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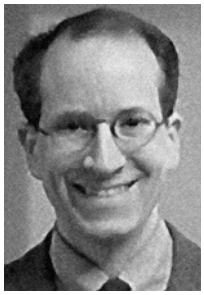
A Method for Identifying Mechanisms of Neurologic Injury from Cardiac Surgery



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ABSTRACT

Background: A method for linking discrete surgical and perfusion-related processes of care with cerebral emboli, cerebral oxyhemoglobin desaturation, and hemodynamic changes may offer opportunities for reducing overall neurologic injury for patients undergoing cardiac surgery.

Methods: An intensive intraoperative neurologic and physiologic monitoring approach was developed and implemented. Mechanisms likely to produce embolic (cerebral emboli), hypoperfusion (oxyhemoglobin desaturation), and hypotensive (hemodynamic changes) neurologic injuries were monitored and synchronized with the occurrence of surgical and perfusion clinical events/techniques using a case video.

Results: The system was tested among 32 cardiac surgery patients. Emboli were measured in the cerebral arteries and outflow of the cardiopulmonary bypass circuit among nearly 75% and 85% of patients, respectively. Oxyhemoglobin desaturation was measured among nearly 70% of patients. Hemodynamic information was recorded in 100% of patients.

Conclusions: We developed and successfully implemented a method for detailed real-time associations between processes of clinical care and precursors of neurologic injury. Knowledge of this linkage will result in the redesign of clinical care to reduce a patient's risk of neurologic injury.

INTRODUCTION

Neurologic complications including global encephalopathy and focal neurologic syndromes have long been reported following cardiac surgery. Pre-, intra-, and postoperative risk factors associated with these deficits have been previously described [Likosky 2003a]. The most commonly reported mechanisms for these injuries are embolism, hypoperfusion, and hemodynamic or metabolic derangements [Likosky 2003b].

In the course of actual clinical care, only symptomatic clinical outcomes are routinely detected. In a study using magnetic resonance imaging to detect new brain lesions after coronary artery bypass grafting (CABG) surgery, Goto and colleagues found that among patients with multiple infarctions 59% were asymptomatic [Goto 2001]. Work done by Newman and colleagues suggests that patients undergoing CABG surgery are at high risk for both short-term (50% at discharge, 24% at 6 months) and long-term (42% at 5 years) cognitive deficits, which are often called subclinical because they have a subtle clinical presentation and thus are not customarily detected through clinical care [Newman 2001]. Intraoperative monitoring studies have identified an association between increased risk of neurologic deficit (whether subtle or overt) and embolization, hypoperfusion, and systemic hypotension [Pugsley 1994, Gold 1995, Edmonds 2000].

Although it is interesting and important, much of the literature surrounding neurologic injury has lacked critical information. Most research has focused on the association between nonmodifiable preoperative risk factors (such as age and gender) and neurologic injury. The clinical outcome usually studied (stroke or cognitive deficit) is too far removed from the process of care to be actionable. Sustainable reduction of neurologic injury requires the identification of the associations between potentially modifiable intraoperative variables and the detection of precursors to neurologic injury.

The Northern New England Cardiovascular Disease Study Group (NNECDSG) is a voluntary research consortium, composed of clinicians, research scientists, and hospital administrators, representing all medical centers in Maine, Vermont, and New Hampshire where CABG surgery is performed. Since 1987 the NNECDSG has maintained a prospective registry of all patients undergoing cardiac surgery in the region. The group fosters continuous improvement in the quality of care of patients with cardiovascular disease in the region through the pooling of process and outcome data and the timely feedback of data to clinicians.

We describe an approach recently developed and implemented at a single medical center for associating intraopera-

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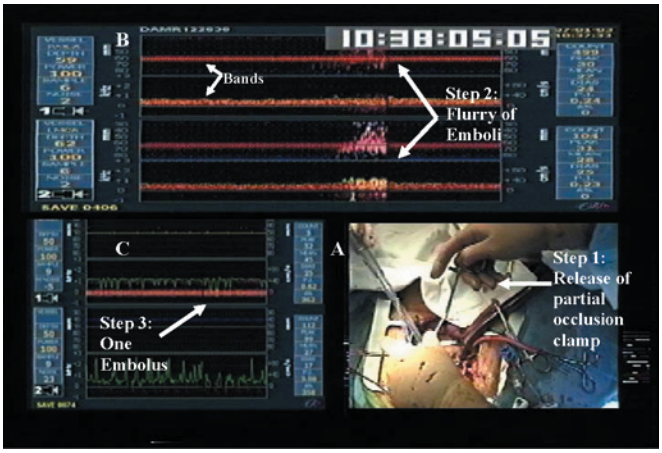

Figure 1. Surgical event/technique: release of partial occlusion clamp. A, Release of the partial occlusion clamp after the completion of the proximal anastomoses (step 1). B, Emboli and blood flow velocity in the right (top 2 bands) and left (bottom 2 bands) middle cerebral arteries. An embolus shows up in both bands because the embolus reflects ultrasound more strongly than the surrounding blood (step 2). C, Emboli and blood flow velocity through the cardiopulmonary bypass circuit (step 3). One embolus is noted.

tive clinical techniques with the onset of emboli, oxyhemoglobin desaturation, and hemodynamic changes.

MATERIALS AND METHODS

The study protocol received approval by the participating institution's internal review board, and informed consent was received from all study patients.

We developed and implemented an intensive intraoperative monitoring approach among 32 patients undergoing cardiac surgical procedures performed at one of our participating medical centers, from October 2002 until November 2003. Patients undergoing cardiac surgery procedures, either coronary (CABG or off-pump) or valvular (CABG/valve replacement or aortic valve replacement/mitral valve repair) were recruited for this study. The aim of this study was to develop and implement a method for linking surgical and perfusion events/techniques with precursors of neurologic injury (Figure 1).

This approach enabled us to associate information regarding discrete clinical events/techniques (Tables 1-3) with measurements of (1) cerebral changes in blood flow and oxyhemoglobin desaturation using near-infrared spectroscopy, (2) emboli as detected via transcranial Doppler ultrasonography of the cerebral arteries and the outflow of the cardiopulmonary bypass circuit, and (3) systemic hemodynamic changes. Equipment used to monitor these parameters was placed in 1 of 2 surgical suites 1/2 hour prior to sternal incision, and surgical case videos were created intraoperatively for each patient. Instrumentation was conducted in parallel to other preoperative monitoring. A digital camcorder, attached to a tripod, recorded audio and video signals during the surgical case. The videotape was aimed at the surgical field to capture surgical events/techniques. Signals from all devices (cerebral blood flow velocity and emboli, oxyhemoglobin desaturation, and hemodynamics) along with digital output from the camcorder were coordinated in several ways. First, all devices were synchronized via a timestamp. Second, a video was created that had the synchronized outputs from the Doppler signal of the cerebral arteries and cardiopulmonary bypass circuit, as well as the surgical video signal. Images were combined onto one screen in real time using a video splitting device (Keywest Technology, Lenexa, KS, USA).

Emboli, oxyhemoglobin desaturation, and hemodynamics were measured on each patient. Blood flow velocity and emboli were measured every 8 milliseconds in the cerebral arteries and cardiopulmonary bypass circuit using transcranial Doppler technology [Moehring 2002]. Doppler probes were

Table 1. Surgical and Perfusion Techniques Collected via Monitoring Approach

Surgical	Perfusion
Sternotomy	Injection of medication into cardiopulmonary bypass circuit
Application of net around heart	Venous line air
Aortic purse strings	Injection of medication
Aortic cannulation and decannulation	Blood sampling
Venous cannulation and decannulation	Occurrence of a low venous reservoir alarm
Onset and cessation of cardiopulmonary bypass	Method of venous drainage
Cardioplegia cannulation	
Aortic palpation and manipulation	
Use of cardiopulmonary suction	
Application and removal of aortic cross clamp	
Application and removal of partial occlusion clamp	
Distal anastomotic technique	
Proximal anastomotic technique	
Reapplication of aortic cross clamp	
Defibrillation	
Application of sternal wires	

Table 2. Key Parameters Collected via Monitoring Approach

Parameter	Frequency of Data Collection
Patient physiologic	
Arterial blood pressure	20 sec
Central venous pressure	20 sec
Pulmonary artery pressure	20 sec
Coronary sinus pressure	Intermittent
Nasal pharyngeal temperature	20 sec
Bladder temperature	20 sec
Cardiopulmonary bypass	
Flow rate	20 sec
Arterial line pressure	20 sec
Venous reservoir pressure	20 sec
Arterial blood temperature	20 sec
Venous blood temperature	20 sec
Inline mixed venous oxygen	20 sec
Inline arterial blood gases	20 sec
Alarm conditions—level sensor, blood sensor, over pressure	20 sec
Cerebral monitoring	
Right hemisphere oxygen saturation	20 sec
Left hemisphere oxygen saturation	20 sec
Right hemisphere embolism/blood flow velocity	8 msec
Left hemisphere embolism/blood flow velocity	8 msec

secured to the patient prior to the induction of anesthesia to identify baseline measurements. Bilateral monitoring of cerebral blood flow velocity and emboli was attempted using the TCD 100M Digital Transcranial Power M-Mode Doppler (Spencer Technologies, Seattle, WA, USA). Once temporal windows were identified, a head-mounted frame was secured on the patient. In order to identify the contribution of the cardiopulmonary circuit to emboli identified in the brain, Doppler probes were affixed on the inflow and outflow of the circuit. With regard to the hypoperfusion mechanism, we recorded bilateral, when possible, regional oxyhemoglobin desaturation (rSO_2) in the frontal cortex at 20-second intervals using the INVOS Cerebral Oximeter (Somanetics, Troy, MI, USA). Patches were placed in the frontotemporal region above the eyebrows. With regard to hypotension, we collected hemodynamic and perfusion data every 20 seconds using a data capturing interface (Stockert-Shiley, Munich, Germany). Hemodynamic data included heart rate, mean arterial pressure, pulmonary artery pressure, and central venous pressure. We also captured information regarding line pressure and degree of vacuum-assisted venous drainage using the same perfusion data-capturing device.

All analyses were performed using the STATA 8.0 program (Stata Corporation, College Station, TX, USA) and SAS 8e Release 2 (SAS Institute, Cary, NC) [SAS Institute 2001, Stata 2003]. Values of each measurement were collapsed to 1-minute intervals. Embolism data were summarized as either median counts or natural log of counts, and other measurements were summarized as means. Associations between discrete clinical events/techniques and each monitored parameter were made through both the surgical case video and patient-level report.

Patient-level reports contained 3 figures (mean arterial pressure, bilateral cerebral embolism count, and bilateral cerebral oxyhemoglobin desaturation) (Figure 2). Physiologic limits were placed at <50 mm Hg on the mean arterial pressure figure, as were 75% of baseline rSO_2 on the right

Table 3. Embolic Signal Counts, Cerebral Oxyhemoglobin Desaturation, and Hemodynamic Changes by Procedure*

	Coronary (n = 28)	Valvular (n = 4)
Left hemisphere cerebral embolic signal counts		
No. of patients	22	3
Counts	448 (117, 698)	1145 (1049, 5783)
Right hemisphere cerebral embolic signal counts		
No. of patients	20	4
Counts	439 (106, 793)	931 (527, 4269)
Perfusion outlet embolic signal counts		
No. of patients	22	4
Counts	840 (275, 1441)	2013 (657, 5150)
Left hemisphere cerebral oxyhemoglobin desaturation (<75% baseline)		
No. of patients	19	3
No. of episodes	1 (1, 1)	1 (1, 14)
Right hemisphere cerebral oxyhemoglobin desaturation (<75% baseline)		
No. of patients	19	3
No. of episodes	12 (4, 18)	3 (2, 13)
Mean arterial pressure (<50 mm Hg)		
No. of patients	28	4
No. of episodes	42 (12, 62)	34 (18, 64)

*Data are expressed as n or median (25th percentile, 75th percentile).

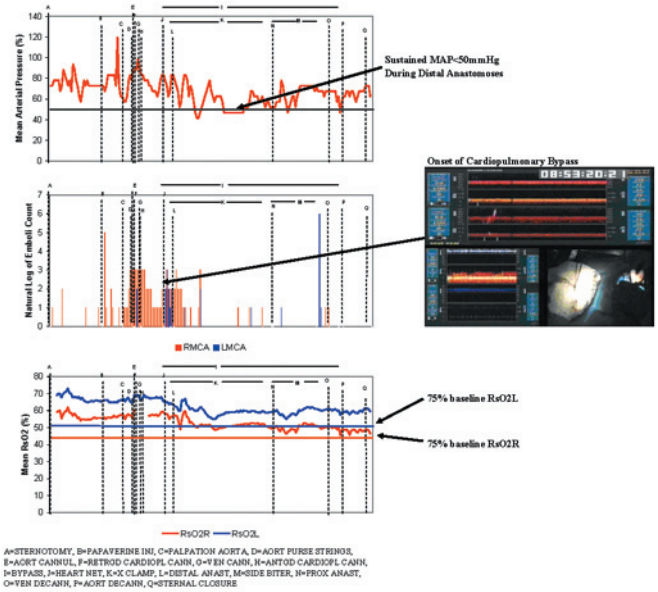

Figure 2. Patient level surgical report. Proposed mechanism: the patient's aorta was assessed with an epiaortic echocardiography probe. Upon inspection, mild diffuse localized thickening of 2 mm on the posterior wall of the aorta was found. Upon removal of the partial occlusion clamp, debris traveled via the common carotid arteries and subsequently into the right and left middle cerebral arteries. A total of 151 embolic signals were detected within 3 minutes of the removal of the partial occlusion clamp.

and left hemispheres in the cerebral oxyhemoglobin desaturation values. Each of the 3 figures shares a common time axis (abscissa), beginning at sternal incision and ending with sternal closure. Vertical and horizontal bars were placed on each of the graphs to depict the occurrence of discrete processes of care. A legend in the footnote of the report is used to reference processes of care. This report enables the user to identify hemodynamic changes, number of cerebral emboli, and cerebral oxyhemoglobin desaturation with each of the studied processes of care, as well as associations between each of these measurements at any particular point in the operative case.

RESULTS

We implemented our monitoring approach on 32 patients undergoing cardiac surgery (28 coronary, 4 valvular). We captured information on cerebral emboli/flow velocity in 27 (22 bilaterally) patients, perfusion emboli in 26 (2 off-pump cases) patients, cerebral oxyhemoglobin desaturation in 23 (22 bilateral) patients, hemodynamics in 32 patients, and video of the surgical site in 32 patients. The 2 predominant reasons for not having Doppler signals were data acquisition errors or the inability to identify a transtemporal window. The most common reason for lack of oxyhemoglobin desaturation signals was data acquisition errors. We have subsequently changed our protocols and worked with the manufacturers of the products used in this study to improve the data acquisition rate. Synchronization of signals was attained in all. This approach has enabled us to identify variation in embolic signal counts, cerebral oxyhemoglobin desaturation, and hemodynamics by patient and procedure. Cerebral embolic signals, regardless of cerebral hemisphere, were highest among coronary patients. Embolic signals were nearly equivalent (among coronary patients) or greater (among valvular patients) in number as counted in the cardiopulmonary bypass circuit versus in the cerebral arteries. Few episodes of regional cerebral oxygen desaturation were noted regardless of type of surgery. Numerous episodes of hypotension were noted among patients, with slightly more occurring among patients undergoing coronary procedures.

Surgical case videos and patient-level reports were created for patients in order to associate processes of care with embolic signal counts, cerebral oxyhemoglobin desaturation, and hemodynamic changes (see movie). As an example (Figure 2), systemic hypotension and cerebral desaturation were noted during the anastomosis of the distal arteries. The highest number of detected cerebral emboli occurred during the removal of the partial occlusion clamp. There were 70 and 151 cerebral embolic signals detected within 3 minutes after the onset of cardiopulmonary bypass (with gravity venous return) and after the removal of the partial occlusion clamp, respectively.

DISCUSSION

We developed and implemented a method for linking in real-time surgical and perfusion-related events/techniques with precursors of neurologic injury. Variations in each of these precursors, as noted in the exam patient-level report (Figure 2) among patients suggest that their occurrence is not inevitable. The redesign of clinical care for reducing these precursors as well as the resulting neurologic injury requires capturing detailed information describing current care processes.

The relationship between neurologic injury and process of care is complex. Our choice of metrics for our monitoring studies is based on an understanding of the principle etiologic mechanisms producing neurologic injuries subsequent to cardiac surgery [Likosky 2003b]. Real and sustainable improvement in the incidence of neurologic injury after cardiac surgery requires the identification of the discrete clinical events/techniques resulting in precursors to neurologic injury and the redesign of clinical care to reduce the incidence of these intraoperative injuries. The approach outlined above offers a mechanism for linking continuously monitored intraoperative cerebral and systemic measurements to discrete clinical events/techniques. Information from this ongoing study is critical to the redesign of clinical care.

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