Stroke is the most significant perioperative risk for patients undergoing carotid endarterectomy (CEA). Patients are accepting a greatly increased short-term risk of stroke in return for a reduced risk over the subsequent years. The causes of perioperative stroke are multiple and include emboli, technical problems such as intimal flap, intraoperative cerebral ischaemia during clamping, and postoperative hyperaemia. Despite a large number of clinical studies over many decades, no particular intervention has been shown to reduce the risk of perioperative stroke. Due to the low rate of perioperative stroke (overall risk <3%) and the multiple different causes, very large trials would be needed to detect a benefit from any particular monitoring technique.

Intraoperative monitoring potentially provides prompt detection of:

- Ischaemia
- Emboli
- Hyperaemia

Although ischaemia during internal carotid artery (ICA) clamping is not necessarily the commonest cause of perioperative stroke, it is the focus of much attention because it is potentially avoidable by insertion of a bypass shunt. Most patients do not suffer significant ischaemia with ICA clamping, therefore not every patient requires a shunt. Furthermore, inserting a shunt is not always technically possible. Shunts increase the technical difficulty of the operation, can dislodge emboli, and can cause intimal damage. Shunt insertion is not always technically possible.

Practice varies markedly from centre to centre and surgeon to surgeon. Some surgeons choose to shunt every patient. The majority shunt selectively but the criteria for shunting is highly variable. Some teams shunt based on preoperative information such as vascular investigations (e.g. disease of the contralateral ICA) or history (e.g. previous stroke). Others teams base the decision to shunt on intraoperative assessment of the adequacy of collateral cerebral blood flow (CBF) during ICA clamping. CBF can be assessed either by detection of symptoms in an awake patient or by various monitoring modalities during general anaesthesia. It is beyond the scope of this presentation to explore the relative merits of local versus general anaesthesia, other than to note that outcome evidence does not support either approach over the other. As the wide variability of practice attests, definitive evidence for any particular shunting practice is lacking.

Some comments on the vascular anatomy.

The terminal ICA divides to form the middle and anterior cerebral arteries (MCA and ACA). Monitoring that specifically targets the MCA territory may be preferable for the following reasons. The MCA is the dominant branch of the circle of Willis, carrying 50% – 70% of the total cortical blood flow, and large
MCA territory strokes are particularly devastating. A not-uncommon abnormality of the circle of Willis is absence or hypoplasia of the first part of the ACA, causing the anterior part of the circle of Willis to be deficient and both ACAs to be supplied by one carotid artery.\(^5\) In this situation, monitoring the ACA territory could be falsely reassuring because loss of supply to the ipsilateral MCA will go undetected while the ACA continues to be supplied by the contralateral carotid artery.

**Stump Pressure.**

The simplest method of assessing adequacy of collateral supply during carotid clamping is measurement of ICA blood pressure. Once clamped, flow along the ICA effectively ceases and stump pressure is equal to the pressure at the origin of the middle cerebral artery (MCA). Hence, stump pressure provides a direct measure of the arterial pressure supplying the largest and most at-risk cerebral vascular territory.

**Limitations:** As with systemic arterial pressure during hypotensive anaesthesia, there is no threshold stump pressure which defines a safe lower limit in all patients. Multiple studies have found stump pressure to be very poorly predictive of cerebral ischaemia as defined by EEG changes and other criteria. Nevertheless, if no other monitoring modality is available, it may be reasonable to measure stump pressure. A low stump pressure, e.g. < 40 mmHg, indicates a higher risk of cerebral ischaemia;\(^6\) albeit with a significant rate of false positives and false negatives.

**Electroencephalography (EEG)**

Cerebral ischaemia causes well-defined EEG changes. Moderate ischaemia causes loss of high frequency activity. More pronounced ischaemia causes large amplitude, low frequency activity and severe ischaemia completely obliterates EEG activity. Older studies demonstrated a correlation between the severity and duration of EEG changes and the risk of post-operative neurological deficit. Ideally, multiple EEG channels should be monitored simultaneously to maximise the chance of detecting ischaemia and to allow comparison with the contralateral side.

**Limitations:** An EEG channel is derived from a pair of scalp electrodes and each channel only detects ischaemia in the vicinity of the electrodes. Furthermore, if only one of the electrode-pair is overlying the ischaemic territory, the EEG changes will be partially obscured by activity under the other electrode. EEG evidence of ischaemia can be quite subtle, requiring vigilance, experience, and properly adjusted signal processing to detect.

EEG-based monitors such as BIS and Entropy can detect cerebral ischaemia. Under general anaesthesia, a sudden reduction in the monitor score following ICA clamping would be highly suggestive of cerebral ischaemia.\(^7\) However, these devices only monitor the frontal area supplied by the ACA and could miss isolated MCA ischaemia. Decreased BIS has been reported in awake patients requiring a shunt, although the BIS did not fall in a patient who developed a pure motor deficit with no change in level of consciousness.\(^8\)
**Somatosensory Evoked Potentials (SSEPs)**

**Technique:** SSEPs are produced by electrically stimulating a peripheral sensory nerve and recording over the appropriate region of the sensory cortex. Because the evoked potential is about one hundredth the amplitude of the spontaneous EEG, it is necessary to average recordings from multiple stimulations to extract the SSEP. For monitoring during CEA, median nerve SSEPs are used because they are reliable and relatively easy to obtain and the relevant sensory cortex is within the MCA vascular territory.

**Advantages:** Median nerve SSEPs are reasonably sensitive and specific for cerebral ischaemia. Unlike EEG monitoring, SSEPs can also detect subcortical ischaemia involving, for example, the internal capsule. A common criteria for diagnosing ischaemia is a ≥50% reduction in SSEP amplitude, although one study found a 30% reduction yielded optimal correlation with symptoms in awake patients.\(^9\) Comparison of SSEPs with onset of clinical symptoms during awake CEA have yielded sensitivities of 85 – 89% and specificities of 89 -90%.\(^9,10\)

**Limitations:** SSEPs are impaired by inhalational anaesthetics (including N\(_2\)O). Electrophysiologic systems capable of acquiring SSEPs are expensive. However, the disposable electrodes for each patient are inexpensive. Setting up the monitoring system is usually performed after induction of anaesthesia and therefore adds to the procedure time. Typically, institutions that monitor SSEPs use the services of an electrophysiologist. However, in our institution we have found it possible for the anaesthetist to set up and run the SSEPs. The crucial time to monitor is at the commencement of ICA clamping. We do not find it difficult for the anaesthetist to attend to the monitor while also managing the anaesthetic care of the patient.

**Transcranial Doppler (TCD)**

**Technique:** Pulsed wave Doppler signals can be obtained from the major intracranial arteries via the temporal window: an area of thin skull above the zygomatic arch in front of the ear. MCA signals are identified as a characteristic waveform arising from blood flowing towards the probe at a depth of around 45 to 55 mm from the skin. TCD equipment outlines the peak velocity envelope of the Doppler spectrum and calculates a time-average of the peak velocity; usually expressed in cm/sec. Providing the diameter of the insonated vessel remains constant, changes in the average velocity have been found to be proportional to changes in actual blood flow.

TCD continuously and non-invasively monitors MCA blood velocity (Vmca). On carotid clamping, it is usual to see an immediate decrease in Vmca followed by a partial recovery over the next 15 seconds as autoregulatory vasodilatation attempts to restore flow. A commonly accepted criterion for intervening is a ≥40% reduction in Vmca.\(^11\)

In addition to monitoring adequacy of cerebral blood flow, TCD is useful for detecting cerebral emboli. Manipulation of the carotid arteries during the dissection phase can occasionally dislodge emboli. Solid emboli detected during dissection or during wound closure are predictive of stroke.\(^12\) If emboli are detected during dissection, the surgeon can adjust their technique in an attempt to minimise disturbance to the plaque or they may choose to protect the brain by
clamping the ICA before completing the dissection. Emboli detected during wound closure or in the immediate postoperative period can be treated with anti-platelet therapy such as dextran. Detection of gaseous emboli is not uncommon after release of the vascular clamp. The incidence of gaseous emboli depends on surgical technique and it has been noted that the audible feedback from the TCD monitor causes surgeons to modify their technique, leading to fewer emboli in subsequent patients. With the aim of sending any embolic material to the facial circulation rather than the brain, it is standard practice to release the external carotid artery before the ICA. However, we have sometimes detected emboli in the MCA immediately on release of the external carotid clamp; presumably due to the presence of ophthalmic artery collateral flow carrying blood from the external circulation in to the circle of Willis.

**Advantages:** TCD is non-invasive and can monitor cerebral blood flow with a better time-resolution than any other technique. After purchase and maintenance of the equipment, there are no additional per-patient costs. In addition to monitoring for ischaemia during clamping, TCD can detect emboli and can detect problems during the closing stage of the surgery such as hyperaemia or sudden loss of flow (e.g. due to an intimal flap).

**Limitations:** The major limitation of TCD is the difficulty obtaining signals in some patients and the difficulty maintaining a robust intraoperative signal in others. Around 10% of patients do not have a temporal window because their bone is too thick to transmit the ultrasound signal. Considerable practice is required to acquire the skills to reliably obtain a TCD signal. Monitoring probes must be firmly held in position and the fixation device needs to be secure enough to minimise movement during surgery. The time taken to locate a signal and secure the probes can add significantly to the procedure time. If the signal is lost during the procedure, attempts to access the probe and regain the signal can be disruptive to the surgeon.

**Near-Infrared Spectroscopy (NIRS)**

Light in the near-infrared spectrum can penetrate the skull and some of the light reaching the brain is scattered back to the surface where it can be detected by a photocell. By utilising multiple wavelengths with differential absorbance by oxygenated and de-oxygenated Hb, NIRS monitors can estimate Hb saturation. The majority of blood in the cerebral tissue is venous, therefore NIRS measurements are closer to cerebral venous saturation (typically 55 - 70%) than arterial saturation. Monitoring venous saturation is useful because venous oxygen content rises and falls depending on the ratio of cerebral blood flow to cerebral metabolism.

Unfortunately, no threshold NIRS values to indicate cerebral ischaemia have been established. Different authors recommend either responding to a decrease relative to the pre-clamp saturation (e.g. >20% fall) or a decrease below an arbitrary saturation (e.g. <60%). In carotid surgery, using bilateral NIRS probes allows comparison with the non-operated side, possibly improving the specificity of the monitor.

**Advantages:** NIRS monitoring is very simple to use and requires no significant training or experience. Adhesive probes are applied to the skin of the forehead. The probes are designed to minimise impingement of extraneous light.
**Limitations:** The main difficulty with NIRS monitoring is that falls in saturation are common but there is no defined threshold to indicate the need for shunting. NIRS probes are placed on the forehead and therefore suffer from the limitation of monitoring the ACA rather than the MCA territory. NIRS probes cost several hundreds of dollars per patient, particularly if bilateral monitoring is used.

*Some comments on studies comparing monitoring modalities.*

The most compelling studies attempting to evaluate the reliability of monitors for detecting ischaemia are those performed in patients undergoing awake CEA. These studies assess a monitor’s ability to distinguish patients who develop neurological symptoms on clamping. Unfortunately, most of the published studies include only a small number of patients with ischaemic symptoms, making firm conclusions difficult.

It is often stated that neurological assessment of the awake patient is the gold standard for detecting ischaemia during CEA; an assertion that is, in the opinion of this author, open to question. The rate of shunting in awake CEA is around 15%. In this author’s series under general anaesthesia we have shunted <4% of cases with a <2% rate of new neurological deficit on emergence (unpublished data). Furthermore, similarly low rates of intraoperative stroke have been achieved in large series of CEA under general anaesthesia with a policy of not shunting any patient. Clearly, most patients who develop symptoms when awake would not suffer a stroke if they were operated under general anaesthesia without being shunted. The explanation for this discrepancy is uncertain. Ischaemia may be less frequent under general anaesthesia; perhaps due to effects of anaesthetic agents or effects of blood pressure and CO₂ management. Alternatively, undetected ischaemia may be occurring under general anaesthesia without progressing to clinically apparent infarction. Regardless of the explanation, it is at least arguable that awake CEA leads to an unnecessarily high rate of shunting and that symptoms in awake patients are not really a gold standard when evaluating monitoring techniques intended for use with general anaesthesia.

For some monitors, studies in awake patients may not be a realistic test of their reliability in anaesthetised patients. For example, there are reports of patients who deteriorated neurologically, recovered when a shunt was inserted immediately, and who showed no change of SSEP amplitude. As SSEPs take time for acquisition, it is possible that the SSEPs would have changed over time if the shunt had not been placed so promptly. In the case of EEG monitoring, the baseline EEG is clearly different in awake versus anaesthetised patients. Therefore, the results of EEG and BIS in awake patients may not be directly relevant to patients receiving a general anaesthetic.

*Conclusions*

No monitoring modality has been shown to be completely reliable in determining the need for a shunt during CEA under general anaesthesia. TCD appealing because it not only provides information relevant to adequacy of collateral blood supply but also provides information on other causes of stroke such as emboli and hyperaemia. However, TCD has not gained popularity due to the skill and time required and due to the significant number of patients in whom a reliable signal cannot be obtained. Given the various monitors each have
their strengths and weaknesses, a strategy of using two or more monitors seems reasonable. This author’s current preference is to use median nerve SSEP combined with TCD.

References