Hemodynamic and Electrophysiological Monitoring during Craniotomies: Current Practice

I. In order to be able to appreciate the value of monitoring it is first necessary to deal with some conceptual matters.

A. **Definitions**

1. *to monitor (V)*
   "to watch, observe, or check, especially for a special purpose"
2. *monitor (N)*
   "that which warns or instructs"

B. **Monitoring Rules**

1. **All monitors are dangerous**
   this varies from the real eg pulmonary artery ruptures, to the theoretical eg micro-electrocution, or the practical eg it might fall on your foot!
2. **Monitors are, of themselves, no benefit.**
   A monitor provides information about physiological variables in a patient ideally about information that is not able to be obtained clinically. They may process this in some way and may provide alarm functions when certain limits are reached. It is very rare for a monitor itself to do anything active.
3. **Benefit is gained from the appropriate use of the information obtained**
   a) **The benefit relates to closing the loop.**
   That is taking some action. This is most commonly us but rarely might be a computer system (e.g. as a microprocessor controlled relaxant infusion). The critical point is this: any benefit from monitoring can ***only*** occur as a result of appropriate clinical decisions in response to the information obtained. It also carries with it the risk that if inappropriate decisions are made the patient may be harmed rather than benefited.
   b) **The more complex the monitor, and the more abstract the parameter being measured, the less likely the appropriate clinical decisions are to be made and the more likely harm will result.**
4. **Only if the benefits exceeds the risks should the monitoring be used**

C. **Monitoring is essential for *rational* decision making**
What this all means is that we monitor to provide the information that we need to make appropriate decisions. i.e. if you arn't going to change the management of the patient then unless when is do a research project you should not be using the monitors.

D. **Monitoring brings with it the problems of, and solutions to "limits"**
The key question when one monitors is what are the acceptable ranges for the variables I am monitoring (of course it may be a pattern of changes that triggers a management change rather than a change in a single variable). At what point will I change the management of the patient. This is a complex *clinical* problem but is often reduced to arbitrary decision making when a given number becomes the sole decider. For example if a patient came to you in your rooms and you measured their saturation and it was 92% you would be unlikely to recommend home oxygen however if this was a patient in the recovery room you would almost certainly give oxygen. One of the main reasons for monitoring is to reduce the arbitary nature of decison making to a minimum however it is, paradoxically, one of the major causes of arbitrary decision making.
These problems become amplified when you are changing the patients state to optimise the surgical conditions e.g. induced hypotension. The further one wishes to move the patients physiology away from their usual state the more one needs monitoring to gain the information necessary to determine whether the changes are likely to harm the paitient.

E. **What are we trying to achieve during Craniotomies?**
Neuroanaesthesia differs from many types of Anaesthesia in that we frequently seek to make substantial changes to the patients physiology in order to allow optimal surgical management. We must therefore balance the benefits to be gained from our manouvers with the risks involved in obtaining these benefits.

1. **Optimise Surgical Conditions**
2. **Minimise harm to the patient**
   a) **Surgical**
   b) **Anaesthetic**
      (1) **Induced hypotension**
      (2) **Induced hypocarbia**
      (3) **Diuretics**
      (4) **Specific side effects of anaesthetic agents**
      e.g cerebral vasodilation with volatile agents

F. **What can we monitor during Craniotomies?**
In this paper I am assuming that the minimum monitoring standards that we are using our usual anaesthetic monitors. In particular Oximetry, Capnography, ECG, Neuromuscular junction function, and temperature. I would also add that all patients should have an oesophageal stethoscope because of the limited access one has

to the patients head and the catastrophic complications that occur when the airway is compromised in these patients.

The various devices that we use allow us to determine the limits to which one can change the patients physiology before harm occurs. Functional monitors can only assess certain parts of the neuroaxis and whilst they provide a more accurate assessment of the ulitimate limits that can be reached (in the areas monitored) the changes that they detect will lag behind other physiological changes. They will also not give us much idea about the adequacy of the conditions that we provide for surgery (ie a slack brain) unless they are grossly inadequate. What they do do however is provide an insight into the effects of the surgical procedure hopefully before they become irreversible.

1. **Absolute levels of pressures**
   eg bleeding, rupture of aneurysms, oedema, brain swelling
   a) Slack Brain (so as to minimise retractor damage)
   b) Minimise Cerebral Oedema
   c) Minimise bleeding
   d) Reduce intra-aneurysmal pressure (prevent rupture)
      (1) Pre-dissection
      (2) During dissection/Clipping
2. **Cerebral Blood Flow**
   a) Assess limits to the physiological changes
   b) Guide management of raised ICP
   c) Determine the limits of surgical procedure
3. **Cerebral Oxygenation**
   a) Assess limits to the physiological changes
   b) Determine the limits of surgical procedure
4. **Cellular function**
   This is the critical factor. Even more interesting would be to the ability to assess the point of irreversible cellular dysfunction but at this stage the best we can say is whether function is impaired or not.
   a) Assess limits to the physiological changes
   b) Determine the limits of surgical procedure
      (1) temporary
         (a) deliberate (vascular clips, retraction)
         (b) accidental (retraction etc)
      (2) permanent
         (a) Feeding vessel ligation
         (b) vessel caught in clip when it is felt that repositioning may be very difficut
            i.e. An A. Com A. Aneurysm where one A2 *may* be felt to be compromised and the presence of unchanged LLSSEPs allows the surgeon not to attempt repositioning.
   c) Assess drug dosage

G. **<u>Function depends on:</u>**
   1. **Cellular mechanics**
      this may be effected by
      a) **Drugs**
         eg barbiturates
      b) **Temperature**
         (1) **hypothermia - reversible**
         (2) **hyperthermia - irreversible**
      c) **Toxins**
         eg cyanide
   2. **Adequate supply of nutrients**
      a) **Cerebral Perfusion**
         this is proportional to:
         (1) **Cerebral Perfusion Pressure**
         (2) **Cerebrovascular Resistance**
            (a) **Vessel diameter**
            (b) **Viscosity**
               This is affected by both haematocrit and the plasma viscosity
      b) **Blood contents of nutrients**
      c) **Metabolic demands**
         eg in seizures “normal” perfusion would be grossly inadequate

H. **<u>Cerebral Perfusion Pressure</u>**
   1. **Definition**: **The difference between the cerebral arterial pressure and the greater of the cerebral venous or tissue pressures**

This is slightly different to the usual way in which it is phrased. Usually the last item is intracranial pressure (ICP) but this does not cover situations where direct pressure occurs eg under retractors. In the majority of situations tissue pressure = ICP.

2. **Problems:** We are all familiar with the autoregulatory curve for cerebral perfusion and that, *on average,* normal individuals perfusion remains constant until CPP falls below 60mmhg. There is obviously a biological variation and commonly our patients have abnormal curves. This leaves us with the situation where even if we can measure all the components of CPP we still can not accurately predict from this, in any given patient, what the perfusion will be. Furthermore tissue demand must be known as well before we can say if perfusion is adequate. Nonetheless if the CPP is very low for extended periods then we can be almost certain harm will follow.
3. **Conclusions:** Measurements of pressure is therefore quite removed from what we are really interested in. An analogy can be drawn between trying to diagnose myocardial ischaemia by measuring the arterial pressure, an important factor but not diagnostic. This does not mean that we should not be interested in the CPP concept just that we should understand it's limitations. The one thing that must be said is that changes in CPP preceed changes in flow and function and may therefore allow earlier intervention than might otherwise occur so it is always necessary to have meaurement of arterial pressures.

II. Haemodynamic Monitors

A. Pressures

1. Arterial Pressure

Intra-arterial pressure monitoring is mandatory. It should almost be always be instituted pre-induction but common sense dictates that if great distress is incurred in insertion of the line, it may be prudent to wait until after induction. In all cases it should be placed prior to intubation.

a) **Cerebral Perfusion Pressure**
MAP is the major component (transducer should be zero referenced to the highest point of brain during induced hypotension.)

b) **Hypertension produces:**
(1) - Cerebral blood volume
if past the right hand edge of the autoregulatory plateau
(2) Cerebral oedema formation
(3) Aneurysm rupture
(- transmural pressure)
(4) - bleeding
not proven but observation seems to confirm the idea at least if -- BP

2. Central Venous Pressure

Central venous catheters are very useful for administration of vasoactive drugs eg SNP or Trimetaphan. Cubital fossa access is the most sensible route (minimal risk of major structure damage, no head down during placement, much more pleasant for the patient, no danger of obstruction of venous drainage of head). With good catheters the success rate is >90% of patients.

a) Volume status
CVP accurately reflects this if normal cardiac function. Mandatory to measure CVP if induced hypotension is planned. If one is interested in cardiac filling pressures then the transducer must be zero referenced to the mid-thoracic level ie a different level to the arterial transducer.

b) **Cerebral Blood volume**
Unlike the arterial system there is little resitance on the venous side. The rises in venous pressure, once the column of veous blood reaches the level of the cranium, will cause the cerebral veins to distend increasing the apparent bulk of the brain and worsen the surgical conditions. A given rise in venous pressure will be much more important than a given rise in arterial pressure.

c) Cerebral Perfusion Pressure
CVP is not usually a component as the head is nearly always elevated above the venous column.

d) Aspiration of Air
A multiple orifice single lumen catheter placed at the junction between the SVC and RA is best. Correct placement is most easily done with ECG control. I will not discuss air embolus further. If the venous transducer is set to the highest point of the cranium the risk of air embolus can be clearly established. If the pressure is positive than there is very little risk of air entrainment. If it is negative especially substantially negative the risk is much greater.

3. Intracranial Pressure

It would obviously be desirable to be able to measure this during the induction of anaesthesia and prior to craniotomy. The problem is that it can not be done non-invasively at present. Those patients with an ICP monitor already in should be monitored. Occasionally a patient will present who has had a CSF reservoir inserted in the past, in these patients it is easy to put a needle into the reservoir and measure ICP. Some authors have suggested ICP monitors be placed pre-induction in craniotomies but it is moderately distressing to the patient, quite time consuming, and carries with it not a small risk of infection. I would

imagine that only a small number of patients might have the critically compromised compliance to make this worthwhile and in these circumstances I would generally prefer to give an anaesthetic avoiding all cerebral vasodilators rather than run the risks of electively placing an ICP monitor pre-induction.
The other practical problem is that due to the shape of the cerebral elastance curve a "normal" size brain and a "shrunken" brain will have the same ICP (that is, not raised) but one needs a "shrunken" brain for non-surface surgery so that a normal ICP in no way guaranties adequate surgical conditions. On the other hand, of course, an elevated ICP guaranties inadequate conditions for *all* surgery.

ICP monitoring has much more use in situations where the patient is neurologically impaired, the cranium is closed, and one can not use the mental state of the patient as an indicator of compromised cerebral function. Electrophysiological monitors have been shown to correlate with ICPs but I image this is more with brain stem shift rather than absolute levels of ICP. They are also much more complex and difficult to organise over long periods. There is little indication for ICP monitors (except for diagnositc evaluation of patients with disordered CSF systems) in the conscious, oriented patient.

a) **Brain Shift**
I suspect that much of the problem with raised ICP relates to compression of the brainstem against the foramen magnum rather than a general compromise of CPP ie a greater rise in brainstem tissue pressure than the general ICP rise. Evidence exists that BAEPs will deteriorate as ICP rises but before compromise of global cerebral perfusion occurs. It is very clear that patients with a uniform rise in ICP (communicating hydrocephalus) can tolerate very high (>40 mmHg) with only minimal symptoms. Patients with non-uniform rises (eg head injuries) become critical when the ICP rises much above 20 mmHg. The only logical mechanism for this is brain shift. In the former cases there is no brain shift and changes in CPP explain symptoms. In a normal people the CPP falls by 30 mmHg every time we stand up and this produces absolutely no symptoms. In the situations where non-uniform rises in pressure occur brain shift produces kinking of blood vessels and this produces ischaemia at much lower pressures. This brain shift occurs across meningeal barriers or down the spinal canal (occasionally also out of the brain in skull fractures). This brain shift also means that if one needs to monitor the ICP where the primary pathology is (i.e. on the side of the tumour) otherwise you can seriously underestimate the real pressues causing the brain shift. The majority of clinical situations it is the absolute level of the ICP that is important and not its effect on CPP.

b) **Cerebral Perfusion Pressure**

c) **Transmural aneurysm pressure**
ICP is important in aneurysmal rupture but is an order of magnitude less than arterial pressure. I suspect that the major time it is of importance is when ICP is quite elevated and is suddenly reduced by CSF drainage.

4. **<u>Pulmonary Artery Pressure/CO</u>**
Indications for placement are **cardiac** not neurosurgical. It is commonly stated as being indicated for SAH patients but there is good evidence that the vast majority of patients with a normal ECG have good cardiac function and that even the presence of ECG abnormalities does not absolutely indicate cardiac dysfunction. That can be assessed non-invasively pre-operatively to delineate the group needing a pulmonary artery catheter (PAC). Certainly there is no evidence that in the absence of cardiac disease they will develop cardiac dysfunction during surgery. Stated indications are:
a) **Optimise volume status to prevent/treat vasospasm**
Current definitive treatment is induced hypertension. Part of this is ensuring adequate cardiac filling and except for those with poor cardiac function a CVP is adequate. There is no published literature to support the routine use of a PAC in these patients. Also, there is no evidence that hyervolaemia **prevents** vasospasm. For a fixed resistance, arterial pressure is the fundamental factor determining perfusion.
b) **Ensure adequate CO with induced hypotension**
Critical organ perfusion is not assessed by cardiac output measurements. This is assessed by individually monitoring organ function. Furthermore CO can now be assessed much less invasively with a trans-tracheal or oesophageal doppler.

B. **<u>Cardiac Output measurement</u>**
1. **<u>Trans-tracheal Doppler</u>**
New technology that non-invasively measures CO via a sensor attached to the end of an endotracheal tube. It could be used as a replacement for a PAC in patients having induced hypotension without cardiac dysfunction. It will not replace PACs completely as no information of left sided filling pressures is provided.
2. **<u>Swan-Ganz Catheters</u>**

C. **<u>Cerebral Blood Flow</u>**
1. Trans-cranial Doppler

May be of some benefit but suffers from the critical problem that velocity is only proportional to flow if the cross-sectional area of the vessel is constant. This area has been shown to vary under a variety of circumstances. A flat probe that can be attached to the side of the head is available but TCDs exact role during craniotomies is unclear. The one area that it does seem to be useful in is the diagnosis of cerebral vasospasm but this is not an intra-operative use. I might point out that the diagnositic finding is an increase in the velocity as the vessel narrows!
No recent evidence that it is vary useful during craniotomies.

2. **Laser Doppler**
   This uses a probe that is placed directly onto an area of cerebral cortex. It uses the reflection of light from RBCs in the area of cortex immediately below the probe to calculate a rCBF equivalent. The banwidth of the reflected light is proportional to the flow. The amount of activity at these shifted frequancies gives an indication of the number of RBCs in the volume of cortex looked at (i.e. a measure of blood volume) It monitors a small (several mm deep) area of tissue. It must be positioned over an area of cortical tissue and not a major vessel for accurate results. Gives relative values but it is claimed it can be calibrated against some other technique to give an absolute value. It looks at flow in very small vessels and probe orientation is not a problem and is therefore much more reliable than ultrasound doppler.
   A problem is that it only looks at one small area of cortex.
3. **Thermal Diffusion**
   CBF is measured by thermal diffusion. The thermal diffusion is constant without blood flow, but with an increased blood flow, the thermal conductivity increment was a linear function of the rate of flow in the tissue. A distal circular gold plate (6mm) is heated (with a fixed amount of power) while a smaller proximal gold plate (2mm) is held at a neutral temperature by the cortex it is resting on. The temperature gradient between the two plates is inversely proportional to CBF. An alternative type exists where the temperature difference between the two plates is held constant and the amount of power needed to do this is proportional to the flow.
   The device is placed subdurally and may contain facility to monitor ICP as well. It should be over an area of cortex not a major vessel for accurate measurement. It correlates well with other techniques. It cortical blood flow (in the top 2-3mm) not global flow.
   It appears that it still needs to be calibrated against some other device however it seems to be possible to do this in a given probe on an experimental animal.
   Has the same problem as Laser doppler in that it only looks at one small area of cortex.

D. **Conclusions**
   Haemodynamic monitors provide information about the supply side of the supply/demand equation only.
   1. **Infer but do not guaranty optimal surgical conditions**
      We are often aiming for a "shrunken" brain but we have no objective measure of this. We can only rely on what the surgeon says once the bone flap is raised. The major mechanism we commonly use to acheive this (diuretics) will have been given some time before this on empiracl grounds. One of the advantages of lumbar CSF drains is that they are able to be used whenever you need it with little lag (they have other costs of course).
   2. **Provide information that may proceed changes in function.**
      This may therefore allow action to be taken in advance to provent functional impairment.
   3. **Acceptable limits are empirical**
      The further one gets from the actual function of the cell the less acurately one can predict acceptable ranges for the variable one is monitoring. It is usually this problem of "limits" that drives us to more detailed monitoring.

III. **Cerebral Oxygenation**

A. **Jugular Venous Oxygen sampling**
   This requires the insertion of a jugular venous bulb catheter and is quite invasive. It runs the risk of compromising venous drainage if a haematoma developes in the neck during insertion. They is a lot of literature about its use in neuro-intensive care situations but little during surgery except in experimental situations.

B. **Near-infrared Spectroscopy**
   Fascinating technology that allows non-invasive assessment of the cerebral haemoglobin oxygenation state as well as oxidised cytochrome c oxidase. Works via the reflectance of near-infrared light through the intact skin/skull. There are several studies in surgery but mostly in situations of global compromised perfusion eg bypass. Some work has been done in Carotids but the current commercially available system has quite a large probe and in order to monitor the area of maximal ischaemia one would need to shave quite a lot of hair would need to be shaved off. At present it is primarily of research interest but it has the potential to revolutionise the management of compromised cerebral perfusion. A problem with it that we are only looking at one are of cortex and it tells us nothing about what is happening in sub-cortical structures. Despite being around for over 10 years now it has not fulfilled it's potential and remains primarily a research tool.

IV. **Electrophysiological Monitors: "Functional" Monitoring**

A. EEG

1. **Cortical Ischaemia detection**

surface electrodes record the majority of their electrical activity ≈2.5 cm around each lead so that for maximal sensitivity they should be placed over the sites of maximal ischaemia. If a site > 2.5 cm away becomes ischaemic it may be detected but the effect will be less due to the persistence of the electrical activity beneath the recording electrode. What we are looking for is a change in the random cortical electrical activity not simply a complete loss of activity.

a) **Indications**

(1) Vessel Occlusion

(a) Temporary clips

eg aneurysms. Tempelhoff et al have reported 2 channel EEG monitoring in middle cerebral artery (MCA) temporary clipping. They used electrodes in fronto-mastoid locations (ie across the ischaemia areas) and found it helped guide intra-operative manipulations and gave no false negatives. Young et al have successfully used strip electrodes placed directly onto the cortical surface to detect ischaemia. If surgery allows lead placement over the appropriate area of cortex I would use the EEG as it is more sensitive to ischaemia than EPs. The MCA and posterior cerebral artery (PCA) territories can usually be monitored in this way.

(b) Permanent clips/ligation

eg aneurysms/tumours. If carotid artery ligation is planned, the EEG can be used to indicate if the patient will tolerate this. The MCA territory is monitored in this situation.

(c) Accidental vessel compression

Permanent Clip misplacement or retractors: we have detected this occurring on several occasions when the surgeons were happy with the clip placement.

(2) Induced hypotension

Myers et al have demonstrated its use for this purpose and lesser EEG changes have been used to justify the use of SNP vs Trimetaphan when the MAP is <50 mmhg.

b) **Problems**

(1) lead placement

The operative site often precludes lead placement in areas where maximum ischaemia will occur. Jones et al found scalp EEGs not to be useful in aneurysms surgery because their leads were on the non-operated hemisphere and over both occipital lobes. The greater the number of electrodes the more sensitive the system becomes but the more cumbersome it is to use. In practical terms, 2 channels are the minimum number that should be used (one on each side) and if appropriately placed they will detect nearly all clinically important ischaemia.

(2) Isolated Subcortical Ischaemia not detected

eg anterior cerebral artery (ACA) or sub-cortical ischaemia eg basilar artery (BA).

2. **Drug Dosing**

a) Cerebral Protection

Maximal "Protection" occurs at the point of maximal depression of CMR and only the EEG allows one to accurately titrate barbiturate loading so as to achieve the maximal effect with minimal side effects. McDermott et al have reported on this use.

There is some recent controversy about this. It appears that perhaps the cerebral protection seen with Barbituates in focal incomplete ischeamia relates more to an inverse steal phenomenon by causing vasoconstriction in the non-ischaemic brain than any effect on the ischaemic brain. Despite the exact mechanism the maximum effect should still relate to the maximum effect on electrical activity which can only be monitored with the EEG.

b) **Epilepsy Control**

The end point being control of the seizures.

c) **ICP Control**

Whilst the dose of barbituates should use the ICP as the initial endpoint a problem arises in knowing when one has given the maximum dose. The maximum effect on ICP is reached when the EEG is isoelectric and therefore the EEG is useful in limiting the dose of barbituates. If the ICP is controllable at a dose that is less than that which produces an isoelectric EEG then the ICP becomes the endpoint.

B. Evoked Potentials

1. **Pathway Ischaemia**

For accurate prediction of postoperative deficits the following must be fulfilled:

a) The pathway monitored must fall within the vascular territory of the vessel at risk.
b) The blood flow at some point within that pathway must fall below the threshold for synaptic activity.

2. **Types**

a) Somatosensory: differing sites of stimulus

(1) Lower limb

stimulate the posterior tibial nerve. EPs are monitored at a peripheral site eg popliteal fossa to determine if the signal enters the system and then at C2 (Brainstem response) and finally over the vertex for the cortical response. The ACA supplies the cortical area.

(2) Upper Limb
stimulate the median nerve. Monitor over the brachial plexus, C2, and sensory cortex. The MCA supplies the cortical area.

b) Brainstem Auditory
only the VIII nerve and the pons are monitored. This is because the generators of the waves that are usually evaluated (I-V) are the VIII nerve (I,II), the ponto-medullary junction (III), body of pons (IV), and ponto-midbrain junction (V).

c) **Motor**
The pathways for the motor and somatosensory systems are different and whilst one can usually predict changes in motor pathways from changes in the somatosensory system (because the ischaemia is usually covers both pathways) this is not always true. We are also usually much more interested in motor defecits rather than sensory ones as they provide much greater functional limitation.
There is essentially no literature dealing with the use of motor evoked potentials in craniotomies outside of the use of stimulation in awake patients for localisation of the motor cortex. There is quite a lot of literature dealing with motor evoked potentials in spinal cord surgery. One of the problems is likely to be their greater sensitivity to depression by anaesthetic agents and the stated need to have the patient unparalysed (most authors use EMG responses rather than the responses in nerves).

(1) **Clinically more relevant**
(2) **Technically more difficult**
(a) **Easily depressed by anaesthetic agents**
(b) **Usually requires an unparalysed patient**

3. **Uses**
more work has been done with EPs due to the problems of lead placement with EEGs and the need for sub-cortical ischaemia detection.

a) **Cortical Ischaemia Detection**
Leads can be placed further away from critical site than with EEGs because of signal conduction. These can be recorded because we are looking for an induced response not an effect on the background random electrical activity.

(1) **Aneurysms**
(a) MCA: ULSSEPs
An extensive body of literature demonstrates its value for clip placement, both for temporary and permanent clipping. The biggest problem with aneurysm surgery is damage to perforators which may produce isolated motor defecits or damage to deep midline structures producing coma in the absence of EP changes.
(b) ACA: LLSSEPs
permit accurate monitoring of this area. This is a difficult area of the cortex for EEG monitoring. The recurent artery of Hubener which supplies part of the internal capsule may produce upper limb weakness if damage is not monitored with LLSSEPs.

(2) **Induced Hypotension**
EP change indicates the true point of synaptic failure. This may occur at higher pressures than otherwise expected due to sub-clinical vasospasm. Conversely lower pressures than might otherwise be used may be tolerated. One problem is that that only one or two parts of the cortex are being monitored. In general the EEG is probably a better guide due to its greater sensitivity to ischaemia. If we are using EPs then we can use them to indicate if the MAP we go down to (≈50 mmhg) is safe.

b) **Brainstem Ischaemia Detection**
(1) **Basilar artery aneurysms**
(a) SSEPs traverse the whole brainstem
(b) BAEPs generated predominantly from the pons
therefore can not give indications of midbrain ischaemia. Only useful for pontine ischaemia.
(c) Midline midbrain ischaemia may not be detected
Most basilar aneurysms are at the basilar tip and therefore clip ischaemia is most likely in the medial perforator (midline midbrain) or posterior cerebral distribution.
ULSSEPs useful, but may give false negatives but not false positives. This is because whilst they traverse the entire brainstem, in the midbrain the pathways have moved laterally and may not be effected by midline midbrain ischaemia.

(2) **Compression**
for the above reasons if I am interested in the brainstem as a whole then I use SSEPs (usually UL as the signals are larger).

(a) Surgical Dissection
eg brainstem haemangiomas
(b) Retractors
(c) Haematomas

c) **VIII Nerve**

(1) **Microvascular decompressions**: **1-4% incidence of deafness**
Monitoring the latency of wave V allows the detection of critical degree of nerve stretch. Many authors have looked at this and demonstrated its usefulness. In fact this is almost the only area where the appropriate use of monitoring has been shown to provide clinical benefit.

(2) **C/P angle tumours**

(a) **Meningiomas**
hearing is commonly preserved and is at risk due to brain stem retraction. BAEPs allows guidance in the amount of medial brain stem retraction tolerable.

(b) **Acoustic Neuromas**
hearing is sometimes present pre-operatively and can be preserved post-operatively. A number of authors have used BAEPs to aid nerve preservation.

d) **Cortical Localisation**
There are a number of centers that use direct recording and stimulation over the cortex to directly localise the sensorimotor cortex for AVM and tumour resection.

e) **Formal demonstration of benefit is unlikely**
If we assumed that the true neurological deficit rate was 5% and the appropriate use of a particular type of monitoring information would reduce this by 50%, and that we accept a level of type 1 error of 5% and Type II error of 20%, we would need a study of 874 patients to demonstrate a real benefit!! The only way we can assess the situation is to look at the published series and cases and try and amalgamate the information and decide where there are benefits. The **False Negative** and **False Positive** rates are important
One could also make the general comment that almost none of our standard monitors have ever had studies done to inidicate their beneficial use (blood pressure, heart rate, ECG, oximtry, capnography, nerve stimulators etc).

(1) **False negatives**
these are the most worrying. Most relate to inappropriate monitoring and sometimes to the termination of monitoring before the end of the case. Continuing the monitoring until the patient is awake and able to be clinically assessed helps delineate situations where changes in physiological parameters may reduce the perfusion below the critical threshold. ie normal at the end of the clipping but with small decreases in BP at end of case the EPs fail thus allowing recognition and appropriate management.

(2) **False positive**
the rate can not be reduced to zero as EEG/EPs fail at rCBF greater than that necessary for irreversible damage ie one can not tell if the deficit present at the end of the case will be permanent. There is also no way to determine the duration a temporary clip may be placed when the EP is clearly abnormal. The prudent approach is to assume no flow and limit clipping, if at all, to less than 3 minutes (37°C).

(3) **Variation in latency and amplitude:**

(a) **Normal variation:** The exact degree to which this occurs is unclear because so few authors control anesthetic and temperature variables. It is also apparent that changes in brain conformation with diuretics and CSF drainage will cause changes (brain moving away from inner table of skull). The temporal relationship of changes to major surgical events helps clarify the cause of changes. Comparisons with the other, hopefully normal side also helps. Unfortunately absolute guidelines do not exist to indicate what is abnormal.

(b) **Anesthetic:** it is critical to control technique to minimise EP variability.
i) SSEPs most effected
ii) BAEPs more resistant
iii) Volatile agents greater effect than intravenous

(c) **Temperature:** > 2° Ø Æ - latency and Ø amplitude of EPs

(d) **Ischaemia:** produces - latency and Ø amplitude.

C. **Facial Nerve Monitoring**
Operations around the C/P angle run the risk of damaging the VII nerve and this is quite a disabling condition. The acurate identificaion by looking for the facial motor response reduces the likelyhood of injury.

1. **Continuous with EMG/Accelerometer**
The facial nerve responds to direct trauma by releasing a volley of impulses. The use of an EMG and an accelerometer (to a lesser extent) allows the surgeon to be aware if he accidentally comes near the nerve (before it is formally indentified)

2. **Intermittent with Accelerometers/observation/hands**
   This allows the formal identificaion of the nerve but this implies that they have clinically identified a sturcture that might be the nerve and that has the problem that the surgeon may not realise he is near the nerve until it is too late. Despite this problem this is a useful technique. The systems of either watching the face or intermittantly feeling for facial contractions are much inferior to devices that allow continuous monitoring. The intermittant system with disposable stimulators and clincial monitoring of contractions is very cheap.

D. Difference between EEG and EPs

1. **Anatomical: Cortical vs Pathway**
   The EEG is the collection of spontaneous electrical potentials generated by the cerebral cortex alone. This is predominantly made up of excitatory and inhibitory post-synaptic potentials **not** action potentials (AP). EPs are summations of synchronised APs. Hence EPs are generated in a variety of anatomical locations from the stimulus site to the final cortical response. Different EPs therefore have different pathways and specificity relates to the stimulus site and type. We monitor different points on these pathways with strategically placed electrodes and by looking at specific time periods.
2. **EEG: spontaneous & random, EP: event related**
3. **EEG: large amplitude, EP: small**
   you need averaging to extract EPs from the background noise, therefore it is a discontinuous monitor. The time for a new trace varies from 30 to 120 seconds depending on the stimulus rate and the background noise.
4. **Threshold for changes**
   the EEG begins to change at ≈20 mls/100 gms/m vs 15 for somatosensory evoked potentials (SSEP). The SSEPs do not change until the point at which synaptic failure begins to occur (isoelectric EEG) and therefore have been said to be a more appropriate measure of function. On the other hand EPs are therefore less sensitive detectors of ischaemia
   The relationship between CBF and electrophysiological changes is markedly non-linear, there are no changes until CBF is quite markedly reduced however once changes begin to occur there is rapid deterioration with further small decrements in flow.
5. **You an still record EPs with barbiturate induced isoelectric EEG**
   Brain stem auditory evoked potentials (BAEP) are least affected but SSEPs are also still recordable.

V. What do I actually do

A. **Machinery/Personnel:** I use a 2 channel Biologic Navigator (≈$40-50,000) which allows EEG (raw plus compressed spectral analysis) and SSEP/BAEP. I do not have a technician and I place all the electrodes and run the machinery myself! It is highly desirable to have someone else present during the anesthetic (ie a resident) so that the patient isn't ignored during the set up period. For electrophysiological monitoring to be usable without a dedicated technician the equipment requires a high degree of automation. The above system is essentially fully automatic and only requires observation of the waveforms produced. It is thus possible to provide an electrophysiological monitoring service for much less than is commonly thought. 2 channel processed EEG is much easier than EPs and can be done without an assistant as long as one places the electrodes pre-induction. We also use a Silverstein (accelerometer based) system for VII N monitoring.

B. **Aneurysms**
   the decision as what to technique to use depends on the expected site of maximal ischaemia. The surgeons will be able to give a reasonable idea where this is.
   1. **MCA**: EEG if electrode placement possible otherwise ULSSEPs
   2. **PCA**: EEG nb visual evoked potentials are of no value intra-operatively
   3. **ACA**: LLSSEPs
   4. **Basilar**: ULSSEPs

C. **Brainstem tumours (non-C/P Angle or when hearing gone):** ULSSEPs

D. **C/P Angle tumours:** BAEPs (unilateral) if hearing present otherwise ULSSEPs

VI. Conclusions

The take home message is:

A. **If there is potentially reversible ischaemia of neural tissue that can be monitored, then it should be**.
   In consultation with the surgeons a protocol should be established outlining how the information that is then available should be managed. Each group who utilises these monitors should endeavour to collect as much data as possible on their cases so as to allow an ongoing assessment of the utility of the procedure.

B. **Anaesthetists can do it!**
   Despite the complexity of the systems being monitored there is equipment available that allows the non-neurologist to successfully undertake this type of monitoring and to gather useful information which cannot be obtained by any other means and rapidly becomes in invaluable aid to optimising patient care.

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