Anaesthesia for Cerebral Aneurysm Repair

Epidemiology

Exact incidence is unclear but probably about 4%. An annual incidence rupture is about 15-20 per 100,000 population.

Age : At any age but peaks at 40 - 60.
Sex : Male to Female 2 : 3 but more males below 40 and more females after.
Sites : 30% ICA
40% ACA and Anterior Communicating
20% MCA
10% Vertebro-basilar systems
Rupture : 90% < 12mm, 5% 12-15mm, 5% > 15mm.

History

First aneurysm clipped in 1931, Operating microscope first used for clipping in 1960.

Aetiology

It had been thought to be a congenital disease but is now thought to be primarily acquired (although there is a congenital component in some cases). Most are of the saccular or berry aneurysms and this type of aneurysm is unique to the cerebral circulation. Rupture is thought to relate to the size of the lesion. This clearly is due to La Places Law T = 2PR and the fact that the wall also becomes thinner as the aneurysm enlarges. The lesions almost always occur at the bifurcation of vessels. It is thought that the process starts with degeneration of the internal elastic membrane at the apex of a bifurcation. This is the site of the maximum haemodynamic stress. The pressure within the aneurysm is at systemic levels and thus rupture is most likely to occur at moments of raised BP. The larger the aneurysm the greater the likelihood of rupture, however most rupture when they are <12mm.

Presentation

Whilst traditionally the vast majority present with a SAH or occasionally with pressure symptoms related to direct pressure on nerves, eg 3, 4, or 6 nerve palsies, many are not presenting with incidental findings on MRI or CT scans.

Those patients who present with a SAH will in fact have had some type of symptoms in the few days prior to the major bleed.

The S&S of SAH are due to :

i) raised ICP
ii) damage due to intra-cerebral bleeds
iii)regional vasospasm
iv)generalised vasospasm

Minor bleeds precede the major haemorrhage in ≈50% of patients. The patients may have hypertension either as the cause or the result of the bleed. Brainstem ischaemia is thought to be the cause of the secondary form.

Signs and Symptoms

1) Result of Initial Rupture: There is an initial abrupt rise in ICP that produces a severe headache ± loss of consciousness. Blood in the CSF produces meningism (photophobia, neck stiffness, and headache).

2) Focal signs: The most commonly occurs 5-9/7 later from spasm but if occurring initially may be due to the direct effects of the jet of blood, intracerebral bleeding, or from herniation.

3) Hydrocephalus: Common, usually due to impaired CSF passage through the basal cisterns. Occasionally large basilar aneurysms may produce IV ventricular obstruction and hydrocephalus.

Pre-operative Management

Classification (Botteral) [perioperative mortality%]

1) Asymptomatic or have mild headache or neck stiffness. [0-5]
2) Headache and neck stiffness but no neurological abnormalities other than cranial nerve palsies. [2-10]
3) Drowsy, confused or have mild focal deficits. [10-15]
4) Stuporosed and have moderate or severe hemiparesis. [60-70]
5) Comatosed, non-responsive, and moribund with decerebrate rigidity. [70-100]

Mortality after SAH

50% dead within 4 weeks
70% from the initial bleed 30% from re-bleeding and other complications.
Re-bleeds do very badly: Mortality after first re-bleed 64%, second 96%
Of those who died of the initial bleed 74% died within the first 24 hrs.
The patient's condition on arrival is very important with 90% 10 day survival if in good condition and 20% if comatosed.
Only about 30% will survive without neurological deficits.

Diagnosis of SAH

This is usually on CT scan (SAH or haematoma mainly but you may be able to see the aneurysm on a plain CT. MRI or CT angiography will have a much better detection rate. Ultimately cerebral angiography will be done to determine whether there is an aneurysm and how it should best be treated.

Management

As the patients who have survived the initial bleed are at risk of re-bleeding it would seem sensible to repair the aneurysm as soon as possible. This is still somewhat controversial but almost all neurosurgeons will now operate as soon as is practical except in GIV and V patients. Those with severe spasm unresponsive to treatment may also be clipped to allow the use of more marked induced hypertension. The other big argument in favour of early clipping is the reduction in hospital stays. Obviously the patient who has a major intracranial bleed with mass effects or obstructive hydrocephalus (blockage of the arachnoid villi or the basal cisterns) will require urgent surgery. In these cases the surgery may be restricted to relieving these problems rather than the definitive repair.

Hydrocephalus requires the placement of an external ventricular drain to allow CSF drainage and ICP measurement.

The traditional approach was to delay the angiography until 7-10 days after the initial bleed provided that the patient continues to improve. Surgery followed soon after this. Nowdays, regardless of whether or not one plans early surgery, an angiogram is done as soon as possible to allow rational management decisions to be made (aneurysms are not always demonstrated in presumed subarachnoid bleeds and the absence of an aneurysm obviously has a major impact on the patient's management).

This intervening period is a dangerous one with 20% suffering a re-bleed, with a >60% mortality. The risk of a re-bleed is higher in those in the worse condition patients.

The goals of this period

i) prevent re-bleeds
ii) prevent or treat vasospasm
ii) detect hydrocephalus
iv) avoid respiratory problems
v) optimise the general medical state of the patient

The patient is confined to bed and they are to avoid things that may increase their BP eg straining at stool (given laxatives). Their BP control is difficult and depends on their clinical status. As a rule hypertension should be controlled however if they have vasospasm a common treatment is to raise their BP. It also depends if there is a prior history of hypertension as they may have disturbed auto-regulation. If there is no obvious vasospasm then the BP is controlled on empirical grounds to about 160 systolic and 100 diastolic. It would seem logical that if there are no neurological signs then the BP should be controlled to normal levels. There is no hard evidence to guide us in this area.

Epsilon aminocaproic acid was used in the past to prevent the clot around the aneurysm from being dissolved. In general it has not proved useful and is now not available in Australia.

Generally the patient's fluid status needs to be very carefully assessed and it may be necessary to measure the CVP, of course the U/O and fluid balance should be carefully measured. The prolonged bed rest predisposes to respiratory problems and these should be aggressively treated.

Vasospasm

This is clinically significant in ≈33% and is one of the worst problems in this condition causing ischaemia and subsequent cerebral oedema that further compromises the cerebral circulation. Radiological evidence of large vessel vasoconstriction is present in about 60-80% of patients but is not always haemodynamically significant. The peak incidence is about day 7. The exact mechanism for it is unclear but appears to be due to the presence of blood breakdown products. FFA derivatives such as the prostaglandins and leukotrienes, oxyhaemoglobin and the oxygen radicals released as it breaks down are all candidates. The end result is narrowing of the vessel but it does not seem to be simply medial smooth muscle contraction. There are structural alterations seen to the vessel wall involving all layers with severe intimal damage, platelet deposits, and endothelial thickening.

Treatment of this condition is unsatisfactory with many agents tried. Direct vasodilators as well as alpha and beta blockade appear to be of no benefit. Phosphodiesterase inhibitors such as Aminophylline have also been used. The traditional treatment is maintenance of an adequate blood volume and cardiac output with induced hypertension if necessary. It is difficult to know exactly how much to do this but it should be titrated against the patient's response. It would also be desirable to be able to titrate the response against actual cerebral blood flow. This is done in some units but is not widely available. The traditional regime is "triple therapy" with hypervolaemia, haemodilution, and hypertension. The aim of the haemodilution is to reduce the haemoglobin to ≈100gm/l thereby reducing viscosity and improving flow.

Overall I do not think hypervolaemia is rational because the problem is a fixed resistance and the management requires increased perfusion pressure (hypertension) or reduced cerebro-vascular resistance (haemodilution). Maintenance of normal blood volume is important so as to prevent hypotension occurring.

Nimodipine has been shown to be effective in the management of the cerebral ischaemia associated with spasm. It is the first line treatment (given by infusion). The exact mechanism of action is unclear because angiographic studies do not show any vasodilation. If nimodipine proves insufficient then induced hypertension is used. It may be that rather than improving flow it simply improves the brains tolerance to ischaemia or improves the microcirculation.

Cerebral Oedema

The treatment of this does not differ from that which is normally used however its presence is a poor prognostic sign. It is rational if the patient requires aggressive treatment for this to monitor the ICP directly. In this case it is probably best to do it via a Interventricular Catheter as this allows the drainage of CSF as needed (obstructive hydrocephalus is common in those developing cerebral oedema).

Angiography

This is usually done as soon as is practical to determine whether an aneurysm is present and allow early surgery. At RPAH it is done under minimal sedation (by the Radiologists) with pre-medication size doses of a Narcotic (provided that the patient has no overt raised ICP). It is done via a femoral catheter and therefore allows good 4-vessel angiography. Patients who are uncooperative need a GA. If so they need the same level of vascular monitoring as for an aneurysm clipping.

Pre-operative Assessment

These patients need very careful pre-op assessment. In particular

i) CVS : Baseline BP (to give some idea of the safe level of hypotension)
CCF
Fluid Status (the fluid balance of these patients is very important, they should not be overloaded otherwise they may develop CCF but if they are dry cerebral circulation may be compromised and vasospasm, if present, may be rendered haemodynamically significant. If hyponatremia is allowed to develop cerebral oedema may result.

ii) RESP: Prolonged bed rest runs the risk of atelectasis and pneumonia if this is present it should be cleared up prior to surgery. Pt's with COAD may also need a higher FiO2 intra-op.

iii) CNS: A simple pre-op assessment will help in the rapid post-op assessment of the patient. Pupil size, gross motor weakness, presence of aphasia's and any specific cranial nerve signs are sufficient.

iv) GEN: As for any pre-op examination.

Pre-op Test's

i) E/LTS/Plus: Electrolyte abnormalities are common and should be corrected pre-op. Renal function should be assessed as this be a consideration in reducing the BP if induced hypotension is used. BGL should be normalised as hyperglycaemia has been shown to worsen neurological deficits in the presence of cerebral ischaemia.

ii) FBC: Anaemia should be corrected pre-op.

iii) Coags: INR/APTT

iv) ECG: This should be done on the day prior to surgery as these patients have a high incidence of ECG abnormalities and these are changeable over the time that surgery is delayed.

v) CXR: As a general assessment of RESP and CVS pathology.

vi) Echo: Patients with questionable cardiac function should have a pre-operative echocardiography. This can be done in the ICU if necessary (this does not assess the cardiac ischaemic potential).

vii) Other tests may be indicated in specific situations.

Pre-Medication

It must never be underestimated that adequate explanation is the best pre-med as you will probably be placing the "a" line, drip, under LA then clearly explaining what is going to happen will reduce their anxiety and help prevent unwanted rises in BP. The aim of pre-med is to sedate the patient so that no rises in BP occur. All the usual medications should continue till immediately pre-op, this is to control BP in the post op period and in the case of B-blockers prevent peri-operative infarcts. Most of these patients do not have raised ICP (in whom no premed is appropriate), For patients who have had a SAH I prefer to use Temazepam 20-30mg PO for an average patient (older patients and those with hypertension have more BP increases with anxiety). As more and more of these cases are detected before aneurysm ruptures, most of these cases are now elective and are admitted on the Day of Surgery (DOSA). Except if the patient is very anxious, most will not get a sedative premedicant.

Two units of blood should be available.

Radiological Management of Aneurysms

Evidence now suggests that where possible Angiographic Coiling of an Aneurysm has fewer risks than surgery. As coiling takes between 2-4 hours and the patient must remain absolutely still during this time, they are always done under General Anaesthesia.

Coiling works by placing extremely coiled material inside the Aneurysm. The material coating the coils is extremely thrombogenic and the aim is for the aneurysm to thrombose and a new intima to grow over the inlet to the aneurysm. A variable number of the coils are placed until such time as the aneurysm if full of them. There are a number of risks to this. Firstly the aneurysm may rupture with the angiographic manipulation, secondly part of the coil could embolise out of the aneurysm into a more distal artery and lastly, the thrombus formation may extend out of the aneurysm and cause thrombus formation in the feeding vessels.

The ideal aneurysm is one with a narrow neck but the use of stents with side holes can convert a wide neck aneurysm into a narrow opening.

Rupture can usually be controlled angiographically but if this fails the patient may need to go to theatre (although the prognosis is poor).

The usual anaesthetic is similar that for operative neurosurgery with relaxation, intubation and ventilation. Propofol, Remifentanil ± N_2O are typically used (see below). An arterial line is always used with a central line a good idea but not essential. At least one large bore peripheral cannula should be placed in case of rupture.

The patients will develop a diuresis from all the contrast material and should be catheterised after induction. The use of a warming blanket will help prevent hypothermia as well.

The cases usually take from three to six hours and the patient is usually quite stable during this time.

Aneurysm rupture is uncommon and can vary from a small leak to a full-blown rupture. The radiologist will be able to tell you the extent of the bleed. In this situation it is wise to revert to pure TIVA anaesthetic (switch off the nitrous) if not already doing so.

If such a bleed does occur then you will need to discuss with the radiologist whether or not you want the BP raised or lowered. It is imperative however not to let the BP become elevate and BP should be controlled with Propofol in the first instance. DO NOT used vasodilators to control the BP.

Unless the bleed is very small the patient will remain intubated and will often need to go to theatres for an ICP monitor or surgical decompression.

Unless there are complications the patient will awaken quickly and be extubated. There is minimal pain, as the groin angiogram site should have had local inserted so there is no need to administer long acting analgesics. Once the patient is stable they should then be taken back to a high dependency area.

Surgical Management

Intra-operative

The principles are :
- i) Avoid increases in transmural aneurysm pressure
- ii) Provide good conditions for the aneurysm surgery
  - a) "slack" brain
  - b) reduce aneurysmal pressure during clipping
    - 1) Induced hypotension
    - 2) Temporary clips
- iii) Avoid damage to the brain during this

Checked your equipment and have a sucker ready.

Monitoring

i) CVS : ECG, Arterial line, CVP (cubital fossa)

ii) RESP : SpO2, End tidal CO2, oesophageal stethoscope

iii)NEUROMUSCULAR : Train of 4 (it is essential that these patients do not move)

iv) CNS : All of my cases get either EEG or EPs (usually the latter)

v)RENAL : U/O all these patients are catheterised the U/O provides an indication that the diuretics are working.

Except in those patients that are extremely nervous (rare) or in whom access is likely to be very difficult the arterial line should be placed pre-induction under LA. This is usually easy. In the very nervous patient or if one is having difficulties it is acceptable to induce the patient but not intubate until the “a” line is placed.

Induction

Rises in BP and falls in ICP should be avoided at this stage as that increases the transmural aneurysm pressure (the pressure in the aneurysm is at arterial levels) and makes rupture more likely; this is a disaster if it occurs.

How one achieves this is an individual choice but I induce the patient with a TCI Propofol infusion (starting at 4-6μg/ml and reducing to 1 once unconscious) Paralyse the patient with Cis-Atracurium 10mg (they are usually on anticonvulsants which markedly increases the metabolism of steroidal muscle relaxants). The patient is then hand ventilated with 67% Nitrous Oxide and Oxygen. I usually start a Remifentanil infusion once the patient is unconscious and adjust to as high a rate as possible ($\leq$10μg/kg/hr) depending on BP.

I monitor the degree of relaxation with a "Train of 4” nerve stimulator on the Common Peroneal nerve and do not intubate until full relaxation achieved. Prior to intubation I give a bolus of 1μg/kg of Remifentantil. If the BP rises unacceptably with intubation further doses of Remifentanil are given until it is controlled. My limits on BP are set using systolic BP prior to induced hypotension (if used), the upper limit is set at the mean of the ward systolic BPs and the lower limit is 30% less than this or 90mmHg (whichever is greater).

If you are using TCI Remifentanil then set the target at 2-4ng/ml at induction (using Effect Site rather than Plasma targeting). To cover Intubation and Mayfield Pins transiently raised the target to 6ng/ml. During the case one runs between 1-6ng/ml.

The patient is hyperventilated to produce a $ETCO_2$ in the order of the mid 20's, to counteract any increased cerebral perfusion due to the nitrous and to improve surgical exposure.

The endotracheal tube (RAE oral 8.0mm in males, 7.0mm in females) should be very securely fixed, the eyes protected and the patient positioned carefully so as to avoid nerve compression. Except for Basilar aneurysms these cases are usually done supine. Basilar aneurysms are usually done on the side, in which case a standard ETT is used in order to get the connectors out of the way of the chest.

I give a further 1μg/kg bolus of Remifentanil to block the response to Pins.

One then adjusts the Remifentanil infusion depending on the BP.

Maintenance

Maintenance is with Nitrous, Remifentanil, and a Propofol infusion. Avoidance of inhalational agents and the use of an intravenous agent that causes cerebral vasoconstriction will help to optimise cerebral conditions.

Nitrous is used at 50-67% (in order to maintain SpO2 >97%). If I am using SNP then I fix the nitrous at 50% once the skin incision occurs. SNP commonly worsens pulmonary shunting and I want to avoid changing the nitrous concentrations when the BP decreases (I am using electrophysiological monitoring which is effected by such changes).

Propofol is maintained at about 2.0μg/ml if the nitrous is reduced to 50%, I usually fix it at this level so as to provide a stable base for my electrophysiological monitoring. Remifentanil is adjusted to control the BP. Once the dura is opened I prefer to restrict the Remifentanil to ≤10μg/kg/hr (cost). I usually add Metoprolol <0.3mg/kg if this is insufficient to control the BP. It is a good idea to get some Metoprolol in before any induced hypotension to reduce the reflex tachycardia and rebound hypertension associated with SNP. Beta blockade also reduces the amount of SNP needed.

The BP should be kept within the previously defined range, aiming for ≈20 below baseline SBP until the aneurysm is approached.

There are several critical periods prior to the aneurysm being clipped. We have already dealt with intubation. It is usual for the head to be secured with pins when these are inserted the BP may increase. A bolus of Remifentanil or propofol helps prevent this or LA can be used in the sites of positioning. Movement of the head may also raise the BP but if the previously mentioned dose of narcotic are used this rarely occurs. The skin incision may also raise the BP. We usually infiltrate the skin incision line with 0.5% Bupivacaine and 1/200,000 Adrenaline which helps but does not prevent the response. Periosteal work and cutting the bone flap are also very stimulating.

Many centres in the world use Lumbar drains to remove CSF and help with operative conditions. If these are used it is important that they are not released prior to the dura being opened as the sudden fall in ICP may cause the aneurysm to rupture. They are not used at RPA for most cases because of this danger and as good conditions are obtainable without it. If marked brain retraction is needed a lumbar drain is used. If operative conditions are not satisfactory at dural opening (and no obvious cause is apparent) then a ventricular catheter can be placed and CSF drained.

Mannitol (1.5 gm/kg) combined with Frusemide (0.3mg/kg) is given to shrink the brain and provide good conditions. Mannitol acts as an osmotic agent and Frusemide acts both as a diuretic and to decrease CSF production. These are not given until the head is secured as mannitol given fast commonly causes hypotension (not hypertension as the textbooks say). I give it over ≈20mins and do not commonly see BP

changes. The Frusemide is given at the end of the infusion, as this is the time that it will produce the maximal effect on brain shrinkage. There are some who believe that very large doses (2gm/kg) actually protect the brain but this is anecdotal. It takes about 1 hour for the maximal effect so the mannitol should be started soon after the BP has settled down from intubation.

Of the volatile agents Sevoflurane produces the least increases in CBF/ICP but the exact effects in an individual patient are somewhat unpredictable and depend on the baseline ICP. It will, however, always cause a greater increase in CBF than propofol (although this may be clinically unimportant). For this reason I prefer Propofol as an adjuvant to nitrous. If the ICP is elevated pre-op then I use a pure intravenous technique without nitrous or volatile agents.

Muscle relaxation is maintained with an infusion of Cisatracurium in adjusted to provide a T4 count of 0 but with a post-tetanic count of >10.

Induced Hypotension

As the pressure inside the aneurysm is at arterial levels it is usual to induce hypotension during the time of dissection and clipping. It has been found that the aneurysm and it's vascular tree are more mobile at MAP's of around 50 torr. The exact degree of hypotension depends on the aneurysm and the general condition of the patient. In general cerebral autoregulation preserves flow at MAP's of 50 torr (providing that there is not raised ICP or cerebral venous pressure) but in patients with pre-existing uncontrolled hypertension this lower limit may be considerable raised. Ideally, there should be monitoring of cerebral function to allow more rational decisions however despite the usual empirical usage in practice it is quite safe. It is important that the MAP is measured at the level of the brain and not at some point below this, obviously at these low levels any small error such as this may be catastrophic.

The choice of agents lie between the inhalational agents, Sodium Nitroprusside and Nitroglycerine. Other agents have been used but there is no general usage of them. As we have discussed the inhalational agents have problems and are therefore rarely used as the prime agents. Nitroglycerine would be attractive due to its minimal toxicity however it is also not nearly as easy to use as SNP and there is not a small incidence of refractory patients. Thus I use SNP in a 0.1% solution. Provided that the dose is kept below 8 ug/kg/min there is little risk of toxicity. If it is possible I try and give at least 10mg Metoprolol before using SNP as this markedly reduces the dose of SNP needed, minimises the reflex tachycardia, and blunts the rebound hypertension.

The usual thing to due is to reduce the MAP to about 60 torr as they are approaching the aneurysm and only go to 50 with clipping. I have seen very little EP evidence that this causes cerebral ischaemia.

Commonly if the patient has had a SAH they may be on a Nimodipine infusion. If only mild reductions in pressures are needed (60 or so), I will often just use this infusion.

The other problem is that the vasodilators cause loss of reflex pulmonary vaso-constriction and may produce hypoxia, I aim for an SpO2 > 95% (ideally >98%).

Monitoring Cerebral Function

This is a complete talk by itself. Ideally all patients in this situation should have some type of this monitoring. It has been shown that there is not irreversible loss of cell function until rCBF is <10 mls/100gm/min (N 54) and that EEG and Evoked Potential changes do not begin to occur until about 18 mls/100gm/min. These are at levels of MAP of about 20-30 torr in the normal person. Of course it depends also on the duration or the decreased flows, at rCBF's of 10mls/100gm/min they may have to be maintained for 15mins or longer.

There are two areas that are liable to ischaemia during hypotension. Firstly, the boundary areas between the main arteries. Secondly, the areas of brain under the retractors where the local pressure from the retractor

means the local perfusion pressure is lowered. The use of mechanical retractors and the provision of a slack brain minimise the problems in this area.

The types of monitoring devices used are i) EEG either raw or in a processed form such as the Cerebral Function Monitor or the Compressed Spectral Array ii) Evoked Potentials eg Auditory or Somatosensory.

It is obviously important if you are going to use such equipment that the anaesthetic does not change during it's use otherwise one won't know what is causing any observed changes.

Hypothermia

As the survival of tissues has been shown to be prolonged by profound hypothermia (20-25°C) it was felt that this might be beneficial in this type of surgery. There are several reasons that it is now only very rarely used these days. Firstly for the average case it did not seem to cause a decrease in the mortality. This is presumable because there usually is not a problem with adequate blood supply as the degree and period of hypotension is not usually great. Secondly, it appears that the incidence of vasospasm is greater than when it is not used. Thirdly it is a major hassle if done via surface cooling and there are not insignificant complications in it's use. To obtain extensive prolongation of brain survival very low temperatures need to be used (ie 15-20°C) and this is only achievable with cardio-pulmonary bypass. This is occasionally indicated when very low pressures or total circulatory arrest are needed.

Recently it has been shown in animals that there is marked cerebral protection from mild hypothermia (2-3°C). This is thought to be due to inhibition of release of excitatory neurotransmitters (glycine and glutamate). These AAs are release with ischaemia and are lead to increased cellular metabolic demands and earlier cell damage. No human studies exist but it is quite easy to have the patient at 34-35°C at the time of clipping/hypotension. Rewarming is also not too difficult if one uses a Bair Hugger.

Post Clipping

Once the aneurysm is clipped the BP is raised to the pre-op level. This should be done slowly over about 10 mins as SNP causes overshoot due to renin-angiotensin activation. All the fluid loss including calculated insensible losses should be replaced prior to the end of surgery unless there was marked Cerebral Oedema pre-op. The fluid used should be N/Saline so as to avoid hypo-osmolality occurring. The maintenance of adequate filling pressures and BP will help prevent an vasospasm becoming clinically significant.

Once the BP has returned to baseline and the surgeons are beginning to close I will increase the nitrous to as high one can whilst the SpO2 is >95%. I turn the absorber off or reduce the ventilation and adjust the FGF to allow the ETCO2 to be in the mid 40s (or reduce the ventilation if you don't have a switchable absorber).

The relaxant infusion is decreased in order to have a T4 count of 1 by the time the head bandage is put on. This prevents coughing with the head movement.

The patient is not extubated until they are awake and breathing well. I usually continue the propofol until about 10 minutes from the end and then cease. I control the BP from then on with Remifentanil

Once the head bandage is on reversal is given, all the infusions are ceased, the nitrous is turned off and the patient put on 100% oxygen.

I keep the patient paralysed (1 twitch of the TO4) until after the head bandage is on and then reverse with Neostigmine (70μg/kg) and Glycopyrrolate (0.8mg per 5.0mg Neostigmine).

I add further Metoprolol and if insufficient Diazoxide (75mg boluses, ≤ 600mg) to control the BP during emergence.

If Remifentanil has been used then Fentanyl (50-100μg) should be given AFTER the patient is awake and appears neurologically normal (Morphine acts too slowly to work well in this situation). You will need to give this whilst the patient is in the theatre as Remifentanil wears off very quickly.

I sit the patient up at 20° and place the arterial transducer at the level of the head.

Carotid Ligation

Occasionally there are aneurysms that are untreatable due to their location eg cavernous sinus aneurysms. These may be treated with embolisation techniques but sometimes come to theatre for carotid ligation. The management of these patients is very similar to carotid endarterectomies. The problem is to pick those who can tolerate the loss of flow. Pre-op compression of the carotid helps but is not infallible (often done now by radiological balloon occlusion of the carotid). It is ideal to have some form of cerebral function monitoring and to measure the stump pressure as a guide. Unfortunately there is an incidence of delayed hemiplegia probably due to extending thrombosis from the occluded vessel, many are therefore heparinised for a period post operatively.

Post Operative Management

The worries post-op are

i) Vasospasm

ii) Re-bleeds

iii) Infarction either due to the clip occluding a vessel or to thrombosis

iv) In the higher risk groups there may be continual decreased level of consciousness and the usual complication occur, eg pulmonary.

The patients should be assessed in the recovery ward prior to their return to ICU as there may be need to re-operate if major new neurological signs have occured. It is for this reason that a simple pre-op neurological examination is useful.

The higher risk groups obviously have the greatest risk of these. Vasospasm is the greatest fear and if a patient goes off after surgery a CT scan needs to be done to exclude a haematoma. If this is normal then an angiogram should be done. Ideally rCBF's would allow more rational use of treatments. The BP is raised until the neurological deficit goes or an arbitrary limit is reached. This vasospasm may not present until several days after an apparently successful operation.

Post op the fluid status needs to be very carefully looked at with enough fluids given to maintain an adequate U/O but not to much as to cause cerebral or pulmonary oedema. There needs to be very close monitoring of their neurological status and it is therefore usual for them to be monitored in an intensive care environment. I personally feel that all their monitoring lines should not be removed until the patient has been stable for at least 12-24 hrs.

They need a close eye kept on their electrolyte status as hyponatraemia, if it occurs, can be catastrophic (causing cerebral oedema)

It is usual for a repeat angiography to be done to see if the aneurysm has been properly dealt with.

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