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Multi-Modality Neurophysiologic Monitoring for Cardiac Surgery

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ABSTRACT

Background: A high percentage of patients who undergo cardiac surgery experience persistent cognitive decline. The costs to insurers from brain injury associated with cardiac surgery is enormous. Furthermore, the same processes that injure the brain also appear to cause dysfunction of other vital organs. Therefore, there are great clinical and economic incentives to improve brain protection during cardiac surgery. This article discusses the methods of monitoring neurophysiologic function during heart surgery, including electroencephalography (EEG), near-infrared spectroscopy (NIRS), transcranial doppler (TCD) ultrasound, and cerebral oximetry, and analyzes the effectiveness of multi-modality neuromonitoring.

Methods: Neurophysiologic studies have implicated hypoperfusion and dysoxygenation as major causative factors for brain injury during cardiac surgery. Since these functional disturbances are often detectable and correctable, there is a new impetus to examine the role of neurophysiologic monitoring in brain protection. We have used a retrospective, single-surgeon case-control study to examine the influence on outcome following myocardial revascularization of multi-modality neuromonitoring, with modalities that include 4-channel EEG, bilateral cerebral oximetry, and single channel TCD.

Results: The majority of noteworthy functional disturbances detected by neuromonitoring can be corrected by simple adjustments in perfusion, oxygenation, or anesthetic administration. In more recalcitrant cases, pharmacological neuroprotection has proven effective. In addition to the substantial reductions in length of hospital stay, costs, and neurologic complications, the results of neuromonitoring suggest a possible benefit to other vital organ systems. Future studies of neuromonitoring efficacy should not overlook these important accessory benefits.

Conclusion: This study provides the clearest evidence to date that multi-modality neuromonitoring for cardiac surgery is safe, clinically beneficial, and cost-effective. Although neuromonitoring involves negligible risk and modest costs, it's benefits for patient outcome and cost control are substantial.

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INTRODUCTION

Nearly half of the one million patients worldwide undergoing cardiac surgery each year will experience persistent cognitive decline [Newman 2001]. The direct annual cost to insurers for brain injury from just one type of cardiac surgery, myocardial revascularization, is estimated at $4 billion [Roach 1996]. Furthermore, the same processes that injure the brain also appear to cause dysfunction of other vital organs. Therefore, there are enormous clinical and economic incentives to improve brain protection during cardiac surgery.

Historically, there has been little enthusiasm for neurophysiologic monitoring during cardiac surgery because of the presumed key role of macroembolization. It is widely assumed that most adult cardiac surgery brain injury results from cerebral embolization of atheromatous or calcified material dislodged from sclerotic blood vessels during their manipulation. Until the emergence of coronary artery bypass grafting (CABG) without cardiopulmonary bypass (CPB), the so-called "off-pump" CABG (OPCAB), this injury was thus viewed as unavoidable.

Recent developments have begun to alter this perception. First, despite reductions in both aortic manipulation and cognitive decline with OPCAB surgery [Kilo 2001], brain injury still occurs. Second, neurophysiologic studies have implicated hypoperfusion [Edmonds 1996] and dysoxygenation [Edmonds 2000] as major causative factors. Since these functional disturbances are often detectable and correctable, there is new impetus to examine the role of neurophysiologic monitoring in brain protection.

Neuromonitoring Techniques

Electroencephalography (EEG)

EEG monitoring for ischemic detection has been performed since the first days of cardiac surgery with CPB [Theye 1956]. However, this long experience has not developed into a broad experience. In contrast to its widespread use during carotid endarterectomy, EEG monitoring for cardiac surgery is generally performed only in a few academic centers. The low usage seems to be based on several common perceptions, or perhaps misperceptions.

First, earlier reports described the technical difficulty or impossibility of EEG recording during CPB because of artifactual contamination, deep anesthesia, and brain cooling [Levy 1992]. Fortunately, modern perfusion and anesthetic practice have generally eliminated each of these sources of artifact. Troublesome roller pumps have been replaced with either centrifugal pumps or no pump at all. Moderate-to-deep hypothermia is now usually applied only in aortic arch reconstruction or other procedures that may require a period of circulatory arrest. Fast track anesthetic protocols rely on much lower anesthetic doses and avoid the near-obliteration of EEG activity that was seen with earlier high dose techniques.

Second, neurologists trained in diagnostic EEG have often advocated 16-channel recordings to identify focal ischemic changes [Blume 1999]. This requirement substantially adds to the complexity and cost of intraoperative monitoring by requiring the added presence of a neurophysiologist or technologist. However, Craft et al. [Craft 1994] showed that a 4-channel recording performed as well in identifying clamp-related ischemic changes during carotid endarterectomy. In support of this observation, during 2000-2001 we relied on a 4-channel computer-processed EEG monitor designed for intraoperative use to record 759 adult and pediatric cardiac surgery patients [Sehic 2002]. As in our earlier 1999-2000 experience [Ganzel 2002], noteworthy EEG changes prompting change in patient management occurred in 22% of the cases. A new neurologic deficit upon initial recovery from anesthesia without intraoperative EEG change occurred in only one case. Thus, the 4-channel EEG failed to detect developing brain injury in only 0.14% of the cases.

Third, the techniques that transform EEG information into actual brain injury prevention are not well described. However, our recent evidence-based studies offer some suggestions. Of the 22% noteworthy EEG changes in our 2000-2001 cardiac surgery database, all but 6% were corrected by simple adjustments in perfusion, oxygenation, or anesthetic administration. In the remaining 44 cases with resistant EEG abnormalities, we recommended a neuroprotectant cocktail composed of fosphenytoin 15 mg/kg plus methylprednisolone 10 mg/kg. The recommendation was based on both laboratory [Chan 1998, Edmonds 2001] and clinical [Schurr 2001] studies. Neuroprotection was instituted in 34 cases and all treated patients experienced an uneventful recovery. In contrast, of the 10 patients with EEG abnormalities that did not receive the cocktail, 90% awoke with new neurologic deficits ($p < .001$). These results illustrate both the neuroprotectant potential of the cocktail and the predictive value of persistent EEG changes in the absence of pharmacologic neuroprotection.

The EEG is exquisitely sensitive to deficiencies in either cerebral oxygenation or perfusion. Our results confirm the many earlier observations that the EEG is very effective in identifying regional physiologic imbalance. However, many changes are diffuse and may thus reflect the benign influence of general anesthesia rather than potentially injurious hypoperfusion or dysoxygenation. The EEG is sensitive to altered synaptic function, but it is not specific for pathology. Additional physiologic monitoring is needed to overcome this limitation. There are two available technologies for this purpose that are well-suited to surgical monitoring—near-infrared spectroscopy and ultrasound.

Near-infrared Spectroscopy (NIRS)

NIRS is familiar to all operating room personnel because of the ubiquitous presence of pulse oximetry. Since the skull is transparent to near-infrared light, a similar approach can be used to measure the intravascular oxyhemoglobin fraction in a small sample of cerebral cortex. The unique infrared absorption spectra of oxy- and deoxyhemoglobin also permit calculation of changes in total hemoglobin. In the absence of sudden hemodilution, total hemoglobin change is proportional to change in blood volume. Therefore, this simple non-invasive device can provide useful clinical information regarding both cerebral cortical oxygenation and perfusion.

Exclusive measurement of brain tissue is accomplished by means of an infrared light source and two sensors. The mean path of infrared light is elliptical as it passes from the skin through the skull, the underlying brain, and back again to the skin at a distant point. Some of these photons are reflected by the skull to the surface at a very acute angle. This extracranial reflection is detected by a sensor located closer to the light source. Light traveling to the deeper cortex is reflected to the surface at a less acute angle and is detected by a more distant sensor. Thus, the difference signal measures brain tissue with minimal extracranial contamination.

To avoid artifactual contamination of the cerebral signal by extraneous light, the infrared source and detectors are held in place on non-hairy skin by an opaque adhesive patch. In general, this requirement limits sensor placement to the forehead above each eye. This location permits monitoring of the perfusion-sensitive watershed region between the anterior and middle cerebral artery distributions of each hemisphere.

Pulse oximetry relies on pulsation to measure oxygen saturation within the arterial circulation. In contrast, cerebral oximetry examines all reflected light, both pulsatile arterial and non-pulsatile venous. Since about 75% of the cerebral blood is in the venous compartment, the signal represents a venous-weighted average. Without a requirement for pulsatility, cerebral oximetry can continue to monitor brain oxygenation during both CPB and circulatory arrest.

We established the threshold value for intervention during tilt-table studies of conscious patients with an implanted cardioverter-defibrillator (ICD) or those complaining of syncope [Singer 1998]. Function of the ICD was tested by induction of ventricular fibrillation. Thus, we examined the magnitude of brain oxygen desaturation accompanying syncope from either ventricular fibrillation or neurally mediated hypotension. Unconsciousness, signifying clinically important cerebral ischemia, was always accompanied by at least a 20% decline in brain oxygen saturation.

Several groups have now shown that changes in cerebral oxygenation may be important in brain injury prevention [Yao 1999, Edmonds 2000, Madsen 2000]. In our recent studies previously cited, we observed clinically significant desaturation in slightly over half of the monitored myocardial revascularization cases. All but 7% of these desaturations were corrected by the simple interventions described below in Table 1 (◉).

Table 1. Neuromonitoring-based Intervention Algorithm for Cardiac Surgery.

Stage	EEG	Temp	BP	TCD	CVOS	Problem	Intervention
Anytime	–	0	0	0	+	anesthesia excess	– anesthetic
	+	0	0,+	0,+	–	inadequate anesthesia	+ anesthetic
	–	0	–	–	–	dysautoregulation	+ BP, flow
Pre-bypass	–	0	0	– sys	–	aortic cannula malposition	reposition cannula
	–	0	0	– dia	–	venous cannula malposition	reposition cannula
Bypass onset	–	0	0,–	0	–	pump prime transient	none
During bypass	–	0	0	HITS	–	embolization	retrograde brain perfusion
	–	–	0	–	0	flow-metabolism coupling	none
	–	+	0	0	–	flow-metabolism uncoupling	+ anesthetic, relaxant
After bypass	–	0	0	– dia	–	cerebral edema	ultrafiltration
Decannulation	–	0	0	HITS	–	particulate embolization	fosphenytoin, + BP
		–		HITS		gas embolization	hyperbaric chamber
ICU	–	0	0	HITS	–	thromboembolization	anti-platelet therapy
	–	+	0,–	0	–	hypermetabolism	O_2 supplement

Abbreviation key: (+) = increase; (–) = decrease; (0) = no change; (sys) = systolic; (dia) = diastolic; EEG = electroencephalography; Temp = nasopharyngeal temperature; BP = mean arterial blood pressure; TCD = transcranial Doppler middle cerebral artery mean flow velocity; CVOS = cerebral venous oxygen saturation; HITS = transcranial Doppler High-intensity Transient Signals (e.g., emboli); ICU = intensive care unit.

Transcranial Doppler (TCD) Ultrasound

Relying on the Doppler principle, the frequency shift of ultrasonic echo reflections from moving red blood cells can be used to calculate the direction and velocity of their movement. In most patients, this determination can be made on large intracranial arteries and veins through paper-thin regions of temporal bone called ultrasonic windows. Since the velocity and flow of red cells through blood vessels are influenced in different ways by vessel diameter, TCD does not provide a direct measure of cerebral blood flow. However, in the absence of hemodilution, *change* in TCD velocity does correlate closely with *change* in blood flow. Sudden large change in velocity or direction is readily detected by continuous TCD monitoring.

Tilt-table testing allowed us to establish the clinical significance of velocity changes. Syncope resulting from systemic hypotension was accompanied by a mean velocity decline of >70% and the absence of diastolic flow. This finding formed the basis of our TCD-based intervention threshold whereby velocity reductions of greater than 70% prompt attempts to enhance cerebral blood flow. These measures include pump flow increase, pressor administration, blood volume increase, and elevation of arterial carbon dioxide tension.

Both gas bubbles and particulate matter reflect sound much better than red blood cells. Thus, TCD can provide a quantitative description of embolic processes—high-intensity transient signals (HITS)—their magnitude and time course. Information on the presence of cerebral microemboli is useful for individual patient management (for instance, repair of an air leak) and general improvement of perfusion technique. For example, between 1993 and 2001, we reduced the average aggregate number of HITS per CABG case from 10,000 to 86. HITS reduction strategies included the introduction of well-designed venous reservoirs and membrane oxygenators, elimination of the partially occluding aortic clamp for proximal graft anastomosis, and the discontinuation of cardiotomy suction re-infusion.

Multi-Modality Neuromonitoring and Outcome

Most clinical studies of neurophysiologic monitoring have concentrated on process variables, such as describing occurrences of ischemic EEG changes, cerebral oxygen desaturation, or number of HITS. Thus, there is as yet no single, adequately powered prospective randomized study demonstrating either the clinical or economic benefit of neurophysiologic monitoring. The primary impediment to these studies is financial, since the cost of a single such study would substantially exceed $1 million.

The next best alternative is a retrospective, single-surgeon case-control study. This design eliminates the variation introduced by multiple surgeons and minimizes systematic group heterogeneity. We have used such a study design to examine the influence of multi-modality neuromonitoring (i.e., 4-channel EEG, bilateral cerebral oximetry, and single channel TCD) on outcome following myocardial revascularization [Ganzel

Table 2. Effect of Neuromonitoring on CABG Outcome.

	Monitored	Unmonitored	p value
Number (N)	78	386	
Hospital stay (days)	6.6	9.0	.02
Neurologic complication (%)	0.0	6.2	.05
Ventilator support >24 hr (%)	5.1	11.6	.09
Pulmonary complication (%)	6.4	9.3	.41
Renal complication (%)	2.6	4.9	.37
Death (%)	2.6	3.6	.64
30-day readmission (%)	5.1	8.7	.29

Monitored group hospital charges 11% less (p = .03)

2002]. The only neurophysiologic monitoring used in the "unmonitored" (UNMON) group was a single-channel EEG bispectral index (BIS) monitor of anesthetic effect. A standardized intervention algorithm (Table 1, ◎) was used in the monitored group to correct physiologic imbalances. Results of the outcome study are shown in Table 2 (◎).

In addition to the substantial reductions in length of hospital stay, costs, and neurologic complications, the results suggest a possible benefit to other vital organ systems. This is not unexpected, since the same processes that injure the brain may also injure other organs. Future studies of neuromonitoring efficacy should not overlook these important accessory benefits.

CONCLUSION

This study provides the clearest evidence to date that multi-modality neuromonitoring for cardiac surgery is safe, clinically beneficial, and cost-effective. Since the risk of neuromonitoring is negligible, the costs modest, and the benefits substantial, it is becoming increasingly difficult to justify a failure to use it.

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