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NEWSLETTER

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SPECIAL EDITORIAL

Cerebral Perfusion: Err on the Side of Caution

As controversy continues regarding the hemodynamic management of patients in the head-up or beach chair position, the **APSF Newsletter** *turns to Dr. William Lanier for editorial perspective. Dr. Lanier is Editor-in-Chief of* **Mayo Clinic Proceedings** *as well as a highly regarded neuroanesthesiologist and neurophysiology investigator.*

In the Summer 2007 issue of the *APSF Newsletter*, Cullen and Kirby reported on 2 patients in whom a catastrophic, new-onset brain injury was discovered after surgery in the beach chair (barbershop) position.[1] The authors presented views on the effect that blood pressure monitoring and management may have had on neurologic injury and provided a formula for correcting hydrostatic blood pressure gradients from the site of measurement to the site of vulnerable brain tissues. This publication generated a series of letters to the *Newsletter*, either supporting or challenging the need for the blood pressure corrections suggested by Cullen and Kirby. Notable among those letters was that of Munis who argued that a correction for hydrostatic gradients was not needed because, in the head-up position, the circulation above the heart functions as a siphon.[2] Cucchiara took another approach and chided practitioners to place an arterial catheter in head-up patients and measure blood pressure at the level of the head to avoid the need for arithmetically corrected measurements altogether.[3] This debate continues in the current issue of the *Newsletter* with letters from Drummond et al. who argue that clinical management of head-up patients must account for hydrostatic gradients,[4] and Kirby and Cullen[5] who expand on concepts raised in their earlier publication.[1]

This debate about blood pressure monitoring and management in head-up patients is unavoidable because of inadequate empirical data involving anesthetized, head-up patients who are at risk for rare, but debilitating, postoperative neurologic deficits.[1,6] Various forms of head-up positioning are used not only for neurosurgical procedures (e.g., posterior fossa craniectomy and cervical laminectomies) where the effects on hemodynamics have been more intensely pondered, but also for surgery to the thyroid gland, shoulder, and other non-neurosurgical sites where debate about blood pressure management has been less common. Placing the patient supine or prone to avoid physiologic challenges imposed by a head-up position is not always an option, as the sitting position for posterior fossa craniotomy is reported to diminish operative blood loss and significantly improve postoperative cranial nerve function.[7] With cervical spine surgery or posterior fossa intracranial surgery, converting from the sitting to prone position may potentially worsen pulmonary gas exchange in patients having medically complicated obesity, or may contribute to the risk of postoperative visual impairment in rare instances. Other surgeries (e.g., thyroid and shoulder surgery) are simply made more technically difficult by varying from an ideal head-up position. As such, it appears that the head-up position during anesthesia and surgery is here to stay, even though ideal blood pressure monitoring and management in these patients is controversial.

One of the core features of the current debate about blood pressure management in the head-up position revolves around whether the circulation above the heart functions as a siphon system[2] or as a waterfall system.[1,4,5] Based on the available evidence, either scenario is probably an oversimplification in anesthetized, surgically positioned patients. The siphon concept is very appealing when speaking of the physiology of unanesthetized healthy humans or giraffes; however, anesthetized surgical patients placed head up—often with the head position deviating considerably from neutral—may introduce more complex physiology. As we will see later, these head-position variations, independent of a gravity effect, have a bearing on cerebral circulation. Further, the siphon analogy assumes that vessels will function in series, when in fact the vessels connecting the heart to the most remote areas of the brain tissues and spinal cord have some elements in series and

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Inside:

APSF GOES ALL GREEN

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Correction:

The Winter 2008-209 issue of the *APSF Newsletter* lead article "Does Anesthetic Management Affect Cancer Outcome?" was authored by Anije Gottschalke, MD, Research Fellow; Marcel E. Durleux, MD, PhD, Professor of Anesthesiology and Neurolgoical Surgery; Mohamed Tiouririne, MD, Assistant Professor of Anesthesiology at the University of Virginia Health System, Charlottesville, VA. Dr. Gottschalk and Dr. Tiouririne were omitted from the original credits.

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NEWSLETTER

The Official Journal of the Anesthesia Patient Safety Foundation

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Too High Likely Safer Than Too Low

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some in parallel. These parallel aspects of the circulation may place tissues within remote watershed regions at risk for ischemic injury coincident with global cerebral and spinal cord blood flow remaining adequate. It is not so simple to model the cerebral circulation as a waterfall either, because a waterfall analogy dictates that the hydrostatic gradient of the column of blood in vessels meaningfully influences the relationship between the pressure at the aortic root and the remote regions of the brain. This analysis, too, overlooks the input of vessels in parallel, some of which may be occluded at baseline (e.g., from atherosclerosis) or as a result of surgical positioning. Some examples are in order:

Toole and Tucker[8,9] reviewed the literature concerning awake patients who acquire new-onset neurologic symptoms related to changes in head position, and they identified multiple contributing factors such as: 1) intraluminal atherosclerosis, 2) deviations from classic vessel configurations within the neck (most commonly involving a diminutive or non-functioning vertebral artery unilaterally), 3) changing relationships between the geography of the brainstem and vertebral vessels during head flexion, and 4) external compression of the carotid and vertebral arteries by osteophytes or normal vertebral anatomy. In a prospective study,[9] they examined the effect of head flexion/extension, rotation, and tilt on blood flow through the carotid and vertebral arteries in 20 fresh cadavers. They determined that, if a change in flow was to occur at all, it occurred at flexion/extension of <45°, rotation of <45°, or tilt of <30°. A positive response was manifested as simultaneous cessation of blood flow in both vertebral arteries in 30% of cadavers, and in both internal carotid arteries (but not simultaneously) in 45% of cadavers. This research also determined that the diminution or ablation of blood flow in these vessels was not linear with head movement, but instead developed precipitously over an incremental 5-10° change. Additionally, they determined that it was not possible to predict in which vessel, or even on which side of the body, vessel occlusion would occur during head rotation. Elsewhere Perkins et al.[10] reported on 2 patients who underwent right carotid endarterectomy while the patients were supine with the head rotated to the left. Inadvertent lidocaine injection into the right carotid arteries (during attempted local anesthesia of the carotid sinus baroreceptors) produced electroencephalographic (EEG) changes in both patients, but the EEG patterns varied greatly for reasons made clear by the preoperative angiogram. In 1 patient, atherosclerotic changes limited the contributions of the right carotid artery to the right side of the brain. Not surprisingly, EEG changes in this patient were unilateral and ipsilateral to the site of lidocaine injection. In contrast, the other patient had simultaneous EEG changes in both cerebral hemispheres, though more prominent in the right. Angiography revealed that, because of widespread atherosclerosis, the left carotid artery contributed nothing to the circulation of either cerebral hemisphere; however, the right carotid artery supplied blood for both hemispheres. Clearly these collective observations of Toole and Tucker[8,9] and Perkins et al.[10] speak to the fact that the plumbing of the human brain can be variable, dependent on changes in head positioning, and conceptually quite different from household plumbing.

Parallel Plumbing Important

If this is the case, one should examine the extremes of blood pressure required to prevent permanent neurologic injury. At the lower end of this range, we could assume a young, healthy, normotensive patient, with classic vessel anatomy, and an intracranial pressure never deviating from 0 mmHg or regional cerebral blood flow distribution never deviating from parity. Assuming a siphon based physiology, then it should be possible to measure blood pressure at the level of the heart, and maintain blood pressure at the lower limit of autoregulation without causing ischemic neurologic injury. Any small errors created by deviations from a pure siphon system, and some uncertainty as to whether there is a precise lower limit of autoregulation and where it might occur in this patient,[11] would be somewhat offset by the fact that, even as perfusion pressure declines below the lower limits of autoregulation, blood flow does not fall into the abyss but instead declines gradually, perhaps still leaving enough circulation to prevent permanent neurologic injury. At the other extreme, if we assume a waterfall-based physiology, we must not only account for a hydrostatic gradient imposed by the sitting position, but we must also take into account the parallel plumbing feeding the waterfall, and the effects that regional variations in intracranial pressure, surgical retractor pressure, head positioning, atherosclerosis, geographic variants of blood vessel distribution, and other factors may have on the flow through contributing vessels, some of which may be critical to patient well-being. Clearly there is a considerable difference between the physiologies described by these 2 extremes.

Simple Study May Not Yield Simple Answer

It is tempting to rush to the animal laboratory to try to mimic and study the exact patterns of physiology during anesthesia and patient positioning. However, such studies will likely reflect the physiology of healthy animals in which the various combinations of heart and head positioning, species-related anatomic variations, and other factors, will not accurately reproduce the conditions of the rare, highest-risk humans. If such studies are eventually performed in animals to better explore the issue of monitoring site versus cerebral well-being as related to siphon versus waterfall hemodynamic models, it must be remembered that measurements of well-being must take into account the watershed regions of brain, eyes, and spinal cord, using techniques such as microspheres, laser Doppler flowmetry, or multidimensional radiologic imaging to quantify regional blood flows, and multiple-lead electrical recordings to assess electrical well-being. Crude assessments of well-being, using transcranial Doppler sonography of conducting vessels, and processed or geographically non-discriminating eletrophysiologic measurements, will simply not address the root of the problem. Unfortunately, attempting to monitor and assess individual patients will be problematic, if for no other reason than that the patients at greatest risk of injury during the head-up position are probably those with some atypical anatomy or baseline physiology. Such patients will be hard to identify, the influence of variations in patient positioning may be impossible to explore in the clinical environment, and data from these patients will be hard to generalize to other high-risk patients.

Absent such evidence, it is tempting to instead analyze and rationalize blood pressure monitoring and management in individual patients, based on core principles. However, we anesthesiologists should be reluctant to choose this approach, recognizing how such a process has ill served us in the past. We need not be reminded that for a period of 3 or more decades, this type analysis of a possible intracranial pressure increase in response to intravenous succinylcholine,[12,13] or to "bucking" and coughing in tracheally intubated subjects,[14,15] erroneously ascribed increases in intrathoracic pressure and central venous pressure as the operant mechanisms. However, when such concepts were first tested experimentally in the 1980s and '90s, neither clinical condition was even remotely related to the long-touted operant mechanism.[12-15] Instead, other altogether different mechanisms appeared to be responsible, and the onset, magnitude, and duration of the intracranial pressure increases were not at all what anesthesiologists had long envisioned. There are a sufficient number of similar, faulty analyses in the history of anesthesiology to make us fearful of introducing new errors in management, based on core-principle analysis absent empirical support. However, unlike previous examples involving transient increases in intracranial pressure, the end result of the current discussion of blood pressure management in head-up patients is not to declare a winner of some innocuous academic pillow fight, but instead to optimize patient management for the purpose of avoiding irreversible neurologic injury.

Without the data we need to definitively identify ideal blood pressure monitoring and management in

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Goal Should Be to Avoid Harm

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head-up, anesthetized patients, what should we do for contemporary blood pressure measurement and management? It would seem appropriate that our practices should err on the side of providing excessive blood pressure to non-critical tissues, and adequate blood pressure to critical tissues. Such an approach has merit not because we have proven that a modified watershed model of cerebral circulation is operant in head-up patients or that core principles have led us to an unimpeachable conclusion, but instead because such an approach moves us in a management direction away from hypoperfusion (whatever the cause). This approach also has merit because experience tells us that small reductions from normal blood pressure are statistically more likely to produce long-term injury (e.g., from ischemia) than are small elevations in blood pressure (e.g., from hemorrhage or edema formation). Risk of cerebral aneurysm rupture is a notable exception.

In the face of inadequate information, pursuing good outcomes primarily by avoiding bad outcomes is not new to anesthesiologists and nurse anesthetists. Indeed, with an ongoing, decades-long debate about alpha-stat versus pH-stat management of blood gases and pH during clinically induced hypothermia,[16] the most commonly accepted management philosophy is directed toward avoiding harm, not pursuing perfection.

It should be remembered that invoking a siphon-related analysis of cerebral perfusion is basically an exploration of the minimal blood pressure required to provide adequate blood flow from the heart, through the brain, and back to the heart, and does not adequately account for the distribution of that blood flow within the brain. It is an analysis of extremes, to determine how far we can push our management approach yet not do harm. Indeed, we are sometimes called upon to transiently push the extremes of systemic blood pressure, to permit the clipping of a cerebral aneurysm, allow the placement of a suture in a critical cardiovascular structure, or ensure adequate perfusion and oxygenation of a fetus. However, these infrequent instances are different from the discussion of blood pressure management in head-up patients. Here, we are not exploring the transient, extreme manipulation of physiology to permit benefit (as in the aforementioned examples), but the prolonged management of blood pressure to avoid harm (e.g., watershed cerebral ischemia).

As such, until we have definitive data proving otherwise, it seems prudent to direct our blood pressure management in head-up patients in a manner that will accommodate for hydrostatic gradients, patient's baseline blood pressure (with its implications for cerebral autoregulation), and the impact of atherosclerotic and other vascular anomalies, regional intracranial pressure, and head positioning. Such an analysis dictates measuring blood pressure at the level of the most vulnerable tissue (i.e., the brain), and maintaining blood pressure well within the patient's normal range of blood pressures observed while unanesthetized. This management philosophy is consistent with our historic role as the vulnerable patient's last homeostatic defense for avoiding injury during anesthesia and surgery.

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Letter to the Editor

Lower Limit of Cerebral Autoregulation Questioned

To the Editor:

In the Summer 2007 issue of the *APSF Newsletter*, the editors published our communication concerning catastrophic neurologic outcomes in patients having shoulder surgery in the beach chair position.[1] We were gratified that the publication generated interest in some readers and several letters to the editor. While a couple of letters[2,3] seemed to agree with our thesis (corrections for blood pressure should be made to account for different values in the brain compared to the usual sites of measurement in the arm: "open" model) and provided additional insights,[4] others subsequently took us to task because of the authors' hypothesis that the site of pressure measurement is irrelevant because gravity has little or no effect on blood flow to and from the brain ("closed model").[5-7]

Although the original thrust of our article did not dwell on cerebral blood flow (CBF) according to the open or closed models,[5] that is a topic of immense interest to us and to those who have commented on our publication.[3,5,7-9]

The presence or absence of a siphon effect is the key point of differentiation in closed and open systems. A vascular siphon depends on the presence of a continuous column of blood in both the arterial and venous limbs of the loop. With respect to brain perfusion, this loop includes the thoracic aorta, brain arteries and arterioles, cerebral and jugular veins, vertebral venous plexus, the superior vena cava, and the right atrium. According to the siphon concept, no work is done by gravity *against* blood flow to the brain, and none is performed in the return of blood from the brain, because gravitational effects are identical on the ascending and descending limbs of the vascular loop.[5,8,9] The proponents of this system state that no correction is needed for blood pressure in the brain versus that in the arm, because the afferent and efferent effects of gravity cancel each other.[5-9]

The siphon concept is not accepted by all investigators. Opponents state that collapsible veins prevent gravitational pressure gradients from being matched on the arterial and venous sides of the vascular loop above the heart, thus preventing the siphon from operating. Fluid in the descending limb "falls" (waterfall concept) and as a result does not aid the ascending limb. If the siphon concept is invalid, the heart alone is responsible for pumping blood to the brain and overcoming viscous resistance to blood flow through the brain, and in the upright patient the descending limb does not aid ascending flow. In this case, a pressure gradient will exist from the heart to the brain, and mean arterial pressure (MAP) in the brain will be lower than that in the arm according to the difference in height of the brain above the arm (and the heart).[1]

Blood flow through the brain is determined by the driving pressure from the left ventricle to overcome cerebrovascular resistance, intracranial pressure, cerebral autoregulation, arterial PCO_2, and venous outflow resistance. If the internal jugular veins are collapsed, a parallel route, the vertebral venous plexus, still exists and can be a conduit to maintain the descending limb of the siphon. This system is thought to be protected from collapse, because of its attachments to rigid structures. However, regardless of which outflow tract is operational, the vessels of the brain are likely to act as a "baffle" and to prevent a siphon effect from being operational at all times in upright patients.

Independent of the siphon or waterfall concepts, the lower limit of autoregulation (LLA) also is critically important. For as long as we can remember, articles and textbooks almost uniformly have quoted this value as a cerebral perfusion pressure (CPP) of 50 mm Hg, where CPP=MAP-ICP (normal ICP should be assumed to be 5-15 mm Hg). Most anesthesiologists and anesthetists have been taught this value, and many have employed it clinically, reasoning that as long as they keep the lower value for CPP at 50 mmHg, CBF will remain constant, and hypoxic ischemic encephalopathy (HIE) will not occur. However, most work over the past 35 years has demonstrated significantly higher values for the LLA, perhaps as high as 80 ± 8 mmHg.[10-18] Assuming this is correct, and current evidence supports the view that it is, an anesthesiologist or nurse anesthetist who persists in adhering to the 50 mmHg value (particularly in the beach chair position) runs the risk of inducing the potentially catastrophic complication of HIE.

Drummond[18] noted that values lower than the LLA do not necessarily mean that patients will develop HIE, but some of them do. Unfortunately, which of these individuals will is unknown preoperatively. Since publication of the initial article by Pohl and Cullen,[19] several additional cases of severe brain damage occurring in healthy patients undergoing shoulder surgery in the beach chair position have come to our attention. Why then should we assume that the closed concept of CBF is always correct and, therefore, not bother to correct for MAP at the brain level? If, in the future, this concept is validated as absolute and always true, so be it, and such corrections will be unnecessary. However, because we are dealing with hypotheses rather than established facts regarding open versus closed (siphon) concepts for CBF, our feeling is that we shouldn't bet on patients' well-being by adhering to an unproven hypothesis and an antiquated value of the LLA that should have been retired years ago.

If we have to undergo shoulder surgery in the beach chair position, we'll make every effort to ensure that our anesthesiologist maintains a safe and appropriate CPP well above 50 mm Hg. In so doing, we would rather be safe than sorry. Other patients deserve no less.

Robert R. Kirby, MD
David J. Cullen, MD

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Letter to the Editor

Hydrostatic Gradient is Important—Blood Pressure Should be Corrected

To the Editor:

In the Summer 2007 issue of the *APSF Newsletter*, Cullen and Kirby described 4 instances of catastrophic neurologic outcomes after surgical procedures performed in the beach chair position.[1] They surmised that the combination of the failure to make allowance for the hydrostatic mean arterial pressure (MAP) gradient between the heart and the head with the clinicians' acceptance of relatively low MAPs at heart level had resulted in cerebral hypoperfusion and ischemic injury. They advocated that clinicians manage patients undergoing procedures in the beach chair position on the basis of MAP measured at the level of the head or corrected to head level by imposing an arithmetic adjustment to MAPs recorded at other sites (*see footnote for illustration).

Dr. James Munis responded with a commentary in which he disputed the necessity to measure MAP at (or correct it to) head level.[2] The essence of his argument (in very brief summary of a lengthy submission) is that the cerebral circulation is a closed system that functions like a siphon. While agreeing that intra-luminal pressures decrease above heart level, he argued that the same reduction in pressure occurs on both the venous and arterial sides of the circulation with, therefore, no net change in the driving (perfusion) pressure across the cerebral vascular bed and, therefore, no change in cerebral blood flow (CBF). The author of a subsequent letter to the editor of the *APSF Newsletter* applauded Dr. Munis's dismissal of "the nonissues of transmural pressures, altering transducer height and 'correction' formulas."[3]

We disagree with Dr. Munis. Furthermore, the possibility that clinicians might broadly accept this dismissal of the significance of the hydrostatic pressure gradient is of substantial concern to us. We believe that corrections for hydrostatic pressure gradients (also referred to as "gravitational pressure gradients") in head-up positions are necessary and appropriate. One approach to convincing the readers of the *APSF Newsletter* of our position might be to take issue with some or all of the many arguments in favor of the closed model of the cerebral circulation that Dr. Munis has laid out in various publications.[2,4,5] However, out of the concern that readers will be unconvinced (or simply confused) by such a complicated discussion, and because these arguments have been set forth in detail previously,[6,7] it seems preferable to argue first that even if the cerebral circulation is a closed system in some circumstances (functioning in the manner of a siphon with balanced hydrostatic pressures in the ascending and descending limbs of closed loop), it will not be so in *all* situations. The closed circulation/siphon theory posits that CBF is a function of the arterial to venous pressure difference across the brain (the "perfusion pressure") and that head elevation leads to equivalent hydrostatic pressure changes in arterial and venous pressure with, therefore, no net change in perfusion pressure or in CBF. However, the argument for parallel (and therefore compensating) changes in venous and arterial hydrostatic pressure becomes irrelevant in a circumstance in which there is direct compression of nervous tissue. In those circumstances, which arise when brain parenchyma is compressed by retractors or in the context of cervical spinal stenosis when the spinal cord is compressed by protruding discs or a hypertrophied posterior longitudinal ligament, it will be the transmural pressure, i.e., the gradient between the intraluminal and extraluminal (tissue) pressures that will be the principal determinant of flow. The siphon model would be similarly invalid when intraluminal pressure on the arterial side of the circulation has decreased more than venous pressure because of a stenosis in the arterial tree. We take the position that even if the closed (siphon) model of the circulation is in effect some, or even most of the time (which we do not accept), the "rules" are likely to be different in the context of the compression or arterial stenoses just mentioned. It would be a slim consolation to the 4 patients who sustained ischemic injuries in the beach chair position (or to their families) to know that hydrostatically reduced intraluminal pressures at the level of the head are *sometimes* not a matter of consequence.

Furthermore, we believe that the non-applicability of the closed circulation/siphon model goes beyond circumstances of CNS compression or vascular stenoses. In a thought experiment used by Dr. Munis to illustrate the non-importance of absolute intraluminal pressure,[2,4] he asks that we imagine an intravenous (IV) infusion system with a fluid bag in its typical position some distance higher than the venous access site. Would the IV continue to flow, he asks rhetorically, if a loop of the IV tubing were raised to the level of the top of the IV bag, and what would the intraluminal pressure be at the apex of that loop of tubing? The answer to the former, from experience, is, "Yes, it would flow" and to the latter, that the pressure would be very close to atmospheric. Voila! Q.E.D! The theorem is proven. The hydrostatic gradient is unimportant! And, those who have used a siphon might accept this. Flow *will* continue even in the presence of the hypothetical loop.

But the shortcoming of this analogy is that the siphons that the readers have used were invariably composed of relatively rigid tubing. What if some portion of that loop at the apex of the system was composed of collapsible tubing with a consistency similar to that of a Penrose drain? As previously argued in detail,[6] the tubing would collapse; flow would cease; and the siphon would not function. The cerebral vessels are not all rigid like the tubing in the IV analogy. At least some of the cerebral veins, venules, and capillaries are non-rigid. Munis and Lozado acknowledge this, but argue that at least some vessels within the nervous system will be patent at any given moment in time and that at least a portion of flow will continue.[4] Perhaps, but we cannot take reassurance from the notion that at any given time "some" of the brain is not ischemic. Again, it would be a slim consolation to the 4 devastated patients (or to their families) to know that blood flow had continued to some portions of their nervous systems while disabling damage was evolving in others.

We cannot assert that Munis and associates' belief in a closed model of the cerebral circulation in which hydrostatic pressure gradients are of no physiologic consequence has yet been refuted definitively. However, we take the position that such a model is highly improbable and at best unproven. We are concerned that there is a substantial potential for the occurrence of additional neurologic injuries if clinicians accept Kleinman's opinion that "transmural pressures, altering transducer height and 'correction' formulas" are "nonissues."[3] We hold the view that clinicians managing patients in significantly head-up postures should continue to measure blood pressure at (or correct it to) head level. We think that if unbiased observers with no prior knowledge of the elements of this debate were to visit and examine the existing body of information, they would be intrigued by the intricacies of the physiologic discussions. But we suspect that they would conclude, in the absence of definitive proof that hydrostatic pressure gradients were never of consequence in determining blood flow to the nervous system, that blood pressure should always be measured at, or corrected to, head level.

** As blood flows vertically from the heart, there will be a reduction in arterial pressure that is related to the weight of a column of blood. That reduction will be approximately 2 mm Hg for each inch (2.54 cm) of vertical displacement. For illustration, consider a patient in a semi-recumbent position such that the external auditory canal (EAC) is 12 inches above the mid-point of a blood pressure cuff on the upper arm. If MAP as measured by the cuff were 65 mmHg, the MAP at the EAC would not be greater than 41 mm Hg.*

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Cerebral Oximetry May Provide Helpful Information

by Christopher A. Troianos, MD

Perioperative neurologic injury is a devastating complication that is not always predictable, but contributes to significant morbidity, mortality, and consumption of health care dollars.[1] The etiologies for stroke and neurocognitive dysfunction following cardiac and non-cardiac surgeries are broadly divided into embolic and perfusion related insults. Epiaortic ultrasound, transcranial Doppler, and screening carotid ultrasonography may reduce the incidence of perioperative neurologic injury by targeting larger arterial vessels (aorta, middle cerebral, and carotid arteries, respectively) for embolic sources of cerebral injury using ultrasound technology. Diffuse insults and subtle neuro-cognitive deficits suggest an etiology related to regional cerebral microcirculation perfusion imbalance. In their prospective study of over 11,000 patients, Likosky and colleagues found that 75% of strokes occur among the 90% of patients with low to intermediate risk undergoing coronary artery bypass grafting (CABG) surgery.[2] These neurologic insults often occur despite well-maintained global circulatory and perfusion parameters of blood pressure and cardiac output.

The adequacy of cerebral hemispheric oxygenation can be estimated by sampling oxygen content of blood in the internal jugular vein, but requires the invasive placement of a jugular venous catheter for repeated measurements. Additionally, these data merely reflect global hemispheric oxygenation, exposing the risk of unrecognized regional malperfusion despite adequate global cerebral oxygenation. Commercially available cerebral oximeters estimate regional tissue oxygenation by transcutaneous measurement of areas most vulnerable to changes in oxygen supply and demand (frontal cerebral cortex). These technologies exploit the ability of light to penetrate the skull and determine hemoglobin oxygenation according to the amount of light absorbed by hemoglobin. Cerebral oximetry differs from pulse oximetry by utilizing 2 photo-detectors with each light source, thereby allowing selective sampling of tissue beyond a specified depth beneath the skin. Near-field photo-detection is subtracted from far-field photo-detection to provide selective tissue oxygenation measurement beyond a pre-defined depth (Figure 1).

Cerebral oximetry differs from pulse oximetry in that tissue sampling represents primarily (70-75%) venous, and less (20-25%) arterial blood. Cerebral oximetric monitoring is also not dependent upon pulsatile flow. Regional estimates of cerebral oxygenation in the vulnerable watershed region of the frontal cerebral cortex provide a sensitive method of detecting changes in oxygen delivery due to the limited oxygen reserve of this area. Cerebral oximetric monitoring thus may serve as an "early warning" of decreased oxygen delivery to the rest of the brain and other major organs. These estimates of regional cerebral oximetry may be used to reverse decreasing cerebral perfusion and avert prolonged ischemia of the brain and other major organs by instituting a strategy that optimizes factors that affect cerebral oxygen supply and demand.[3] Several recent articles have demonstrated the association between decreased cerebral oximetric measurements and neurocognitive decline, increased major organ morbidity, and increased hospital length of stay (LOS). Interventions utilized to improve regional cerebral oximetry depend on the clinical situation and generally follow less invasive manipulations of physiologic parameters before transfusion of packed red blood cells. These interventions include correction of patient or cannula malpositioning, increasing blood pressure, increasing cardiac output (or cardiopulmonary bypass (CPB) flow) above 2.5 L/m^2/min, increasing FiO_2, increasing $PaCO_2$ to >40 mmHg (if <40 mmHg) by decreasing minute ventilation (or decreasing oxygenator fresh gas sweep flows during CPB), administering anesthesia and/or muscle relaxants as indicated, and finally administering a red blood cell transfusion if the hematocrit is <20%.

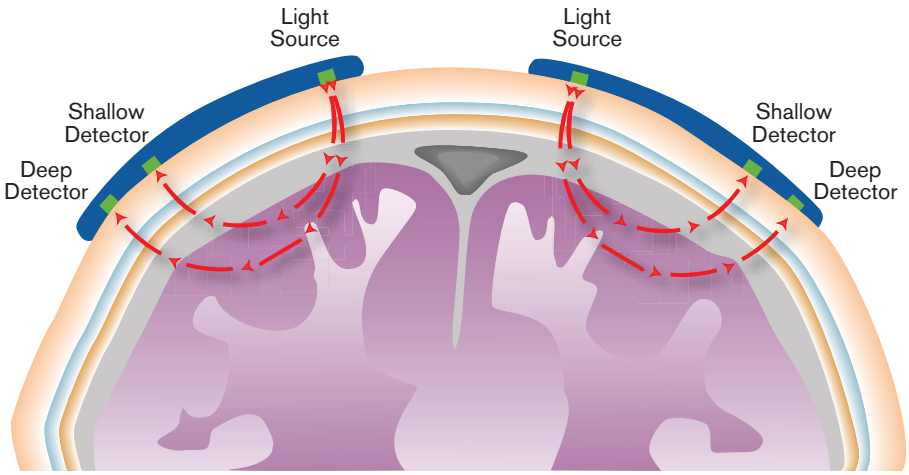

Figure 1: Diagram of cerebral oximetry illustrating a deep and shallow photo detector paired with each light source.

Clinical Applications

The value of measuring cerebral oximetry during surgery is illustrated in Figure 2 (which shows cerebral oximetry changes during a proximal humerus repair in a 94-year-old female with multiple co-morbidities) and in Figure 3 (which represents changes in cerebral oximetry during cardiopulmonary bypass events).

Indeed, a number of recent articles have demonstrated the benefits of cerebral oximetry among patients undergoing both cardiac and non-cardiac surgery. Slater et al. demonstrated the association between intraoperative cerebral oxygen desaturation and an increased risk of cognitive decline and prolonged hospital stay by assessing cognitive function in adult patients undergoing CABG surgery preoperatively, postoperatively, and 3 months after surgery, using a battery of neurocognitive tests. Patients were prospectively randomized to a blinded control group or to an unblinded interventional group. The area under the curve indicating the degree of desaturation below a 50% threshold over time accounted for both

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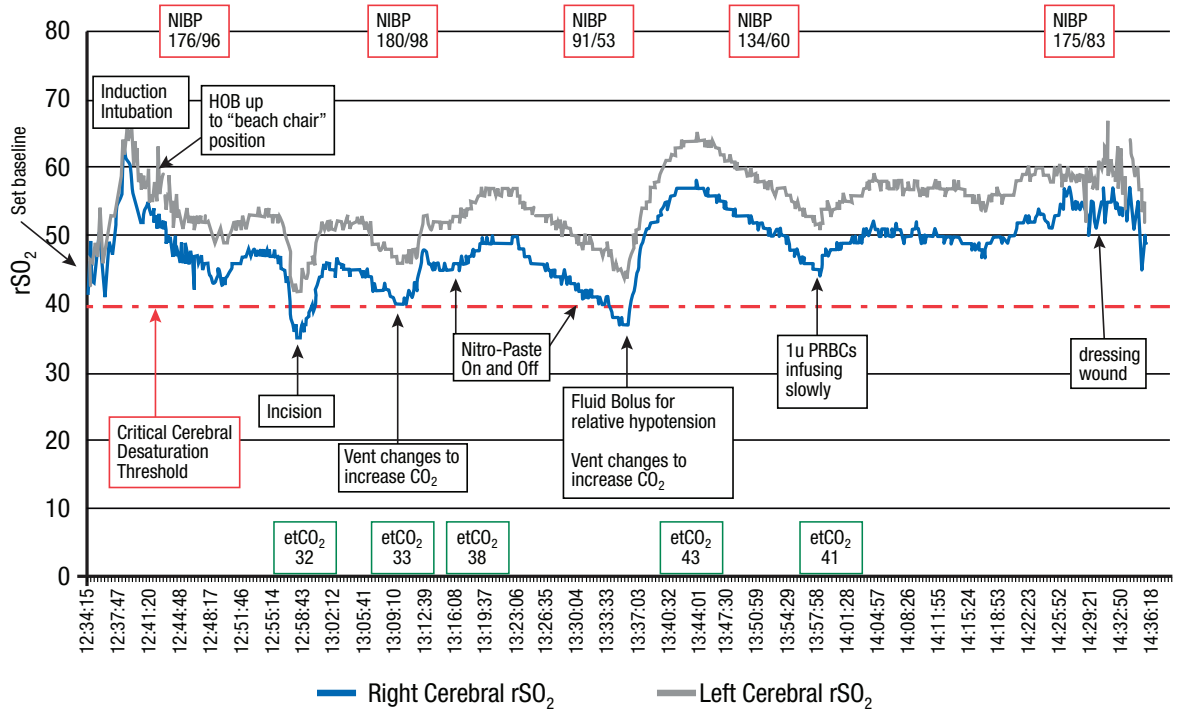

Figure 2. Time line of events and cerebral oximetry changes during a proximal humerus fracture repair in a 94-year-old female who presented with a history of hypertension, atrial fibrillation, COPD, and TIAs.

the depth and duration of desaturation below this 50% threshold. Patients with rSO_2 desaturation score >3,000%-second had a significantly higher risk of early postoperative cognitive decline [p=0.024]. Patients with rSO_2 desaturation score >3,000%-second also had a near 3-fold increased risk of prolonged hospital stay (>6 days) [p=0.007].[4] Similarly, Yao et al. found a significant correlation between the degree and duration of cerebral oximetry desaturation and early postoperative neuropsychological dysfunction among patients undergoing elective cardiac surgery with CPB.[5]

Patients with cerebral desaturation during non-cardiac surgery have also been shown to exhibit declines in their Mini Mental State Examination (MMSE). Casati and colleagues studied 122 patients older than 65 years, scheduled for major abdominal, nonvascular surgery under general anesthesia (with an expected duration >2 hr). Surgical procedures represented by major abdominal surgery with a xiphopubic skin incision included gastric resection, colonic resection, hepatic resection, and duodenocephalo-pancreatectomy. Patients were randomly assigned to an intervention group (the monitor was visible and rSO_2 maintained at >75% of pre-induction values) or a control group (the monitor was blinded and anesthesia was managed routinely). There was a significant correlation between the area under the curve of rSO_2 <75% of baseline and postoperative decrease in MMSE score from preoperative values. Control group patients with intraoperative cerebral desaturation also experienced a longer time to post-anesthesia care unit (PACU) discharge (47 min [13–56 min]) and hospital LOS (24 days [7–53] days) compared with patients of the treatment group (25 min [15–35 min] and 10 days [7–23 days], respectively; p = 0.01 and p = 0.007).[6]

Cullen and Kirby reported an unexpected and devastating complication of neurologic injury in this *Newsletter*, occurring in a healthy 47-year-old woman undergoing shoulder surgery in the "beach chair" position.[7] They identified the lack of appreciation of cerebral hypoperfusion that occurs with blood pressure measurement in the non-operative arm that is positioned well below the level of the brain, and the physiologic and anatomic changes that occur with the beach chair position. These can include decreased venous return, vasodilation, and head flexion, which may impede jugular venous flow and thus decrease cerebral perfusion. Deliberate hypotension coincident with these physiologic changes, requires enhanced vigilance on the part of the clinicians caring for these patients. Use of a cerebral oximeter in this setting provides an additional tool in assessing adequate oxygen delivery to vulnerable cerebral tissue.

Cerebral oximetry has been shown to reduce major organ dysfunction following cardiac surgery. Murkin et al. prospectively randomized 200 patients undergoing CABG surgery to intraoperative cerebral regional oxygen saturation monitoring with active display and treatment intervention protocol, or blinded rSO_2 monitoring.[8] Significantly more patients in the blinded group had prolonged cerebral desaturation (p=0.014) and a longer ICU LOS (p=0.029) versus intervention patients. Significantly more patients in the blinded group had major organ morbidity or mortality (death, ventilation >48 hr, stroke, myocardial infarction, return for re-exploration) versus patients in the intervention group (p=0.048). Patients experiencing major organ morbidity or mortality had lower baseline and mean rSO_2 measurements, more cerebral desaturation, longer ICU LOSs, and longer postoperative hospitalization than patients without major organ complications. There was a significant inverse correlation between intraoperative rSO_2 measurements and duration of postoperative hospitalization among patients requiring more than 10 postoperative days.

Discussion

The clinical studies described above demonstrate the potential benefits of cerebral oximetric monitoring in a variety of clinical situations. Although the vast majority of clinical studies have been conducted among cardiac surgical patients, the application of cerebral oximetry to non-cardiac surgical patients is compelling in certain clinical situations. The previously described work by Casati demonstrated the benefits of using cerebral oximetry among elderly patients undergoing major abdominal surgery.[6] The use of intraoperative cerebral regional oxygen saturation monitoring for patients undergoing carotid artery surgery can guide surgical and physiologic

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intervention in terms of shunt use and blood pressure management, as bilateral measurements are compared with each other and with baseline measurements. An emerging area of cerebral oxygen saturation monitoring is the patient undergoing shoulder surgery in the beach chair position for the reasons described above. Cerebral malperfusion may be unappreciated in this setting, where blood pressure monitoring may not be optimal, head position may impede cerebral venous drainage, and positive pressure ventilation impedes an already compromised decreased venous return to the heart because of the beach chair positioning.

The reduction of major organ morbidity (death, ventilation >48 hr, stroke, myocardial infarction, return for re-exploration) associated with intraoperative cerebral oximetric monitoring in patients undergoing CABG surgery is another very important aspect of cerebral oximetric monitoring. Maintaining an adequate oxygen balance in the most vulnerable watershed tissue of the frontal cerebral cortex apparently provides an "early warning" of decreased oxygen delivery to the rest of the brain and other major organs. Interventions that reverse decreasing cerebral perfusion may avert prolonged ischemia of the brain and thus minimize oxygen desaturation in other major organs (Figure 3). The early warning aspect of cerebral oximetry was further demonstrated by a pediatric study that examined the time to a 5% and 10% reduction in baseline oxygen saturation measurements in 10 children subjected to apnea during laser surgery of their airway. The average time for their pulse oximeter to exhibit a 5% and 10% reduction from pre-apnea levels was 146 ± 49 and 189 ± 64 seconds, respectively, while the cerebral oximeter exhibited an earlier warning of 5% and 10% cerebral desaturation at 94 ± 8 and 138 ± 89 sec, respectively. Cerebral desaturation thus occurred 1 min before the pulse oximeter indicated desaturation among these children.[9]

The benefits of cerebral oximetric monitoring are continually emerging as more work is published demonstrating improved outcome and enhanced patient safety. Although the majority of published data have demonstrated improved outcomes among cardiac surgical patients, the studies performed thus far among non-cardiac surgical patients are beginning to identify its utility in other clinical scenarios. Future research to identify and validate the benefits of cerebral oximetry monitoring in improving patient outcomes among non-cardiac surgical patients (as well as cardiac surgical patients) represents an exciting and important opportunity to explore and utilize this recent technology.

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Disclosures: Dr. Troianos has had no financial relationship with any manufacturer of cerebral oximetry technology. Somanetics, a manufacturer of this technology, is a corporate donor to the APSF.

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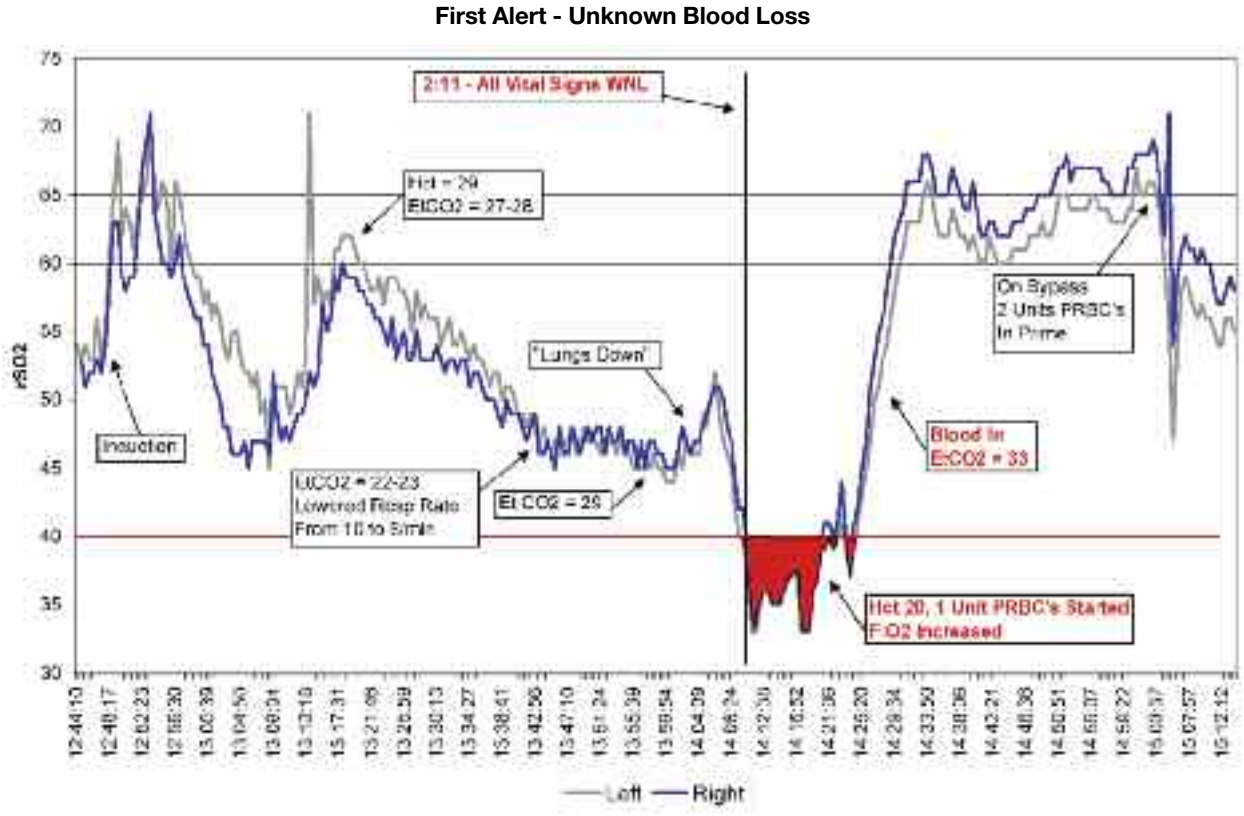

Figure 3. Cerebral oximetry values and time-line of events during a cardiac surgical procedure.

Can Processed EEG Monitoring Detect Postural Cerebral Ischemia or Cerebral Hypoperfusion?

by Mindaugas Pranevicius, MD, and Osvaldas Pranevicius, MD

Recent reports about unrecognized cerebral ischemia under general anesthesia are alarming,[1,2] and reveal that it may start insidiously, progress covertly, and result in devastating outcomes. Although much of the emphasis for anesthesia management is focused on maintenance of adequate cerebral perfusion pressure (CPP), does this ensure sufficient perfusion of the brain? Perfusion pressure by itself is the propulsion force only; it does not determine distribution of cerebral blood flow (CBF), ensure adequate collateral circulation, nor account for variations in the venous outflow path. Here we present a case that demonstrates the utility as well as limitations of processed EEG monitoring to assess adequacy of the CBF during an episode of postural cerebral venous steal.

To measure CPP, an arterial catheter is placed and zeroed at the level of the external acoustic meatus to approximate the circle of Willis. Most anesthesiologists and nurse anesthetists aim to maintain CPP within 15-20% of the baseline value. To calculate CPP in the sitting position one needs to know both arterial (inflow) and venous (effective outflow) pressures.[3] We can easily measure the arterial (inflow) pressure, but we usually only estimate the effective outflow pressure. When outflow through the vertebral venous plexus is not adequate, atmospheric pressure assumes the role of effective backpressure due to jugular vein compression at the skull base.[3,4] In this context pressure measurement at the external acoustic meatus estimates the CPP, assuming that the effective outflow pressure is zero (atmospheric) at the scull base.[3]

We also can only assume that the effective outflow pressure is uniform throughout the brain. Regional differences in the effective outflow pressure in the brain can cause a cerebral venous steal phenomenon diverting blood flow to the pathway of least resistance.[5] During head-up tilt, atmospheric pressure hinders outflow from the upper body with zero venous pressure and diverts the flow to the lower body where venous pressure exceeds atmospheric (postural "steal"). Similarly, alveolar pressure diverts pulmonary blood flow into dependent parts of pulmonary circulation.[6,7] Thus the adequacy of cerebral perfusion can not be determined solely from the arterial pressure, but requires consideration of the effects of vascular anatomy, autoregulation, $PaCO_2$, anesthetic, viscosity, vascular tone, and regional variation of these factors on the CBF. More direct assessment of cerebral perfusion would be desirable.

Ideally, a sensitive neurological exam can assess potential compromise of cerebral function occurring during periods of inadequate cerebral perfusion. However, under general anesthesia assessment of cerebral function is more challenging. Although many options are available, no single monitoring modality has been demonstrated as superior.[8]

Neurophysiologic monitoring with evoked potentials requires dedicated trained staff and equipment and is typically limited to select cases. Transcranial Doppler monitoring of middle cerebral artery flow velocity is operator-dependent, very sensitive to the acoustic contact, and fails when the acoustic "window" cannot be found. Jugular vein oximetry is invasive. Near infrared oximetry monitors tissue oxygenation in hair-free areas of the head but is not broadly available. In contrast, processed EEG monitoring devices are widespread, and although typically utilized to help manage intraoperative anesthetic dosing, they could help to detect cerebral hypoperfusion.

Case Report

A 34-year-old female, 5'8", 76.2 kg underwent laparoscopic cholecystectomy under general endotracheal anesthesia. Medical history was significant for hypertension, controlled with metoprolol. She had previously undergone gastric bypass surgery for morbid obesity. Following induction, anesthesia was maintained with sevoflurane 2% inspired in air/oxygen. The patient's baseline blood pressure was 143/97 mmHg; the immediate preinduction value was 167/113 mmHg; and following induction was 130/80 mmHg. Approximately 40 min after induction, with pneumoperitoneum and reverse Trendelenburg position, blood pressure was noted to decrease acutely to 95/50. At this point, a BIS monitor was applied and the initial BIS value was 20 with sevoflurane end-tidal value at 1 MAC. Two doses of ephedrine (5 mg + 10 mg) were administered. Blood pressure promptly returned to 130/80 and BIS simultaneously increased to 40-50 without decreasing the MAC (see Figure 1). The case was completed and the patient recovered uneventfully.

We submitted the BIS trend data for review by the manufacturer (Aspect Medical Systems) who confirmed that BIS was functioning as designed. Interrogation of the trend recording revealed the BIS system was detecting delta wave activity at the beginning of the record and the BIS values were appropriately low during the initial readings.

Discussion

Although the BIS Index was originally developed to measure the effects of anesthetic and sedative agents, multiple case reports have described the ability of BIS monitoring to detect episodes of cerebral ischemia.[9-17] In the absence of larger validating studies, sensitivity and specificity of BIS for this

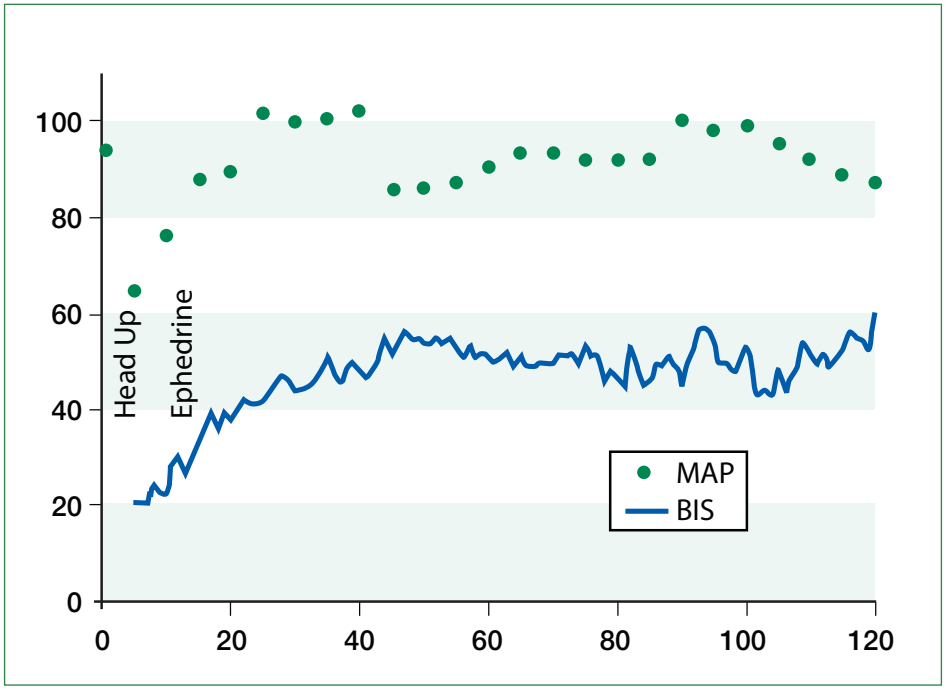

Figure 1. 120 min BIS and MAP trend during orthostatic hypotension treated with ephedrine

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BIS and MAP Trend Up After Treatment

"EEG," From Preceding Page

application has been questioned.[13,18,19] Although we believe our case demonstrates the ability of processed EEG monitoring to detect cerebral hypoperfusion, it also highlights important limitations. Without a baseline record prior to and following induction of anesthesia, a low BIS value during orthostatic hypotension may reflect the effect of the anesthetic on the EEG with or without the additive effects of hypoperfusion. In our patient, we observed rapid resolution of the low BIS values following ephedrine administration. Although ephedrine increases blood pressure and CBF, it has also been reported to increase BIS values directly.[20] Similar to cerebral oximetry, BIS and other processed EEG monitors analyze EEG signals from the frontal lobe only, and consequently may miss regional CBF abnormalities. Although our patient exhibited low BIS values during the period of postural hypotension, it should be noted that a variety of artifact conditions may cause spuriously elevated BIS values.[13]

Conclusion

Processed EEG monitoring systems are operator-independent, widely available, relatively inexpensive technologies to use during the intraoperative period. Although these technologies are not specifically developed as monitors for cerebral perfusion, they may help detect otherwise unrecognized global cerebral ischemia. If anesthetic dosing and surgical stimulation are stable, and postural changes or acute hypotension result in a precipitous decrease in the brain function values, clinicians may consider correcting the hypotension till brain function values improve.

The recent case reports should remind us that the brain is a critical and fragile organ that we rarely monitor directly during general anesthesia. We do not believe that the absence of the "perfect" cerebral monitor should discourage us from obtaining the most information from available modalities. We believe that available processed EEG monitoring devices could help in this regard. Larger scale validation studies to determine processed EEG and CBF correlates could advance functional cerebral monitoring and improve patient safety.

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Dear SIRS

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R ESPONSE
S YSTEM

Dear SIRS refers to the Safety Information Response System. The purpose of this column is to allow expeditious communication of technology-related safety concerns raised by our readers, with input and responses from manufacturers and industry representatives. This process was developed by Drs. Michael Olympio, Chair of the Committee on Technology, and Robert Morell, Editor of this newsletter. Dr. Olympio is overseeing the column and coordinating the readers' inquiries and the responses from industry. **Dear SIRS** made its debut in the Spring 2004 issue.

Dear SIRS:

Our hospital has recently purchased new IV fluid warmers and installed them in our operating rooms. Although the device is very effective at warming fluid, we have found that it can produce a significant amount of electrical interference in the operating room. This has rendered the device unusable in cases when neurologic monitoring with evoked potentials is required. As another safety concern, we have found that the device can also interfere with the ECG, making it appear as though the patient is having PVCs. We have provided in Figures 1 and 2 an example illustrating this ECG artifact. This particular patient had atrial fibrillation, and the multiple artifacts in lead V could potentially be misinterpreted as PVCs (Figure 1). As soon as the power button on the device was switched off, the artifacts disappeared (Figure 2). We are pleased to inform the anesthesia community that we have addressed this issue with the manufacturer, and they have promptly created a solution in which all devices subsequently produced will have reduced electrical interference. Additionally, the manufacturer is modifying our previously purchased devices so that they too will also produce less electrical interference.

David L. Saliba, MD
John E. Reynolds, MD
Winston-Salem, NC

In Response:

Thank you for sharing your observations. We have previously had some minor and sporadic ECG interference reported by users of the enFlow® IV Fluid/ Blood Warming System. Some interference with neuro evoked potential monitoring has also been reported. No previous reports have involved clinical misinterpretation.

The enFlow system has been used clinically over 250,000 times, and most customer feedback has been extremely positive. The product has been tested extensively and complies with applicable national and international standards, including those having requirements relating to electromagnetic compatibility with other monitors.

On previous occasions when interference has been reported, we have frequently been successful in resolving the issue using standard troubleshooting suggestions (e.g., checking ECG monitoring electrode impedances, repositioning intertwined cables). We also initiated engineering efforts to further investigate and address possible sources for interference. Our efforts determined that in some unanticipated circumstances, depending on the particular characteristics of monitors in use, an electrical transient associated with the on/off cycling of the warming system could produce an artifact. Drs. Saliba and

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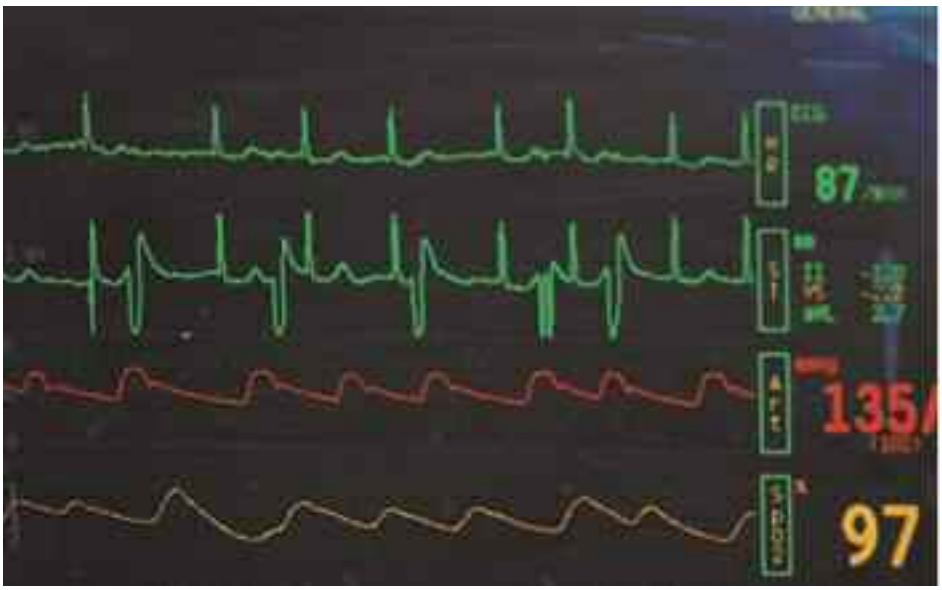

Figure 1. Artifact in lead V could be mistaken for PVCs.

Manufacturer Engineers Solution to Problem

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Reynolds show this in the photographs they provided. The artifact seen primarily in lead V is asynchronous with the heart rate, and other parameters are displayed normally.

Our engineering efforts have led to design improvements that further mitigate the potential for interference. These improvements are the demonstrated "solution" referred to by Drs. Saliba and Reynolds in their letter. We are in the final stages of implementing these improvements in production. We intend to proactively communicate with other enFlow users.

Sincerely,
Vital Signs – A GE Healthcare Company
David Cassidy
Executive Vice President
Enginivity LLC, a subsidiary of Vital Signs, Inc.

This Dear SIRS column is an excellent example of the positive results that can come from a patient safety dialogue between clinicians and manufacturers—Thank you all!!

Figure 2. Artifact disappears when fluid warmer is turned off.

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APSF gratefully acknowledges the support of Eisai Inc. in the full funding ($150,000) of a 2009 APSF Research Grant that will be designated the

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Q&A
SPECIAL EDITION

Cross-Contamination Via Anesthesia Equipment?

The APSF has received numerous questions regarding cross-contamination of one patient to another via the anesthetic machine and breathing circuit. They concern the need for and efficacy of various breathing circuit filters, their optimal location, the cleaning of the machine, and the reuse of disposable circuits and/or filters. These questions are interrelated and the Committee on Technology would like to address them in the next few issues of the *Newsletter*.

First, we'd like to refer the readership and question writers to a previous *Dear SIRS* column in the Spring 2007 issue of the *Newsletter*, pages 12-14, entitled "Can Soda Lime Canisters Spread MRSA?" (http://www.apsf.org/assets/Documents/spring 2007.pdf). Within that article are important references regarding filtration efficacy, filtration for *M. tuberculosis*, and the latest CDC recommendations for cleaning the anesthesia machine. Next, a few general issues surrounding contamination of the circle breathing system will be presented in this column, followed by specific questions and answers. Considerations of circuit re-use and specific manufacturer recommendations for cleaning the machine will be presented in a subsequent issue of the *Newsletter*.

The Committee on Technology was asked whether bacteria and/or viruses live through the highly alkaline pH of soda lime. In 1941 an overly simplistic model was proposed that bacteria became trapped in the breathing circuit and never reached the inspiratory limb, possibly due to the bactericidal action of soda lime (pH 11-14). Therefore, cross-contamination of patients was thought highly unlikely.[1] When CO_2 enters the reaction with soda lime, heat and water are released and soda lime forms a highly alkaline solution. This alkaline solution appears bactericidal for pathogens like *Staphylococcus aureus* and *Pseudomonas aeruginosa* but not for others, including *Mycobacterium tuberculosis*.[2,3] Infectious particles aerosolized by patients have a wide range of particle size and mass which together with their velocity influences their ability to either 1) remain in the gas flow stream, 2) become trapped in the highly alkaline liquid surrounding the soda lime granules, or 3) deposit themselves on various surfaces.[4] CO_2 absorber canisters would see variable amounts of bacterial load dependent upon the fresh gas flow from the anesthesia machine, the inspiratory flow rate, and tidal volume of the delivered breath. For example, in the case of high fresh gas inflow, there would be redirection of exhaled gas outwards through the scavenger, and retrograde filling of the absorber with fresh gas, instead of recirculation of patient gas through the absorber. Conversely, in situations of low fresh gas flow and/or smaller CO_2 canisters, there could theoretically be an increase in the pathogens eluted from the inspiratory limb. Soda lime is not a true barrier for bacteria or viruses.

One reason for so many conflicting studies regarding the cross-contamination of patients, relates to the large number of variables that contribute to transmission of virulent pathogens in sufficient numbers to the inspiratory limb of the breathing circuit, where they may be delivered to the next patient. Variables that are likely to influence this ability of pathogens to be transmitted include

1. Number of organisms aerosolized
2. Virulence of the organism
3. Resistance of the organism
4. Electrical charge of the organism and aerosol[4]
5. Size and distribution of particles entering the breathing system[4]
6. Fresh gas flow[4]
7. Tidal volume, inspiratory flow rate, and I:E ratio
8. Volume of the CO_2 absorber canister
9. Granule size of the CO_2 absorbent
10. Frequency of use of the machine[5]
11. Time between cases[5]
12. Immunity of the patient.

A single study that controls all of these variables is highly unlikely given the complexity of anesthesia care in a clinical setting. It must also be remembered that most studies are carried out under standardized conditions and can only approximate the clinical scenario of a busy operating room.

Q Dear Q&A,

Are filters a necessary precaution we need to use for the purpose of protecting patients, or are they not really performing any useful function? I am interested in the patient safety function of the filters and not the intent to re-use the circuit on another patient.

Pam Krueger, CRNA
Taylor, TX

A Dear Ms. Krueger,

Protecting patients from undesirable pathogens via the anesthesia circle breathing system has been the topic of many studies, often with divergent conclusions.[1-3,5-10] Many of the contaminations found were non-pathogenic skin flora.[9,11] Before a breathing circuit can serve as a vector for respiratory infections:

1. A patient must aerosolize a sufficient number of pathogens to contaminate the anesthesia machine
2. The pathogen must remain viable from one case to the next

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Numerous questions to the Committee on Technology are individually and quickly answered each quarter by knowledgeable committee members. Many of those responses would be of value to the general readership, but are not suitable for the Dear SIRS column. Therefore, we have created this simple column to address the needs of our readership.

More Q&A

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3. The pathogen must be eluted from the machine in sufficient numbers and with sufficient virulence to infect other patients.[5]

Infected patients can aerosolize large numbers of bacteria and efficiently transmit them to the anesthesia machine when intubated.[5,6,12] These pathogens can reside in the anesthesia machine for prolonged periods of time.[7] In particular, the mask, Y-piece, and breathing hoses, which can become readily contaminated with patient secretions, must be cleaned and subjected to high level disinfection, according to the Centers for Disease Control and Prevention,[13,14] as well as the ASA Committee on Occupational Health of Operating Room Personnel as described in their *Recommendations for Infection Control for the Practice of Anesthesiology*.[15] Many clinicians prefer single-use disposable breathing hoses instead. The low frequency of documented transmission of infections through use of the anesthesia equipment suggests that these policies are effective.[6,16,17] There are, however, reports of cross-contamination despite such guidelines.[18] Filters in the breathing circuit may be an approach to manage this complex situation; however, opinions regarding their use remain ambiguous.[19,20] The current recommendations from the Centers for Disease Control and Prevention state that use of filters is an unresolved issue[13,14] except in the case of patients with active tuberculosis.[21] The ASA recommendations[15] state that routine use of filters is not supported by current evidence, except in the prevention of transmission of *M. Tuberculosis*.

Filters bring their own set of potential dangers. Case reports described distal occlusion[22] and filter obstruction from secretions,[23] hypoxia,[24] toxic metabolite production,[25] increases in dead space for pediatric circuits,[26] and undetectable changes in filter resistance leading to decreased tidal volumes, increased airway pressures[25] and even bilateral pneumothoraces.[27] These problems may not be immediately apparent and can lead to delay in diagnoses with dire consequences for patients.

Q Dear Q&A,

What is the current status on which limb of the anesthesia machine the viral filter of the circuit should attach? Expiration or inspiration limb of the machine? Any reasons?

Chuck Klos, CRNA, APNP
Menomonie, WI

A Dear Mr. Klos,

Breathing filters are assessed according to their bacterial filtration efficiency (BFE) and viral filtration efficiency (VFE). Protection of the anesthesia machine from the patient would suggest that a high efficiency filter be placed in the expiratory limb. Protecting the patient from the anesthesia machine suggests that a high efficiency filter be used on the inspiratory limb of the breathing circuit. Placing the breathing filter between the endotracheal tube and the Y-piece will protect the patient and the anesthesia machine from contamination,[6,8,9,11,16,28-30] and is probably the most logical placement. The ASA recommends this location in cases of *M. Tuberculosis*.[15] Interposition of a filter would lead some to suggest reuse of the breathing circuit tubing, thus saving the cost of a new circuit for each patient,[31] but the issues related to reuse of single-use anesthesia equipment are complicated and will be discussed in a later issue of the *Newsletter* (see "On the Horizon" below).

In summary

Nosocomial pneumonia is one of the most costly hospital acquired infections. The magnitude of cross-contamination via the anesthesia machine or breathing circuit is very small in comparison to other modes of nosocomial infections. With the use of bacterial/viral filter devices this risk is further reduced and most experimental and clinical studies confirm the high efficiency of such devices. Viral filtration rate is most likely not as effective as bacterial filtration.[6] Filters should be interposed between the endotracheal tube and the Y-piece. Thus, for cross-contamination to occur, a pathogen has to bridge a filter device twice, making the event even less likely. Routine filter use is currently not supported by CDC guidelines or ASA recommendations, except for *M. Tuberculosis*. Many advocates wish to use these filters in order to reuse breathing circuits and save costs. This practice, even though widespread in the US, Canada, and Europe, is not yet proven to be safe and is thus controversial.[20]

A. William Paulsen, MMSc, PhD, CCE, AAC
Vice-Chair, Committee on Technology
with
Gunnar Klauss, MD, MS
Resident, Department of Anesthesiology
Wake Forest University School of Medicine

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A Statement by the Executive Committee of the APSF

From time to time, the Anesthesia Patient Safety Foundation reconfirms its commitment of working with all who devote their energies to making anesthesia as safe as humanly possible. Thus, the Foundation invites collaboration from all who administer anesthesia, all who supply the tools of anesthesia, and all who provide the settings in which anesthesia is practiced, all individuals and all organizations who, through their work, affect the safety of patients receiving anesthesia. All will find us eager to listen to their suggestions and to work with them toward the common goal of safe anesthesia for all patients.

More Q&A

"Q&A," From Preceding Page

On the Horizon: Questions Regarding Circuit Reuse and Filters

Q Dear Q&A,

We are trying to determine 1) a proper cleaning procedure for our machines, 2) the parts of these machines that are considered "reusable breathing components," and 3) the necessity and costs associated with a routine disinfection protocol.

Randy Blessing, BMET
Augusta, GA

Q Dear Q&A,

We have been approached about using a filter device placed at the end of the Y-piece which supposedly allows you to protect the circuit from contamination and REUSE it! This is supposed to save costs and minimize storage and waste. Is this safe?

Steve Kimatian, MD
Hershey, PA

Our facility is considering the use of a filter that the manufacturer states can allow reuse of the circuit for the entire day. The filter is changed at the end of each day. The facility is strongly encouraging the use of this device. Are you aware of any independent data on these filters?

Robert Ponte, MD
Orange Park, FL

In an effort to decrease costs, the facility director of our ASC would like us to consider using filters on our anesthesia circuits, changing only masks between patients. Is this an acceptable practice? Any recommendations for what to look for in a filter? Can they be used for pediatric patients as well as adults? How often do the filters have to be changed? Other than resistance are there other patient risks? Infections? Do many other groups do this?

Mark J. Shulkosky, MD
Erie, PA

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Appendix

In order to access the referenced CDC Guidelines in PDF format please use the following convenient hyperlink:

http://www.premierinc.com/safety/topics/guidelines/cdc_guidelines.jsp

The cited ASA Recommendations, also in PDF format, can be accessed through:

http://www.asahq.org/publicationsAndServices/infectioncontrol.pdf

Letter to the Editor

Anesthesia Distractions

To the Editor:

The concern of reading during the administration of anesthesia has been debated.[1] There is agreement that there are many distractions in the operating room during the administration of an anesthetic. One area of concern I have noticed is anesthesia providers entering the OR with a laptop computer or personal digital assistant (PDA). They often turn on the device and surf the web during the conduct of the anesthetic. A lively debate occurred with the subject of reading, but I wonder if the use of a personal computer adds a new dimension to the topic of distractions during surgery.

Steven Dean, CRNA, MS
McKinney, TX

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Perioperative Coronary Stent Thrombosis: *A Continuing Safety Concern*

by Steven B. Greenberg, MD; Eric C. Matten, MD; Glenn S. Murphy, MD; and Jeffery S. Vender, MD FCCM, FCCP

Today, more patients are presenting for non-cardiac surgery (NCS) after recent placement of either Bare Metal (BMS) or Drug-eluting (DES) stents. Stent thrombosis is an infrequent but devastating complication after stent implantation. This complication accounts for up to a 60% acute myocardial infarction rate and up to a 45% mortality rate.[1] Some notable risk factors for stent thrombosis include the presence of bifurcation and small vessel lesions, suboptimal angiographic results, high-risk patients (i.e., diabetes mellitus and renal failure), and most importantly, cessation of dual antiplatelet therapy (i.e., aspirin and clopidogrel).[2] Non-cardiac surgery performed following recent stent placement poses an additional risk of thrombosis due to the inherent hypercoagulable state of surgery as well as the common practice of discontinuing antiplatelet therapy perioperatively. Most perioperative stent thromboses occur intra- or postoperatively, as opposed to the preoperative "drug free" period. Proposed reasons include the prothrombotic nature of surgery, the time needed to synthesize new "aspirin-free platelets," the wash-out of thienopyridine, or a combination of all of the above.[2,3]

Given the complexity of this newly recognized perioperative phenomenon, it is not surprising that one survey reported that 63% of Canadian anesthesiologists did not know the published guidelines regarding the timing of elective surgery following stent placement.[4] Part of the reason for this lack of knowledge is the paucity of definitive data to guide physician perioperative decision-making. This review will focus on some of the issues facing healthcare practitioners including the recommended timing of elective NCS following stent placement, the perioperative management of antiplatelet therapy prior to elective NCS, and the perioperative management of antiplatelet therapy prior to urgent/emergent surgery.

In June 2008, the American College of Chest Physicians (ACCP) published guidelines related to this topic.[5] The emerging consensus suggests that, when possible, surgery should be delayed for at least 1 week following percutaneous transluminal coronary angioplasty (PTCA) without stent placement, with the recommended delay being extended to at least 4-6 weeks for BMS and for at least 12 months following DES placement.[5,6] The difference in recommended waiting periods for BMS versus DES is due to the slower rate of endothelialization for DES. These guidelines also state that patients who undergo surgery within 6 weeks of BMS placement or within 1 year of DES placement should continue perioperative dual antiplatelet therapy (Class IC).[5,6] However, these guidelines are based on expert opinion, case series, and small retrospective studies.

Two recently published large retrospective analyses by Nuttall et al. and Rabbitts et al. support these recommendations.[7,8] They investigated the rates of major adverse cardiac events (MACE-defined by a composite of death, myocardial infarction, stent thrombosis, and repeat revascularization) in patients undergoing NCS following either BMS[7] or DES[8] placement. The authors found that MACE occurred in 10.5% of patients when surgery was performed within 30 days of BMS placement, but dropped to 3.8% and 2.8% when performed between 30 and 90 days, and >90 days after BMS, respectively.[7] Non-cardiac surgery within 90 days of DES placement resulted in a 6.4% rate of MACE, but declined to rates comparable to BMS (3.3%) only when surgery was delayed for at least 1 year.[8] Avoiding elective surgery during these vulnerable periods is the optimal way to mitigate this perioperative complication.

The risk of perioperative bleeding associated with antiplatelet therapy must be weighed against the catastrophic event of stent thrombosis from discontinuation of antiplatelet therapy. A review by Chassot et al. recommended that all patients continue aspirin throughout the perioperative period, except in cases where excess bleeding could have irreparable consequences (i.e., intracranial surgery).[9] The authors further state that when feasible, patients should continue dual antiplatelet therapy perioperatively. Both recent retrospective studies by Nutall et al. and Rabbitts et al. reported that perioperative bleeding was not associated with perioperative antiplatelet therapy.[7,8] Therefore, many cardiologists endorse continuing dual antiplatelet therapy indefinitely, especially during low risk bleeding procedures.[2] For those patients who must undergo urgent or emergent high risk bleeding procedures, bridging therapies such as unfractionated heparin, low molecular weight heparin, direct thrombin inhibitors, or glycoprotein IIb/IIIa inhibitors have been proposed for utilization. Presently, there is a lack of evidence supporting bridging therapy, and, therefore, the ACCP guidelines echoed by the recent practice alert in *Anesthesiology* do not suggest its routine use.[5,6] If surgery must be performed in patients that must have their thienopyridine therapy interrupted, it is recommended that aspirin be continued and the thienopyridine be started as soon as possible after the procedure.[6] Further studies need to be performed to investigate alternative therapies for reducing the risk of stent thrombosis in patients undergoing emergent high risk bleeding surgeries.

In the absence of universally accepted protocols for management of patients who present for NCS following recent stent placement, it is necessary for collaborative decision making to take place between the patient, internist, surgeon, anesthesiologist, and cardiologist. We strongly encourage this multidisciplinary discussion to include the type and timing of stent placed, the importance of the type of surgery being considered, the management of perioperative antiplatelet therapy, and the choice of facility at which to perform the surgery. If surgery needs to be performed in patients with recent stent placement, if possible, it should take place where a 24-hour interventional cardiologist is available, as emergent PCI remains the best treatment option.[2]

Acknowledgments

The authors wish to thank John G. T. Augoustides, MD, FASE, FAHA (University of Pennsylvania Medical Center, Philadelphia) for sharing his expert opinion on this matter.

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Dr. Greenberg is an associate professor, Dr. Matten is an instructor, Dr. Murphy is an associate professor, and Dr. Vender is a professor, all at the Northwestern University Feinberg School of Medicine, NorthShore University Healthsystem, Evanston, IL.

Intraoperative Hypercapnea During Thoracoscopy—A Case Report

by Ann Musgjerd, MD; David Beebe, MD; and Richard C. Prielipp, MD

Introduction

Thoracoscopy results in less blood loss, less postoperative pain, less respiratory dysfunction, and a faster recovery than open thoracotomy. To facilitate visualization, one-lung ventilation (OLV) and carbon dioxide insufflation into the pleural space optimize the surgical-visual field.[1] While carbon dioxide (CO_2) is usually well tolerated, CO_2 can be readily absorbed through inflamed or disrupted membranes such as the peritoneum or pleura. Anesthesia professionals must be aware of the conditions under which CO_2 may be absorbed in large volumes, as well as its potent biological effects.

We report a patient with extensive pleural adhesions who developed abrupt, extreme hypercapnea following CO_2 insufflation prior to instituting OLV. The cause of this hypercapnea was identified as a surgical rent in the lung producing a bronchopleural fistula—functionally adding large volumes of exogenous CO_2 gas to the "inspired fresh gas" in the trachea. Urgent treatment required placement of a bronchial blocker to isolate the afflicted lung. End-tidal CO_2 concentrations immediately decreased. *We recommend providers routinely consider OLV for thoracoscopy patients where raw pleura is present or there is substantial risk for transection of blebs and bronchioles.*

Case Findings and Management

A 73-year-old male with a long history of tobacco use and chronic obstructive pulmonary disease (COPD) was brought to surgery for thoracoscopic-assisted esophagectomy for esophageal cancer. Seven years prior, he had undergone a right upper lobectomy for cancer. His pulmonary function tests showed an FEV_1/FVC and FEF_{25-75} at 54% and 11% of the predicted values, respectively, along with a significant diffusion defect. Additional significant previous medical history included hypertension, hypothyroidism, gastroesophageal reflux disease, and a moderate size aortic aneurysm.

The surgeon thought insufflation of CO_2 into the pleural space would provide adequate surgical exposure for the esophagectomy without the additional limitations of OLV. Therefore, after intravenous induction of anesthesia with etomidate, fentanyl, and rocuronium, the patient's trachea was intubated with a single-lumen, 8.0-mm endotracheal tube. A left radial arterial catheter and a right internal jugular central venous catheter were also inserted, and the patient was positioned prone for the thoracic portion of the procedure. Anesthesia was maintained with isoflurane, fentanyl, and rocuronium.

The right thorax was initially insufflated to 8 mmHg pressure with CO_2, and all hemodynamic and respiratory parameters were stable. Significant, extensive adhesions were encountered from the previous right upper lobectomy. Lysis of these adhesions and lung mobilization created extensive areas of raw pleural surface and several lung blebs were torn. To improve exposure, the insufflation pressure was increased to 14 mmHg. Less than a minute later the end-tidal CO_2 increased from 33 to 111 mmHg. The central venous pressure increased to a peak value of 20 mmHg and systolic blood pressure decreased to 90 mmHg, which required treatment with phenylephrine. The arterial blood gases demonstrated the following values: pH, 6.98; $PaCO_2$, 131 mmHg; PaO_2, 145 mmHg, HCO_3, 24 mmol/L (see Table 1). Insufflation was terminated and all thoracic CO_2 released, resulting in an immediate reduction in the patient's end-tidal CO_2 and stabilization of the blood pressure.

An Arndt™ bronchial blocker was placed using fiberoptic bronchoscopy in the right mainstem bronchus to isolate the left lung. A second attempt to insufflate the pleural to a pressure of 8 mmHg with CO_2 was well-tolerated, and the procedure continued ventilating only the left lung for the remainder of the thoracic part of the procedure. Although the end-tidal CO_2 remained normal (28-36 mmHg), successive arterial blood gas samples showed a persistent acidosis (pH: 7.11-7.25) and hypercarbia ($PaCO_2$; 60-76 mmHg) despite doubling the minute volume. The patient was turned supine upon completion of the thoracic portion of the procedure and again, both lungs ventilated. The patient's subsequent arterial gases showed near final resolution of the acidosis and hypercarbia following completion of the thoracoscopy: pH, 7.28; $PaCO_2$, 49 mmHg; PaO_2, 320 mmHg; HCO_3, 22 mmol/L. The remainder of the procedure was uneventful. The patient's trachea was extubated the following day and made an uneventful recovery.

Table 1. Vital Signs and ABG during Thoracoscopy in OR

Elapsed Time (min)	$etCO_2$ (mm Hg)	$PaCO_2$ (mm Hg)	pH	PaO_2 (mm Hg)	Vent Settings Vt/rate/PIP	CVP (mm Hg)	BP (mm Hg)	HR (beats/ min)
0 (base)	33	N.A.	N.A.	N.A.	500/12/18	12	145/75	60
30	111	131	6.98	145	500/20/26	14	100/60	64
45	33	76	7.11	220	400/22/34	13	126/62	72
75	32	66	7.16	253	450/26/41	14	120/60	72
105	36	66	7.12	108	400/28/42	13	130/80	80
165	28	60	7.25	100	560/16/42	15	110/60	70
240	24	49	7.28	320	600/16/41	15	110/65	70

Discussion

Thoracoscopy results in less blood loss, less postoperative pain, less respiratory dysfunction, and a faster recovery than open thoracotomy.[2-4] In addition, insufflation of CO_2 optimizes surgical exposure, which may be used with or without lung isolation and OLV. Occasionally, patients with limited pulmonary reserve may require thoracoscopy with only CO_2 insufflation (while ventilating both lungs) to maintain respiratory homeostasis.[5] We report a case of thoracoscopic-assisted esophagectomy in which CO_2 insufflation while maintaining two-lung ventilation was the initial approach for surgical exposure in the right chest. However, extreme hypercapnea and hypercarbia developed unexpectedly following a surgical request to increase CO_2 insufflation pressure. Severe respiratory acidosis required urgent placement of a bronchial blocker and institution of OLV.

Carbon dioxide is the gas used most commonly for insufflation for both laparoscopy and thoracoscopy. It is nonflammable and, in comparison to other gases, extremely soluble. Soluble gases such as CO_2 are much safer in the event of inadvertent gas embolism than less soluble gases such as air or helium.[6] However, the high solubility increases its rate of absorption across membranes such as the pleura or peritoneum. Significant absorption of CO_2 can occur during thoracoscopy and laparoscopy and result in hypercarbia if ventilation is not increased appropriately. High insufflation pressures can increase the rate of CO_2 absorption by increasing the

See "Hypercapnea," Page 23

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Intralipid Treatment of Bupicavaine Toxicity

by Pete Stiles, BA, and
Richard C. Prielipp MD, MBA, FCCM

Background

Cardiac toxicity associated with overdose of intravascular injection of local anesthetic is characterized by hypotension, atrioventricular conduction delay, idioventricular rhythms, and eventual cardiovascular collapse. Although all local anesthetics potentially shorten the myocardial refractory period, bupivacaine avidly blocks the cardiac sodium channels, thereby making it most likely to precipitate malignant arrhythmias. Even levobupivacaine and ropivacaine (single-enantiomer derivatives), developed to ameliorate cardiovascular side effects, still harbor the potential to disrupt cardiac function.

Data suggest up to 20 out of 10,000 peripheral nerve blocks and 4 per 10,000 epidural blocks result in systemic local anesthetic toxicity. As current practice often favors the addition of regional anesthesia and major plexus blocks to supplement or substitute for general anesthesia, all anesthesia professionals must be familiar with signs of local anesthetic cardiotoxicity—and current treatment options.

Lipid to the Rescue?

While *pretreatment* with a lipid infusion in rats was found to increase the dose of *i.v.* bupivacaine required to induce asystole, subsequent studies examined resuscitation in dogs with lipid emulsion *after* an intravenous dose of bupivacaine. Researchers found substantially improved hemodynamics and myocardial metabolism.[1] Thus, by 2006, many touted "lipid rescue" for local anesthetic cardiotoxicty and suggested that anesthesiologists routinely stock lipid emulsions wherever regional anesthesia was practiced. Some challenged these conclusions on grounds that severe systemic toxicity from local anesthetics occurs with far greater frequency than is published in medical literature, and that the most appropriate way to limit the hazards of local anesthetics is to prevent complications with proper injection techniques and careful dosing.

Case Reports Document Lipid Rescue

One case report describes a 58-year-old man who, 30 seconds after an interscalene injection, developed a tonic-clonic seizure and cardiac arrest. Prolonged ACLS failed to restore a perfusing rhythm, so 100 mL of 20% Intralipid was rapidly infused while maintaining cardiac compressions and preparing for cardiopulmonary bypass. Remarkably, the first defibrillation after lipid administration restored a sinus rhythm, and cardiovascular performance now responded to inotropes and vasopressors. Intralipid 0.5 mL/kg/min was infused for 2 hours, during which time the patient regained full consciousness and recovered without neurological sequelae.[2] While this case suggests lipids might be routinely stocked in areas in which peripheral nerve blocks are performed[2], the high-dose safely profile of Intralipid is unknown, and other questions also remain:

1. What is the mechanism of action of lipid rescue?
2. Is the beneficial effect of Intralipid promoted or hindered by concurrent drug therapy administered via ACLS protocol?

Currently, 12 published cases support lipid rescue in the setting of local anesthetic cardiotoxicity, where early administration of Intralipid is emphasized. Fortunately, it appears that the beneficial effect of Intralipid administration also includes local anesthetics other than bupivacaine.[3]

Proposed Mechanisms

The mechanism by which lipids reverse local anesthetic cardiotoxicity may be increasing clearance from cardiac tissue. This nonspecific, observed extraction of local anesthetics from aqueous plasma or cardiac tissues is termed a "lipid sink."[4] Another proposed mechanism is that lipids counteract local anesthetic inhibition of myocardial fatty acid oxidation, thereby enabling energy production and reversing cardiac depression.

Caution is Still Prudent

The ultimate role of lipid rescue is still debated as some suggest that successful resuscitation could be due to spontaneous clearance of the instigating local anesthetic within 20 minutes of routine ACLS. Others caution that prevention is always more appropriate—and the concept of a "remedy" could make some practitioners less careful.[5] Moreover, while lipid rescue may be the driving force behind successful cardiac resuscitation, the risk to the brain from prolonged circulatory collapse remains.[5] Thus we emphasize that primary therapy remains adherence to proven guidelines—cardiac and SpO_2 monitoring, proper availability and dosing of all local anesthetics, immediate means to support ventilation, proper cardiac compressions during CPR, and application of proven advanced life support techniques. Only then should lipid rescue be considered in the therapeutic algorithm.

What Should Clinicians Conclude?

Assertion of a unique role for Intralipid with new ACLS protocol guidelines[6] must be tempered by awareness that the appropriate dose of Intralipid for resuscitation remains unknown and that excess lipid may interfere with lipophilic ACLS drugs. Current doses vary widely, and pediatric dosing recommendations are even more elusive. Nonetheless, in a survey completed in 2006, respondents from 90 academic anesthesiology departments revealed that 26% would consider using lipid rescue in the setting of local anesthetic toxicity—and that the more major nerve blocks performed at an institution, the more likely they were to use lipid rescue.[7] Thirty-nine percent of institutions stored Intralipid in the OR pharmacy, 35% in the hospital pharmacy, 22% in the "code box," and 4% in a drug-dispensing device in the OR. More than half of the centers specified that the drug was accessible in less than 10 minutes. The Association of Anaesthetists of Great Britain and Ireland recently provided members with protocols to treat local anesthetic cardiotoxicity that include an infusion of lipid emulsion.[8] In an editorial published in *Anesthesia & Analgesia*, Brull explains, "based on the available data, it would seem reasonable to have a [lipid] rescue kit available in any setting in which regional anesthesia is practiced—and, in fact, in any location where local anesthetics are administered by any professional, by any route, and in almost any dose."[9] Moreover, it will be critical to support further investigation of lipid rescue.

Thus, anesthesia professionals should consider this alternative when a patient shows signs and symptoms of local anesthetic toxicity with, or even before, failing CPR. A useful website, www.lipidrescue.org, is dedicated to the discussion and promotion of lipid emulsion reversal of local anesthetic systemic toxicity. Here, the latest data and case reports are synthesized. Readers are cautioned that human prospective studies have not yet been reported, so a registry of local anesthetic-associated cardiac arrests is being planned. Indeed, acknowledging the limited understanding of lipid therapy, many questions remain:

- Should the lipid dose be titrated, by patient weight, local anesthetic dose, or the symptoms/signs/severity of toxicity?
- What is the best rate and total dose of the infusion that follows bolus dosing? Is there a safe upper limit of lipid dosing?
- How long should the patient receive the lipid infusion?
- What is the risk of reoccurrence of toxicity once the lipid infusion is stopped?
- Should lipid emulsion be used for patients exhibiting signs of CNS toxicity, or should intralipid only be used for cardiac toxicity?
- What are the possible complications or adverse effects of lipid infusion?
- Should lipid be used alone or in combination with epinephrine, and other components of standard resuscitative measures?
- What is better, 20% or 30% lipid? What formulation is best?
- Intralipid has been used predominantly so far, but is there a better choice?

See "Lipids," Next Page

Reasonable Dosing Algorithm Proposed

"Lipids," From Preceding Page

- Do the other available lipid emulsions work as well?

With all the limitations noted above, one plausible dosing application to consider after "all standard resuscitation methods fail to re-establish sufficient circulatory stability" would be as follows:

20% Intralipid:

1. Administer 1.5 mL/kg as an initial bolus; the bolus can be repeated 1- 2 times for persistent asystole.
2. Start an infusion at 0.25 mL/kg/min for 30-60 minutes; increase infusion rate up to 0.50 mL/kg/min for refractory hypotension.[3,10]

Pete Stiles, BA, is a senior medical student at the University of Minnesota Medical School. He is expecting to be awarded his medical degree in May 2009. Dr. Prielipp is the JJ Buckley Professor and Chair of the Department of Anesthesiology at the University of Minnesota Medical School, Minneapolis, MN

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Letter to the Editor

In a Specified Context, Ketamine is Non-Adrenergic and Provides Preemptive Analgesia

To the Editor:

Dr. Durieux's article "Does anesthetic management affect cancer outcome?" in the winter 2008-9 *APSF Newsletter* was really very interesting. However, his non-contextual inclusion of ketamine as a "bad" drug might discourage APSF readers from administering BIS-monitored propofol ketamine (PK) anesthesia[1] to their patients. In the context in which Dr. Durieux cites the use of ketamine,[2] as well as its historically established reputation,[3] it is unquestionably an adrenergic stimulator. However, that context ignores earlier (and later) published work that demonstrates a lack of hypertension, tachycardia, and hallucinations.[4-7] These publications demonstrate a context in which ketamine is not an adrenergic stimulator; namely that in which propofol is incrementally titrated to BIS <75 prior to the administration of a 50 mg dissociative dose of ketamine and 2-3 minutes prior to injection or incision.[1]

BIS monitoring was added to PK anesthesia in 1997. It serves a 2-fold purpose. First, BIS defines a level of propofol sedation at which ketamine can be given without the adrenergic side effects. Second, during the case, it helps the anesthesiologist educate the surgeon (when possible) when the patient requires re-injection of small amounts of local anesthesia, despite the appearance of a blanched field. Conducting a case in this manner prevents the patient from experiencing pain during the initial injection of local and, subsequently, during the surgery itself. This, plus the decreased catecholamine state from the preoperative oral clonidine premedication, sets the stage for minimal postoperative discomfort. Not only is this specific context devoid of adrenergic stimulation, but also it provides the patient with the benefits of preemptive analgesia, avoiding the postoperative pain Dr. Durieux says "may play a very important role in metastasis after cancer surgery."

Postoperatively, patients receiving BIS/PK MAC have only required oral Tylenol®, Tylenol PM®, or IV Toradol®, even for abdominoplasties, not morphine PCA. No hospital admissions have resulted for unmanageable pain of PONV since inception of PK MAC in 1992. Absent a Level I study to establish reproducibility, BIS/PK MAC has been administered for more than 100 different surgeons over a decade in >2,500 patients. This clinical experience should strongly suggest reproducibility. Other anesthesiologists have also reported similar outcomes when following the clinical pathway referenced herein. Interested readers may access the pertinent algorithms from the home page of my website (www.drfriedberg.com).

Barry L. Friedberg, MD

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Letter to the Editor

A Few Suggestions for Reduction of Medication Errors

In a consumer update dated April 2008, the FDA stated that they have received reports of more than 95,000 medication errors since the year 2000.[1] This corresponds to more than 33 medication errors per day. Poor packaging design has been identified as one of the causative factors.

Studies have indicated that most medication errors in anesthesia are due to either preparation or administration of the wrong drug.[2] Suggestions to minimize these errors have included careful reading of labels, standardization of labels, and formal organization of drug drawers and workspaces.[3]

It seems that the vial caps have been ignored. On opening an anesthesia drug cart, the anesthesiologist may get a view of medications similar to that in Figure 1. Although the vial cap is the most visible part of the vial, it typically contains no information about the vial contents. This is due in part to a lack of coordination among different suppliers.

Table 1. Sample list of look-alike drug packaging

	Naloxone (N) vs. Clindamycin (C)
	Phentolamine (P) vs. Ketorolac (K)
	Ephedrine (E) vs Phenyleprine (P) vs Adenosine (A) vs. Metoclopramide (M)
	Diphenhydramine (D) vs Verapamil (V) vs. Gentamicin (G)

Figure 2. Photo of 2 similar sized vials of atropine with similar appearance but differing concentrations.

Figure 1. Anesthesia drug cart showing misplaced vials denoted by red letters -refer to Table 1.

With most anesthesiologists experiencing at least one drug error in their career,[4] we suggest that vial cap color be standardized similar to the current syringe labeling system (e.g., bright red for muscle relaxants, purple for vasoactive drugs, etc.). Similar color coding could be used for the label, or for the ink on the label, as well.

See "Errors," Next Page

Letters to Editor

Similar Vial Size Potentially Confusing

"Errors," From Preceding Page

Another potential source of confusion is the supplying of varying doses and concentrations of a medication in the same size vial (Figure 2). We suggest that, whenever possible, larger doses and higher concentrations of a medication be supplied in a larger size vial.

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Reader Notes Uncommonly Large Dose of Glycopyrrolate

To the Editor:

Regarding the previous letter to the editor by Dr. Li in the Winter 2008-2009 issue of this *Newsletter*, glycopyrrolate and dexamethasone come in vials with concentrations of 0.4 mg/ml and 4 mg/ml, respectively, for a good reason. Typical doses of glycopyrrolate and dexamethasone are 0.01 mg/kg and 0.1 mg/kg respectively. A 15-kg girl would more commonly be given dexamethasone 1.5-2 mg IV; 20 mg is excessive and not commonly given in that dose.

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Pleural Adhesions Prompt Caution

"Hypercapnea," From Page 18

CO_2 gradient across the biological membrane surface. In addition, large increases in intrapleural pressures can directly diminish venous return, cardiac output, and blood pressure.[7]

This patient's previous chest surgery (right lobectomy) produced significant adhesions that required significant surgical dissection. In addition, large lung blebs were disturbed and produced one or more direct broncho-pleural fistula(s). Thus, insufflated CO_2 gas had a direct pathway into the conducting bronchioles, resulting in dramatic hypercapnea. This diagnosis was confirmed by immediate correction of the increased $etCO_2$ after inflation of the bronchial blocker. The persistent elevation of $PaCO_2$ and acidosis despite OLV involved multiple factors. The pleural surface was newly dissected, enhancing CO_2 absorption. Moreover, CO_2 elimination is also impaired in patients with increased dead space. In a report of 10 patients undergoing series of thoracoscopic assisted esophagectomy where OLV with a double lumen tracheal tube and CO_2 insufflation in the right pleural cavity was used, a 10 mmHg rise in the end-tidal CO_2 was noted in all patients that lasted until the end of surgery in spite of increasing the minute volume ventilation.[7]

Conclusion

The airway in patients with extensive pleural adhesions scheduled for thoracoscopy procedures should be secured with a double-lumen tracheal tube or at least with provision for blockade of the affected bronchus during CO_2 insufflation. OLV prior to CO_2 insufflation will prevent the inflow of exogenous CO_2 gas through iatrogenic broncho-pleural fistulas created during dissection.

Affilated with the University of Minnesota, Minneapolis, MN: Ann Musgjerd, M.D., CA-1 resident in Anesthesiology; David Beebe, M.D., Professor of Anesthesiology and Program Director; and Richard C. Prielipp, MD, MBA, JJ Buckley Professor and Chair, Department of Anesthesiology

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