Intraoperative Monitoring for Carotid Endarterectomy

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Scott Francis Davis and Jeremy Andrew Bamford

Introduction

Carotid endarterectomy (CEA) is the most frequently performed procedure for the prevention of stroke. Strict selection criteria are applied to determine surgical candidates for CEA as indicated for the treatment of moderate to severe carotid stenosis. Carotid endarterectomy is associated with procedural and periprocedural risks including stroke (embolic or hemodynamic), myocardial infarction, as well as cranial nerve palsy resulting from traction on the recurrent laryngeal nerve. Recent attention has turned to a less invasive surgical approach to treat carotid stenosis, carotid stenting. Stenting and endarterectomy have shown comparable efficacy, but more randomized studies are needed [1].

Carotid revascularization by endarterectomy involves clamping the common, external, and internal arteries so that the vessel can be incised and the plaque removed. The ability of the patient to tolerate the cross-clamp depends on the sufficiency of collateral flow through the circle of Willis. Prior to routine intraoperative monitoring of cerebral perfusion, the surgeon would place an intraluminal shunt in all patients for the purposes of maintaining blood flow around the clamp. Routine shunting has been largely abandoned in favor of selective shunting [2–5]. In selective shunting, the need for a shunt is determined by intraoperative electrophysiological monitoring data [6]. The incidence of procedural embolic stroke is possibly correlated with the use of intraluminal shunts [4, 7]. This could be explained by the increased chance of introducing particulate emboli when the shunt is inserted through a diseased arterial wall. However, the literature is not in agreement that selective shunting reduces intraoperative stroke complications over routine shunting and more randomized studies are called for [8]. The monitoring community, nevertheless, advocates selective shunting, because the need for a shunt can be determined with high sensitivity and specificity with the use of electrophysiological monitoring methods. In addition, continuous monitoring can detect ischemic changes during other critical phases of the procedure as well as monitor the function of an intraluminal shunt if placed. In order for selective shunting to be safely performed, a means for assessing collateral flow and monitoring ongoing cerebral perfusion must be utilized. Older methods of monitoring, such as measurement of carotid stump pressure and cerebral oximetry, have either been replaced or become adjunct to the

S. F. Davis (✉)
Department of Anesthesiology, Louisiana State University School of Medicine, Tulane University School of Medicine, New Orleans, LA, USA

J. A. Bamford
Department of Anesthesiology, Louisiana State, University Health Sciences Center, New Orleans, LA, USA

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modalities of EEG and median nerve SSEP [9–12]. Transcranial Doppler studies may be added to monitor for particulate emboli associated with clamp release and reperfusion as well as intraoperative ischemia [13–15].

It is essential that the neuromonitorist understand the critical phases of the endarterectomy procedure and the risks associated with each phase. Determining the likely cause of intraoperative changes, such as whether a stroke is embolic or hemodynamic in nature, is critical to providing relevant information that may be used by the surgeon or anesthesiologist to formulate an intraoperative treatment plan and prevent a negative outcome.

Intraoperative monitoring of CEA should include multiple modalities including EEG and median nerve SSEP [16, 17]. Continuous monitoring is advised even once a shunt has been placed as the integrity of the shunt may fail and go undetected by the surgeon. An appreciation for the endarterectomy procedure is necessary to insure appropriate attention is paid to all times of increased risk of neurological injury as complications are not restricted to clamping [18]. Both technical and professional monitoring personnel must be well trained and familiar with alarm criteria as well as recording parameters for monitored modalities.

Carotid Stenosis

Stroke is one of the leading causes of death and disability in the United States, and carotid stenosis is one of the leading causes of stroke [19]. Stenosis can occur in any artery in the body and is a result of the accumulation of atherosclerotic plaque buildup on the arterial wall. The most common sites for stenosis are arterial bifurcations. At an arterial bifurcation, blood flow is turbulent and there is more opportunity for plaque accumulation. A good analogy for this process is a fork in a river. The fork is the point along the course of a river where you are most likely to encounter "white water" and find debris along the riverbanks. Carotid stenosis occurs most often at the bifurcation of the common carotid into the internal and external carotid arteries (Fig. 15.1).

Carotid endarterectomy is the surgical option for the treatment of carotid stenosis. Stenosis that occurs much higher near the intracranial segment of the internal carotid artery cannot be treated

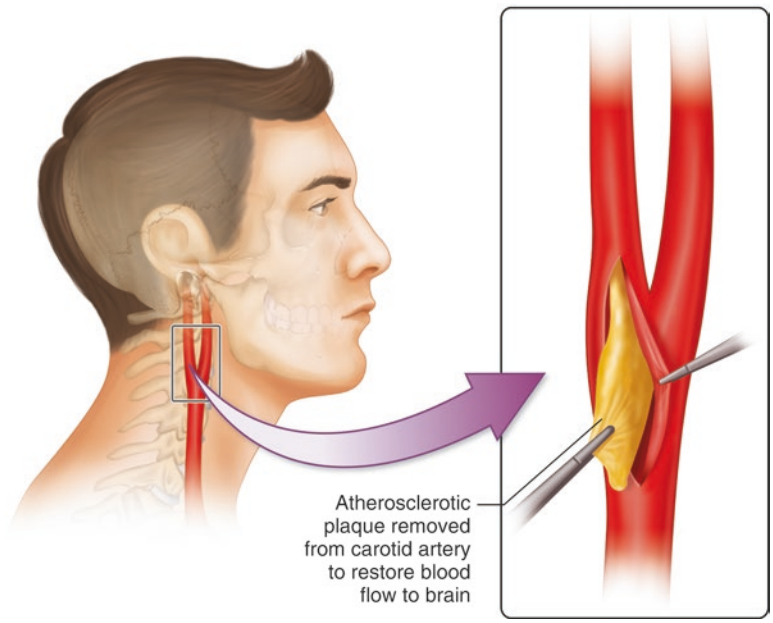

Fig. 15.1 Illustration showing the carotid bifurcation and the removal of plaque at this site by endarterectomy

with endarterectomy, and carotid artery stenting must be considered [20].

Selection Criteria for CEA

Carotid endarterectomy carries with it the risk of stroke and death along with the risks associated with general anesthesia [21–23]. For this reason, the risk to benefit ratio should favor surgical intervention. Recent studies have led to strict selection criteria for patients undergoing CEA. Current selection criteria support CEA for symptomatic patients with severe (>70%) and moderate (50–69%) stenosis as well as asymptomatic patients with severe stenosis. Other factors taken into consideration include comorbidities that may increase the perioperative complication rate, history of ipsilateral stroke, and life expectancy [24–26].

Preoperative Testing

EEG and SSEP testing may be performed on a patient prior to the day of surgery. This is not required for accurate intraoperative neurophysiological monitoring of the patient but may be useful in determining whether any abnormalities or asymmetries may be expected in the operating room. The existence of preoperative asymmetries should heighten the awareness of the monitorist of an increased potential for change during cross-clamping especially if there are any residual neurological symptoms following a prior stroke [27]. It is important to utilize the results of preoperative testing for the purposes of planning while remembering that the patient's intraoperative (post-induction) baselines will be the only data that matter during the monitoring procedure.

Anesthesia for Monitoring of CEA

The anesthetic regimen for intraoperative neurophysiological monitoring of any surgical case is determined based on the modalities to be monitored. For monitoring of most endarterectomies, the anesthetic requirements for SSEP and EEG recordings are to be considered [28]. Anesthesia and intraoperative monitoring is reviewed elsewhere in this volume. When monitoring of the recurrent laryngeal nerve is included in the monitoring protocol, the avoidance of muscle relaxants would also be essential. In the absence of preoperative EEG and SSEP testing, a preinduction baseline can illuminate any asymmetries due to a prior ischemic event. No further importance should be given to preinduction data, as the post-induction baseline will be the data against which changes are judged.

The pattern of EEG will change as the patient proceeds through the various states of anesthesia [28, 29]. Rapid induction, especially with barbiturates, will result in an alpha/beta pattern dominant in the frontal channels. As the stage of anesthesia moves toward the surgical plane, this activity will generalize and then begin to slow. Increases in volatile anesthetics beyond 1 MAC may result in a burst suppression pattern in the EEG, which is not conducive to monitoring EEG. If the EEG is in burst suppression, it is important for the monitoring team to inform the surgeon that EEG monitoring is currently unreliable and then begin to work with the anesthesia team to adjust the regimen to one more permissive of EEG monitoring. Anesthetic protocols may involve the use of minimal inhalants with the addition of a propofol infusion. In many instances, it is preferable to have the volatile agent higher as long as it does not exceed 1 MAC and the propofol infusion rate lower. It would be better to avoid a propofol infusion altogether since propofol can lead to a concentration-dependent burst suppression of the EEG. While it is optimal to have data from multiple modalities available when making interpretations, it is worth noting that SSEPs can still be reliably monitored even when the EEG is in burst suppression [30, 31]. Good communication with the anesthesia team prior to the case will help insure that such interruptions in monitoring are kept to a minimum.

Changes in the anesthetic load will also affect the reliability of SSEP data. Symmetric changes in the cortical potential (N20) can be suggestive

of anesthetic change, but the possibility of a surgical or peri-surgical cause cannot be ruled out. An asymmetric reduction in the amplitude or latency increase of the N20, however, is suggestive of a clinically significant change over an anesthetic-induced change. It is important that the anesthesia team be aware that changes in anesthetic load (e.g., delivering a bolus) are undesirable, especially near the time of or during an important surgical step.

Monitoring the patient's physiological status is an important job of the anesthesia team. The neurophysiological monitoring clinician can aid the anesthesia team by correlating change in physiological status with cerebral perfusion. One of the most important functions of the anesthesia team during the procedure is the regulation of the mean arterial pressure (MAP). Unlike most spine procedures, the CEA requires that the patients' MAP be carefully regulated at different points during the procedure [32]. For example, the MAP is increased during clamp to facilitate collateral circulation but reduced just before unclamping to avoid reperfusion injury. In addition, many patients undergoing CEA have a history of cardiovascular disease and hypertension, which may impede the ability of the arterial system to autoregulate. The consequence of this is that the patient may not tolerate the mean arterial pressure that they are being maintained at by the anesthesia team. Changes in neurophysiological data not correlating with a surgical step may be a result of changes in MAP. This becomes even more critical during both clamping and reperfusion (clamp release) when MAP must be carefully regulated.

Procedure Details and Critical Phases for Monitoring

While continuous neurophysiological monitoring is essential, there are critical phases of the procedure that warrant specific consideration due to the increased risk (Fig. 15.2). Thompson and Talkington [32] provide a good review of the procedural details of carotid endarterectomy. For the purposes of intraoperative monitoring of the procedure, it is important that the monitorist establishes quality baseline data for all modalities

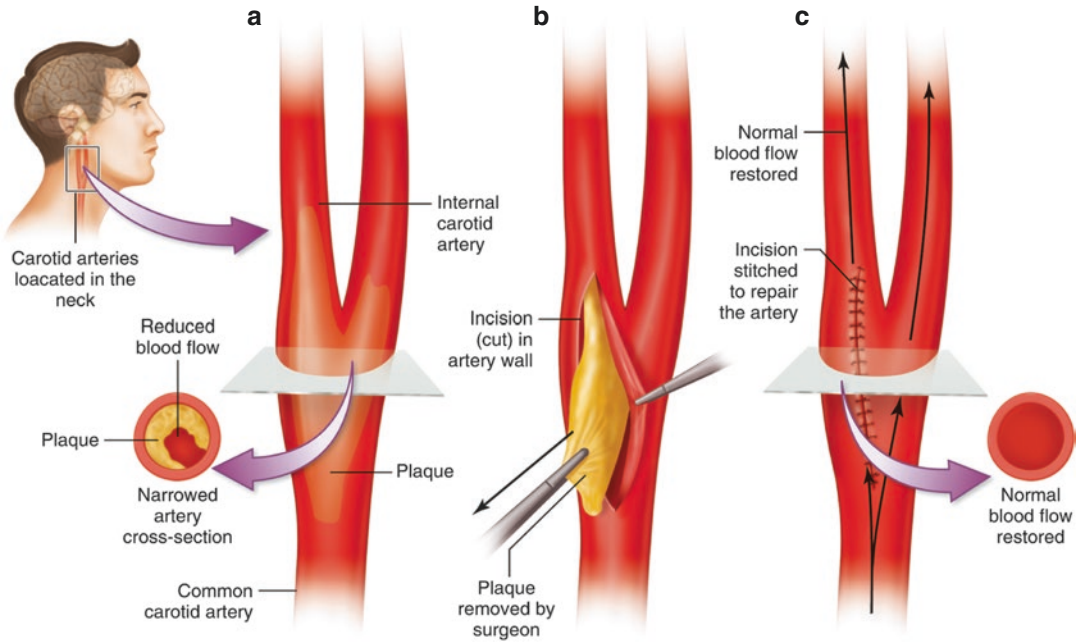

Fig. 15.2 The surgical steps of carotid endarterectomy

monitored after induction but well before cross-clamp. Premedicated baselines should be considered when possible solely for the purposes of revealing any preoperative asymmetries. At least a post-induction 10-min pre-clamp baseline should then be established for the purposes of comparing testing results throughout the procedure [33].

The first critical event is the administration of heparin. Heparin, an anticoagulant, is given prior to carotid cross-clamp for the purpose of preventing thrombus formation that may lead to embolic stroke on reperfusion. By the same mechanism, heparin may re-aggravate any bleeds that may have occurred from aneurysms or other disorders. It takes 4–5 min on average for heparin to raise the active clotting time sufficiently to proceed with carotid cross-clamping.

The next critical event, carotid artery cross-clamping, is likely the reason the surgeon has ordered monitoring to begin with. As you recall, the carotid arteries feed the ipsilateral anterior circulation of the brain. In most healthy patients, the contralateral circulation compensates for the loss of blood flow from one carotid artery. This compensation occurs by virtue of collateral circulation through the circle of Willis. A majority of people have an incomplete circle of Willis, of which there are many variants (Fig. 15.3) [34]. Although incomplete, the circle of Willis is still adequate to provide sufficient collateral circulation in most people. There are, however, certain anatomic variants or pathological conditions (including prior stroke) that result in the inability of the contralateral circulation to compensate for a unilateral carotid occlusion such as occurs during carotid clamping [35]. Changes in electrophysiological data that correlate with carotid cross-clamping should be taken as an alarm that collateral circulation is inadequate to perfuse the brain. A further discussion of alarm criteria will be presented below. In order to facilitate endarterectomy, the common, external, and internal carotid arteries must all be clamped. When collateral circulation is judged inadequate by changes in electrophysiological data, the surgeon will place an intraluminal shunt whose purpose is to reroute blood around the clamp maintaining

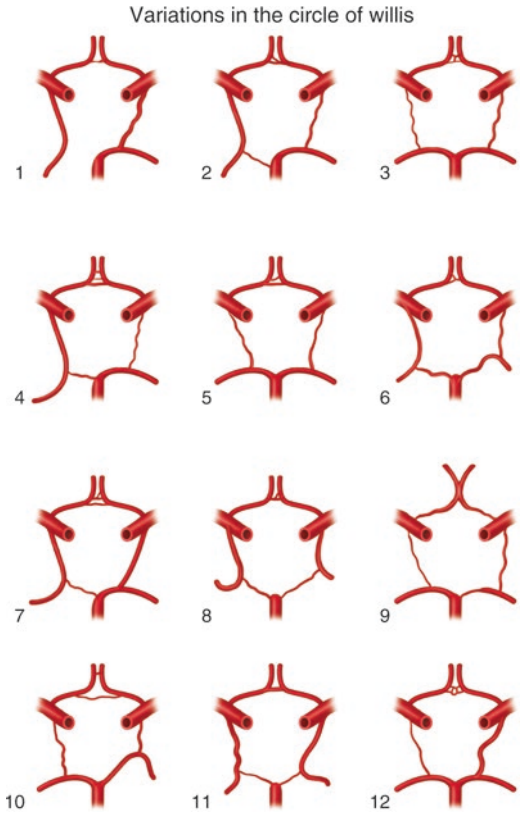

Fig. 15.3 Illustration of 12 variations seen in the circle of Willis

flow to the brain. Due to the increased risk of embolic stroke with shunt placement, the current standard is to shunt selectively as determined by changes in the monitoring data [4, 6, 7]. The anesthesia team must carefully manage the patient's blood pressure during cross-clamp. In order to support collateral circulation, the blood pressure is elevated above normal pre-clamp levels. Sufficient blood pressure can be titrated by carefully observing electrophysiological data from SSEPs and the EEG. Insufficient perfusion will result in a loss of amplitude from recorded signals providing a functional assay that can be used to determine the best blood pressure for the patient.

While carotid cross-clamping is largely considered the most critical phase of the endarterectomy procedure by many, reperfusion is the phase during which the patient is most at risk of suffering a stroke. When the carotid cross-clamp is

released, particulate emboli are released into the circulation. Most of these emboli are too small to cause a problem, but occasionally larger emboli may become lodged in a smaller vessel creating an obstruction [36]. If the obstruction occurs in a cerebral vessel, the resulting ischemia will likely be detectable as a change in SSEP or EEG data prompting intervention. A subcortical obstruction, however, will likely go undetected by routine monitoring modalities. Figure 15.4 shows an example of a clamp-related change in SSEP and EEG data and recovery of these data following insertion of an intraluminal shunt.

Reperfusion injury may occur secondary to a condition known as cerebral hyperemia [37]. Hyperemia can happen in any organ and is the result of too much blood flow. Hyperemia commonly known as reactive hyperemia may occur after a period of ischemia, which, in the case of CEA, may occur during carotid cross-clamp [38]. Hyperemia may develop in the postoperative period and occasionally develops intraoperatively sometime after clamp release. The increase in blood flow seen in hyperemia may cause an increase in intracranial pressure (ICP) that can compress the brain resulting in injury. Transcranial Doppler is the most useful modality in detecting postoperative hyperemia.

EEG Monitoring

Continuous EEG monitoring is used intraoperatively to assess the adequacy of cerebral perfusion and help determine the need for a shunt during carotid endarterectomy [39]. Intraoperative EEG monitoring for carotid endarterectomy does not necessitate recording as many channels as diagnostic EEG. A minimum of eight channels is required for intraoperative monitoring, while the use of more channels is encouraged [40]. The generator of the EEG signal is the cerebral cortex, and as such only cortical perfusion may be monitored with this modality. Subcortical events, such as embolic stroke, are unlikely to be detected with EEG.

EEG monitoring has the advantage of allowing direct monitoring of cerebral function as opposed to modalities such as stump pressure or TCD that only provide an indirect measure of cerebral function. Only SSEPs have demonstrated equal sensitivity to EEG [16]. The addition of median nerve SSEPs, thus, provides a necessary redundancy to EEG monitoring. Hemodynamic changes that do not affect the EEG can usually be assumed to be clinically insignificant, unless an effect is seen in the SSEP recording. EEG monitoring has largely replaced cerebral oximetry for carotid monitoring; however, oximetry may still be used as an adjunct in some centers. Cerebral oximetry measures regional oxygen saturation from the frontal lobes and primarily samples venous blood [9, 11, 12]. The effect of changes in oximetry on cerebral function must be inferred in contrast to the direct information provided by EEG. The following sections provide technical information on setting up and running the intraoperative EEG for monitoring a carotid endarterectomy. The reader is encouraged to become familiar with professional practice guidelines and position statements [41, 42].

Electrode Placement

Stainless steel subdermal needle electrodes are most commonly used for intraoperative EEG with some centers still opting for cup electrodes. The use of needles facilitates a safe and efficient recording setup without the use of adhesives. Electrodes should have an impedance of less than 5 kΩ. A minimum of eight channels of EEG should be recorded for monitoring of carotid endarterectomy. There are several acceptable montages for EEG monitoring of CEA. Table 15.1 shows one of the more commonly used montages often referred to as the modified double banana. A referential montage refers all active leads to a common cephalic reference (usually Cz). In a bipolar montage, active leads are referenced to each other giving the added advantage of increased specificity or ability to more easily locate the area of change. Since efficiency is required in the operative setting, many monitorists make use of their SSEP scalp leads in their EEG montage. The most important considerations are that the choice of recording sites contains areas from frontal to occipital and that leads are placed symmetrically on the left and right side.

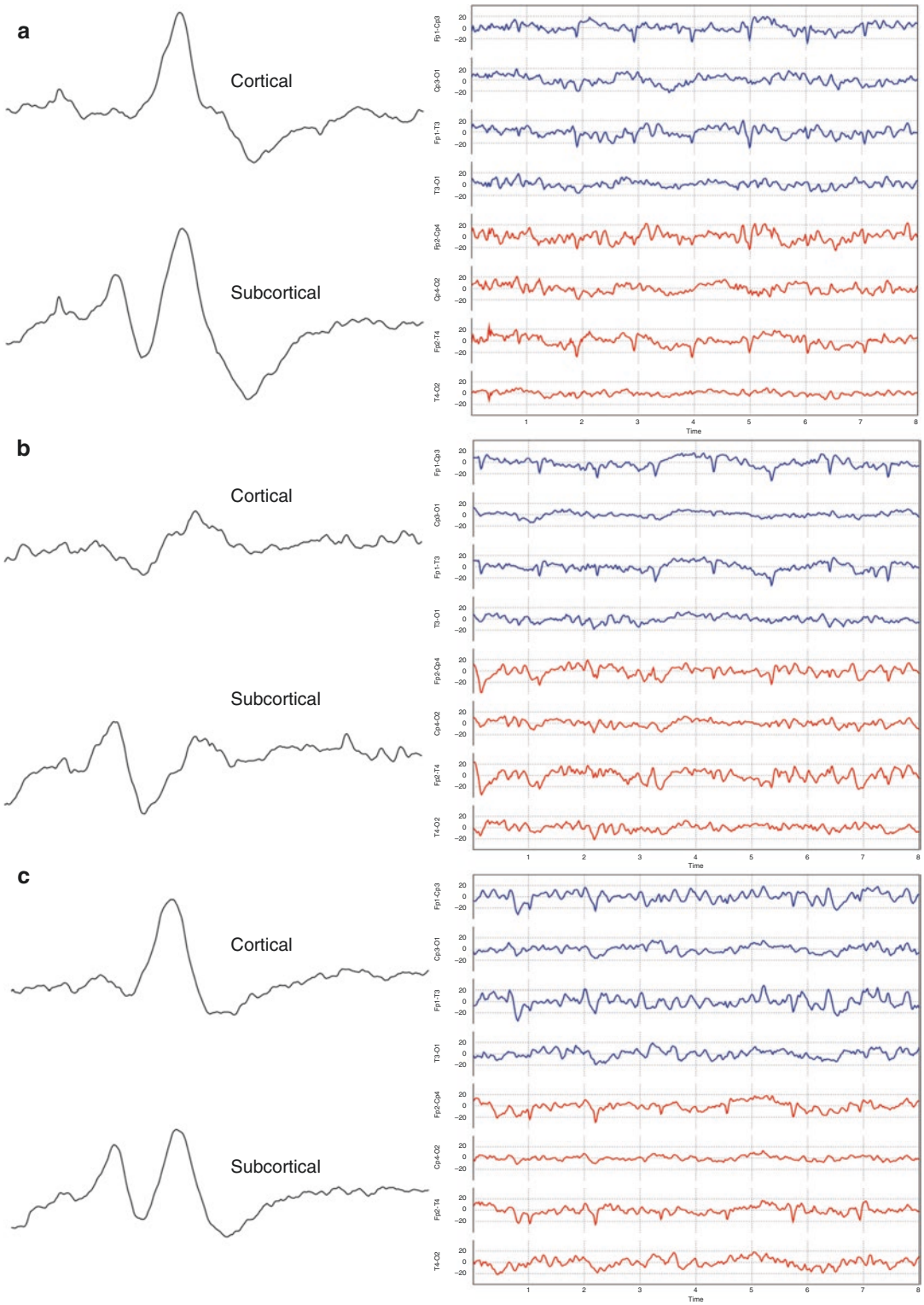

Fig. 15.4 Clamp-related SSEP and EEG change (**a**) SSEP and EEG baseline data established prior to carotid cross-clamp. (**b**) Data taken immediately after carotid cross-clamp showing amplitude reductions in the left cortical SSEP and left EEG. Note no change in the subcortical SSEP data. The generator of this potential is supplied by the posterior cerebral circulation. (**c**) Data taken after shunt placement showing recovery of all amplitudes

Table 15.1 Modified double banana electrode placement

Left	Right
Fp1-Cp3	Fp2-Cp4
Cp3-O1	Cp4-O2
Fp1-T3	Fp2-T4
T3-O1	T4-O2

Recording Parameters

Intraoperative EEG recording should have a bandpass of 0.5–70 Hz. Higher frequency signals such as the gamma band are not seen intraoperatively since they are associated with cognitive function. A notch filter may be used, but only when all attempts at eliminating the source of 60 cycle noise have failed.

Sweep speed (time base) may be set according to the preference of the monitorist with equivalent paper speeds of 10–30 mm/s being the most common. Shorter time bases make it easier to detect changes in the fast beta activity. This activity is generally the first to disappear in an ischemic event.

Sensitivity should be set such that the waveforms are not clipped (sensitivity too high) or appear to be flat (sensitivity too low). Intraoperative EEG is generally of lower amplitude than diagnostic EEG and thus is best viewed between 30 and 50 μV/cm.

Analyzed EEG

The advent of digital EEG has led to the ability to instantly analyze the raw EEG waveform and represent the composite waveform as a spectrum of its component frequencies. This type of analysis is termed spectral analysis and is accomplished with a fast Fourier transform (FFT) algorithm. To perform spectral analysis, the raw EEG waveform is sampled at a desired rate that is set by the user. The composite waveform (sample) is deconstructed into its component frequencies using FFT. The results are displayed graphically showing the power of each frequency band in the composite signal.

Spectral analysis can be useful during a carotid endarterectomy to confirm suspected changes in frequency detected by visual interpretation of raw EEG. It is important to note that the analyzed EEG is not a substitute for the raw EEG and that the raw data should be used as the primary source for interpretation [43].

Alarm Criteria

Alarm criteria for EEG are not widely agreed upon. Correlating different degrees of EEG changes with the postoperative outcome and assigning a weight to the type of change (amplitude reduction, general slowing, reduced fast activity, etc.) is problematic. One commonly used set of criteria include a 50% or greater reduction in amplitude associated with slowing. When less significant changes are judged to be clinically significant, the specificity of the EEG decreases. In spite of the possibility of decreased specificity, it is reasonable to take as clinically significant any change that correlates with a critical surgical event (such as clamping). Future studies may better define safe windows for change. Most clamp-related changes in the EEG recording occur within the first 20 s in most patients with the remainder of patients showing changes within the first minute. Occasionally clamp-related changes may be seen as late as 4 min post-clamp. There are data correlating changes in analyzed EEG with the postoperative outcome; however, one should be cautioned about using analyzed EEG to predict outcome in most practical settings.

SSEP Monitoring

The use of median nerve SSEPs has become a standard adjunct to continuous EEG monitoring during carotid endarterectomy. While MN-SSEPs provide specific protection to the somatosensory cortex, they have demonstrated remarkable sensitivity to cerebral ischemia resulting from carotid cross-clamp. It has been argued that SSEPs are even more sensitive to ischemia than EEG. The ease of SSEP interpretation compared with that of EEG may result in fewer missed occurrences when monitored by personnel less comfortable

with EEG interpretation. Such events cannot be attributed to a failure of EEG monitoring, but rather interpretive error.

Stimulation Parameters

Adhesive surface electrodes are predominantly used for stimulation of the median nerve. Placement of the stimulating electrodes is between the tendons of the palmaris longus and flexor carpi radialis muscle (approximately 2 cm proximal to the wrist crease). Care should be taken to make sure the cathode (stimulating pole) is proximal to the anode in order to prevent the phenomenon of anodal blocking. In rare instances, subdermal needle electrodes may be used when there is a patient history of peripheral neuropathy, body habitus, or edema.

A square-wave monophasic pulse with a pulse width of 200–300 μs is used as the stimulus. The pulse should be delivered at a frequency of approximately 3–5 pulses per second, taking care that the exact frequency is not divisible evenly by 60 so as not to average in-line noise. The intensity of stimulation should be supramaximal. To titrate the supramaximal intensity, the current is increased stepwise until no additional increases in the amplitude of the response are measured and then 10% is added to this intensity.

Recording Parameters

Median nerve SSEPs are recorded using a peripheral, subcortical, and cortical channel. The peripheral potential is recorded with the active electrode in the ipsilateral Erb's point and referenced to the contralateral Erb's point. The resulting signal is a peak of negative polarity and a latency near 9 ms. The generator is the brachial plexus. The N9 is most useful in determining the adequacy of stimulation as well as for monitoring the brachial plexus for positional issues. The subcortical (often called cervical) potential is recorded with an electrode usually placed around the C5 vertebrae. Alternate active electrode sites include over the mastoid bone, the earlobe, and the chin. The negative peak recorded at 13 ms and the corresponding trough at 14 ms are generated by the dorsal column nuclei and caudal medial lemniscus respectively. These potentials, similar to the N9, are not affected by anesthesia and are located caudal to the tissue at risk. The cortical potential is of greatest interest during a CEA. It is most commonly recorded with the active electrode at Cpc referenced to Fpz. Some monitorists prefer a non-cephalic reference such as the contralateral Erb's point if the fast frontal EEG commonly recorded from Fpz becomes problematic. The N18 is another peak of interest. Generated by the thalamus, this peak is recorded with the active electrode at Cpi referenced to the contralateral Erb's point. The thalamic potential is supplied by the posterior circulation. Monitoring this thalamic potential may be useful in detecting ischemia resulting from the phenomenon of posterior steal where too much blood is provided to the anterior circulation from the circle of Willis at the expense of posterior perfusion.

Alarm Criteria

Alarm criteria for SSEP monitoring are well agreed upon in general. For spinal cord monitoring, the widely accepted alarm criteria are a 50% reduction in amplitude and/or 10% increase in latency. Lam et al. [16] found that a reduction of 50% or greater in amplitude proved as sensitive as EEG monitoring for monitoring carotid endarterectomy. Similar to EEG changes, a minor or moderate change in SSEPs may or may not indicate an impending neurological deficit. It is clear that if minor SSEP changes are taken as an alarm, the overall specificity of SSEP monitoring will decrease significantly (more false positives). Until more research is done to define the significance level for SSEP monitoring for carotid surgery, many monitorists are more conservative with their approach to alarm criteria and report any change that correlates with a surgical event such as clamping or unclamping as significant.

Recent work by Reddy et al. [44] found that intraoperative SSEP changes correlated with an increased risk of postoperative stroke over

30 days after surgery. The postoperative stroke risk increases in a stepwise manner with the severity of the SSEP change.

Conclusion

Carotid endarterectomy is becoming one of the most commonly monitored surgical procedures. There are many opportunities for ischemic injury during the procedure, and the surgical and anesthesia teams must walk a fine line when regulating mean arterial pressure throughout the various phases of this surgery. Prior to the advent of patient monitoring, surgeons would place an intraluminal shunt in every patient. As it became evident that the use of a shunt increases the risk of an embolic stroke, surgeons began to look for ways to select patients for shunting based on the adequacy of collateral flow. Initial techniques used for this purpose were limited to measuring carotid stump pressure during clamping and possibly continuous monitoring of cerebral oximetry. Neither of these modalities provides both a continuous and direct measure of cortical function during surgery. Later on, intraoperative EEG became standard protocol for monitoring CEA. The addition of neurophysiological monitoring to the procedure provides assurance to the surgeon that the brain is being adequately perfused during the entire procedure. Although the sensitivity and specificity of EEG monitoring is quite good, many intraoperative monitorists lacked formal training in EEG making them uncomfortable or unqualified to interpret real-time EEG data for the purposes of assessing the adequacy of collateral flow. The addition of median nerve SSEPs to the monitoring protocol provided a familiar redundancy that could be used as an adjunct to EEG monitoring. With equal (if not greater) sensitivity and specificity to EEG, SSEPs have become a mainstay for intraoperative monitoring of carotid endarterectomy. Many centers now include transcranial Doppler monitoring to measure mean flow velocity in the middle cerebral artery and to detect emboli upon clamp release. The use of TCD for measurement of flow velocity does not provide the type of direct information on cortical function that EEG and SSEPs provide. In addition, the detection of emboli has not correlated well with clinical outcome.

Review Questions

1. Discuss three differences between routine and selective shunting. What role does IOM play?
2. What is the best course of action when noting a minor change in monitoring data that correlates with cross-clamping?
3. What is posterior steal and how can it be monitored?

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