Review Article

Neuromonitoring in the Cardiopulmonary Bypass Surgical Patient: Clinical Applications
David A. Moehle, BS, CCT, CCP

United Extracorporeal Support Inc., Louisville, Kentucky

Abstract: Intraoperative multimodality neurodiagnostic monitoring utilizing a transcranial Doppler, electroencephalography (EEG), and cerebral oximetry has been shown to reduce the incidence of neurological complications greatly. Potential problems affecting the brain can be detected and resolved or managed before disastrous consequences result. The use of multimodality neuromonitoring combined with traditional hemodynamic monitoring provides the clinician with additional data to manage the cardiac/vascular surgical patient better. Because the brain has the highest blood flow and metabolic demand of any organ, it seems the ideal and most logical place to monitor the adequacy of body perfusion. This review discusses our experiences and research over the last decade with the use of multimodality EEG monitoring on cardiopulmonary bypass surgical patients. Key words: Multimodality neuromonitoring, oximetry, EEG, transcranial Doppler. JECT. 2001;33:126–134

INTRODUCTION

Cerebral complications represent the leading cause of morbidity and disability after cardiac surgery (1). Brain damage, stroke, and awareness deficits represent the single largest fraction (17%) of malpractice claims (2). Incidence of perioperative stroke in cardiac surgery is 3–5% and greater than 8.9% in patients above 75 years of age (1). Cardiac transplantation carries a risk of 50% for a cerebral event (1). Transient neurological deficits are frequently detected with comprehensive neurological testing of postcardiotomy surgical patients. Unfortunately, the source of these deficits and time of injury are generally unknown. Multimodality neuromonitoring has shown that early detection and aggressive intervention can significantly reduce perioperative cerebral deficits (3). This review discusses our experiences and research over the last decade with the use of multimodality neuromonitoring on cardiopulmonary bypass surgical patients.

NEUROMONITORING METHODS (MULTIMODALITY NEUROMONITORING)

To understand the cause and time of injury of perioperative cerebral insults better, a multimodality cerebral-monitoring program was implemented for our cardiac surgical patients (4). A cerebral oximeter (INOS 4100, Somanetics, Troy, MI) was employed that utilizes an adhesive patch containing an infrared light-emitting diode and two distal sensors on the left and right sides of the patient’s forehead. This measured regional cerebral hemoglobin oxygen saturation (rSO2 index) within the patient’s frontal cortex. Regional cerebrovenous oxygen saturation (rCOVS) was calculated from the differential signal obtained from these two sensors. Transcranial near-infrared spectroscopy enables continuous noninvasive measurement of oxy- and deoxyhemoglobin within the cerebral vasculature of the frontal cortex. Since the human skull is transparent to near-infrared light, and the brain’s hemoglobin concentration is high, cerebral oximetry is possible (3). Due to intravascular compartment volumes of the cortex normally consisting of 25% arterial, 5% capillary and 70% venous blood, the rSO2 index is a venous-weighted percentage of the oxygenated hemoglobin (3).

In addition to rCOVS, cerebrocortical function was observed by using 4-channel quantitative EEG (A-1000, Aspect Medical Systems, Newton, MA). Biopotentials were recorded with gold cup electrodes placed at the Fp1-T3, Fp2-T4, C3-O1, and C4-O2 sites of the International 10–20 electrode placement system. A nylon skullcap (Electro-Cap International, Eaton, OH) was used for the placement and securement of the EEG electrodes.

Cerebral blood flow was also noninvasively monitored...
through the right and left middle cerebral artery (MCA) by transcranial Doppler ultrasound (TCD). This was achieved with a 2MHz pulse-wave ultrasound transducer fixed above the zygomatic arch with a soft rubber holder. Because the middle cerebral arteries typically carry about 40% of the entire hemispheric blood flow, they are the single most important vessels to give an overview of cerebral perfusion. A Doppler probe was also frequently placed distal to the perfusion circuit arterial filter to monitor extracorporeal-generated emboli. A color Fourier analyzer (NeuroGuard, Medisonics, Fremont, CA) was utilized to display the flow velocity spectral profile on a monitor. Cerebral blood flow velocity was displayed on the upper edge of the velocity spectrum. Reversal of the blood flow direction was seen as an inversion of the velocity spectrum. Figure 1 illustrates the typical placement of these three neuromonitoring devices on a surgical patient.

**PATIENT MANAGEMENT USING MULTIMODALITY NEUROMONITORING**

The goal of neuromonitoring is to detect potential problems in the cardiac surgical patient management and determine an effective response. Basic problems observed were abnormalities in cerebral perfusion/oxygenation, insufficient or excess anesthetic depth, and gaseous or particulate emboli (4). This goal was achieved by continuously observing the EEG, rCOVS, and the TCD in addition to the traditional monitoring devices. Traditional monitoring in our institution consists of the electrocardiogram, arterial and pulmonary artery pressures, systemic oxygen saturation, blood gases, and standard heart–lung bypass pump flows with venous blood oxygen saturation monitoring. An intervention algorithm was devised to facilitate a standardized corrective intervention initiated by EEG slowing and/or cerebral oxygen desaturation (Table 1)(4).

The 4-channel EEG employs digital quantitative signal analyzers that were designed specifically for intraoperative use. These analyzers graphically display the frequency composition of successive brief samples of EEG activity. The analyzers also provide numerical indices to track trends in the amplitude and frequency composition of the cerebral biopotentials. The bispectral index (BIS) display on the (A-1000, Aspect Medical Systems, Newton, MA) monitor represents a complex parameter composed of a combination of time domain, frequency domain, and high-order spectral subparameters of the EEG (5). Decreasing BIS values indicates decreasing synaptic activity with increasing patient anesthesia. Patients who exhibit a BIS value of less than 65% had a 0% of surgical recall (6). Anesthetized patients with BIS value greater than 65% increased the probability of surgical recall as defined by the BIS number (6). Figure 2 illustrates the decreasing amplitude and high-frequency content of the EEG waveform in a surgical an awake patient progressing to deep anesthesia.

Assessment of the EEG before anesthesia induction is essential in order to establish baseline EEG indices. The effects of endotracheal intubation, anesthetic induction, induced hypocapnea, and surgical interventions are then observed as changes from baseline EEG indices. In patients with bilateral carotid disease and an incomplete circle of Willis, regional changes in cerebral perfusion and depressed EEG activity were occasionally seen produced by head rotation for jugular cannulation (3). Return of the head to midline position usually corrected the cerebral perfusion abnormality with the return of EEG activity to baseline.

Increasing synaptic activity perioperatively generally indicated decreasing anesthetic effect. Conversely, depression or slowing activity may signify excessive anesthesia (4).

Current trends in cardiac anesthesia utilize low dose narcotic anesthesia and an early extubation protocol (Fast Tracking)(7). Perioperative EEG monitoring provides the anesthesiologist additional information on the patient’s depth of anesthesia. Monitoring BIS values can aid the anesthesiologist in possibly reducing the patient’s total an-
esthetic drug exposure and decrease postoperative recovery time (5). Better patient care with a significant hospital cost savings is the ultimate goal.

The addition of cerebral oximetry monitoring helps distinguish between EEG slowing caused by ischemia and that of excessive anesthesia (4). Cerebral ischemia was characterized by inadequate oxygen delivery causing synaptic depression and cerebral oxygen desaturation (8). Inadequate cerebral blood flow was rapidly detected by decreasing transcranial Doppler signals followed by quickly decreasing cerebral oxygen saturation. Cerebral oxygen desaturation was defined as a rCOVS decrease >20% from precannulation baseline for greater than 3 min. The value of 20% was determined empirically from a previous study of cerebral ischemia of implantable cardioverter-defibrillator testing in normothermic unanesthetized adults (9). Absolute ideal cerebral saturation values have not currently been determined for the anesthetized adult or pediatric patients. Although, recent data by Edmonds showed the incidence of neurocomplications in patients with saturation values <40 scale units was 23%, as compared to 0.3% in those with minimum values >50 scale units (10). A 25% decline from baseline rCOVS was deemed noteworthy (10). Any decline greater than 25% resulted in loss of consciousness and neurological dysfunction (10). The Somanetics oximeter has shown to be useful as a trend monitor to detect declining cerebral perfusion resulting from progressive intracranial hypertension in both adults (11) and children (12). Cerebral desaturation was frequently seen when anemic patients were further hemodiluted by the extracorporeal circuit prime. Despite a high blood flow rate, high FiO2, reasonable arterial blood pressure, and increasing arterial CO2 content, the cerebral desaturation continued. Transfusion of packed red blood cells usually reversed the desaturation trend. Conversely, excessive anesthesia was observed by an increase in cerebral oxygen saturation and decrease in synaptic activity (EEG) secondary to the decrease in metabolic demand.

Throughout our studies, it became obvious that mixed venous saturation values measured at the perfusion circuit’s venous line did not correlate with the cerebral oximeter values (13, 14). Mixed venous saturation (i.e., 75%) measured on the extracorporeal circuit’s venous line did not always ensure adequate cerebral oxygenation or perfusion.

A wide range of perfusion abnormalities can be detected with transcranial Doppler (TCD) ultrasonography. TCD is capable of determining the existence and direction of blood flow within the ultrasonic window. The end diastolic velocity change is inversely related to change in the cerebrovascular resistance (15). End diastolic velocity is related to the same position on the arterial waveform as the end diastolic pressure. Although TCD provides valuable peak flow, diastolic flow, and mean blood flow velocity measurements, it cannot provide information on cerebral oxygen saturation or desaturation. Therefore, the addition of cerebral oximetry monitoring is essential to ensure adequate cerebral oxygenation and perfusion.

Table 1. Intervention algorithm initiated by EEG slowing and/or cerebral venous oxygen desaturation.

<table>
<thead>
<tr>
<th>Temp</th>
<th>BP</th>
<th>TCD</th>
<th>Notification</th>
<th>Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prebypass</td>
<td>0 0</td>
<td>−Peak velocity</td>
<td>Aorta obstructed</td>
<td>Adjust aortic cannula</td>
</tr>
<tr>
<td></td>
<td>0 0</td>
<td>−Diastolic velocity</td>
<td>Cava obstructed</td>
<td>Adjust venous cannula</td>
</tr>
<tr>
<td>Cardiopulmonary bypass</td>
<td>0 0</td>
<td>+Peak velocity</td>
<td>Hyperemia</td>
<td>−Pump flow</td>
</tr>
<tr>
<td></td>
<td>0 0</td>
<td>Gas emboli</td>
<td>Gas emboli</td>
<td>Deair; repair circuit</td>
</tr>
<tr>
<td></td>
<td>+ 0</td>
<td>−Peak velocity</td>
<td>Flow-metabolism uncoupling</td>
<td>+BP; −metabolic demand</td>
</tr>
<tr>
<td></td>
<td>0 0</td>
<td>−Peak</td>
<td>Cerebral flow low</td>
<td>+Pump flow/fix cannula</td>
</tr>
<tr>
<td>Postbypass</td>
<td>0 0</td>
<td>−Diastolic velocity</td>
<td>Cerebral edema</td>
<td>Ultrafiltration</td>
</tr>
<tr>
<td>Anytime</td>
<td>0 0</td>
<td>−Peak velocity</td>
<td>Dysauto-regulation</td>
<td>+BP; fosphenytoin</td>
</tr>
<tr>
<td></td>
<td>0 0</td>
<td>0</td>
<td>Excess anesthesia</td>
<td>−Anesthesia</td>
</tr>
<tr>
<td>With + EEG frequency</td>
<td>0 0, +</td>
<td>0, +Peak velocity</td>
<td>Insufficient anesthesia</td>
<td>+Anesthesia</td>
</tr>
</tbody>
</table>

0 = no change; + = increase; − = decrease; BP = blood pressure; TCD = transcranial Doppler; Temp = tympanic or nasopharyngeal temperature.

Figure 2. The above EEG tracing represents an awake patient at (1) progressing to deep anesthesia at (6). Note the reduction in high-frequency content and decreasing amplitude as anesthesia increases. (Figure courtesy of Harvey Edmonds, Jr., Ph.D.)
locity data, velocity changes alone do not necessarily indicate a serious problem. Detection of perfusion abnormalities with TCD is corroborated with the EEG synaptic depression and/or cerebral venous oxygen desaturation. Signs of inadequate anesthetic delivery can be seen by decreased cerebrovenous oxygen saturation, increased synaptic activity, and no change in TCD flow velocities (8).

Furthermore, TCD is extremely sensitive to the detection of particulate and gaseous emboli. The presence of TCD high-intensity transient signals (HITS) indicates emboli within the vessel. Figure 3 illustrates the ultrasonic signatures of gaseous (bubbles) cerebral microemboli. Particles appear as HITS that reside totally within the flow velocity spectrum (i.e., area under the white curved line outlining the waveform tracing). In contrast, the much more ultrasound-reflective air bubbles appear as full-scale high-intensity spikes. The maximal signal intensity at all velocities is attributable to amplifier saturation. Microemboli appeared as a result of laser firing during a transmyocardial revascularization. Particles presumably are the result of heat-coagulated proteins from the ventricular lining and blood in the laser beam path. Bubbles are thought to be from steam generated within the ventricular chamber by the high-power laser beam.

Flow patterns can also distinguish between aortic and caval obstructions. The effects of aortic and venous obstruction on middle cerebral artery flow velocity are shown in Figure 4. Note the major decrease in systolic (i.e., peak) flow velocity with aortic cannula malposition. In contrast, increased cerebral vascular resistance attributable to compromised venous outflow from the brain leads primarily to a diminished flow velocity during end-diastole (15). Cannula repositioning leads to an immediate normalization of flow, as shown in the left tracing. Immediate detection of hypervelocity patterns were quickly reduced to decrease the potential for an intracerebral hemorrhage. Anesthetized patient baseline Doppler tracings were used to compare and determine the existence of later hypervelocity and hypovelocity flow patterns. Excessively high peak velocities were defined as a value twice the baseline persisting for more than 3 min (4). A low diastolic velocity was defined as a velocity below the level of detection (i.e., 4 cm/sec with a 75-Hz low-pass filter) (4). During decreased perfusion, the TCD shows low flow with EEG depression and/or decreased oxygen saturation. Rapid detection of decreased flow allows time for corrective intervention before ischemic changes develop. This was seen with a malpositioned aortic cannula, occlusion of the great vessels in aortic aneurysm repair, congenital abnormalities, or during a hypotensive episode (15).

Several authors have documented the autoregulation of cerebral pressure during cardiopulmonary bypass (16,17). These studies were conducted on relatively healthy adult and pediatric patients. TCD has demonstrated that in cardiac patients cerebral autoregulation is not within a set pressure range, and its threshold may change during the course of the surgery (8). Unfortunately, each patient possesses a unique set of variables that determine cerebral autoregulation threshold for any given time (8). These variables may include the patient’s age, vascular anatomy, temperature, anesthetic depth, hypertension history, diabetes, or nicotine use (15).

Blood flow dynamics have historically been described in relation to water flow dynamics in a fixed diameter rigid pipe. To the contrary, TCD has shown blood flow dynamics possess the characteristics of “sludge” flowing through an expandable tube (18). Well known to engineers, the behavior of sludge or Bingham fluids, have their own distinct properties. An increase in sludge velocity decreases the viscosity and resistance (18, 19). The “yield pressure,” as described by engineers, is the critical pressure needed to initiate sludge flow (18, 19). Physiologically, a similar phenomenon has been observed but at higher pressures relating to autoregulation. This phenomenon called the
vascular waterfall has been seen in low-flow and low-pressure situations in pediatric hypothermic perfusions (8). The vascular waterfall describes the point at which systemic pressure falls to the yield pressure (20). Any further decrease in pressure will result in complete cessation of cerebral blood flow. This has been seen with an adjustment as small as 200 mL/min of reduction in flow and/or a 2-mmHg pressure drop (8).

TCD has also been invaluable in the management of our adult circulatory arrest/cerebral retrograde perfusion (RCP) cases. The often recommended maximum retrograde pressure of 20 mmHg or flow rate of 500 mL/min (21) has shown to be insufficient to produce ultrasonically detected bilateral flow through the left and right middle temporal cerebral arteries (22). Our experience has shown that a high flow rate up to 2 L/min and a venous pressure of 50 mmHg may briefly be required to refill the collapsed venous system and overcome venous vascular resistance to produce a detectable bilateral MCA retrograde flow (22). Figure 5 depicts two transcranial Doppler ultrasonic flow spectra flow changes in the MCA associated with circulatory arrest and the initiation of retrograde cerebral perfusion. The left panel indicates the termination of antegrade (spectrum above the baseline) perfusion, and the right panel illustrates establishment of selective low-level retrograde (spectrum below the baseline) cerebral perfusion through the superior vena cava. Despite the fact that both antegrade and retrograde pump flows were 2.0 L/min, it is clear that retrograde is less effective.

The upper trend line in Figure 6 displays changes in cerebral blood flow velocity before, during, and after cardiopulmonary bypass in a neonate. The left arrow indicates the period of selective antegrade cerebral perfusion with systemic circulatory arrest. Cerebral flow rates of 15–30 mL/kg/min are typically required in neonates to prevent cerebral oxygen desaturation (4). The arrow also points to the transcranial Doppler waveform, made pulsatile by the action of the roller pump. This nonphysiological pulsatility is compared with the patient’s native pulsatile flow as seen after bypass (right arrow).

Once adequate bilateral retrograde flow was established according to EEG, cerebral saturation, and cerebral Doppler indices, flow and pressure were decreased to the point of maintaining acceptable MCA bilateral flow and cerebral oxygen saturation. During the cooling phase, pH stat blood gas management strategy was employed. A gradient of 10°C was maintained between arterial blood temperature and bladder temperature to ensure even cerebral perfusion and cooling. Cooling was stopped as soon as the EEG became flat line. This temperature varies widely from 14–22°C (nasopharyngeal) (23). During circulatory arrest the brain was kept cold with the help of ice bags surrounding the head. In addition, tourniquets previously placed on the upper arms were expanded to reduce extracranial venous sources from the cerebral retrograde perfusion.

On select adult circulatory arrest cases, we utilized a slightly different perfusion protocol. When anticipating short periods of circulatory arrest times (i.e. >20 min) in adults, the systemic temperature is slowly dropped and maintained at 25°C (bladder temperature). A propofol bolus is given just before circulatory arrest to aid in cerebral protection by decreasing synaptic activity as described by Laylock (24). Upon commencement of circulatory arrest and retrograde cerebral perfusion, the retrograde inflow blood temperature is decreased to 18°C. Within seconds the EEG becomes flatline. This inflow temperature is maintained for the duration of the arrest time. When systemic antegrade reperfusion has been reestablished, a cold 10–15 min reperfusion time is allowed at the current systemic temperature before rewarming. The cold reperfusion technique is used to limit flow-metabolism decoupling seen when rewarming quickly following deep hypothermic arrest (25). Previous research by Kern has dem-

![Figure 5](https://via.placeholder.com/150)

**Figure 5.** The arrest TCD panel shows MCA flow at 2 L/min on CPB decreasing to no flow upon circulatory arrest. The retrograde TCD panel illustrates the commencement of retrograde cerebral flow at 2 L/min via the superior vena cava. Both the arrest and retrograde TCD tracings are 30-sec in duration. (Figure courtesy of Harvey Edmonds, Jr., Ph.D.).

![Figure 6](https://via.placeholder.com/150)

**Figure 6.** The above illustrates a TCD signature trend within the MCA before, during, and after CPB. Expanded views of selective cerebral perfusion and post bypass TCD samplings are shown in the bottom two panels. (Figure courtesy of Harvey Edmonds, Jr., Ph.D.).
onstrated that cerebral blood flow velocities should be linearly related to cranial temperature (26). Delayed EEG recovery secondary to cerebral ischemia is seen when warm-induced elevated metabolic activity is not matched with increases in blood flow velocities (27). The return of the predicted linear relationship and improved outcome were achieved by utilizing a 10–15-min period of cold reperfusion before rewarming (25, 27). Upon warming, pH-stat management is progressively weaned to the alpha-stat blood gas management for the remainder of the pump run. Attempting to rewarm quickly with high inflow blood temperatures can dramatically increase the risk of a cerebral ischemic event (8). Cerebral temperatures (tympanic) should be limited as close to 37°C as possible. Maintaining the patient at 37°C with heart ejecting on bypass for several minutes helps to ensure thorough systemic rewarming. The upper trace in Figure 7 represents a 4-hour trend plot of middle cerebral artery flow velocity in a pediatric cardiac case using a period of deep hypothermic circulatory arrest. (Note a rapid decline from baseline flow on CPB is attributable to rapid cooling that preceeds the loss of flow during circulatory arrest). Absent flow-metabolism coupling attributable to cold-induced vasoparesis was evident during rewarming. Tight coupling of cerebral blood flow with cerebral metabolism would have resulted in a temperature-related increase in flow velocity. Instead, flow remained low during rewarming. This supply-demand mismatch caused cerebral oxygen desaturation and retarded the return of EEG activity. Uncoupling was the cause of cerebral ischemia in the face of “adequate” systemic arterial pressure and pump flow (25). The TCD waveform in the bottom panel taken at the end of the case shows a highly abnormal pattern suggestive of cerebral edema (i.e., increased cerebrovascular resistance with low diastolic flow velocity).

TCD is also extremely sensitive to emboli present within the vascular lumen. Recent studies have correlated the number of HITS with postoperative evidence of neurobehavioral, pulmonary, and renal complications (28, 29). The clinical significance of low numbers of HITS is still unclear (28). However, the sudden appearance of a large number of HITS (>10,000) in conjunction with the deterioration of the EEG parameters indicates the need for immediate interventional maneuvers (15). In the case of a significant embolic event, there are several responses possible. Slight cooling will decrease the brain’s susceptibility to injury by blocking the release of excitotoxins (30). Decreased temperature also decreases the diameter of gaseous emboli facilitating easier passage through the microcirculation (15). Increased perfusion pressure and pulsatility will improve flow through unobstructed collateral circulation (26). In addition, suppression of neuronal activity with isoflurane and/or propofol may increase the neuronal tolerance to ischemia (24, 31). If available, hyperbaric therapy has shown to further facilitate the passage of the gaseous microemboli (32).

During the course of our studies, we discovered an extremely high number of HITS being produced from the extracorporeal circuit at one particular hospital. The source of the presumed gaseous microemboli was quickly traced to the venous reservoir used exclusively at that institution. The reservoir’s (BMR1500, Bentley Laboratories, Irvine, CA) design lacked a tube that gently directs the venous blood to the bottom of the reservoir like other reservoirs in our studies. Blood entering the reservoir was dropped into an open chamber mixing with room air at high flow rates. We assumed the microemboli seen were nitrogen bubbles entering the blood in the reservoir and passing through the perfusion circuit. The right Doppler trace in Figure 8 was obtained from a 4 MHz continuous wave probe placed on the perfusion circuit arterial outflow.
line between the arterial filter and the patient. The left trace represents flow within the patient’s middle cerebral artery. Emboli forming high-intensity transient signals (white spots) are apparent in both, providing evidence of an extracorporeal source for at least a portion of the cerebral microemboli. Also note the effect of pump flow change on cerebral perfusion. A step increase in pump flow velocity from 40 to 80 cm/sec (200% increase) was associated with a cerebral artery velocity change from 20 to 30 cm/sec (150% increase).

The reservoir study results for the mean total embolic load generated were: 9,894 ± 5,418 for the Bentley Model BMR 1500 (N = 32), 359 ± 406 for the Sarns Model 9438 (N = 57), and 207 ± 344 for the Terumo Capiox-SX (N = 87) reservoir oxygenator combination (p < .001) (8). It appears that venous reservoir design can effect the number of HITS produced from the extracorporeal circuit.

Recent use of vacuum-assisted venous drainage (VAVD) has raised the issue of increased gaseous microemboli generation from the extracorporeal circuit. Our preliminary observations indicate that VAVD does not create more gaseous microemboli with a low HITS generation leading to a possible cerebral event.

In the repair of complex congenital anomalies, the reduction of cerebral flow and residual air were major concerns. Figure 9 dramatically demonstrates flow changes in the middle cerebral artery associated with a stroke-in-progress. A relatively normal pulsatile waveform recorded 1 min after bypass changes into a progressively hyperpulsatile (i.e., no diastolic flow) tracing following a blast of emboli detected on the TCD. Neurological parameters soon deteriorated leading to eventual irreversible brain injury.

Neonatal and pediatric cardiopulmonary bypass carries an additional risk of cannula malposition. Venous cannula malposition results in intracranial hypertension caused by venous congestion. This was detected on the TCD spectrum as a decrease in diastolic velocity (Figure 4). An aortic cannula inadvertently positioned away from the head vessels can result in cerebral ischemia with an otherwise normal systemic pressure. Alternatively, a large portion of blood flow directed toward the head vessels may lead to a cerebral infarct. Without TCD, the EEG would still detect a problem but with a longer delay in identification leading to a possible cerebral event.

Recent trends toward minimally invasive procedures utilizing a ministernotomy, femoral-femoral cannulation, and systems such as the Heartport® increase the risk of a cerebral perfusion abnormality. Aortic dissection and occlusion of the innominate artery are concerns when using the Heartport system (Heartport, Inc., Redwood City, CA). In addition, an increased risk of arterial or venous cannula malposition is possible with ministernotomy cannulation. Off-pump coronary artery bypass (OPCAB) surgery pose a challenge for the anesthesiologist in managing hypotension and hypertension episodes resulting from heart manipulation. Decreased cerebral perfusion and hypotension resulting from heart manipulation may cause cerebral ischemia and/or synaptic depression. Successful corrective management of these problems requires coordination between the surgeon, anesthesiologist, perfusionist, and neuromonitoring technicians. Repositioning of vascular clamps, adjusting cannula positions, increasing or decreasing pump flows, increasing arterial pCO₂, and additional deairing maneuvers are frequently recommended by our neuromonitoring technicians.

**PATIENT OUTCOMES**

Assessment of neurological deficits incurred perioperatively were based on changes from preoperative and postoperative neurodiagnostic testing. Testing included short-term memory, long term memory, eye movements, visual identification of objects, letter cancellation test, reading, and response to commands. Antisaccadic eye movements revealed frontal and parietal cortical deficits (33, 34). The antisaccadic eye movement test involves the Conscious movement of the eyes laterally in the direction opposite to a presented cue. In addition, the Folstein Mini-Mental Status Exam includes the assessment of orientation and cognition (35). Saccadic eye movements test the function of the extraocular muscles, cranial nerves, and brainstem oculomotor pathways (36). A 10% decline from preoperative baseline testing was defined as noteworthy (8).
Primitive reflexes and ophthalmologic abnormalities were the most common new postoperative neurological deficits seen (8).

The benefits of neuromonitoring were plainly evident. Of 250 pediatric patients monitored in one study, a significant change in cerebral perfusion or metabolism was detected in 176 (70%) of them (4). A perfusion imbalance comprised 37% of the problems, and 58% of the problems were cerebral oxygen desaturation (4). Pediatric patients whose care was guided by multimodality neuromonitoring had one-fourth the neurologic complications of those without neuromonitoring (4). In this study, the perfusionist was directly involved in 52% of the intervention adjustments. The survivor’s median length of stay was 6 days in the no change and intervention groups, as compared to 9 days in the no-intervention group (4). Additional convincing data came from a study that compared 900 monitored adult surgical patient outcomes to the outcomes of 209 unmonitored surgical patients in the same hospital over the same time interval (37). Major neurological sequelae appeared immediately postoperatively in 2.8% of the unmonitored and 0.3% of the monitored patients. Only two of the monitored patients experienced a neurological complication without a noteworthy intraoperative EEG change. Unmonitored CABG and/or valvular repair patients were nine times as likely to experience a neurological complication, as compared to monitored patients (37).

Research in 1996 by Ganzel and associates studied the effects of RCP on the patient’s recovery, as compared to non-RCP circulatory arrest patients (22). The influence of RCP was very apparent in the early postoperative period. Patients managed with RCP regained consciousness more quickly than those with arrest only. No patients in the arrest group were awake and alert within the first 12 postoperative hours, as compared to 81% in the RCP group. Furthermore, 42% of the RCP group were extubated within 18 hours versus 13% of those without RCP. Thirty-two percent of the RCP group was discharged from the ICU within 24 hours, as compared to 0% without RCP.

CONCLUSIONS

An aggressive interventional neuromonitoring protocol has shown to improve patient outcomes by quickly detecting, limiting, and/or reversing intraoperative cerebral insults. Otherwise, an undetected intraoperative cerebral deficit could cause a catastrophic patient outcome.

Individually, each of these three neuromonitoring devices is limited in the diagnostic information they provide. When combined, with the addition of traditional systemic hemodynamic and electrocardiographic modalities, they provide a more detailed and accurate picture of the patient’s physiological status. Retrospective data analysis of multimodality neuromonitored patients has shown to reduce the incidence of an expensive cerebral complication benefiting both the patient and the hospital (37). Because the brain has the highest blood flow and metabolic demand of any organ, it seems essential to monitor the brain in addition to traditional modalities to adequately assess total body perfusion and oxygenation.

Venous reservoir emboli research data affirms the belief that perfusion circuit selection should not be based solely on cost, but on overall product performance and safety. Randomized prospective neuromonitored studies are currently being designed to study monitoring techniques and patient outcomes further. Simplified user-friendly neuromonitoring systems are being designed by manufacturers to be easily used by surgical health-care professionals.

The average hospital’s direct cost per neurological complication was reported as approximately $15,000 (10, 37). Neuromonitoring charges per patient, as stated by Pollock were $375/patient (37). The average hospital charge for an average monitored patient was $3,460 less than that of the average unmonitored patient (37). Obviously, these data justify the additional cost incurred for neuromonitoring and patient safety. Unfortunately, most hospital administrators and physicians are reluctant to support a cardiac neuromonitoring program because of lack of current monitoring knowledge. Widespread assumptions consider neuromonitor-guided protective measures futile because of the etiology of cerebral emboli (8). Empirically derived protocols historically used for neurological management fall short of ensuring adequate patient safety. In today’s managed care arena, we as health-care professionals, must demonstrate new methods to increase the patient’s standard of care without increasing the hospital’s total cost.

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REFERENCES


