



# NEWSLETTER

The Official Journal of the Anesthesia Patient Safety Foundation

Volume 22, No. 2, 25-40

Circulation 81,489

Summer 2007

## Beach Chair Position May Decrease Cerebral Perfusion *Catastrophic Outcomes Have Occurred*

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

### Case Presentation

A 47-year-old, healthy female underwent general anesthesia for shoulder arthroscopy. Preoperative blood pressure (BP) was 125/83 mmHg. After premedication with 50 mg of meperidine, 40 mg hydroxyzine, and 0.2 mg glycopyrrolate intramuscularly, anesthesia was induced with 200 mg propofol, 100 mg succinylcholine, and 30 mg lidocaine. Because she was hypertensive, just prior to induction, 50 mg of labetalol was given intravenously. Anesthesia was maintained with 2% isoflurane, 60% nitrous oxide, and oxygen. The patient was placed in the "barbershop" position for the surgery. Twenty minutes into the case, blood pressure decreased to 100/60 mmHg and then remained in the 80-90 mmHg systolic range for the remainder of the case. Oxygen saturation was 100% and end tidal CO<sub>2</sub> values were in the 30s throughout the case. Upon arrival in the post-anesthesia care unit (PACU), her blood pressure was 113/60 mmHg but she did not awaken. Naloxone 0.1 mg was given intravenously, but she remained unresponsive and did not move her extremities. Another 0.1 mg of naloxone was given 35 min after arrival in the PACU followed by 3 more doses of naloxone and 2 doses of physostigmine. During this time, her trachea remained intubated and she was well oxygenated. Neurologic evaluation suggested a diencephalic syndrome, possibly brain infarction. She was unresponsive to voice commands or painful stimuli, and reflexes were decreased bilaterally. A computer axial tomography (CAT) scan of the head was normal initially, but 5 days later suggested brain swelling and obliteration of the cistern. Magnetic res-

onance imaging (MRI) 1 week later showed changes in both cerebral hemispheres suggesting cortical infarcts, involvement of the anterior and medial temporal lobe bilaterally, no significant edema, and no significant herniation. At no time was there any evidence of an intracranial bleed. After 2 weeks, her Glasgow coma scale was 3; her fundi were clear and crisp. She had corneal reflexes, a positive gag, and negative doll's eyes; she was hyperreflexive with increased tone and was unresponsive to noxious stimuli in all 4 extremities. She is expected to remain in a persistent vegetative state.

### Considerations When Using the Beach Chair Position

The beach chair (barbershop) position was developed in the 1980s for orthopedic shoulder arthroscopy procedures. Patients are sat up at angles varying from 30-90° above the horizontal plane with appropriate padding and with the head secured in a headrest. Injuries to the brachial plexus are reduced compared to the lateral decubitus position, and the surgeon has excellent access to the shoulder. The position helps the surgeon because the weight of the arm distracts the shoulder joint while avoiding distortion of the intra-articular anatomy.

However, significant changes can develop when patients are moved to the upright position. Mean arterial pressure (MAP), central venous pressure (CVP), pulmonary artery occlusion pressure (PAOP), stroke volume, cardiac output, and PaO<sub>2</sub> all decrease while the alveolar-arterial oxygen gradient (PAO<sub>2</sub>-PaO<sub>2</sub>), pulmonary vascular resistance, and total peripheral

resistance increase. Under nonanesthetized conditions, these effects are compensated for by an increase in systemic vascular resistance by up to 50-80%. However, this autonomic response is blocked by vasodilating anesthetics, which further exacerbate and compromise cardiac output. Blood pressure remains unchanged or increases slightly in nonanesthetized patients in the sitting position but decreases in the anesthetized state. Cerebral perfusion pressure (CPP) decreases by approximately 15% in the sitting position in non-anesthetized patients and could further decrease under anesthesia because of vasodilation and impaired venous return. Venous return from the cerebral circulation is usually increased by inspiratory subatmospheric pressure during spontaneous ventilation, but this mechanism is nullified by positive pressure ventilation. Obstruction of the internal jugular veins in the sitting position may also impede cerebral venous drainage, especially with unfavorable positions of the head and neck, such as flexion of the head. Pohl and Cullen reported a series of cases in 2005 that documented blood pressure decreases ranging from 28-42%; consequently, hypotension was thought to be a likely cause of ischemic brain injury. Given the potential for peripheral vasodilatation and myocardial depression that can occur in patients who are anesthetized with potent intravenous and inhalational drugs, the effects of the upright position and anesthesia synergize.

Cerebral autoregulation has been thought to maintain cerebral blood flow (CBF) constant between MAP of 50-150 mmHg. However, it must be remembered that in poorly controlled hypertensive patients, autoregulation of CBF is shifted to the right, requiring higher CPP/MAP to ensure adequate cerebral perfusion. In recent years, Drummond and others have emphasized that the quoted value of 50 mmHg for the lower limit of autoregulation (LLA) should be modified upward to reflect a range of values from 70-93 mmHg with a mean value of 80 ± 8 mmHg rather than the specific number of 50 mmHg. Some orthopedic surgeons request deliberate hypotension for shoulder surgery. With the acquiescence of the anesthesiologist or the nurse anesthetist, deliberate hypotension to mean arterial pressures of 50-60 mmHg eliminates any margin for error in case blood pressure falls further. In addition, neither the surgeon nor the anesthesiologist nor the CRNA typically seems to consider the added effect of the beach chair position on cerebral perfusion.

### Inside:

Review of Maternal Arrest Cases .....	Page 28
Cardiology Perspective on Stents .....	Page 32
Dear SIRS: Alarm Requires Selection of Heart Rate Source .....	Page 34
Donor List .....	Page 35
Case Report: Wrap Delays Detection of Disconnect .....	Page 36
Q&A: Pipeline Pressure Primer .....	Page 38

See "Perfusion," Page 27

# Physicians Lee and Lofsky Named Consultants to Executive Committee

Dr. Lorri Lee and Dr. Ann Lofsky have recently been appointed consultants to the APSF Executive Committee. Their experience and expertise will certainly make them important additions to our organization.

Lorri A. Lee, MD, received her training in anesthesiology at the University of Washington where she is currently an associate professor in the Departments of Anesthesiology and Neurological Surgery (adjunct). As an investigator for the ASA Closed Claims Project, her research is focused on evaluating trends and associated factors in anesthesia-related patient injuries and medical liability. Dr. Lee is director of the ASA Postoperative Visual Loss Reg-

istry and served on the ASA Perioperative Blindness Advisory Task Force

Ann Lofsky, MD, completed both internal medicine and anesthesiology residencies at UCLA before entering private practice in anesthesiology. She is currently a partner in the anesthesia group at Saint John's Hospital in Santa Monica. For 13 years, Dr. Lofsky served on the board of directors of The Doctors Company, a physician-owned medical malpractice insurer. In that capacity, she has written and lectured extensively on risk management and patient safety concerns for anesthesia providers. She is now a governor emeritus and anesthesia consultant for The Doctors Company.



Lorri Lee, MD (left), and Ann Lofsky, MD (right), with Dr. Robert Stoelting, president of the APSF.



www.apsf.org

## NEWSLETTER

The Official Journal of the Anesthesia Patient Safety Foundation



The **Anesthesia Patient Safety Foundation Newsletter** is the official publication of the nonprofit Anesthesia Patient Safety Foundation and is published quarterly at Wilmington, Delaware. Annual contributor status: Individual-\$100, Corporate-\$500. This and any additional contributions to the Foundation are tax deductible. © Copyright, Anesthesia Patient Safety Foundation, 2007.

The opinions expressed in this *Newsletter* are not necessarily those of the Anesthesia Patient Safety Foundation or its members or board of directors. Validity of opinions presented, drug dosages, accuracy, and completeness of content are not guaranteed by the APSF.

**APSF Executive Committee:**  
 Robert K. Stoelting, MD, President; Jeffrey B. Cooper, PhD, Executive Vice President; George A. Schapiro, Executive Vice President; Paul A. Baumgart, Vice President; David M. Gaba, MD, Secretary; Casey D. Blitt, MD, Treasurer; Sorin J. Brull, MD; Robert A. Caplan, MD; Nassib G. Chamoun; Robert C. Morell, MD; Michael A. Olympio, MD; Richard C. Prielipp, MD; Matthew B. Weinger, MD. Consultants to the Executive Committee: John H. Eichhorn, MD; Lorri A. Lee, MD; and Ann S. Lofsky, MD.

**Newsletter Editorial Board:**  
 Robert C. Morell, MD, Editor; Sorin J. Brull, MD; Joan Christie, MD; Jan Ehrenwerth, MD; John H. Eichhorn, MD; Lorri A. Lee, MD; Rodney C. Lester, PhD, CRNA; Glenn S. Murphy, MD; Denise O'Brien, MSN, RN; Karen Posner, PhD; Andrew F. Smith, MRCP FRCA; Wilson Somerville, PhD; Jeffery Vender, MD.

Address all *general, contributor, and subscription* correspondence to:  
**Administrator, Deanna Walker**  
 Anesthesia Patient Safety Foundation  
 Building One, Suite Two  
 8007 South Meridian Street  
 Indianapolis, IN 46217-2922  
 e-mail address: walker@apsf.org  
 FAX: (317) 888-1482

Address *Newsletter* editorial comments, questions, letters, and suggestions to:  
**Robert C. Morell, MD**  
 Editor, *APSF Newsletter*  
 c/o Addie Larimore, Editorial Assistant  
 Department of Anesthesiology  
 Wake Forest University School of Medicine  
 9th Floor CSB  
 Medical Center Boulevard  
 Winston-Salem, NC 27157-1009  
 e-mail: apsfeditor@yahoo.com



www.apsf.org

## Anesthesia Patient Safety Foundation

is pleased to announce the  
**APSF/American Society of Anesthesiologists (ASA)  
 Endowed Research Award**  
 in full support (\$150,000) of a grant to  
 be awarded in October 2007 for initiation in January 2008.

The funds for this named grant will be provided  
 from the APSF Endowment Fund, which was made possible  
 by the generous contributions of ASA to APSF over  
 the last several years.

# Consider Correction for Cuff Location

## “Perfusion,” From Page 25

In the upright position, MAP at the brain is very different when compared to the site at which the BP is actually measured, usually the arm. Unfortunately this difference may be overlooked. In the supine position, BP measured in the arm and BP perfusing the brain are essentially the same. However, if the patient is upright in the beach chair position, BP will be less in the brain than at the heart or arm. The BP difference will be equal to the hydrostatic pressure gradient between the heart/arm and the brain. For example, suppose the BP at the heart/arm is 120/80 mmHg, (MAP 93 mmHg). If the height of the external auditory meatus (representing the base of the brain) is 20 cm above the heart, the difference in BP at the heart compared to the brain will be 15 mmHg. Thus, the BP at the base of the brain will be 105/65 mmHg (MAP 78 mmHg). Most patients undergoing relatively straightforward procedures such as shoulder arthroscopy or even open shoulder surgery do not have intra-arterial BP monitoring available. Therefore, they do not have a transducer placed at the level of the external auditory meatus to monitor BP at the base of the brain. Instead, it is up to the anesthesiologist and/or nurse anesthetist to correct the BP readings at the arm to account for the height of the brain above the arm. Even accounting for the hydrostatic gradient between the external auditory meatus (base of brain) and the arm does not take into account the added distance from the base of the brain, at the Circle of Willis, to the most cephalic portion of the cerebral cortex, an additional distance of 10-12 cm (depending on the patient's height), which represents a further gradient of about 9 mmHg.

The case presented at the beginning of this article suggests that the gradient between the arm and brain was not appreciated. Blood pressure was measured at the arm with a non-invasive cuff, but was not adjusted upward to maintain an adequate MAP at the level of the brain during the procedure. Systolic BPs of 80-100 mmHg probably corresponded to MAPs of 50-80 mmHg, but in the beach chair or upright position, MAP at the base of the brain was probably 15-20 mmHg lower, and at the top of the cerebral cortex, another 9 mmHg lower. It is reasonable to estimate a MAP of 30-40 mmHg at the cerebral cortex and a little higher at the brainstem. CAT scan on the fifth postoperative day showed brain swelling and obliteration of the cistern. MRI 1 week later showed cortical infarcts in both cerebral hemispheres and no intracranial bleed. The injury was consistent with the hypoperfusion that occurred intraoperatively.

Estimates of the MAP at the head can be made once the patient is in the beach chair position. The critical variable is the vertical distance between the external auditory meatus and the BP cuff. Once that distance is known, it should be converted to a hydrostatic pressure gradient that then must be incorporated into BP management during the procedure.

To quantitate the hydrostatic gradient, there is a 0.77 mmHg decrease for every centimeter gradient (1 mmHg for each 1.25 cm). In general, the approximate distance between the brain and the site of the BP cuff on the arm in the seated position will be 10-30 cm depending on the angle of the sitting position and the height of the patient; hence the brain MAP will be 8-24 mmHg lower than the measured mean brachial artery

pressure. If the beach chair position is combined with the use of deliberate hypotension, cerebral perfusion will be severely compromised. An even more exaggerated occurrence may develop when the BP cuff must be placed on the leg because the contralateral arm is not available for BP measurement, e.g., in a patient with prior lymph node dissection for breast cancer. In the beach chair position, the legs are considerably lower than the trunk, therefore the BP difference between the BP cuff measured on the leg and the BP in the brain will be even greater than the gradient between the arm and the brain.

The following case illustrates this point. A 54-year-old woman underwent left shoulder replacement surgery in the beach chair position. The patient had no history of hypertension or myocardial infarction. Preoperative electrocardiogram, echocardiogram, thallium scan, and exercise tolerance test were normal. The patient received an interscalene block with 40 ml of 0.5% bupivacaine with epinephrine (1:200,000). Because of a previous mastectomy, a 20-gauge intravenous catheter was placed in the right foot and a noninvasive BP cuff was placed on the calf. There was no documentation that the calf BP was validated to the left arm BP before the patient was anesthetized and the left arm became unavailable. No arterial catheter was placed although deliberate hypotension was used. Anesthesia was induced with 100 mg propofol, 250 mg sodium pentothal, 50 mg rocuronium, and 250 mcg fentanyl. Anesthesia was maintained with 3.5% sevoflurane and 67% nitrous oxide in oxygen. Nitroglycerin, 50 mcg times 3 doses, and labetalol, 5 mg times 4 doses, were used to produce deliberate hypotension. One hour after induction, her systolic BP was between 85-100 mmHg. Two hours later, her BP was 70/40 mmHg and then remained around 90/60 mmHg for the next 40 min, when it decreased to 50/25 and was treated with phenylephrine. Electrocardiography showed sinus rhythm throughout, oxygen saturations were always high, and end-tidal CO<sub>2</sub> was in the high 20s for most of the case. In the PACU, emergence was delayed, and she did not breathe spontaneously. A radial artery catheter was finally placed while she was in the PACU, and BPs were normal. Apnea persisted and her pupils were fixed and dilated. Blood gas analysis on controlled ventilation showed a PaO<sub>2</sub> of 236 mmHg, PaCO<sub>2</sub> of 35 mmHg, and pH of 7.4, with glucose of 92 mg/dl. Neurologic evaluation revealed brain death with no CBF, flat electroencephalogram, no reflexes, no response to pain, and no lesions on the CAT scan. At autopsy the upper spinal cord and medulla were infarcted. The anesthesia equipment tested normal. In this case, not only was deliberate hypotension used to very low values, but BP was measured in the leg while the patient was in the sitting position. One can only imagine how low the BP was in the brain when BP in the leg was 70/40 or 90/60.

In addition to avoiding deliberate hypotension, one must be extremely vigilant and treat aggressively the unexpected hypotension that often occurs during anesthesia in the beach chair position for the reasons enumerated above. These treatments are well known to all anesthesia providers and include careful control of the inhalation anesthetic concentration, adequate and timely fluid administration, and vasopressor infu-

sion, as needed during the time of the procedure when the patient is upright and at risk.

Head position is also important because some degree of head manipulation is required when positioning the patient in the seated position. Most surgeons use a headrest to immobilize the head. Several studies suggest that CBF can be compromised by mechanical obstruction and injury to major veins or arteries. Blood flow reduction in the vertebral artery caused by extension and rotation or tilt of the head may result in posterior brain circulation infarcts.

Finally, hypotension and generalized circulatory instability can result from gas embolism. This rare complication has been reported with both air and carbon dioxide distension of the joint capsule followed by pressurized injection of irrigation fluid. Thus, anesthesiologists and CRNAs should keep the possibility of venous gas embolism in mind during shoulder arthroscopy in the sitting position if sudden cardiovascular collapse occurs.

## Summary

Despite its low incidence, intraoperative stroke associated with shoulder surgery, particularly in healthy patients at no risk for stroke, is a totally unexpected and devastating complication. Patients in the beach chair position are at risk for an intraoperative stroke if borderline low BPs, as measured in the arm, are used without appreciating the effect on CPP and CBF. Because of the specific physiologic changes associated with the sitting position, great care should be applied when using and interpreting BP cuff measurements in the nonoperative arm or, even more so, if leg measurements of BP must be used. Blood pressure values <80% of preoperative resting values should be treated aggressively to enhance the margin of safety. Deliberate hypotension must be avoided. A thorough understanding of the physiologic changes associated with the upright position, and the physical effects of gravity on BP in the brain is crucial to prevent catastrophic neurologic outcome during shoulder surgery in the sitting position.

*Dr. Cullen is formerly Chair of the Department of Anesthesia and Pain Medicine, Caritas St. Elizabeth's Medical Center and former Professor of Anesthesiology at Tufts University School of Medicine in Boston, MA.*

*Dr. Kirby is an Emeritus Professor of Anesthesiology at the University of Florida College of Medicine in Gainesville.*

## General References

1. Pohl A, Cullen DJ. Cerebral ischemia during shoulder surgery in the upright position: a case series. *J Clin Anesth* 2005;17:463-469.
2. Drummond JC. The lower limit of autoregulation: time to revise our thinking? *Anesthesiology* 1997;86:1431-1433.
3. Drummond JC, Patel PM. Neurosurgical anesthesia. In: Miller RD, Cucchiara RF, Miller ED, Jr., et al., eds. *Anesthesia*, 5th ed. Philadelphia: Churchill Livingstone, 2000:1903-5.
4. Faure EA, Cook RI, Miles D. Air embolism during anesthesia for shoulder arthroscopy. *Anesthesiology* 1998;89:805-6.

# Doctors Company Reviews Maternal Arrests Cases

## (Reprinted with permission from The Doctors Company)

by Ann S. Lofsky, MD

Anesthesia-related maternal arrest is a feared complication that places the lives of both mother and baby at risk. Literature reviews on the subject have been traditionally hampered by a lack of specifics regarding the care provided and the aging of data by the time it could be collected and analyzed. Valid concerns about the privacy of stricken families, the confidentiality of the healthcare providers involved, and liability risks have likely acted together to prevent the wider dissemination of case specifics in an open forum.

Despite recent advances and changing practice patterns in obstetrical anesthesia, malpractice claim reviews indicate that maternal arrest on labor and delivery continues to result in major morbidity and mortality. The Doctors Company recently reviewed 22 anesthesiology claims that were filed after maternal arrests on labor and delivery wards between 1998 and 2006. Anesthesia care was analyzed at the time of initial medical record review from both Standard of Care and patient safety viewpoints. Characteristics of these claims and expert reviewer comments regarding suggested practice changes that might possibly have avoided the arrests or improved the outcomes are presented here with an aim toward improving maternal safety.

### Overall Outcome

The mothers, aged 17 to 41, suffered the most severe complications post-arrest. Ten out of the 22 died, including 3 who were declared brain dead and removed from ventilators. Eleven suffered degrees of anoxic brain damage, ranging from minimal to severe, but with neurological deficits deemed to be permanent at the time of final claim resolution. Only 1 mother out of the 22 had no apparent residuals post-arrest and was suing primarily for emotional distress.

Surprisingly, the infant outcome in these cases appeared quite different. In all cases where information was available, infants (or children) had been evaluated as developmentally normal at the time of final medical records review. This was true even for the babies born with low Apgar scores, and included not only the 10 babies delivered prior to the maternal arrests, but also the 12 born after their mothers had sustained significant periods of circulatory and/or respiratory arrest.

This disparity suggests that the fetus may well be more resistant to periods of hypoxia and hypotension than is the parturient, and it reaffirms the importance of the anesthesiologist's primary focus being the welfare of the mother. This raises the question as to whether maternal resuscitation should ever be intentionally delayed in order to expedite delivery of the fetus.

### Respiratory Arrests after Regional Anesthetics

The most common scenario in this series (13 patients) was a respiratory arrest following epidural or spinal block. Included in this group were 11 patients who developed unintentionally high neuraxial blockade with resultant apnea and 2 patients who arrested after intravenous sedation was administered post-cesarean section delivery under spinal anesthesia. None of these patients were attached to a maternal monitor with audible alarms at the time of the arrest, making delay in response and resuscitation a frequent reviewer concern.

### Labor Epidurals

There were 8 patients in this series who arrested in labor rooms following attempted insertion and dosing of epidural catheters to relieve labor pains. Of these, 7 had subsequent evidence of unintentional subarachnoid blocks, either by positive aspiration of the catheter for cerebrospinal fluid (CSF) (3 patients) or air in the ventricles on CT scanning (4 patients), presumably introduced into the CSF during injection through the needle or catheter.

All 8 of these arrests occurred within the first 30 minutes of initial catheter placement. In half of the cases, anesthesia providers were not in the room at the time, and the arrest was noted first by other healthcare workers. Reasons given by anesthesia providers for leaving the room after catheter placement included wanting to chart at the nursing desk, being called away to place another labor epidural or to attend to another labor patient's needs, or needing to locate drugs or airway equipment.

In the 1 case in which the mother recovered without obvious neurologic impairment, she was placed supine immediately by the anesthesiologist on initial complaint of difficulty breathing and ventilated with oxygen by Ambu-bag as soon as respirations appeared inadequate. The obstetrician accomplished a crash cesarean section within minutes while still in the labor room, with only a benzodiazepine provided before incision. Blood pressure was supported with IV fluid infusion.

The other 7 cases involved the transfer of a mother in respiratory and/or circulatory arrest from the labor room to the operating room for STAT cesarean section due to fetal distress. In 4 of these cases, there were documented delays in the ventilation of the mother for reasons including initial failure to notice maternal arrest, desire to wait for more optimal intubating conditions in the OR, difficulty locating an Ambu-bag or airway device, or an anesthesia provider not being in attendance. The improved outcome in the 1 case involving immediate resuscitation suggests that the rapid establishment of adequate ventilation and blood pressure support might be crucial factors after unintentionally high spinal blockade.

### Cesarean Sections

There were 5 cases of maternal respiratory arrest following regional anesthesia administered for elective cesarean-section delivery. These all involved spinal anesthetics, possibly because this is a preferred anesthesia choice for purely elective cases. In 2 instances, the mothers received an intravenous benzodiazepine or opioid after delivery; both had also received spinal opioids. Maternal respiratory arrests occurred after delivery in these cases, although there were possible delays in recognition of the arrests.

In the other 3 cases, the mothers received no intravenous anesthetics. One mother arrested immediately after the spinal was placed, with suspected preeclampsia and volume depletion as contributing factors. The other 2 cases involved apparent high spinals, with delay in recognition and/or resuscitation also potential problems.

### Contributing Factors

Morbid obesity, which is known to complicate regional anesthesia, was documented in 3 out of the 8 labor epidural cases and 1 out of the 5 cesarean sections. These proportions would appear to be higher than those present in most labor and delivery populations and suggest that morbid obesity may be a significant relative risk factor for maternal arrest following regional blocks.

Three mothers in this series carried the diagnosis of preeclampsia. Two arrested at the time of induction of anesthesia for cesarean section (1 spinal, 1 general anesthetic). Reviewers raised the possibility of relative hypovolemia in these cases and questioned whether invasive monitoring might have provided useful additional information.

### Arrests after Maternal Hemorrhage

There were 7 cases involving arrests in mothers after massive postpartum hemorrhage—3 after normal spontaneous vaginal deliveries and 4 after cesarean section births. Predisposing diagnoses, when available, included placenta accreta, placental abruption, and traumatic arterial laceration. Knowing there had been a maternal arrest due to hemorrhage, reviewers attempted to identify ways in which the treatment might have been optimized, although it was acknowledged that the size and facilities of the obstetric units involved were varied.

A frequent reviewer impression was that the hemorrhage was so excessive by the time it was diagnosed, it was extremely difficult for the anesthesia provider to "catch up" with the continuing blood loss. Postpartum hemorrhage was not always initially apparent through vaginal bleeding, as it was often primarily internal. The initial presentation was frequently hypotension and/or tachycardia in the

See "Arrest," Next Page

# Respiratory Arrests After Epidural Occurred Within 30 Minutes

## “Arrest,” From Preceding Page

mother, which was usually treated first with intravenous crystalloid and pressors. It was not always clear when continued bleeding should have been suspected as the cause of the maternal vital sign instability.

In some instances, delays in transfusing mothers were related to problems obtaining or transporting blood products from the blood bank, or to an inability to run the blood products through available intravenous lines more rapidly. Some cases involved delays in waiting for crossmatched blood when possibly O-negative or type-specific blood might have been available. Reviewers commented that several patients might have benefited from earlier consideration of additional blood components—including fresh frozen plasma, platelets, or cryoprecipitate. Laboratory tests of serial hemoglobin and hematocrits, coagulation panels, or disseminated intravascular coagulation (DIC) screens were not always ordered.

Better communication might have facilitated transfusion in some cases. On retrospective reviews, potentially improvable delays were identified in informing blood banks of the need for products, in calling for additional medical assistance, or in notifying obstetricians that postpartum patients were hemorrhaging and that surgical intervention (such as exploration, uterine ligation, or hysterectomy) might be required. Potentially useful equipment, such as central line kits or rapid infusion devices, were sometimes available in the facility, but labor and delivery ward personnel might not have known how or where to obtain them.

## General Anesthesia

Included in these maternal arrest cases were 5 general anesthetics and 17 regional blocks. This likely reflects an overall shift toward the use of regional anesthesia in obstetrics. In 3 of the general anesthetics cases, the arrest followed severe postpartum hemorrhage. In those cases, the choice of anesthesia did not likely affect the ultimate outcome significantly. General anesthesia was chosen in 2 cases for postpartum hysterectomy for patients who were already severely bleeding. Only 1 general anesthetic involved a difficult intubation and loss of the airway—traditionally one of the more feared complications of emergency cesarean sections. Interestingly, aspiration of gastric contents, traditionally listed as a leading cause of death in obstetric anesthesia,<sup>1</sup> was not seen in this series of arrests.

## Discussion

The physiological changes of pregnancy undoubtedly contribute to the high incidence of anoxic brain damage and death cases after maternal arrests. The size of the full-term uterus decreases functional residual capacity (FRC) in the mother,

leading to a much more rapid development of hypoxia during periods of apnea than would be expected in the woman's non-pregnant state. The increased oxygen demand of pregnancy further shortens the interval of apnea tolerated before arterial desaturation results. Although a pre-oxygenated, non-pregnant woman may sustain a several-minute period of apnea without desaturating, that same patient at 9 months' gestation breathing room air might not.

Maternal circulation is compromised in the supine position due to compression of the vena cava and aorta by the uterus, decreasing venous return and cardiac output. The necessity of placing an already unstable mother supine, to combat rising spinal levels, to transport her to the operating room, or to manage the airway, may further complicate successful resuscitation.

## Monitoring

Since all respiratory arrests after labor epidurals in these cases occurred within the first 30 minutes after catheter insertion, increased monitoring during this time period would seem a worthwhile consideration. This could be visual—with the anesthesia provider, nurse, or a designee in the room with the patient—or through electronic monitoring of pulse oximetry, capnography, or ventilation, with an alarm audible to responsible personnel. Some birthing facilities have labor and delivery rooms equipped with pulse oximeters that read continuously at the nursing stations, yet that is not currently standard. Many of the maternal arrests following labor epidurals occurred on wards in which only fetal monitoring was transmitted continuously to nurses. The outcomes in those cases suggest that by the time hypoxia due to apnea becomes apparent on a fetal tracing, it might already be too late to prevent anoxic brain damage in the mother.

In 4 cases of planned labor epidurals, the anesthesiologist or CRNA observed symptoms or signs consistent with unintentional spinal blockade prior to the arrest (such as positive aspiration for CSF or maternal complaints of sudden headache or difficulty breathing). Since most labor epidural patients in this series arrested after unplanned subarachnoid blocks, patients for whom there are suspicions of “wet tap” may be at increased risk and might benefit from closer observation and/or monitoring.

Case reviews suggest that keeping pulse oximeter or end-tidal carbon dioxide monitor alarms in an audible mode continuously during cesarean sections is advisable, even after delivery of the newborn. As of October 2005, the American Society of Anesthesiologists (ASA) standards for basic anesthesia monitoring include the statement that whenever pulse oximeters or capnometers are utilized, the low threshold alarms should be audible.<sup>2</sup>

## Ventilation

Rapid recognition of maternal respiratory arrest and restoration of oxygenation and ventilation should be key goals. Airway devices such as self-inflating bag/mask systems, oral and nasal airways, laryngeal mask airways (LMAs), and intubation equipment should be immediately available if needed in labor rooms, with all nurses and anesthesia providers acquainted with their location. The recently revised ASA Practice Guidelines for Obstetrical Anesthesia contain more complete lists of potentially useful equipment.<sup>3</sup>

Because an anesthesia provider may not always be the first to arrive on the scene, labor and delivery nurses should also be able to assess the adequacy of ventilation, establish an airway, and begin ventilation if necessary. Supplemental oxygen should be available in the labor room and immediately available in portable tank form, should transportation of an apneic patient become necessary. Since hypoxia will likely develop rapidly in a full-term apneic patient, adequate ventilation and oxygenation of the mother should ideally be established before transporting her to another location.

## Circulation

As with any resuscitation, maternal blood pressure and circulation should be evaluated and supported, if necessary, with fluids and pressors. CPR should be started as soon as maternal circulation appears inadequate. Since aortocaval compression and an elevated hemidiaphragm can complicate standard CPR, the American Heart Association suggests displacing the uterus to the left by tilting the patient, and performing chest compressions higher on the sternum (slightly above the center).<sup>4</sup>

## Transfusion

Massive hemorrhage on labor and delivery is a rare occurrence, and as a result, many anesthesia providers have little or no experience managing it. Yet, the incidence of major hemorrhage in the obstetric population appears to be increasing over time. The increased rate of repeat cesarean sections, with the associated rise in incidence of placenta previa and placenta accreta, may largely account for this.<sup>5</sup> One New York hospital, after experiencing 2 maternal hemorrhage-related deaths, created a multidisciplinary patient safety team specifically designed to handle labor and delivery patients experiencing major bleeding episodes.<sup>5</sup>

Their obstetric rapid response team includes members of the trauma team, as the individuals identified in that hospital with the most experience in establishing large-bore intravenous lines and massive volume and blood replacement. Efforts were made to identify high-risk patients, who were advised about auto-donation of blood and type and

See “Arrest,” Next Page

# Plan for Worst-Case Scenario

## “Arrest,” From Preceding Page

screened in advance of delivery. A cell saver blood scavenging device was used, when necessary, after the fetus was delivered and after peritoneal lavage.<sup>5</sup>

With these and other interventions, that hospital was able to significantly decrease the number of maternal deaths, despite an increase in the total number of cases of major obstetrical hemorrhage. This suggests that having a pre-planned and coordinated multi-departmental approach to maternal hemorrhage may well advance patient safety.

## Patient Safety

While maternal arrest is, fortunately, a very rare complication, the above cases are a testament to the fact that it still can and does occur—even when currently acceptable anesthesia practices are followed. Anesthesia providers and labor and delivery staff should consider planning their own response to a “worst-case scenario” before it happens to them. A few questions that those providing obstetrical anesthesia may wish to consider:

- If most maternal arrests occur within 30 minutes of the placement of a regional block, how will your patient be monitored during that time period, and who will respond if required?
- If a patient were discovered apneic in a labor and delivery room, where is all potentially necessary airway equipment kept? Would you have access to all the drugs that you might need?
- Are a portable oxygen tank and a bag/mask immediately available for transferring labor patients for crash cesarean sections? Would you need a portable monitor?
- During cesarean sections: As you currently use them, would a monitor alarm notify you if a patient developed apnea at any time?
- How would you and your facility handle an unexpected massive hemorrhage on the labor and delivery ward?
- Who is available to help you with a maternal arrest on labor and delivery, and how would they be notified if needed?

Every case included here was devastating on many levels to the patients, families, and healthcare providers involved. While it is tempting to search for “mistakes” in each individual scenario, the major issues identified were rarely unique. It is hoped that through taking a “systems” approach and focusing instead on the common factors that these cases share, similar occurrences might be prevented and maternal safety improved.

*Ann Lofsky, MD is currently a partner in the anesthesia group at Saint John’s Hospital in Santa Monica, CA, and a governor emeritus and anesthesia consultant for The Doctors Company.*

## References

1. Hawkins JL. Anesthesia-related maternal mortality. *Clin Obstet Gynecol* 2003;46:679-87.
2. Standards for basic anesthetic monitoring. Amended by the ASA House of Delegates October 25, 2005. Available online at: <http://www.asahq.org/publicationsAndServices/standards/02.pdf>. Accessed May 18, 2007.
3. American Society of Anesthesiologists Task Force on Obstetric Anesthesia. Practice guidelines for obstetric anesthesia: an updated report by the American Society of Anesthesiologists Task Force on Obstetric Anesthesia. *Anesthesiology* 2007;106:843-63. Available online at: <http://www.asahq.org/publicationsAndServices/OBguide.pdf>. Accessed May 18, 2007.
4. 2005 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Available online at: [http://circ.ahajournals.org/content/vol112/24\\_suppl/](http://circ.ahajournals.org/content/vol112/24_suppl/). Accessed May 18, 2007.
5. Skupski DW, Lowenwirt IP, Weinbaum FI, et al. Improving hospital systems for the care of women with major obstetric hemorrhage. *Obstet Gynecol* 2006;107:977-83.

## Vision

*The vision of the Anesthesia Patient Safety Foundation is to ensure that no patient shall be harmed by anesthesia.*



## Mission

The APSF’s Mission is to improve continually the safety of patients during anesthesia care by encouraging and conducting

- safety research and education
- patient safety programs and campaigns
- national and international exchange of information and ideas.

# Intralipid Might Save Lives As a Rescue from Bupivacaine Toxicity

*by Robert C. Morell, MD*

The intravascular injection or excessive absorption of bupivacaine can lead to cardiac depression, severe arrhythmias, and/or cardiac arrest, from which resuscitation may be difficult, prolonged, and even impossible. A past issue of this *Newsletter* highlighted the perspective of a patient and her anesthesiologist following a bupivacaine cardiac arrest after a popliteal nerve block.<sup>1</sup> The only reason that the patient survived to tell her story was the heroic and quick action of the anesthesiologist and the resuscitation team at utilizing an available cardiac operating room to institute cardiopulmonary bypass after conventional resuscitative measures were not successful. A new alternative therapy appears to exist. This new and important therapy was emphasized by one of our readers, Dr. Baumgarten, in a Letter to the Editor in the fall 2006 issue of this *Newsletter*. Several case reports (including recent personal communication) and research data consistently indicate that the intravenous administration of intralipid may facilitate and permit successful resuscitation from bupivacaine cardiotoxicity where conventional advanced cardiac life support protocols may fail. Weinberg and colleagues demonstrated that lipid infusion shifted the dose response to bupivacaine-induced asystole in rats and improved survival of dogs from bupivacaine cardiac toxicity.<sup>2,3</sup> As Dr. Baumgarten noted, Dr. Rosenblatt and colleagues published a case report of a successful resuscitation using a 20% lipid emulsion (intralipid), after a bupivacaine-induced cardiac arrest.<sup>4</sup>

Readers should note that other lipid containing medications have not demonstrated such efficacy, and

one should be particularly careful not to assume that propofol would be a safe or effective alternative. Propofol has negative inotropic properties that may cause additional cardiac depression in the setting of bupivacaine-induced cardiac decompensation.<sup>5</sup>

Along with standard resuscitative drugs, it would seem wise to insure the rapid availability of intralipid where regional anesthesia is performed involving the administration of significant quantities of bupivacaine. Certainly, further study is warranted to answer a number of questions including the relationship of intralipid to local anesthetic toxicity caused by agents other than bupivacaine, the optimum dose of lipid emulsion, the potential advantages of lipid infusions vs. bolus dosing, and the optimal interval for redosing.

## References

1. van Pelt FA, Kenney L. Patient and doctor reconcile for greater good. *APSF Newsletter* 2006;21:9-10.
2. Weinberg G, et al. Pretreatment or resuscitation with a lipid infusion shifts the dose-response to bupivacaine-induced asystole in rats. *Anesthesiology* 1998;88:1071-1075
3. Weinberg G, Ripper R, Feinstein D, Hoffman W. Lipid emulsion infusion rescues dogs from bupivacaine-induced cardiac toxicity. *Reg Anesth Pain Med* 2003;28:198-202.
4. Rosenblatt MA, Abel M, Fischer GW, Itzkovich CJ, Eisenkraft JB. Successful use of a 20% lipid emulsion to resuscitate a patient after a presumed bupivacaine-related cardiac arrest. *Anesthesiology* 2006;105:217-8.
5. Groban L, Butterworth J. Lipid reversal of bupivacaine toxicity: has the silver bullet been identified? *Reg Anesth Pain Med* 2003;28:167-9.

# Propofol Safety Review

by Tricia A. Meyer, PharmD

On June 15, 2007, the FDA released a safety alert concerning reports over the past few months of cases of fever, chills, and body aches in several clusters of patients shortly after the administration of propofol. These new cases involved patients undergoing procedures in gastrointestinal suites. The FDA noted that the symptoms were similar to those reported when propofol was first introduced in the US. The postoperative infection in these early cases was attributed to lapses in aseptic technique with risk factors that included "batch" preparation of propofol syringes for use throughout the day, reuse of syringes or infusion pump lines on different patients, use of propofol syringes prepared more than 24 hours in advance, transfer of prepared syringes between operating rooms or facilities, failure to wear gloves during insertion of intravenous catheters, and failure to disinfect the stoppers of the propofol vials. It was also noted that 50-ml and 100-ml vials were used as multi-dose vials. The formulation at that time did not contain preservatives.

In the most recently reported cases, investigators also found use of the single-use vials for multiple patients. To date, tests performed on multiple units of propofol vials and lots by the FDA have not identified any units contaminated with bacteria or endotoxins. Testing of other possible sources such as the lidocaine coadministered with propofol and the instrumentation sterilization systems have not identified any potentially causative agents.

Propofol is marketed as Diprivan® and is also available as a generic disodium edetate. Sodium metabisulfite or benzyl alcohol is added to the propofol to **retard** the rate of microbial growth. Even though the product contains preservatives, microbial growth is still possible and it is not an antimicrobially preserved product under USP standards. The emulsion is capable of supporting microbial growth in the event of contamination during administration due to the level of soybean oil and egg lecithin or egg yolk phospholipids contained in the product.

Recommendations and considerations by the FDA are:

- Both the vial and prefilled syringe formulation must be used on only 1 patient.
- Administration must commence immediately after the vial or syringe has been opened.
- In general anesthesia or procedural sedation: administration from a single vial or syringe must be completed within 6 hours of opening.
- In ICU sedation: propofol administered directly from a vial must be limited to only 1 patient, must commence immediately on opening the vial

and must be completed within 12 hrs of opening the vial.

## Package Insert Guidelines:

- Strict aseptic technique must always be used during handling, including hand washing prior to use.
- Propofol should be visually inspected prior to use for:
  - particulate matter
  - discoloration
  - evidence of separation of the phases of the emulsion.
- Do not use if contaminated.
- Prepare for use just prior to administration to each patient.
- The vial rubber stopper should be disinfected using 70% isopropyl alcohol.
- Discard unused portions within the required time limits.

The FDA urges individuals to report adverse events to the MedWatch Adverse Event Reporting Program.

*Tricia A. Meyer, PharmD, MS, is Director of the Department of Pharmacy and Assistant Professor, Department of Anesthesiology, Scott and White Hospital, Texas A&M Health Science Center, Temple, TX.*

## General References

1. FDA alert/information for healthcare professionals: propofol (marketed as Diprivan and as generic products) Information. U.S. Food and Drug Administration, Center for Drug Evaluation and Research. Available at: <http://www.fda.gov/cder/drug/infopage/propofol/default.htm>. Accessed June 17, 2007.
2. Bennett SN, McNeil MM, Bland LA, et al. Postoperative infections traced to contamination of an intravenous anesthetic, propofol. *N Engl J Med* 1995;333:147-54.
3. Infections linked to lax handling of propofol. *Am J Health Syst Pharm* 1995;52:2061, 2066.
4. Propofol injection. Deerfield, IL, Baxter Healthcare Corporation, Sept. 2004.
5. Propofol injection. Bedford, OH., Bedford Laboratories, July 2005.
6. Diprivan®. Wilmington, DE, AstraZeneca, 2005 (currently Abraxis).

## ANESTHESIA PATIENT SAFETY FOUNDATION

### CORPORATE ADVISORY COUNCIL

- George A. Schapiro, Chair*  
*APSF Executive Vice President*
- Elizabeth F. Holland .....*Abbott Laboratories*  
 Sean Lynch .....*Anesthesia Healthcare Partners*  
 Cliff Rapp .....*Anesthesiologists Professional Insurance Company*  
 Rick Eagle .....*Arrow International*  
 Nassib G. Chamoun .....*Aspect Medical System*  
 Stanley Horton, PhD .....*Bayer Pharmaceuticals*  
 Raul A. Trillo, MD .....*Baxter Healthcare*  
 Michael S. Ferrara .....*Becton Dickinson*  
 Timothy W. Vanderveen, PharmD .....*Cardinal Healthcare*  
 Sarah Hopkins .....*Cerner Corporation*  
 Roger S. Mecca, MD .....*Covidien*  
 Thomas W. Barford .....*Datascope Corporation*  
 Robert Clark .....*Dräger Medical*  
 Mike Gustafson .....*Ethicon Endo-Surgery*  
 Paul A. Baumgart .....*GE Healthcare*  
 Steven Pregulman, MD .....*Hospira*  
 Steven R. Block .....*LMA of North America*  
 Dana Capocaccia .....*Luminetx*  
 Joe Kiana .....*Masimo*  
 Nancy Stahulak .....*Medical Protective*  
 Anne Young .....*Merck & Co.*  
 Bert de Jong, MD .....*Organon*  
 Dominic Corsale .....*Oridion*  
 Walter Huehn .....*Philips Medical Systems*  
 Edward C. Mills .....*Preferred Physicians Medical Risk Retention Group*  
 Ron Richard .....*ResMed*  
 Michael Stabile, MD .....*Safer Sleep*  
 Andrew Rose .....*Smiths Medical*  
 Joseph Davin .....*Spacelabs*  
 Ann S. Lofsky, MD .....*The Doctors Company*  
 Terry Wall .....*Vital Signs*  
 Abe Abramovich  
 Casey D. Blitt, MD  
 Robert K. Stoelting, MD

# Cardiology Experts Share Perspective on Stents

## Response to Letter to the Editor: Antiplatelet Therapy Should Not Be Stopped

We thank Dr. Kempen for his letter highlighting the challenges of caring for patients with coronary drug-eluting stents (DES). We have discussed some of his questions and other issues related to perioperative management of patients with coronary stents with noted national experts in this area from cardiology. Below is a synopsis of the perspective of Cindy L. Grines, MD, FACC, and chair of the 2007 AHA/ACC/SCAI/ACS/ADA Science Advisory committee report on stent management, with her permission.

The purpose of the 2007 AHA/ACC/SCAI/ACS/ADA Science Advisory was to alert both patients and healthcare professionals that stent thrombosis (ST) is a serious medical issue.<sup>1</sup> The guidelines for dual antiplatelet therapy have been changed to say emphatically that patients should receive a minimum of 12 months of dual antiplatelet therapy—and the minimum means, in many cases—physicians may want to go even further than that period of time. There are several patient subsets that would likely benefit much longer than 12 months. These include patients with acute coronary syndromes, long stents, multiple stents, overlapping stents, diabetes, renal failure, all of which are additional risk factors for stent thrombosis. In these patients, indefinite clopidogrel (Plavix®) use may be recommended. The guidelines were co-written by the American Heart Association, the American College of Cardiology, as well as the American College of Physicians, Surgeons and Dentistry. The Science Advisory was an attempt to avoid the premature discontinuation of dual antiplatelet therapy; providers must realize stopping antiplatelet therapy in patients with DES without discussing it with their cardiologist could result in their patients having a fatal heart attack. The leading adverse event associated with early discontinuation of antiplatelet therapy is ST. Stent thrombosis occurs in up to 29% of patients who discontinue antiplatelet therapy early. The mortality rate in patients with ST ranges from 20-45%.<sup>1,2</sup> Premature discontinuation of dual antiplatelet therapy is the greatest predictor of ST. In a single-site study of 652 patients treated with sirolimus DES, premature discontinuation of clopidogrel was associated with a 30-fold greater risk of ST, with >25% of patients who discontinued clopidogrel therapy within the first month suffering ST.<sup>1,3</sup> In a study of 500 patients who received DES after an acute myocardial infarction (MI), the death rate over the next 11 months was 7.5% for those who stopped taking their thienopyridine medication compared with 0.7% in those who had not stopped therapy.<sup>1,4</sup>

With regard to antithrombin agents, if you look

at the studies done on comparing aspirin with warfarin compared to aspirin with thienopyridine, the warfarin is a big loser. I mean, a 5-fold increased risk of ST; so in my mind, I've extrapolated other antithrombin agents to the warfarin situation . . . . I tend to lean more in the camp of the antiplatelet agents. But again, I would rather just keep the patient on the aspirin and the clopidogrel because I'm very concerned about the abrupt stopping of the antiplatelet agent, and then at the same time you're inciting an inflammatory and hypercoagulable state by performing surgery.

Discontinuation of dual antiplatelet therapy with clopidogrel and aspirin does occur because of cost, perceived risk of bleeding, patients and physicians unaware of the benefits and time of therapy required, and the discontinuation of these drugs by physicians, dentists, surgeons, or their staff before surgical procedures. We avoid elective surgery for more than 1 year after DES, more than 3 months after bare metal stents (BMS), as the risk of ST in both stent types remains high during the postoperative period. With more urgent procedures, if any provider—a physician, dentist, or surgeon—feels that stopping the antiplatelet medicines is absolutely necessary, then there should first be a consultation with the patient's cardiologist. We have changed our practice to never allow the discontinuation of aspirin within the first year of a DES, and would probably extend that to longer duration in stents at high-risk of thrombosis. Importantly, clopidogrel should be restarted as soon as possible. In general, we see more surgeons willing to operate on dual antiplatelet therapies, or at least aspirin. If dual antiplatelet therapy has been discontinued for whatever reason, aspirin should be restarted immediately. Non-enteric coated aspirin (4 baby aspirins) may be given, and the patient should have antiplatelet effects within 2 hours.

Drug-eluting stents prevent the tissue growth that causes restenosis and reduce the need for angioplasty or bypass surgery, but it should be noted that the reduced tissue growth means the stent is exposed to blood for a longer time; this increases the risk of clotting. Dual antiplatelet therapy is also important in patients receiving bare metal stents, but the risk of thrombosis in these procedures remains high for only 1 to 3 months. Consideration should be given to using a bare metal stent if patients are noncompliant with medications, or cannot afford to take clopidogrel for the full year, or if they absolutely cannot postpone an elective surgery.

Following is a summary of additional commentary from Deepak Bhatt, MD, FACC, associate director at the Cleveland Clinic Foundation and A. Michael Lincoff, MD, FACC, vice chairman of Cardiovascular Medicine at the Cleveland Clinic Foundation.

Although there are no evidence-based data supporting the perioperative use of a short-acting glycoprotein (GP) IIb/IIIa platelet inhibitor ("bridging therapy"), there appears to be a role for this use. There are instances in which the only option perioperatively is fast-acting parenteral antiplatelet inhibition with a GP IIb/IIIa antagonist. There are patients for whom there is increased risk of stent thrombosis (ST); for example, a patient had a complex DES procedure a month ago, and then needed hip surgery after falling and breaking their hip, and the surgeon just absolutely refused to operate. This patient was brought into the hospital and started on a short-acting intravenous IIb/IIIa inhibitor. Though practically speaking it means bringing these patients in hospital, there are many patients for whom that extra 4-day hospitalization is worth it. One has to balance the risks and look at the consequences of thrombosis. In those instances, go ahead and proceed with bridging therapy.<sup>5</sup> Another option is cangrelor, which is being tested in Phase III trials. An intravenous short-acting ADP receptor antagonist, cangrelor may conceptually be a future option in the perioperative period.

We are in agreement with Grines et al.: procedures should only be performed on patients with DES in institutions where 24-hour interventional cardiology coverage is present in the event that immediate percutaneous coronary intervention (PCI) is needed for perioperative ST.<sup>6</sup> Almost all case reports to date have cited ST occurring postoperatively, most commonly in the Post Anesthesia Care Unit, and manifesting as a ST-elevation MI (STEMI). Thus, the importance of having immediate access to a coronary catheterization laboratory must be emphasized. McFadden et al. reported ST and STEMI occurring preoperatively after premature discontinuation of dual antiplatelet therapy, or aspirin, in cases where the patient had completed their prescribed course of clopidogrel.<sup>7</sup> Stent thrombosis and its sequelae occur acutely. Therefore, it would be unlikely that a patient with ST would be asymptomatic. However, in all cases, the anesthesiologist (or a member of the anesthesia care team) and surgeon should speak with the patient's cardiologist in order to reach a consensus as to what the safest course of treatment may be. We agree with Dr. Kempen that *all* patients with coronary stents undergo preoperative screening a week

See "Stents," Next Page

# Stents Require a Multidisciplinary Approach

## “Stents,” From Preceding Page

before scheduled surgery to ensure that the appropriate care has been coordinated, dual antiplatelet therapy has been appropriately managed, and all appropriate parties are involved in this very complex care.

Ideally, patients who have clopidogrel and aspirin discontinued for more than 5 days prior to a procedure who are asymptomatic should have aspirin reinstated for 3-5 days to achieve steady-state before proceeding with surgery. We have cancelled a number of cases over the past few months because of premature discontinuation of dual antiplatelet therapy. If aspirin has been discontinued for 3-5 days, we have given 325 mg of non-enteric coated aspirin, and proceeded with surgery later that day to allow platelet inhibition to take effect. Studies have shown that 160 mg of aspirin will inhibit platelet function.<sup>8</sup> The same effect can be achieved with aspirin 75-81 mg over 3-5 days.

We also agree with Dr. Grines that aspirin should never be discontinued in the perioperative period. However, in cases where the surgeon absolutely refuses to operate with the patient on aspirin, then it is imperative that communication occur between the patient's cardiologist, surgeon, and anesthesia provider, and the risks of ST versus major bleeding be carefully weighed. Again, the risk of ST is 29%, and the mortality rate from ST ranges from 20-45%. The surgeon must be made acutely aware of this when considering whether to operate while the patient remains on aspirin.

The use of platelet infusions intraoperatively should be avoided except in the instance where there is life-threatening bleeding. There are certain states (acute MI, unstable angina, trauma, surgery), in which platelet aggregation and activity are enhanced even without an increase in platelet counts.<sup>9</sup> This

phenomenon occurs as a consequence of sympathetic activation and cytokine release.<sup>10,11</sup> Inflammatory activation from endothelial damage, as in PCI and surgery, exacerbates the prothrombotic state, making the patient highly susceptible for thromboembolic events. Autopsy results have shown this mechanism is responsible for at least half of all perioperative infarctions.<sup>12,13</sup> Theoretically, apheresis platelets administered to patients with stents who have serum levels of clopidogrel and aspirin may not develop antiplatelet effects to provide adequate protection from ST for hours to days. Also, activation of the transfused platelets may occur, further increasing the risk of ST, MI, and death. Direct and autologous donation of any blood component is being discouraged by blood banks at many institutions, including our own, because of increased cost and no proven safety benefit over homologous donation.

The controversy and concern surrounding coronary artery stents, especially in the case of DES, illustrate the importance of instituting a multidisciplinary approach in the care of these patients.

Lisa Newsome, MD

Roger Royster, MD  
Winston-Salem, NC

Richard Prielipp, MD  
Minneapolis, MN

## References

1. Grines CL, Bonow RO, Casey DE Jr; American Heart Association; American College of Cardiology; Society for Cardiovascular Angiography and Interventions; American College of Surgeons; American Dental Association; American College of Physicians. Prevention of premature discontinuation of dual antiplatelet therapy in patients with coronary artery stents: a science advisory from the American Heart Association, American College of Cardiology, Society for Cardiovascular Angiography and Interventions, American College of Surgeons, and American Dental Association, with representation from the American College of Physicians. *Circulation* 2007;115:813-8.
2. Iakovou I, Schmidt T, Bonizzi E, et al. Incidence, predictors, and outcome of thrombosis after successful implantation of drug-eluting stents. *JAMA* 2005; 293:2126-30.
3. Jeremias A, Sylvia B, Bridges J, et al. Stent thrombosis after successful sirolimus-eluting stent implantation. *Circulation* 2004;109:1930-2.
4. Spertus JA, Kettelkamp R, Vance C, et al. Prevalence, predictors, and outcomes of premature discontinuation of thienopyridine therapy after drug-eluting stent placement: results from the PREMIER registry. *Circulation* 2006;113:2803-9.
5. Rabbat MG, Bavry AA, Bhatt DL, Ellis SG. Understanding and minimizing late thrombosis of drug-eluting stents. *Cleve Clin J Med* 2007;74:129-36.
6. Brilakis ES, Banerjee S, Berger PB. Perioperative management of patients with coronary stents. *J Am Coll Cardiol* 2007;49:2145-50.
7. McFadden EP, Stabile E, Regar E, et al. Late thrombosis in drug-eluting coronary stents after discontinuation of antiplatelet therapy. *Lancet* 2004;364:1519-21.
8. Patrono C. Aspirin as an antiplatelet drug. *N Engl J Med* 1994;330:1287-94.
9. Neumann FJ, Ott I, Gawaz M, Puchner G, Schomig A. Neutrophil and platelet activation at balloon-injured coronary artery plaque in patients undergoing angioplasty. *J Am Coll Cardiol* 1996;27:819-24.
10. Mangano DT. Perioperative cardiac morbidity. *Anesthesiology* 1990;72:153-84.
11. Cohen MC, Aretz TH. Histological analysis of coronary artery lesions in fatal postoperative myocardial infarction. *Cardiovasc Pathol* 1999;8:133-9.
12. Dawood MM, Gutpa DK, Southern J, et al. Pathology of fatal perioperative myocardial infarction: implications regarding pathophysiology and prevention. *Int J Cardiol* 1996;57:37-44.
13. Poldermans D, Schouten O, Vidakovic R, et al. A clinical randomized trial to evaluate the safety of a noninvasive approach in high-risk patients undergoing major vascu-

The Anesthesia Patient Safety Foundation is pleased to announce the



## BOARD OF DIRECTORS WORKSHOP

Training Before Using Advanced Medical Devices in the Operating Room—  
Voluntary or Mandatory for the Anesthesia Professional?

Friday, October 12, 2007

1300-1700 (Parc Ballroom II/III)

Parc Fifty Five Hotel, 55 Cyril Magnin Street, San Francisco, CA

If you are interested in attending this Workshop, please email [stoelting@apsf.org](mailto:stoelting@apsf.org) to reserve your place. Space is limited and attendance will be possible only by invitation or a limited number of advanced reservations.

## Dear SIRS

# Alarm Requires Selection of Heart Rate Source

**S**AFETY  
**I**NFORMATION  
**R**ESPONSE  
**S**YSTEM



Michael Olympio, MD,  
Chair of the APSF Committee on Technology  
and Co-Founder of the Dear SIRS Initiative.

**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:

I am a CRNA with 13 years of experience. I never had a problem with a pulse oximeter until I encountered the Datex-Ohmeda Cardiocap 5. I was accustomed to a pulse oximeter alarming loudly anytime it was low or not on the patient. I have had 2 incidences in the last 2 years where the ECG beeped when the pulse oximeter was off, such that one might have thought the pulse oximeter reading was OK when not facing the monitor. While another patient was under monitored anesthesia care, the pulse oximeter initially was working, but ceased to work 3 minutes into the case. Once again the ECG beeped, but without the annoyingly loud alarm from the pulse oximeter; there was a single, short beep about every minute. The probe was not illuminated and had to be switched.

I think it is very dangerous to allow a monitor not to alarm if it is not working. I have worked with many monitors over the course of my career, and feel this is taking a step backward if the software cannot be adjusted. I appreciate any information. Thanks.

Kathleen Piotrowski, MSN, CRNA  
Cuyahoga Falls, Ohio

### In Response:

Thank you for the opportunity to respond. I've reviewed this with our design center in Helsinki, Finland, and have observed the monitor's behavior in order to provide the most thorough response. First, I would like to provide you with my name and contact information and assure you that you are welcome to contact me directly at any time.

The Cardiocap/5 monitor is designed with a 3-tiered approach to alarm logic where advisory alarms are displayed in white with a single beep, serious alarms are in yellow and provide 3 beeps, and life-threatening alarms display in red and give 5 beeps.

In the situation you describe, I believe the monitor performed according to the specifications. The Cardiocap/5 monitor, by default, sources the heart rate automatically looking for ECG, Pleth, or Invasive Pressure for an available heart rate. When the heart rate is sourced from the ECG, you will hear the beat sound also sourced from the ECG, and the heart rate will be displayed in the same color as the ECG waveform and numeric. If the heart rate is sourced from the Pleth, the beat sound will be sourced from Pleth and will provide an audible tone different from the ECG tone. The heart rate will be displayed in the same color as the Pleth waveform and numeric.

If the current source becomes unavailable, for example the SpO<sub>2</sub> probe falls off the patient's finger, the monitor will automatically search for another heart rate source (either ECG or invasive pressure). When the monitor switches to another source, ECG for example, the tone of the beat sound will change, the numeric on the display will change to match the color and value coming from the ECG, and a white advisory alarm will be displayed noting "SpO<sub>2</sub> probe off" or "No SpO<sub>2</sub> pulse," whichever is applicable. After several seconds, the white advisory alarm will escalate to a yellow serious alarm and provide 3 beeps. The yellow alarm remains active until it is either acknowledged, or the alarm event is corrected (i.e., probe returned to the patient's finger).

Based on the information we have available, I would speculate that the monitor is performing according to specifications. I would be more than happy to discuss this situation further and to engage our Field Service team to complete a more thorough investigation of the monitor to test the performance. Please feel free to contact me at the telephone or email address listed above.

Best regards,  
Gina Petry  
Product Manager—Perioperative  
GE Healthcare Technologies  
Madison, Wisconsin

### Editor's Question:

This sounds like the identical mechanism on our Datex-Ohmeda S/5 monitoring system, and it has fooled some of our own clinicians as well. One option your response did not include is to select the heart rate source manually, instead of automatically. At least in the S/5, this would stop the tone, and cause the clinician to look upwards. Is that possible in the Cardiocap/5?

Michael A. Olympio, MD

### In Response:

You are correct—it should be the same mechanism as your S/5 monitor. The logic is almost identical throughout the Datex-Ohmeda family of monitors. You can set the heart rate source manually to Pleth, for example. This setting cannot be saved as a default, however. The loss of a pulse from Pleth will result in the loss of the beat sound and a yellow alarm.

Gina Petry

The information in this column is provided for safety-related educational purposes only, and does not constitute medical or legal advice. Individual or group responses are only commentary, provided for purposes of education or discussion, and are neither statements of advice nor the opinions of APSF. It is not the intention of APSF to provide specific medical or legal advice or to endorse any specific views or recommendations in response to the inquiries posted. In no event shall APSF be responsible or liable, directly or indirectly, for any damage or loss caused or alleged to be caused by or in connection with the reliance on any such information.

# Anesthesia Patient Safety Foundation

## Corporate Donors

### Founding Patron (\$500,000 and higher)

American Society of Anesthesiologists (asahq.org)

### Grand Patron (\$150,000 to \$199,999)

Anesthesia Healthcare Partners, Inc. (AHP) (ahphealthcare.com)



Cardinal Health Foundation (cardinal.com)



### Supporting Patron (\$15,000 to \$24,999)

Baxter Anesthesia and Critical Care (baxter.com)  
 Dräger Medical (draeger.com)  
 Preferred Physicians Medical Risk Retention Group (ppmrrg.com)

### Patron (\$10,000 to \$14,999)

Cardinal Health, Alaris Products (alarismed.com)  
 DocuSys (docusys.net)  
 Ethicon Endo-Surgery (ethiconendo.com)  
 iMDsoft (imd-soft.com)  
 Oridion Capnography (oridion.com)  
 Spacelabs Medical (spacelabs.com)

### Sustaining Donor (\$5,000 to \$9,999)

Anesthesiologists Professional Assurance Company (apacinsurance.com)  
 Arrow International (arrowintl.com)  
 Bayer Healthcare (bayerhealthcare.com)

### Sustaining Patron (\$100,000 to \$149,999)

Merck & Co. (merck.com)



### Sponsoring Patron (\$75,000 to \$99,999)

GE Healthcare (gemedical.com)



### Benefactor Patron (\$25,000 to \$49,999)

Abbott Laboratories (abbott.com)



Aspect Medical Systems (aspectms.com)



Hospira (hospira.com)



Masimo Corporation (masimo.com)



Organon (organon.com)



Philips Medical Systems (medical.philips.com)



Tyco Healthcare (tycohealthcare.com)



## Community Donors

(includes Anesthesia Groups, Individuals, Specialty Organizations, and State Societies)

### Grand Sponsor (\$5,000 and higher)

Alabama State Society of Anesthesiologists  
 American Academy of Anesthesiologist Assistants  
 American Association of Nurse Anesthetists  
 Asheville Anesthesia Associates  
 Florida Society of Anesthesiologists  
 Dr. and Mrs. Thomas R. Hill  
 Indiana Society of Anesthesiologists  
 Iowa Society of Anesthesiologists  
 Frank B. Moya, MD, Charitable Foundation  
 Robert K. Stoelting, MD

### Sustaining Sponsor (\$2,000 to \$4,999)

Academy of Anesthesiology  
 Anaesthesia Associates of Massachusetts  
 Anesthesia Consultants Medical Group  
 Anesthesia Resources Management  
 Arizona Society of Anesthesiologists  
 Nassib G. Chamoun  
 Georgia Society of Anesthesiologists  
 Madison Anesthesiology Consultants  
 Massachusetts Society of Anesthesiologists  
 Michigan Society of Anesthesiologists  
 Minnesota Society of Anesthesiologists  
 Ohio Society of Anesthesiologists  
 Old Pueblo Anesthesia Group  
 Pennsylvania Society of Anesthesiologists  
 Society of Cardiovascular Anesthesiologists  
 Tennessee Society of Anesthesiologists

### Contributing Sponsor (\$750 to \$1,999)

Affiliated Anesthesiologists, Inc.  
 American Association of Oral and Maxillofacial Surgeons  
 American Society of Critical Care Anesthesiologists  
 American Society of PeriAnesthesia Nurses (aspan.org)  
 J. Jeffrey Andrews, MD  
 Anesthesia Associates of Northwest Dayton, Inc.  
 Associated Anesthesiologists of St. Paul, MN  
 Association of Anesthesia Program Directors  
 Sorin J. Brull, MD  
 Frederick W. Cheney, MD

Connecticut State Society of Anesthesiologists  
 Jeffrey B. Cooper, PhD  
 Steven F. Croy, MD  
 Mark T. Destache, MD (Associated Anesthesiologists)  
 District of Columbia Society of Anesthesiologists  
 David M. Gaba, MD  
 John H. Eichhorn, MD  
 Illinois Society of Anesthesiologists  
 Kentucky Society of Anesthesiologists  
 John W. Kinsinger, MD  
 Thomas J. Kunkel, MD  
 Rodney C. Lester, CRNA  
 Edward R. Molina-Lamas, MD  
 Madison Anesthesiology Consultants  
 Maryland Society of Anesthesiologists  
 Michiana Anesthesia Care  
 Missouri Society of Anesthesiologists  
 Robert C. Morell, MD  
 Nebraska Society of Anesthesiologists  
 John B. Neeld, MD; in honor of Orin F. Guidry, MD  
 Northwest Anesthesia Physicians  
 Neshan Ohanian, MD  
 Nevada State Society of Anesthesiologists  
 Nurse Anesthesia of Maine  
 Oklahoma Society of Anesthesiologists  
 Oregon Society of Anesthesiologists  
 Oregon Anesthesiology Group  
 Physician Anesthesia Service  
 Pittsburgh Anesthesia Associates  
 Santa Fe Anesthesia Specialists  
 Society of Academic Anesthesia Chairs  
 Society for Ambulatory Anesthesia  
 Society of Neurosurgical Anesthesia and Critical Care  
 Society for Pediatric Anesthesia  
 South Dakota Society of Anesthesiologists  
 Stockham-Hill Foundation  
 Texas Society of Anesthesiologists  
 Drs. Mary Ellen and Mark Warner  
 Washington State Society of Anesthesiologists  
 Wisconsin Society of Anesthesiologists

### Sponsor (\$100 to \$749)

Robert L. Barth, MD  
 Manuel E. Bonilla  
 California Society of Anesthesiologists  
 Robert A. Caplan, MD

Melvin A. Cohen, MD  
 Colorado Society of Anesthesiologists  
 Kathleen A. Connor, MD  
 David M. Clement, MD  
 Paula A. Craig, MD  
 J. Kenneth Davison, MD  
 John DesMarteau, MD  
 Walter C. Dunwiddie, MD  
 Norig Ellison, MD  
 Jan Ehrenwerth, MD  
 Thomas R. Farrell, MD  
 Anthony Frasca, MD  
 B. L. Friedberg, MD  
 Thomas R. Farrell, MD  
 Jane C. K. Fitch, MD/Carol E. Rose, MD  
 Ian J. Gilmour, MD  
 Gregory E. Ginsburg, MD  
 Barry M. Glazer, MD  
 James D. Grant, MD  
 Griffin Anesthesia Associates  
 William D. Heady, CRNA  
 Alexander A. Hennenberg, MD  
 Peter L. Hendricks, MD  
 James S. Hicks, MD  
 Dr. and Mrs. Glen E. Holley  
 Howard E. Hudson, Jr., MD  
 Indianapolis Society of Anesthesiologists  
 Robert. H. Intress, MD  
 Robert E. Johnstone, MD  
 Tamos Kallos, MD  
 Daniel J. Klemmedson, DDS, MD  
 Kansas Society of Anesthesiologists  
 BettyLou Koffel, MD  
 Maine Society of Anesthesiologists  
 Alan P. Marco, MD  
 Maryland Association of Nurse Anesthetists  
 John P. McGee, MD  
 Tom L. McKibban, CRNA  
 Cora B. McKnight, CRNA  
 Medical Anesthesiology Consultants Corporation  
 Mississippi Society of Anesthesiologists  
 A. J. Montes, MD  
 Roger A. Moore, MD  
 Ervin Moss, MD  
 New Hampshire Society of Anesthesiologists  
 New Jersey State Society of Anesthesiologists

New Mexico Society of Anesthesiologists  
 L. Charles Novak, MD  
 Denise O'Brien, RN  
 Michael A. Olympio, MD  
 Carmelita S. Pablo, MD  
 Pennsylvania Association of Nurse Anesthetists  
 Mukesh K. Patel, MD  
 Gaylon K. Peterson, MD  
 Physician Specialists in Anesthesia  
 Richard C. Prielipp, MD  
 Rhode Island Society of Anesthesiologists  
 Gail I. Randel, MD  
 Henry C. Safford, CRNA  
 Eduardo A. Salcedo, MD (Salmon Medical Innovations)  
 Drs. Chris and David Santamore  
 Muthia Shanmugham, MB  
 Eugene P. Sinclair, MD  
 Society for Obstetric Anesthesia and Perinatology  
 Society for Technology in Anesthesia  
 South County Anesthesia Association  
 Shepard and Marlene Stone  
 Sara L. Strom, AA-C  
 The Woodlands Anesthesia Associates  
 University of Maryland Anesthesiology Associates  
 Vermont Society of Anesthesiologists  
 Virginia Society of Anesthesiologists  
 Matthew B. Weinger, MD  
 West Virginia Association of Nurse Anesthetists  
 West Virginia State Society of Anesthesiologists  
 Andrew S. Weisinger, MD  
 Dr. and Mrs. Wetchler  
 Wichita Anesthesiology, Chartered  
 Lawrence Wobbrock, JD  
 Benjamin and Elizabeth Yoder

### In Memoriam

In memory of Dr. Marc Balin (anonymous)  
 In memory of Maurice Chait, MD (Texas Society of Anesthesiologists)  
 In memory of Oneita M. Hedgecock, MD (Texas Society of Anesthesiologists)  
 In memory of Laurie A. Noll, MD (The Coursin family)  
 In memory of Bonnie J. Slarsky (Jeffrey B. Cooper, PhD)  
 In memory of Rex E. Thomas, MD (Texas Society of Anesthesiologists)

Note: Donations are always welcome. Send to APSF; c/o 520 N. Northwest Highway, Park Ridge, IL 60068-2573 (Donor list current through July 10, 2007)

## Case Report

# Wrap Delays Detection of Disconnect

## To the Editor:

Accidental disconnection of components of the breathing system during general anesthesia with an endotracheal tube persists as an occasional problem. It seems more likely in face surgery where the tube is in the midst of an active surgical field and not accessible by the anesthesiologist. While the potential for great danger during controlled ventilation or the entrainment of room air and/or rebreathing during spontaneous ventilation still exists, modern electronic monitoring will almost always sound alarms and convey information that leads to rapid discovery and re-connection of the wayward breathing system components. We report an unusual case of breathing system disruption with a misleading presentation in that the capnogram appeared normal and there was no gas irregularity.

A 50-year-old female was undergoing a facelift. After routine anesthesia induction and intubation with a standard 7.5 mm endotracheal tube, the operating room (OR) table was turned 180°. The breathing circuit elbow piece was removed and replaced with a straight connector between the tube adapter and the circuit Y-piece. All connections were snugly pushed together. The patient's face, neck, endotracheal tube, and distal breathing circuit were prepped into the

field. A clear plastic 45 × 60 cm Steri-Drape™ 1010 (3M, Minneapolis, MN) was spirally wrapped around the parts of the endotracheal tube, connector, sample tubing, and circuit that were in the surgical field (see photo).

After approximately 1 hour of surgery, with the patient on controlled ventilation, desflurane at 1 MAC in 1 L/min oxygen, the reservoir bag of the Dräger Apollo anesthesia machine was noted to be completely empty, and, at the same time, the "low volume" alarm of the machine sounded. The capnogram showed a pattern characteristic of spontaneous breathing, with the waveform returning to baseline during each breath. There was no decrease in FiO<sub>2</sub>. Oxygen flow was increased from 1 L/min to 10 L/min with no filling of the bag or change in the alarm. Careful inspection revealed that the 15 mm connector had come out of the end of the endotracheal tube. The surgeon quickly grasped the enfolding plastic drape and pushed the enclosed connector back into the endotracheal tube.

Of particular interest in this incident is the fact that the plastic drape wrapped around both the distal circuit tubing and the endotracheal tube prevented significant entrainment of room air and kept the fresh

gas flow from completely dissipating away from the patient. There was no CO<sub>2</sub> rebreathing under the drapes as is often seen with disconnects in facial cases. The discontinuity did cause loss of measured expired volume and some pressure, which activated an alarm. The capnograph alarm was not triggered because the capnogram persisted.

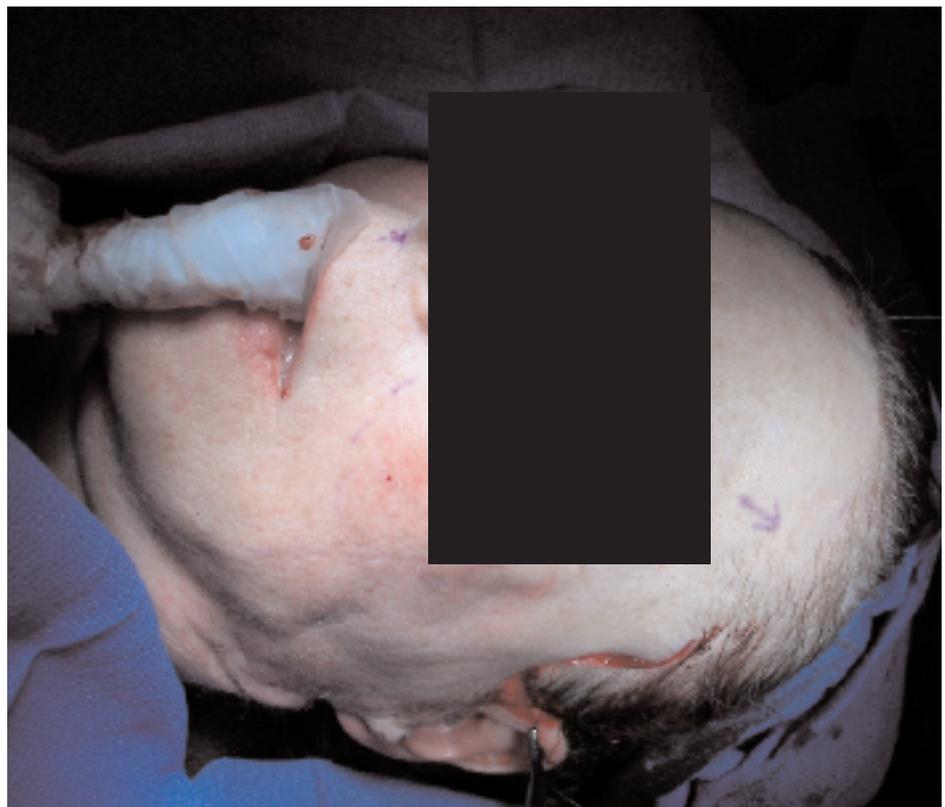
Obviously it was necessary and desirable to replace the 15 mm connector into the endotracheal tube, and this was easily accomplished. The unusual placement of the circumferential plastic drape combined with spontaneous ventilation created a unique circumstance that in one way reduced patient danger (fresh gas remained available), but in other ways increased danger in that the characteristic disappearance of the capnogram did not occur with this disconnect.

Anesthesiologists and nurse anesthetists should have heightened awareness of breathing system continuity during major facial surgery and of the potential unusual implications of encasing all the connections in a wrap of sterile plastic drape.

Gregory Rose, MD  
John Eichhorn, MD  
Lexington, KY

## APSF Executive Committee Invites Collaboration

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, 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.



Photograph of extensive wrapping of tracheal tube and connection, which delayed detection of the disconnect.

Report on the 7th Invited Conference by Cardinal Health

# Intensive Insulin Therapy

June 7-8, 2007, Center for Safety and Clinical Excellence, San Diego, CA

An invited conference sponsored by Cardinal Health Center for Safety and Clinical Excellence was held June 7-8 in San Diego, to review and summarize expert opinion on tight glycemic control (TGC) for acute hospitalized patients. Speakers included Simon Finfer, MBBS (Nice-SUGAR trial, Sydney, Australia) and Philippe Devos, MD (VISEP/Glucontrol trials, Belgium), with another 45 participants from across the United States and Canada. Thought leaders represented the disciplines of anesthesiology, intensive care, endocrinology, surgery, hospitalist medicine, medical genetics, nutrition, nursing, pharmacy, biostatistics, and healthcare biotechnology.

*The central question, which anchored the conference, was whether ICU patients benefit most from “intensive” or “tight” glycemic control (usually defined as blood glucose in the range of 80-110 mg/dL) or “tighter” control (typically translated as blood glucose in the range of 110-150 mg/dL).*

**In the meantime, conference presentations yielded these highlights:**

- Insulin is the most common drug reported as a medication error to the U.S. Pharmacopeia. Moreover, the error harm rate (categories E, F, H, I) = 9.3%.
- Data from hospitals using “smart” IV pumps documented a high frequency of averted insulin dosing errors in addition to high variability in concentrations and mixed use of weight-based and nonweight-based dosing units.
- The landmark ICU study by Dr. Greet Van den Berghe, Catholic University of Leuven in Belgium, targeted blood glucose of 80-110 mg/dL in the treatment group. She noted decreased mortality and morbidity (less renal failure, surgical wound infections, blood transfusions, ICU ventilator days, etc.) in surgical ICU patients.
- Two recent European glucose control studies—VISEP (488 patients in 17 German Centers) and GluControl (1,101 randomized patients across 21 ICU units in 7 European countries)—were both stopped due to unacceptably high rates of hypoglycemia and lack of beneficial effect.
- The on-going NICE-Sugar open-label, randomized stratified study in 25 Australian, 19 Canadian, and 2 American hospitals has a planned enrollment of 7,000 patients! This study will compare 2 target ranges for blood glucose (81-108 mg/dL vs. <180 mg/dL). Conference participants expect this to be the pivotal outcome investigation—and one that will likely define the target glucose for future ICU patients. The study is well on its way to 4,000 enrolled patients.
- Current data are insufficient to mandate TGC for patients in the operating room. A recent randomized study in cardiac surgery patients (*Ann Int Med* 2007;46:233) found no difference in ICU or hospital LOS despite TGC throughout the operative period.

- It is important to note that perioperative hyperglycemia occurring in “non-diabetics” may actually indicate undiagnosed Type II diabetes. Providers should consider hemoglobin A1c determinations in these patients to direct optimal metabolic management.

**Additional insights based on the collective experience of participants included**

- Prevalence of diabetes is increasing rapidly.
- TGC requires an interdisciplinary team approach, a culture of safety, and a focus on professional education. Moreover, benchmarks to evaluate effectiveness are needed.
- Current “paper” ICU protocols for TGC achieve target glucose concentrations about 40% of the time.
- Published TGC protocols differ significantly in insulin dosing recommendations.
- Computerized protocols improve the efficacy to 60%, and have the additional benefit of decreasing hypoglycemic episodes.
- Bedside glucometers rely on a number of different chemical reagents, and users should be aware of potential confounding variables—even including parameters like hematocrit, PaO<sub>2</sub>, and so forth. Interfering substances can generate seriously erroneous meter readings.
- Total nursing time for each point-of-care glucose determination varies from 3.5-9 min (median time = 4.7 min). The aggregate time and RN workload of applying TGC is *substantial*.
- Current continuous glucose monitors (CGM) measure glucose in the interstitial fluid, which lags behind blood concentrations by 3-10 min. Nonetheless,

CGM appears to facilitate smoother, timelier titration of insulin infusions, and patients reach target glucose values more quickly. Coordination of this technology with a computerized protocol minimizes the incidence of hypoglycemia.

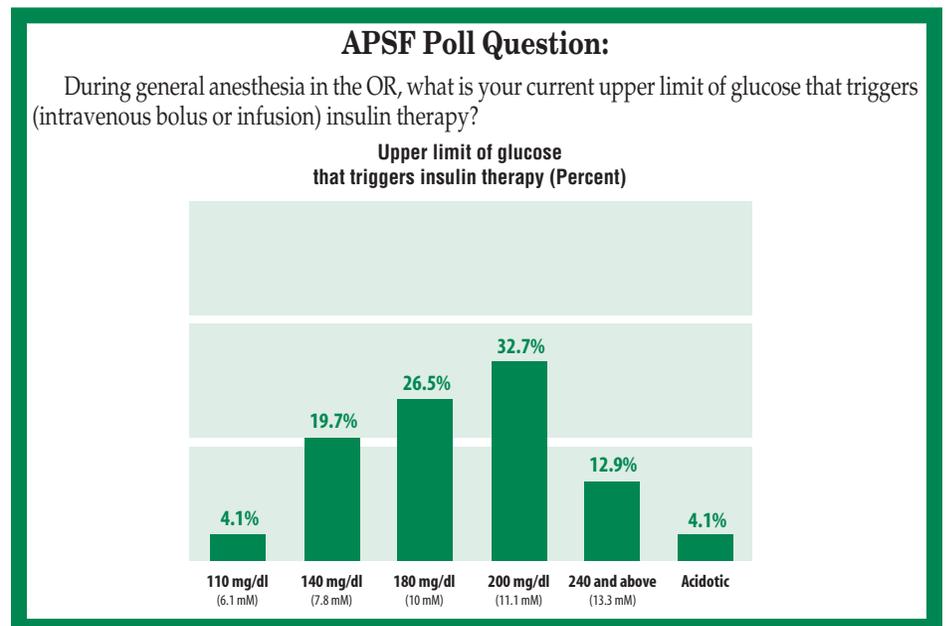
- Clinical experience suggests minimal risk of a single episode of hypoglycemia (FBS ≤ 40 mg/dL), if diagnosed and managed in a timely fashion.
- Transitions from ICUs to Medical/Surgical care units and from IV to oral feedings are especially problematic in maintaining TGC.
- Intensive insulin therapy and TGC is cost effective, but may be population and protocol sensitive.

The optimal glucose threshold for TGC in ICU patients remains under investigation, and anesthesia providers debate how these principles should apply to patients in the operating room. More data are needed. An independent APSF poll regarding triggers for initiation of insulin therapy is also presented below.

*Richard C. Prielipp, MD, MBA, FCCM  
Professor and Chair of Anesthesiology  
University of Minnesota  
Minneapolis, MN*

*Carol S. Manchester, MSN, APRN, BC-ADM, CDE  
Diabetic Clinical Nurse Specialist  
UMMC-Fairview  
Minneapolis, MN*

*Timothy W Vanderveen, PharmD, MS  
Vice President  
Cardinal Health Center for Safety and Clinical Excellence  
San Diego, CA*



Results of an independent APSF Poll regarding readers' triggers for initiation of insulin therapy.

## Letters to the Editor

# Are Patients With Obstructive Sleep Apnea Safer at Home?

### To the Editor:

I would like to respond to Dr. Weinger's lead article, published in the 2006-2007 winter issue of the *APSF Newsletter*, which discusses the dangers of postoperative opioids. My particular interest is in regard to postoperative pain control for the obstructive sleep apnea (OSA) population. It seems much of the data we have related to this safety issue are based on the use of parenteral opioids postoperatively. Specifically, the article mentions Dr Lofsky's report on the effect opioids have on the neural efferent system, which is said to be responsible for depression of

upper airway patency. The opioid delivery of reference in this discussion was patient-controlled analgesia (PCA). My questions are 1) Has our specialty determined the safety of oral opioid-based analgesics for the ambulatory patient with OSA, and 2) What is the impact of the provision of parenteral opioids as part of the general anesthetic or immediate postoperative pain relief in the OSA patient being discharged to home? I ask these questions in light of the fact that there is an alteration in perioperative sleep that is pronounced in the OSA population and observed in the 24-hour postoperative period. This alteration, which is partly due to the exposure to

anesthetics and analgesics, is worsened by the effect of rapid eye movement (REM) sleep rebound<sup>1</sup> and can create a tenuous postoperative period while the patient is home unmonitored.

Drs. Weinger and Morell did provide their personal suggestion that it is likely safer for OSA patients to be discharged home on oral analgesics rather than be admitted and receive parenteral opioids. I can easily agree with this due to all of the potential life-threatening risks of PCA or intermittently dosed opioids when monitoring is substan-

See "OSA," Next Page

## Q & A

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.

### Pipeline Pressure Primer

#### Q Dear Q&A,

I have always thought that, in a hospital central gas supply system, the oxygen pipeline should operate at a slightly higher pressure than the air and nitrous oxide lines in order to mitigate the effects of a possible cross connection. I am working at a new hospital and the pipeline person is asking for documentation. Is this just an informal safety measure or is it mandated by code? Thanks for your help.

Samuel Tirer, MD

#### A Dear Dr. Tirer,

The National Fire Protection Association (NFPA) 99 Standard for Health Care Facilities, 2005 Edition, states, "Piping systems, with the exception of nitrogen systems, shall be capable of maintaining 50-55 psig (345-380 kPa gauge) to all outlets at the maximum flow rate." Reading the NFPA is like reading IRS 1040 instructions, so perhaps there is a qualifier (regarding different oxygen pressure) somewhere else in the document that I missed in my scan. But I have never heard of different wall pressure for O<sub>2</sub>.

I believe there is not a standard driving this, but a local preference. I have heard of hospitals set-

ting oxygen at the top of the allowable range, such that if a check valve failed somewhere in the system, the oxygen would prevent potentially hypoxic scenarios.

Using the highest pressure for oxygen in a pipeline system is a very old practice for 2 reasons:

- 1) It allows one to certify that day-to-day running of a mixed pipeline system is "safe" without using an oxygen analyzer on each outlet. In many parts of the world this is the routine safety check. Where gas-mixing devices are used, as with nitrous/oxygen for analgesia, it could be part of the basic design.
- 2) If there is any sort of link between 2 lines, better that oxygen dilutes the other. I think you'll find this rule originates in old British standard safe practice that preceded people writing specs.

The APSF Committee on Technology

#### A Dear Dr. Tirer,

The pressure ranges listed in Table 5.1.11 from NFPA 99-2005 for medical air, oxygen, nitrous oxide, helium, and carbon dioxide are the same (50-55 psi). I can find nothing that says one should be greater than the other to avoid cross

connections. NFPA 99 also requires stringent initial testing, and specifies testing that is necessary after working on the system.

There are 2 tests acceptable to NFPA to initially verify that there are no cross connections in a system. One is called the *Individual Pressurization Test*, whereby the system being checked shall be pressurized to 50 psi, while all other disconnected, atmospheric lines are simultaneously checked to determine that test gas is being dispensed only from the outlets/inlets of the piping system being tested.

What some of your contacts may be referring to is the other acceptable method, or *Pressure Differential Test*. This test does require that the oxygen system and the medical air system (and others) be pressurized and maintained to different, specified psi (50 psi, 60 psi, respectively for oxygen and medical air), after which a pressure test is made at every outlet to check for cross-connections.

NFPA does NOT say that the system needs to be operated during normal use with those pressures—rather only for the verification test.

Mike Mahan, PE  
North Carolina Baptist Hospital  
Engineering  
Winston-Salem, NC

The information provided in this column is for safety-related educational purposes only, and does not constitute medical or legal advice. Individual or group responses are only commentary, provided for purposes of education or discussion, and are neither statements of advice nor the opinions of APSF. It is not the intention of APSF to provide specific medical or legal advice or to endorse any specific views or recommendations in response to the inquiries posted. In no event shall APSF be responsible or liable, directly or indirectly, for any damage or loss caused or alleged to be caused by or in connection with the reliance on any such information.

## Letters to the Editor, continued

### “OSA,” From Preceding Page

dard, but are there any objective data to substantiate this anecdotal evidence? As a resident physician, I am becoming increasingly aware of the growing ambulatory surgery population as well as an increasing prevalence of OSA as the obesity epidemic continues.<sup>2</sup> I would like to know that as I care for patients in the ambulatory setting, with either formal or presumptive clinical diagnosis of OSA, the provision of perioperative opioids is not placing them at higher risk for incurring a life-threatening respiratory event while recovering at home.

Michelle Lawrence, MD  
Chicago, Illinois

#### References

1. Benumof JL. Obesity, sleep apnea, the airway and anesthesia. *ASA Refresher Courses in Anesthesiology* 2002;30:27-40.
2. Kaw R, Michota F, Jaffer A, et al. Unrecognized sleep apnea in the surgical patient: implications for the perioperative setting. *Chest* 2006;129:198-205.

## Post-Op Hypoxia Multifactorial and Should Be Treated With Supplemental Oxygen

### To the Editor:

In the 2006-07 winter issue of the *APSF Newsletter*, the suggestion was made that pulse oximetry might be an acceptable monitor to assess and avoid opioid overdose—providing patients breathe only room air. We believe that all post-anesthesia care unit (PACU) patients and those given intravenous and neuraxial opioids should initially be given supplemental oxygen regardless of the monitor used. Hypoxia in the postoperative period is often multifactorial in nature (residual anesthetics, splinting, atelectasis, obesity, fluid overload, opioid medication). Patients can be in pain without significant respiratory depression, yet still be hypoxic. Supplemental oxygen can correct this hypoxia and possibly avoid a catastrophic event. To withhold pain medication from patients because their room air saturations are low would only serve to increase complications relating to the stress response. Patients on oxygen receiving opioids will usually have elevated pCO<sub>2</sub>s; however, mild degrees of hypercarbia are well tolerated. It would be ideal to reliably monitor oxygen saturation and expired CO<sub>2</sub> in all patients, but until these monitors become widely available on the hospital wards, we must continue to rely on healthcare providers who are trained to recognize pending opioid toxicity.

Merlin D. Larson, MD  
Andrew Itkin, MD  
John W. Severinghaus, MD  
San Francisco, CA

## Reader Has Low Tolerance for Zero Tolerance

### To the Editor:

I read with amazement the article “Dangers of Postoperative Opioids” (Vol 21, #4). The article states, “We advocate the use of continuous monitoring of oxygenation (generally pulse oximetry) and of ventilation in non-ventilated patients receiving PCA, neuraxial opioids, or serial doses of parenteral opioids.” Basically the APSF is instituting a new standard of care. This would require billions of dollars of equipment that the article acknowledges as being “plagued by false positive . . . and false negative . . . alarms” and thousands of new personnel to monitor the equipment and monitors.

I am surprised that the article did not recommend the abandonment of PCA, neuraxial opioids, or serial doses of parenteral opioids until perfect monitors were developed. This would go along with the desire of the authors to have “zero tolerance” for any respiratory morbidity associated with the use of opioids.

The only people this article helps are malpractice lawyers who can now ask us, “Doctor, are you not aware that your own safety foundation recommends monitoring for EVERY patient receiving opioids?”

Robert F. LaPorta, PhD, MD  
Glen Cove, NY

#### New Feature

### Innovative Technology and Pharmaceuticals (ITP)

This column is dedicated to providing our readers with information regarding new and innovative technological or pharmaceutical developments that may directly or indirectly impact patient safety. By virtue of the unique and long-standing relationship between the APSF and industry, it is inevitable that we will, from time to time, discuss or review products, devices, or pharmaceuticals that may be manufactured, sold, or distributed by corporations or entities that have or continue to supply financial or in-kind support to the APSF. We will strive to disclose those relationships as appropriate.

by Joel Saltzman, MD

Establishing vascular access for administration of medications, or to obtain blood for laboratory tests, is stressful for the patient and can be stressful for the practitioner; limiting the number of sticks is beneficial to both. While most practitioners are adept at venipuncture, patients with extremes in age, habitus, co-morbidities, and multiple punctures present a challenge to even the most experienced. Not uncommonly, the anesthesia team is called upon to obtain venous access after multiple attempts by others in the hospital. By this time, we may be presented with a patient who is stressed, potentially dehydrated, sore, and angry, and with concerned family members. A new technology has been able to “shine a light” on this problem, a green light.

Experienced practitioners have long been able to access the vascular system provided they can see or feel the vessel, or to attempt a blind stick based on anatomical landmarks. Multiple technologies have tried to assist the practitioner to visualize the target. Ultrasound has assisted, but requires the use of gels and a significant learning curve with specialized training. Transillumination with high-intensity light involves direct patient contact, elimination of ambient light, and may result in heating at the site. One infrared device, VeinViewer by Luminetx, uses near-infrared technology to allow the practitioner to visualize the vessel without direct contact, gels, heat, or advanced training. This device projects the image of the vasculature directly onto the patient’s skin, focusing attention on the patient rather than the monitor, and allows both hands to be free to perform the procedure. The practitioner may also visualize arteries as pulsatile structures if they are within the 6-8 mm imaging range of depth.

The near infrared light source is used to differentiate red blood cells from surrounding tissues. The light is reflected back from the surface tissue, but not reflected

from the blood in the vessels. The infrared light photons are received by a detector located in the digital video camera; a computer digitizes these photons, produces an image and projects it onto the patient’s skin. The image is displayed in real-time, and veins



appear as a black road map on a green field. The non-ionizing energy emitted from the LED light sources is many magnitudes under previously established safety limits.

Whenever a clinician or an institution considers new equipment, safety and cost must be part of the equation. Although the machine is somewhat large, it is self-contained, well-balanced, and relatively easy to move.

This device may help alleviate trips to the operating room for central venous access—offsetting costs and risks that may be deferred by placement of a PICC line or simple peripheral vascular access. Anesthesia, Emergency Room, Radiology, Critical Care, phlebotomists, PICC teams, and others can all utilize this technology throughout the hospital. There is also the potential to update and advance the capabilities of the equipment with software updates imported into the machine via the 2 USB ports. In sum, the VeinViewer can potentially improve patient safety by facilitating intravenous access as an alternative to central venous cannulation, while also reducing patient discomfort.

Dr. Saltzman is Chief of Anesthesiology at Le Bonheur Children’s Medical Center, Memphis, TN.

DISCLOSURES: Luminetx Corporation is a financial contributor to the APSF. Dr. Saltzman has no financial relationship to Luminetx.

# Inside This Issue

- ▲ **More About Drug-Eluting Coronary Stents and Late Thrombosis**
- ▲ **Perils of Hypotension in the Beach Chair Position**
- ▲ **Review of Maternal Arrests**
- ▲ **Case Report and Letters to the Editor**

**Anesthesia Patient Safety Foundation**  
Building One, Suite Two  
8007 South Meridian Street  
Indianapolis, IN 46217-2922

NONPROFIT ORG.  
U.S. POSTAGE  
**PAID**  
WILMINGTON, DE  
PERMIT NO. 1387