respiration is assisted, and 50% nitrous oxide in oxygen is introduced at this stage. When complete muscle paralysis sets in, mask ventilation is gently controlled by hand.

An intravenous bolus of lignocaine 1,5 mg/kg is injected 2 minutes before laryngoscopy and endotracheal intubation in order to avoid a rise in blood pressure and intracranial pressure.[15] Although this is not our practice, it seems justified to deepen the level of anaesthesia before intubation with an incremental dose of an intravenous anaesthetic agent. If high arterial pressure before intubation causes concern, labetalol 1,5 mg/kg can be given intravenously.

The patient is then intubated with an armoured endotracheal tube, and an oesophageal stethoscope and a temperature probe are inserted. The endotracheal tube is fixed with adhesive strapping and the oesophagus is sealed off with a throat pack impregnated in a topical steroid ointment. The eyes are protected from abrasions by surgical drapes and antiseptic solutions used to clean the scalp, with gauze covered in petroleum jelly and adhesive plaster. Anaesthesia is maintained with 40% oxygen in nitrous oxide, supplemented with 4 ampoules of alphaxalone and alphadolone intravenously per hour. A mechanical syringe controls the rate of administration of undiluted alphaxalone and alphadolone. If a mechanical syringe is not available, the drug may be diluted in 250 ml 5% dextrose water or a 0,9% saline

solution. This volume can add up to 1 000 ml in 4 hours and should be taken into consideration when planning intravenous fluid requirements during a neurosurgical procedure.

This dose of alphaxalone and alphadolone is adequate for surgical anaesthesia when combined with nitrous oxide and fentanyl. It also offers pharmacological protection of the brain by reducing cerebral blood flow, intracranial pressure and cerebral oxygen uptake, and can increase regional cerebral blood flow in damaged areas of the brain while reducing regional blood flow in the surrounding normal area.[16,17] Administration of this drug is stopped 20 minutes before the end of the procedure; this allows rapid recovery and assessment of neurological function.

Although it is generally accepted that low concentrations of inhalational agents such as 0,5% halothane and controlled hyperventilation can be used for neurosurgical patients, we avoid these.

Ventilation is controlled with a ventilator with a preset volume which is adjusted to ensure hypocapnia with $PaCO_2$ levels of 3,3-3,4 kPa. $PaCO_2$ levels at this range ensure adequate control of cerebral blood flow and intracranial pressure and do not cause cerebral ischaemia.[18]

The tidal volume and inspiratory/expiratory ratio are adjusted to ensure optimal venous drainage and so avoid provoking an increase in intracranial pressure.

Intravenous fluid therapy

Overtransfusion should be avoided. On the other hand, severe dehydration with hypovolaemia and the resulting cardiovascular instability complicate induced hypotension. To compensate for pre-operative deficits, we administer a 10% dextrose-balanced electrolyte solution (Hidroliet) before induction. The volume infused is 1,5 ml/kg up to a maximum of 500 ml in adults. The rate is adjusted to allow infusion of this volume over a 30-minute period while preparing the patient for induction.

During neurosurgical procedures third-space losses are minimal, and a balanced salt solution (Plasmalyte B) is infused after the dextrose solution at a rate of 2 ml/kg/h. A central venous pressure of 0 - 2 cm H_2O is aimed at.

Fluid flow in the central venous line is adjusted so as to keep the line patent and not add to the volume of fluid administered to the patient. Blood loss is minimal, and blood is only replaced when more than 500 ml is lost. This seldom occurs. Blood is ordered on a 'group and hold' basis on the previous day.

Twenty minutes after the administration of furosemide, 20% mannitol 1 g/kg is administered and the rate is adjusted to ensure that the calculated dosage has been given at least 15 minutes before the cranial flap is opened.

We have not encountered raised intracranial pressure or rebound phenomena when using the furosemide-mannitol sequence in this manner. Serum electrolyte levels must be monitored, however, and corrected when necessary.

Neurosurgical patients may on occasion require prolonged intravenous therapy, and everything possible should be done to protect veins. Aseptic venepuncture and care of the puncture site with antiseptic ointment, a gauze dressing and adhesive plaster are mandatory.

Positioning

The operating table is tilted to a 15° head-up position to facilitate venous drainage and hypotension. Neck flexion must be avoided, since this will impair venous drainage and raise intracranial pressure.

Induced hypotension

Induction of hypotension for intracranial aneurysm surgery is widely practised. Hypotension renders the vessels at the site of the aneurysm more mobile, reduces the intraluminal pressure of the aneurysm (thereby reducing the risk of rupture), and facilitates clip application.[19] What is often not appreciated is that hypotension also reduces brain turgor, making the brain more pliable. This facilitates the approach to the aneurysm, and the experienced neurosurgeon can detect arterial pressure changes from the 'feel of the brain'.

Arterial pressure is reduced by 20% from pre-induction levels when skin incision starts. When the craniotomy flap has been removed, arterial pressure is reduced by 40% from pre-induction levels; during the approach to the aneurysm and clip application, mean pressure is reduced to 40 mmHg. When the skull is open this low level of blood pressure seems to be well tolerated for short periods if the brain is protected pharmacologically.[20] Pulsatile flow in small vessels can still be seen through the operating microscope.

Hypotension is induced with labetalol 1,5 mg/kg, followed by an infusion of 0,01% SNP. The rate of SNP infusion is controlled with an infusion pump to achieve the desired pressure levels. The rationale of labetalol administration has been demonstrated previously.[11] After the clip has been applied the SNP is gradually reduced, allowing the arterial pressure to return to the pre-induction level. SNP is not completely withdrawn, and is used to control blood pressure increases of more than 10% above pre-induction levels.

Recovery

The alphaxalone and alphadolone infusion is stopped at the start of closure of the dura. After skin closure and application of bandages the throat pack is removed, the airways are cleared and the patient is extubated. Extubation is preceded by intravenous administration of a lignocaine bolus as for intubation in order to avoid excessive rises in blood pressure and intracranial pressure. After extubation the patient is ventilated by mask, the muscle relaxant effect is reversed with prostigmine and glycopyrrolate, and the nitrous oxide is switched off.

As soon as spontaneous respiration is thought adequate, the patient is transferred on his bed to the intensive care unit for monitoring purposes and control of blood pressure if necessary.

Controlled therapy with 40% oxygen is administered by mask for at least 12 hours.

Results

During the period 1980 - 1982, 47 patients were operated on. Fig. 1 shows the age and sex distribution and morbidity and mortality figures. The sites of the aneurysms are set out in Table II.

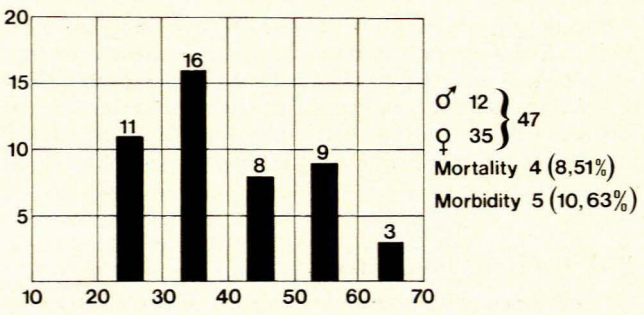

Fig. 1. Age and sex distribution of and morbidity and mortality among 47 patients undergoing surgery for intracranial aneurysms, 1980 - 1982 (number of patients on the vertical axis, age in years on the horizontal axis).

TABLE II. LOCATION AND INCIDENCE OF INTRACRANIAL ANEURYSMS IN 47 PATIENTS

Anatomical site	No.	%
Internal carotid artery	**4**	**8,5**
Anterior cerebral and communicating arteries	**16**	**34,0**
Middle cerebral artery	**8**	**17,0**
Posterior communicating artery	**14**	**29,8**
Other (multiple aneurysms, optical artery, anterior choroidal)	**5**	**10,6**

Discussion

The average age of aneurysm victims was 40 years and the female/male ratio was approximately 3:1. In the under-40 group there were 10 males and 17 females, while in the over-40 group there were 2 males and 18 females. There was therefore a strong female preponderance in the over-40 group. Forty-four patients presented at the height of their productive lives. The importance of early diagnosis, surgical intervention and correct anaesthetic management in enabling these patients to resume a productive life cannot be overemphasized.

The incidence and location of the aneurysms in our series do not correspond with figures found in the literature,[21] possibly because in these respects patients who die before surgical intervention differ from the survivors operated on.

Since this was a retrospective study, we could not demonstrate a correlation between the ASA risk classification, the Botterell neurological grading and final surgical results. Clearly the anaesthetic risk could be evaluated better by a prospective study.

Our mortality rate of 8,5% is slightly lower than and the morbidity figure of 10,6% equal to rates reported for other centres.[1] With a 1% incidence of intracranial aneurysm in the general population, we could expect approximately 3 000 cases of SAH due to this cause to occur annually in the RSA. It is estimated that only 0,04% of patients with a SAH eventually reach the operating theatre, the majority dying before any intervention.[21] The need for early diagnosis, surgical intervention and correct anaesthetic management if this unacceptably high mortality figure is to be improved upon cannot be overemphasized.

REFERENCES

1. Drake CG. Aneurysm surgery: past, present and future. *Int Anesthesiol Clin* 1982; **20:** 1-9.
2. Byer E, Ashman R, Toth LA. Electrocardiograms with large, upright T waves and long Q-T intervals. *Am Heart J* 1947; **33:** 796-806.
3. Levine HD. Nonspecificity of the electrocardiogram associated with coronary artery disease. *Am J Med* 1963; **15:** 344-355.
4. Cropp GJ, Manning GW. Electrocardiographic changes simulating myocardial ischemia and infarction associated with spontaneous intracranial hemorrhage. *Circulation* 1960; **22:** 25-38.
5. Cruickshank JM, Neil-Dwyer G, Stott AW. Possible role of catecholamines, corticosteroids and potassium in production of electrocardiographic abnormalities associated with subarachnoid haemorrhage. *Br Heart J* 1974; **36:** 697-706.
6. Jachuck SJ, Ramani PS, Clark F, Kalbag RM. Electrocardiographic abnormalities associated with raised intracranial pressure. *Br Med J* 1975; **1:** 242-244.
7. Millar K, Abildskov JA. Notched T-waves in young persons with central nervous system lesions. *Circulation* 1968; **37:** 597-603.
8. Rudehill A, Gordon K, Sundquist K, Sylvin G. A study of ECG abnormalities and myocardial specific enzymes in patients with subarachnoid haemorrhage. *Acta Anaesthesiol Scand* 1982; **26:** 344-350.
9. Benedict GR, Loach AB. Clinical significance of plasma adrenaline and noradrenaline in patients with subarachnoid haemorrhage. *J Neurol Neurosurg Psychiatry* 1978; **41:** 113-117.
10. Botterell EH, Lougheed WM, Scott JW, Vandewater SL. Hypothermia and interruption of carotid or carotid and vertebral circulation in the surgical management of intracranial aneurysms. *J Neurosurg* 1956; **13:** 1-42.
11. Erasmus FR, Coetzee AR. Labetalol, hypotension and anaesthesia for surgical repair of intracranial aneurysms (Paper presented at the South African Society of Anaesthetics Bi-annual Congress, Plettenberg Bay, 1981).
12. Campkin TC, Turner JM. Anaesthesia for intracranial vascular lesions. In: Campkin TC, Turner JM, eds. *Neurosurgical Anaesthesia and Intensive Care*. London: Butterworths, 1980: 175-187.
13. Cottrell JE, Robustelli A, Post K, Turndorf H. Furosemide and mannitol induced changes in KP and serum osmolality and electrolytes. *Anesthesiology* 1977; **47:** 28-30.
14. Carlsson C, Keykhah MM, Smith DS, Harp JR. Cerebral blood flow and oxygen consumption after fentanyl. *Anesthesiology* 1981; **55:** suppl, A193.
15. Hamil JF, Bedford RF, Weaver DC, Colohan AR. Lidocaine before endotracheal intubation: intravenous or laryngotracheal? *Anesthesiology* 1981; **55:** 578-583.
16. Pickerodt VWA, McDowall DG, Coroneos NJ, Keaney NP. Effect of althesin on cerebral perfusion, cerebral metabolism and intracranial pressure in the anaesthetized baboon. *Br J Anaesth* 1972; **44:** 751-758.
17. McDowall DG. Neurosurgical anaesthesia and intensive care. In: *Recent Advances in Anaesthesia and Analgesia*, vol 12. Edinburgh: Churchill Livingstone, 1976: 25-26.
18. Campkin TC, Turner JM. Control of cerebral blood flow. In: Campkin TC, Turner JM, eds. *Neurosurgical Anaesthesia and Intensive Care*. London: Butterworths, 1980: 5-11.
19. Ferguson GG. The rationale for controlled hypotension. *Int Anesth Clin* 1982; **20:** 89-93.
20. Spoerel WE. Monitoring the safe levels of hypotension. *Int Anesth Clin* 1982; **20:** 111-119.
21. Kassell NF, Torner JC. Epidemiology of intracranial aneurysms. *Int Anesth Clin* 1982; **20:** 13-17.