American Society of Anesthesiologists

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Ellison C. 'Jeep' Pierce, Jr., M.D. 1928 -2011

As this special edition of the *ASA NEWSLETTER* was being published, news of the death of Ellison "Jeep" Pierce, the driving force behind the patient safety movement and the founder of the Anesthesia Patient Safety Foundation, was received on April 4, 2011. He was 82 years old. In the mid-1980s, in response to a nationally televised news media program on the dangers of anesthesia, and coinciding with his lifelong interest in unrecognized esophageal intubation, Dr. Pierce conceived of the idea of a group devoted to increasing the safety of anesthesia. As president of ASA in 1984, he began discussions on what would become the APSF in 1985. The foundation's vision that "No patient shall be harmed by anesthesia" is both simple and direct, reflective of its founder. As you read this celebration of the accomplishments of APSF, and the national culture it created, remember the simple desire of its founder. Dr. Pierce received the ASA Distinguished Service Award in 1996. His gentle, guiding spirit will be greatly missed.

– D.R.B.

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American Society of Anesthesiologists

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Dr. Atul Gawande

The Ear Piece

In the early 1980s, it was easy to tell the anesthesia staff from the rest of the operating room personnel in the break room. Attached to their scrubs, each had a safety pin, through which was looped a piece of plastic tubing. One end was open, and on the other was a piece of plastic. Some had vibrant colors – reds, deep purple, green and blue were common – others were clear. The function, though, was universal. The plastic was molded to the anesthesia staff's individual ear, and when caring for patients, the earpiece was connected to a precordial stethoscope for continuos monitor-ing of heart tones and breath sounds. Some of the team also had a three-way stop clock that connected to a manual blood pressure cuff. Just before induction, the cuff was inflated and the pressure assessed. It would not be routinely taken again until the airway was secured and the patient's induction complete.

This was the world of anesthesiology I entered as a resident. Pulse oximetry and end-tidal carbon dioxide monitoring were just beginning. With too few monitors for each operating room, the in-room providers fought over equipment. In one hospital there were six end-tidal carbon dioxide and six automated blood pressure monitors for nine rooms. There was one pulse oximeter. It was not uncommon, after getting the anesthesia equipment set up and going to see the patient, to return to the room and find pieces missing. Cases were done without automated blood pressure monitoring, end-tidal carbon dioxide or pulse oximetry – and as a learning experience, it was invaluable. Attending anesthesiologists asked the six ways to check that an endotracheal tube was in proper position. Palpating the pulse and actually touching the patient were important aspects of anesthetic care – and having a "feel" for the case meant more than just interpreting physiologic data.

Douglas R. Bacon, M.D.
Editor, *ASA NEWSLETTER*

During my first year of residency, the Anesthesia Patient Safety Foundation (APSF) was formed. Driven by the vision of Ellison "Jeep" Pierce, M.D. (1928-2011), that no patient will be harmed by anesthesia, monitoring in the operating room advanced quickly. At the same time as the APSF's birth, the Harvard Standards for monitoring were published and the ASA adopted "Standards for Basic Anesthetic Monitoring" (see related article on page 22). This document was first approved in 1986 and has been continually updated and modified to reflect changes in technology and practice.

Photos courtesy of AverySound.com

When the Harvard Standards first came out, our chairman asked each resident to sign a statement that we would abide by them. We had to document that we were listening to our patient's breath sounds and heart tones – thus continuously monitoring respiration and circulation. Tethered to our patients with the connection from the earpiece to the precordial, we never had far to stray in the operating room. And heaven forbid the surgeon felt you were not paying attention to him or the case! A sharp rap on the precordial with a clamp sent shock waves from your ear across the brain. It was not pleasant!

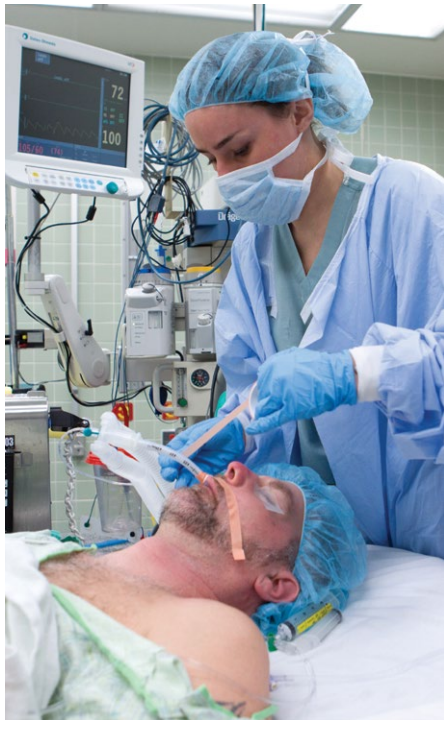
The creation of the APSF occurred at the right time. The "new" technology of pulse oximetry and end-tidal carbon dioxide monitoring allowed new parameters to be measured and watched. In the case of carbon dioxide monitoring, immediate confirmation of correct placement of the endotracheal tube occurred. While each of the observed parameters of correct endotracheal tube placement had false positives, the end-tidal monitoring did not. Running up to the intensive care unit to place or replace an endotracheal tube was "scary." No longer was the comfort of the end-tidal conformation immediately available. If continuously monitored

throughout the case, it was also possible to immediately detect a disconnection of the anesthesia machine circuit from the endotracheal tube. Thus, long before harm ensued, the circuit could be reconnected and the patient saved from the inevitable anoxia.

Likewise, pulse oximetry became a touchstone for how the patient was progressing under anesthesia. Deflate one lung during a thoracic procedure, and quickly learn if the patient's physiology could tolerate such a limitation to ventilation. Likewise, the reassuring pulse oximetry reading has changed a common surgical parlance. No longer on incision do surgeons comment upon the color of the blood coming from the wound. Desaturation is treated rapidly, again before harm has occurred, rather than wait for cyanosis to appear.

APSF has created a new culture in anesthesiology. Over the past 25 years, the organization has sponsored research and heightened awareness on many issues. Consequently, anesthesiology has become less risky for patients. Part of the culture change has been the acceptance of critical incident reporting and an open and free discussion about what can be learned from this particular event.Trying not to assign blame, but rather look for opportunities for improvement, has increased patient safety. Our most recent efforts to encourage each member of the operating room team to speak up and address problems as they see them also is an offshoot of this culture.

At my own institution, one of our anesthesiologists, David Martin, M.D., Ph.D., has set up and monitors a "near miss" site. Short narratives are



presented about errors made in anesthesia care that have not injured patients but had the potential so to do. For example, if the label for a neuromuscular blocker has a similar color to a vasoactive amine, and the wrong drug was about to be administered but the error was recognized prior to the patient receiving the medication, it is a near-miss. Likewise, a problem with calibration of a sensor is written up on this website. Protected from the outside, it is useful to the department and helps generate a healthy discourse and raise awareness.

Jeep Pierce's vision, and the foundation he helped to create, has gone beyond anesthesia. The Institute of Medicine's report *To Err Is Human* has cited the APSF as a force for change in this arena. All of anesthesiology was lauded for the specialty's commitment to patient safety, and other organizations began to set up similar safety groups. The National Patient Safety Foundation is an American Medical Association-sponsored group that patterned itself after the APSF. It is dedicated to decreasing errors in medicine, yet it lacks the driving vision that Dr. Pierce so eloquently stated so long ago.

Much has changed in the years since the APSF was founded. In some ways, however, we have failed Dr. Pierce. Patients are still being harmed by anesthesia despite all our best efforts. So his goal must remain before us. Can it be achieved? I believe it can, but we need to continue to study our errors and learn from them in a non-threatening way. We need to incorporate everyone in the decision-making process, in a manner similar to the way in which the board of APSF is inclusive with safety experts, physicians, advance practice nurses, operating room nurses, anesthesiologist assistants and representatives from industry. To succeed, we need each group to contribute its unique knowledge toward eradication of anesthesia harm.

One of the great honors in my life is that I have come to know Dr. Ellison "Jeep" Pierce as a personal friend. He is a gentle man who has not been changed by his monumental contribution to anesthesiology and the world of medicine. Dr. Pierce remains a role model for those with vision. He had the will to follow his dream and thereby made anesthesiology safer. May each one of us continue this goal, and may we in the coming years finally be able to tell Dr. Pierce that, indeed, no patient has been harmed by anesthesia.

– D.R.B.

Revolutionary and Evolutionary

25 Years of Excellence

Mark A. Warner, M.D.
ASA President

The Anesthesia Patient Safety Foundation (APSF) is now celebrating its 25th anniversary. And what a 25 years the revolution it brought to health care has been! It is not an exaggeration to suggest that the APSF – originating from the imagination of Ellison C. "Jeep" Pierce, Jr., M.D. and his colleagues in anesthesiology – has introduced health care worldwide to the premise, promise and culture of patient safety. ASA and key industry supporters can proudly proclaim the leadership roles they have played in providing the support needed for this unprecedented contribution to advancing medicine and improving the care of patients.

In this *NEWSLETTER*, Robert K. Stoelting, M.D., President of APSF, and colleagues who have been involved in the evolution of the organization provide historical as well as current information on the anesthesia patient safety movement. New knowledge generated from APSF research grants and focused discussions on a broad array of perioperative topics in APSF workshops and meetings have pushed organizations such as the ASA and U.S. administrative entities such as the Agency for Healthcare Research and Quality (AHRQ) to develop guidelines and practice standards that have positively impacted health care. In the most sincere form of flattery, the APSF was used as the model for the creation of the National Patient Safety Foundation.

Ellison C. "Jeep" Pierce, Jr., M.D.
1928-2011

While this 25th anniversary offers a grand opportunity to reflect on the historical and current impact of APSF on patient safety, such reflection might be more appropriately focused on setting an agenda and direction for its next 25 years. The specialty of anesthesiology is evolving, and so too must the vision for APSF. Anesthesiology tomorrow will stay true to the core values of a specialty that provides care for those who suffer acute and chronic pain, including those undergoing painful procedures. However, it will by necessity, talent and purpose evolve to include all aspects of the episodes of care for these patients, ranging from their pre-procedural assessment to their postoperative management and, for those who need it, long-term hospice and palliative care. Thus, the expanse of issues to be considered by the APSF should also spread. The real question for the next 25 years is: "Can APSF have an important positive impact on patient safety across the full spectrum of perioperative care?"

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Mark A. Warner, M.D., is Professor of Anesthesiology, Dean, Mayo School of Graduate Medical Education, Mayo Clinic College of Medicine, Rochester, Minnesota.

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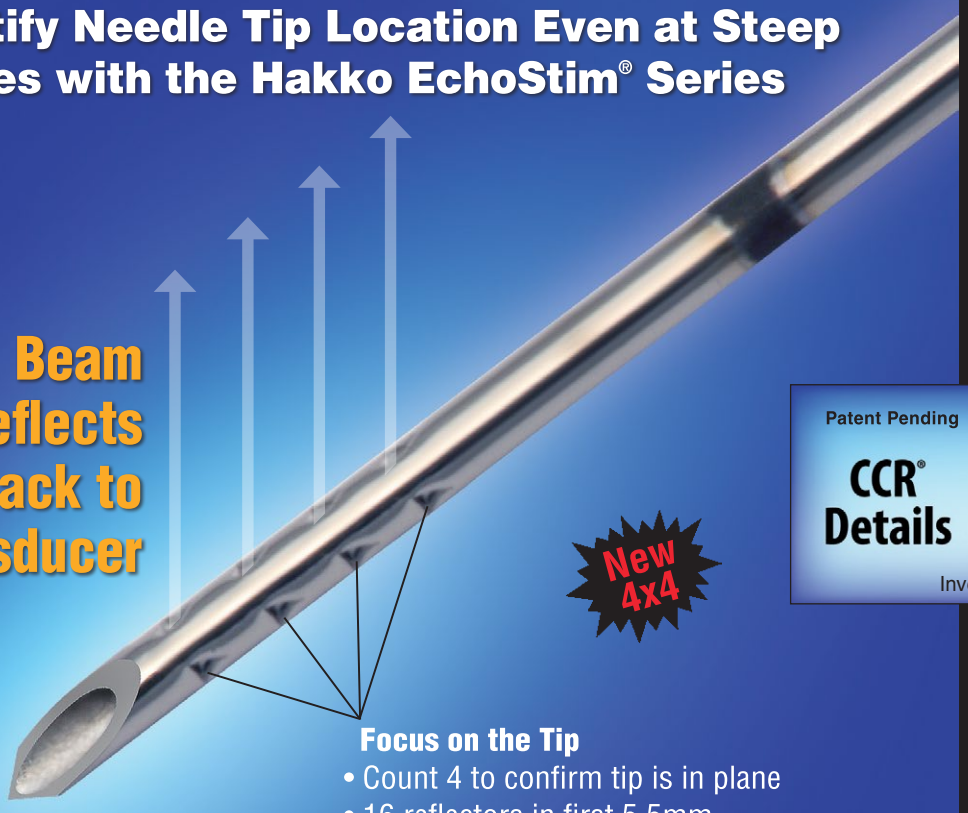

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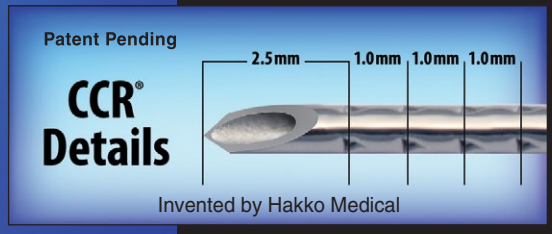

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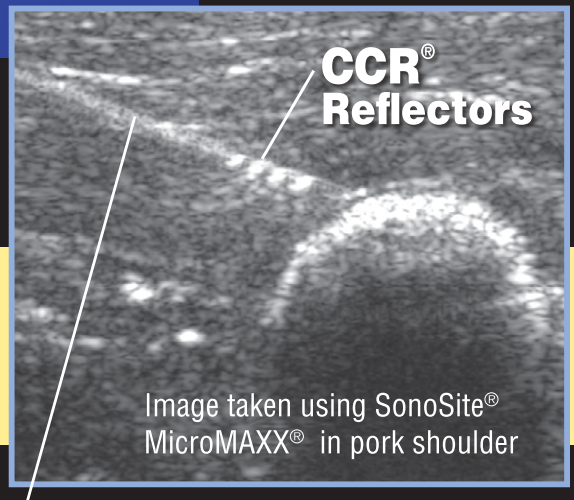

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Continued from page 6

The evolution of APSF to provide impact beyond traditional anesthesiology boundaries will require new insights, occasional uncomfortable alliances, and innovation in technology and pharmaceutical industries. Reaching beyond the operating rooms to extend the positive impact of anesthesiology's patient safety culture will require anesthesiologists to step forward and lead a union of forces who, thus far, have not always been collaborative.

Let's take a simple example: each year, literally hundreds of patients in the U.S. either die or suffer anoxic brain damage from perioperative respiratory failure associated with the use of opioids for analgesia. Many of these events may be preventable. Yet there is no single group that takes ownership of this devastating perioperative problem. Anesthesiologists and other anesthesia caregivers provide intraoperative and some postoperative opioid analgesia. Surgeons and well-meaning hospitalists prescribe opioids for other parts of the perioperative episode. Nurses often administer these opioids. And hospital administrators may (or may not) provide the resources needed for appropriate monitoring of respiration for patients who receive these medications. As an independent foundation, APSF can provide the resources, primarily through grants, to perform the research and inclusive workshops to better understand this phenomenon and stimulate practice standards and industry innovation that will reduce the frequency and severity of respiratory depression's catastrophic affects.

This June in Phoenix, the APSF will sponsor a workshop that will specifically focus electronic monitoring strategies to detect significant opioid-related respiratory depression during the perioperative period. Later in October, it will host a special session on Saturday afternoon of the ASA's Annual Meeting in Chicago. The audience at that session will be asked to provide its ideas and priorities for perioperative patient safety in the coming years. You are all invited to attend and help APSF set its agenda for this next stage in the evolution of patient safety.

In the meantime, please take time to read this *NEWSLETTER* about the remarkable advances that the APSF, with outstanding and sustained ASA and industry support, has generated. Congratulations to the many colleagues in anesthesiology and industry who have made the specialty the national leader in patient safety. It's been exciting to have been swept along in the moment of patient safety's revolution, and it will be gratifying to help push forward the momentum as patient safety and APSF evolve over the next 25 years.

Essential Monitoring Strategies to Detect Clinically Significant Drug-Induced Respiratory Depression in the Postoperative Period

Royal Palms Resort and Spa
Phoenix, Arizona
Wednesday, June 8, 2011

APSF believes that clinically significant drug-induced respiratory depression in the postoperative period is a serious patient safety risk.

APSF further believes that continuous electronic monitoring of oxygenation and ventilation with available and developing technology offer the opportunity for a prompt improvement in patient safety.

The goals of this one-day conference are to define the problem and identify electronic monitoring strategies that will provide early warning of clinically significant postoperative respiratory depression.

Experts from clinical medicine (nursing and physicians), industry (manufacturers of monitoring devices), hospital administration, the insurance industry, regulatory agencies and families of injured patients will provide input.

Speakers and attendees will be asked to focus on the following three questions:

- Should electronic monitoring be utilized to facilitate detection of drug-induced postoperative respiratory depression?
- If "yes" to electronic monitoring, who should be monitored (inclusive or selective), and what monitors/technology should be utilized?
- If "no" to electronic monitoring, why?

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For registration information, contact Robert K. Stoelting, M.D., President, APSF at **stoelting@apsf.org**.

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1. NAROPIN Prescribing Information. Data on file. **2.** Writer WDR, Stienstra R, Eddleston JM, et al. Neonatal outcome and mode of delivery after epidural analgesia for labour with ropivacaine and bupivacaine: a prospective meta-analysis. *Br J Anaesth.* 1998;81:713-717. **3.** Asik I, Göktug A, Gülay I, Alkis N, Uysalel A. Comparison of bupivacaine 0.2% and ropivacaine 0.2% combined with fentanyl for epidural analgesia during labour. *Eur J Anaesthesiol.* 2002;19:263-270.

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INDICATIONS AND USAGE Naropin is indicated for the production of local or regional anesthesia for surgery and for acute pain management. Surgical Anesthesia: epidural block for surgery including cesarean section; major nerve block; local infiltration. Acute Pain Management: epidural continuous infusion or intermittent bolus, e.g., postoperative or labor; local infiltration.

CONTRAINDICATIONS Naropin is contraindicated in patients with a known hypersensitivity to ropivacaine or to any local anesthetic agent of the amide type.

WARNINGS In performing Naropin blocks, unintended intravenous injection is possible and may result in cardiac arrhythmia or cardiac arrest. The potential for successful resuscitation has not been studied in humans. There have been rare reports of cardiac arrest during the use of Naropin for epidural anesthesia or peripheral nerve blockade, the majority of which occurred after unintentional accidental intravascular administration in elderly patients and in patients with concomitant heart disease. In some instances, resuscitation has been difficult. Should cardiac arrest occur, prolonged resuscitative efforts may be required to improve the probability of a successful outcome. Naropin should be administered in incremental doses. It is not recommended for emergency situations, where a fast onset of surgical anesthesia is necessary. Historically, pregnant patients were reported to have a high risk for cardiac arrhythmias, cardiac/circulatory arrest and death when 0.75% bupivacaine (another member of the amino amide class of local anesthetics) was inadvertently rapidly injected intravenously. Prior to receiving major blocks the general condition of the patient should be optimized and the patient should have an i.v. line inserted. All necessary precautions should be taken to avoid intravascular injection. Local anesthetics should only be administered by clinicians who are well versed in the diagnosis and management of dose-related toxicity and other acute emergencies that may arise from the block to be employed, and then only after ensuring the **immediate (without delay)** availability of oxygen, other resuscitative drugs, cardiopulmonary resuscitative equipment, and the personnel resources needed for proper management of toxic reactions and related emergencies (See also ADVERSE REACTIONS, PRECAUTIONS, and MANAGEMENT OF LOCAL ANESTHETIC EMERGENCIES). Delay in proper management of dose-related toxicity, underventilation from any cause, and/or altered sensitivity may lead to the development of acidosis, cardiac arrest and, possibly, death. Solutions of Naropin should not be used for the production of obstetrical paracervical block anesthesia, retrobulbar block, or spinal anesthesia (subarachnoid block) due to insufficient data to support such use. Intravenous regional anesthesia (bier block) should not be performed due to a lack of clinical experience and the risk of attaining toxic blood levels of ropivacaine. Intra-articular infusions of local anesthetics following arthroscopic and other surgical procedures is an unapproved use, and there have been post-marketing reports of chondrolysis in patients receiving such infusions. The majority of reported cases of chondrolysis have involved the shoulder joint; cases of gleno-humeral chondrolysis have been described in pediatric and adult patients following intra-articular infusions of local anesthetics with and without epinephrine for periods of 48 to 72 hours. There is insufficient information to determine whether shorter infusion periods are not associated with these findings. The time of onset of symptoms, such as joint pain, stiffness and loss of motion can be variable, but may begin as early as the 2nd month after surgery. Currently, there is no effective treatment for chondrolysis; patients who experienced chondrolysis have required additional diagnostic and therapeutic procedures and some required arthroplasty or shoulder replacement. It is essential that aspiration for blood, or cerebrospinal fluid (where applicable), be done prior to injecting any local anesthetic, both the original dose and all subsequent doses, to avoid intravascular or subarachnoid injection. However, a negative aspiration does *not* ensure against an intravascular or subarachnoid injection. A well-known risk of epidural anesthesia may be an unintentional subarachnoid injection of local anesthetic. Two clinical studies have been performed to verify the safety of Naropin at a volume of 3 mL injected into the subarachnoid space since this dose represents an incremental epidural volume that could be unintentionally injected. The 15 and 22.5 mg doses injected resulted in sensory levels as high as T5 and T4, respectively. Anesthesia to pinprick started in the sacral dermatomes in 2-3 minutes, extended to the T10 level in 10-13 minutes and lasted for approximately 2 hours. The results of these two clinical studies showed that a 3 mL dose did not produce any serious adverse events when spinal anesthesia blockade was achieved. Naropin should be used with caution in patients receiving other local anesthetics or agents structurally related to amide-type local anesthetics, since the toxic effects of these drugs are additive. Patients treated with class III antiarrhythmic drugs (e.g., amiodarone) should be under close surveillance and ECG monitoring considered, since cardiac effects may be additive.

PRECAUTIONS: General: The safe and effective use of local anesthetics depends on proper dosage, correct technique, adequate precautions and readiness for emergencies. Resuscitative equipment, oxygen and other resuscitative drugs should be available for immediate use. (See WARNINGS and ADVERSE REACTIONS.) The lowest dosage that results in effective anesthesia should be used to avoid high plasma levels and serious adverse events. Injections should be made slowly and incrementally, with frequent aspirations before and during the injection to avoid intravascular injection. When a continuous catheter technique is used, syringe aspirations should also be performed before and during each supplemental injection. During the administration of epidural anesthesia, it is recommended that a test dose of a local anesthetic with a fast onset be administered initially and that the patient be monitored for central nervous system and cardiovascular toxicity, as well as for signs of unintended intrathecal administration before proceeding. When clinical conditions permit, consideration should be given to employing local anesthetic solutions, which contain epinephrine for the test dose because circulatory changes compatible with epinephrine may also serve as a warning sign of unintended intravascular injection. An intravascular injection is still possible even if aspirations for blood are negative. Administration of higher than recommended doses of Naropin to achieve greater motor blockade or increased duration of sensory blockade may result in cardiovascular depression, particularly in the event of inadvertent intravascular injection. Tolerance to elevated blood levels varies with the physical condition of the patient. Debilitated, elderly patients and acutely ill patients should be given reduced doses commensurate with their age and physical condition. Local anesthetics should also be used with caution in patients with hypotension, hypovolemia or heart block. Careful and constant monitoring of cardiovascular and respiratory vital signs (adequacy of ventilation) and the patient's state of consciousness should be performed after each local anesthetic injection. It should be kept in mind at such times that restlessness, anxiety, incoherent speech, light-headedness, numbness and tingling of the mouth and lips, metallic taste, tinnitus, dizziness, blurred vision, tremors, twitching, depression, or drowsiness may be early warning signs of central nervous system toxicity. Because amide-type local anesthetics such as ropivacaine are metabolized by the liver, these drugs, especially repeat doses, should be used cautiously in patients with hepatic disease. Patients with severe hepatic disease, because of their inability to metabolize local anesthetics normally, are at a greater risk of developing toxic plasma concentrations. Local anesthetics should also be used with caution in patients with impaired cardiovascular function because they may be less able to compensate for functional changes associated with the prolongation of A-V conduction produced by these drugs. Many drugs used during the conduct of anesthesia are considered potential triggering agents for malignant hyperthermia (MH). Amide-type local anesthetics are not known to trigger this reaction. However, since the need for supplemental general anesthesia cannot be predicted in advance, it is suggested that a standard protocol for MH management should be available. **Epidural Anesthesia:** During epidural administration, Naropin should be administered in incremental doses of 3 to 5 mL with sufficient time between doses to detect toxic manifestations of unintentional intravascular or intrathecal injection. Syringe aspirations should also be performed before and during each supplemental injection in continuous (intermittent) catheter techniques. An intravascular injection is still possible even if aspirations for blood are negative. During the administration of epidural anesthesia, it is recommended that a test dose be administered initially and the effects monitored before the full dose is given. When clinical conditions permit, the test dose should contain an appropriate dose of epinephrine to serve as a warning of unintentional intravascular injection. If injected into a blood vessel, this amount of epinephrine is likely to produce a transient "epinephrine response" within 45 seconds, consisting of an increase in heart rate and systolic blood pressure, circumoral pallor, palpitations and nervousness in the unsedated patient. The sedated patient may exhibit only a pulse rate increase of 20 or more beats per minute for 15 or more seconds. Therefore, following the test dose, the heart should be continuously monitored for a heart rate increase. Patients on beta-blockers may not manifest changes in heart rate, but blood pressure monitoring can detect a rise in systolic blood pressure. A test dose of a shortacting amide anesthetic such as lidocaine is recommended to detect an unintentional intrathecal administration. This will be manifested within a few minutes by signs of spinal block (e.g., decreased sensation of the buttocks, paresis of the legs, or, in the sedated patient, absent knee jerk). An intravascular or subarachnoid injection is still possible even if results of the test dose are negative. The test dose itself may produce a systemic toxic reaction, high spinal or epinephrine-induced cardiovascular effects. **Use in Brachial Plexus Block:** Ropivacine plasma concentrations may approach the threshold for central nervous system toxicity after the administration of 300 mg of ropivacaine for brachial plexus block. Caution should be exercised when using the 300 mg dose. (See OVERDOSAGE.) The dose for a major nerve block must be adjusted according to the site of administration and patient status. Supraclavicular brachial plexus blocks may be associated with a higher frequency of serious adverse reactions, regardless of the local anesthetic used. **Use in Peripheral Nerve Block:** Major peripheral nerve blocks may result in the administration of a large volume of local anesthetic in highly vascularized areas, often close to large vessels where there is an increased risk of intravascular injection and/or rapid systemic absorption, which can lead to high plasma concentrations. **Use in Head and Neck Area:** Small doses of local anesthetics injected into the head and neck area may produce adverse reactions similar to systemic toxicity seen with unintentional intravascular injections of larger doses. The injection procedures require the utmost care. Confusion, convulsions, respiratory depression, and/or respiratory arrest, and cardiovascular stimulation or depression have been reported. These reactions may be due to intra-arterial injection of the local anesthetic with retrograde flow to the cerebral circulation. Patients receiving these blocks should have their circulation and respiration monitored and be constantly observed. Resuscitative equipment and personnel for treating adverse reactions should be immediately available. Dosage recommendations should not be exceeded. (See DOSAGE AND ADMINISTRATION.) **Use in Ophthalmic Surgery:** The use of Naropin in retrobulbar blocks for ophthalmic surgery has not been studied. Until appropriate experience is gained, the use of Naropin for such surgery is not recommended. **Drug Interactions:** Specific trials studying the interaction between ropivacaine and class III antiarrhythmic drugs (e.g., amiodarone) have not been performed, but caution is advised (see WARNINGS). Naropin should be used with caution in patients receiving other local anesthetics or agents structurally related to amide-type local anesthetics, since the toxic effects of these drugs are additive. Cytochrome P4501A2 is involved in the formation of 3-hydroxy ropivacaine, the major metabolite. *In vivo*, the plasma clearance of ropivacaine was reduced by 70% during coadministration of fluvoxamine (25 mg bid for 2 days), a selective and potent CYP1A2 inhibitor. Thus strong inhibitors of cytochrome P4501A2, such as fluvoxamine, given concomitantly during administration of Naropin, can interact with Naropin leading to increased ropivacaine plasma levels. Caution should be exercised when CYP1A2 inhibitors are coadministered. Possible interactions with drugs known to be metabolized by CYP1A2 via competitive inhibition such as theophylline and imipramine may also occur. Coadministration of a selective and potent inhibitor of CYP3A4, ketoconazole (100 mg bid for 2 days with ropivacaine infusion administered 1 hour after ketoconazole) caused a 15% reduction in *in-vivo* plasma clearance of ropivacaine. **Pregnancy Category B:** There are no adequate or well-controlled studies in pregnant women of the effects of Naropin on the developing fetus. Naropin should only be used during pregnancy if the benefits outweigh the risk. **Labor and Delivery:** Local anesthetics, including ropivacaine, rapidly cross the placenta, and when used for epidural block can cause varying degrees of maternal, fetal and neonatal toxicity (see CLINICAL PHARMACOLOGY and PHARMACOKINETICS). The incidence and degree of toxicity depend upon the procedure performed, the type and amount of drug used, and the technique of drug administration. Adverse reactions in the parturient, fetus and neonate involve alterations of the central nervous system, peripheral vascular tone and cardiac function. Maternal hypotension has resulted from regional anesthesia with Naropin for obstetrical pain relief. Local anesthetics produce vasodilation by blocking sympathetic nerves. Elevating the patient's legs and positioning her on her left side will help prevent decreases in blood pressure. The fetal heart rate also should be monitored continuously, and electronic fetal monitoring is highly advisable. Epidural anesthesia has been reported to prolong the second stage of labor by removing the patient's reflex urge to bear down or by interfering with motor function. Spontaneous vertex delivery occurred more frequently in patients receiving Naropin than in those receiving bupivacaine. **Nursing Mothers:** Some local anesthetic drugs are excreted in human milk and caution should be exercised when they are administered to a nursing woman. The excretion of ropivacaine or its metabolites in human milk has not been studied. Based on the milk/plasma concentration ratio in rats, the estimated daily dose to a pup will be about 4% of the dose given to the mother. Assuming that the milk/plasma concentration in humans is of the same order, the total Naropin dose to which the baby is exposed by breast-feeding is far lower than by exposure *in utero* in pregnant women at term (see Precautions). **Pediatric Use:** The safety and efficacy of Naropin in pediatric patients have not been established. **Geriatric Use:** Of the 2,978 subjects that were administered Naropin Injection in 71 controlled and uncontrolled clinical studies, 803 patients (27%) were 65 years of age or older, which includes 127 patients (4%) 75 years of age and over. Naropin Injection was found to be safe and effective in the patients in these studies. Clinical data in one published article indicate that differences in various pharmacodynamic measures were observed with increasing age. In one study, the upper level of analgesia increased with age, the maximum decrease of mean arterial pressure (MAP) declined with age during the first hour after epidural administration, and the intensity of motor blockade increased with age. This drug and its metabolites are known to be excreted by the kidney, and the risk of toxic reactions to this drug may be greater in patients with impaired renal function. Elderly patients are more likely to have decreased hepatic, renal, or cardiac function, as well as concomitant disease. Therefore, care should be taken in dose selection, starting at the low end of the dosage range, and it may be useful to monitor renal function. (See PHARMACOKINETICS, Elimination.)

ADVERSE REACTIONS Reactions to ropivacaine are characteristic of those associated with other amidetype local anesthetics. A major cause of adverse reactions to this group of drugs may be associated with excessive plasma levels, which may be due to overdosage, unintentional intravascular injection or slow metabolic degradation. The reported adverse events are derived from clinical studies conducted in the U.S. and other countries. The reference drug was usually bupivacaine. The studies used a variety of premedications, sedatives, and surgical procedures of varying length. A total of 3,988 patients have been exposed to Naropin at concentrations up to 1.0% in clinical trials. Each patient was counted once for each type of adverse event. **Incidence ≥5%:** For the indications of epidural administration in surgery, cesarean section, postoperative pain management, peripheral nerve block, and local infiltration, the following treatment-emergent adverse events were reported with an incidence of ≥5% in all clinical studies (N=3988): hypotension (37.0%), nausea (24.8%), vomiting (11.6%), bradycardia (9.3%), fever (9.2%), pain (8.0%), postoperative complications (7.1%), anemia (6.1%), paresthesia (5.6%), headache (5.1%), pruritus (5.1%), and back pain (5.0%). **Incidence 1-5%:** Urinary retention, dizziness, rigors, hypertension, tachycardia, anxiety, oliguria, hypoesthesia, chest pain, hypokalemia, dyspnea, cramps, and urinary tract infection. **Incidence in Controlled Clinical Trials:** The reported adverse events are derived from controlled clinical studies with Naropin (concentrations ranged from 0.125% to 1.0% for Naropin and 0.25% to 0.75% for bupivacaine) in the U.S. and other countries involving 3,094 patients. Tables 3A and 3B list adverse events (number and percentage) that occurred in at least 1% of Naropin-treated patients in these studies. The majority of patients receiving concentrations higher than 5.0 mg/mL (0.5%) were treated with Naropin.

Table 3A
Adverse Events Reported in ≥1% of Adult Patients Receiving Regional or Local Anesthesia
(Surgery, Labor, Cesarean Section, Post-Operative Pain Management, Peripheral Nerve Block and Local Infiltration)

Adverse Reaction	Naropin total N=1661		Bupivacaine total N=1433	
	N	(%)	N	(%)
Hypotension	536	(32.3)	408	(28.5)
Nausea	283	(17.0)	207	(14.4)
Vomiting	117	(7.0)	88	(6.1)
Bradycardia	96	(5.8)	73	(5.1)
Headache	84	(5.1)	68	(4.7)
Paresthesia	82	(4.9)	57	(4.0)
Back pain	73	(4.4)	75	(5.2)
Pain	71	(4.3)	71	(5.0)
Pruritus	63	(3.8)	40	(2.8)
Fever	61	(3.7)	37	(2.6)
Dizziness	42	(2.5)	23	(1.6)
Rigors (Chills)	42	(2.5)	24	(1.7)
Postoperative complications	41	(2.5)	44	(3.1)
Hypoesthesia	27	(1.6)	24	(1.7)
Urinary retention	23	(1.4)	20	(1.4)
Progression of labor poor/failed	23	(1.4)	22	(1.5)
Anxiety	21	(1.3)	11	(0.8)
Breast disorder, breast-feeding	21	(1.3)	12	(0.8)
Rhinitis	18	(1.1)	13	(0.9)

Table 3B
Adverse Events Reported in ≥1% of Fetuses or Neonates of Mothers
Who Received Regional Anesthesia (Cesarean Section and Labor Studies)

Adverse Reaction	Naropin total N=1661		Bupivacaine total N=1433	
	N	(%)	N	(%)
Fetal bradycardia	77	(12.1)	68	(11.9)
Neonatal jaundice	49	(7.7)	47	(8.2)
Neonatal complication-NOS	42	(6.6)	38	(6.6)
Apgar score low	18	(2.8)	14	(2.4)
Neonatal respiratory disorder	17	(2.7)	18	(3.1)
Neonatal tachypnea	14	(2.2)	15	(2.6)
Neonatal fever	13	(2.0)	14	(2.4)
Fetal tachycardia	13	(2.0)	12	(2.1)
Fetal distress	11	(1.7)	10	(1.7)
Neonatal infection	10	(1.6)	8	(1.4)
Neonatal hypoglycemia	8	(1.3)	16	(2.8)

OVERDOSAGE Acute emergencies from local anesthetics are generally related to high plasma levels encountered, or large doses administered, during therapeutic use of local anesthetics or to unintended subarachnoid or intravascular injection of local anesthetic solution. (See ADVERSE REACTIONS, WARNINGS, and PRECAUTIONS.)

MANAGEMENT OF LOCAL ANESTHETIC EMERGENCIES: Therapy with Naropin should be discontinued at the first sign of toxicity. No specific information is available for the treatment of toxicity with Naropin; therefore, treatment should be symptomatic and supportive. The first consideration is prevention, best accomplished by incremental injection of Naropin, careful and constant monitoring of cardiovascular and respiratory vital signs and the patient's state of consciousness after each local anesthetic and during continuous infusion. At the first sign of change in mental status, oxygen should be administered. The first step in the management of systemic toxic reactions, as well as underventilation or apnea due to unintentional subarachnoid injection of drug solution, consists of immediate attention to the establishment and maintenance of a patent airway and effective assisted or controlled ventilation with 100% oxygen with a delivery system capable of permitting immediate positive airway pressure by mask. Circulation should be assisted as necessary. This may prevent convulsions if they have not already occurred. If necessary, use drugs to control convulsions. Intravenous barbiturates, anticonvulsant agents, or muscle relaxants should only be administered by those familiar with their use. Immediately after the institution of these ventilatory measures, the adequacy of the circulation should be evaluated. Supportive treatment of circulatory depression may require administration of intravenous fluids, and, when appropriate, a vasopressor dictated by the clinical situation (such as ephedrine or epinephrine to enhance myocardial contractile force). Should cardiac arrest occur, prolonged resuscitative efforts may be required to improve the probability of a successful outcome. The mean dosages of ropivacaine producing seizures, after intravenous infusion in dogs, nonpregnant and pregnant sheep were 4.9, 6.1 and 5.9 mg/kg, respectively. These doses were associated with peak arterial total plasma concentrations of 11.4, 4.3 and 5.0 µg/mL, respectively. In human volunteers given intravenous Naropin, the mean (min-max) maximum tolerated total and free arterial plasma concentrations were 4.3 (3.4-5.3) and 0.6 (0.3-0.9) µg/mL respectively, at which time moderate CNS symptoms (muscle twitching) were noted. Clinical data from patients experiencing local anesthetic induced convulsions demonstrated rapid development of hypoxia, hypercarbia and acidosis within a minute of the onset of convulsions. These observations suggest that oxygen consumption and carbon dioxide production are greatly increased during local anesthetic convulsions and emphasize the importance of immediate and effective ventilation with oxygen, which may avoid cardiac arrest. If difficulty is encountered in the maintenance of a patent airway or if prolonged ventilatory support (assisted or controlled) is indicated, endotracheal intubation, employing drugs and techniques familiar to the clinician, may be indicated after initial administration of oxygen by mask. The supine position is dangerous in pregnant women at term because of aortocaval compression by the gravid uterus. Therefore, during treatment of systemic toxicity, maternal hypotension or fetal bradycardia following regional block, the parturient should be maintained in the left lateral decubitus position if possible, or manual displacement of the uterus off the great vessels should be accomplished. Resuscitation of obstetrical patients may take longer than resuscitation of nonpregnant patients and closed-chest cardiac compression may be ineffective. Rapid delivery of the fetus may improve the response to resuscitative efforts.

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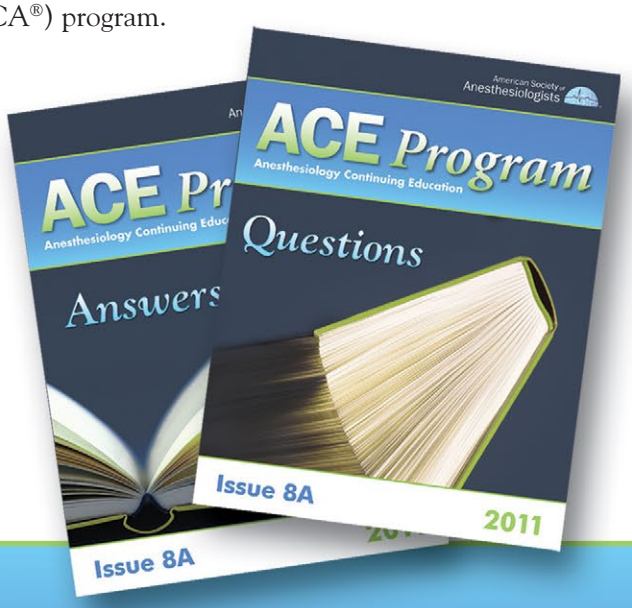

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APSF Celebrates 25 Years

Robert K. Stoelting, M.D., President
APSF

The Anesthesia Patient Safety Foundation (APSF) celebrated its 25th anniversary in October 2010 (Figure 1).[1] A quarter-century after establishing "patient safety" as a specific concept and discipline, APSF's vision remains that *"no patient shall be harmed by anesthesia."* 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; and*
- *National and international exchange of information and ideas.*

Figure 1: APSF 25th Anniversary logo

Anesthesiology was the first medical specialty to champion patient safety as a specific focus.[2,3] The coincidence of multiple factors beginning in the late 1970s led to significant changes in practice that have decreased mortality and catastrophic morbidity caused by anesthesia administration. APSF was the first independent multidisciplinary organization (practitioners, equipment and drug manufacturers, and many related professionals) created expressly to help avoid preventable adverse clinical outcomes, especially those related to human error. Anesthesiology is widely recognized as the pioneering leader in patient safety efforts.

Although reports were anecdotal and imperfect, from the 1950s through the 1970s, there was a widespread impression that anesthesia care itself caused a mortality of 1-2/10,000 anesthetics, which was perceived to be unacceptably high. Anesthesiologists constituted 3 percent of physicians and generated 3 percent of the malpractice claims, but those claims accounted for a disproportionately high 12 percent of medical liability insurance payout.

A seminal publication in 1978 described the use of the aviation-inspired critical incident analysis technique to understand the causes of anesthesia-related mishaps and injuries.[4] In the early 1980s, national media publicity turned a harsh spotlight on anesthesia accidents that injured patients.[5] Thus stimulated, and avoiding the urge to fixate on tort reform, Ellison C. Pierce, Jr., M.D. (1928-2011), the 1984 president of ASA, constituted a new ASA standing Committee on Safety and Risk Management, emphasizing the need to address the causes of patient injury. That same year, Dr. Pierce and his Harvard colleagues convened the International Symposium on the Prevention of Anesthesia Mortality and Morbidity, which constituted the first organized examination of what was soon to be known as "anesthesia patient safety." There the idea for the APSF was born.

Robert K. Stoelting, M.D. is President, Anesthesia Patient Safety Foundation.

Early APSF History

APSF was launched in late 1985 as an independent nonprofit corporation, thus allowing organizational agility and the freedom to tackle openly the sensitive issue of anesthesia accidents.[1,3] Initial financial support came from the ASA and several corporate sponsors. Members of the APSF Board of


ANESTHESIA PATIENT SAFETY FOUNDATION
NEWSLETTER
Volume 1, No. 1, pp 1-8 March, 1986
Safety Foundation Organized
Statement of Purpose

Table 1: Seminal Articles in *APSF Newsletter* *(modified from Eichhorn[1])*

March 1986	Closed Claims Study Seeks Data	Richard J. Ward, M.D.
March 1986	Is There Minimal Essential Monitoring?	J.S. Gravenstein, M.D.
March 1987	Outpatient Anesthesia: No Double Standard	Bernard V. Wetchler, M.D.
March 1989	FDA Applauds APSF Efforts	Joseph S. Arcarese Peter B. Carstensen
Spring 1991	ASA Standards Amended: CO_2 Seen After Intubation now the "Standard of Care"	John H. Eichhorn, M.D.
Fall 1995	Simulator Training in Anesthesia Growing Rapidly	David M. Gaba, M.D.
Spring 1998	Lancet Article Addresses a Different Type of Safety Question: Elderly Suffer Prolonged Postoperative Cognitive Dysfunction	J. S. Gravenstein, M.D.
Winter 2000	Landmark Report Published on Patient Safety	David M. Gaba, M.D. Jeffrey B. Cooper, Ph.D.
Winter 2001	APSF Endorses Use of Automated Record Keepers	APSF Board of Directors
Winter 2003	Virtual Anesthesia Machine Has Worldwide Impact	Sem Lampotang, Ph.D.
Winter 2003	Cannister Fires Become a Hot Safety Concern	Michael A. Olympia, M.D. Robert C. Morell, M.D.
Spring 2004 Summer 2004	Dear Safety Information Response System (SIRS) Debut Column APSF Stresses Use of Audible Monitor Alarms	Michael A. Olympio, M.D. Robert K. Stoelting, M.D.
Spring 2005	Fatigue and the Practice of Anesthesiology	Steve Howard, M.D.
Summer 2007	Beach Chair Position May Decrease Cerebral Perfusion	David J Cullen, M.D. Robert R. Kirby, M.D.
Winter 2007	Dangers of Postoperative Opioids	Matthew B. Weinger, M.D.
Spring 2008	Lipid Emulsion: The Time Has Come for Treating Systemic Local Anesthetic Toxicity	Guy L. Weinberg, M.D. David Mayer, M.D.
Summer 2009	Dangers of Postoperative Opioids – Is There A Cure?	Robert K. Stoelting, M.D. Matthew B. Weinger, M.D.
Spring 2010	APSF Hosts Medication Safety Conference	John H. Eichhorn, M.D.

Directors represent a broad spectrum of stakeholders, including anesthesiologists, nurse anesthetists, nurses, manufacturers of equipment and drugs, regulators, risk managers, attorneys, insurers and engineers.

APSF grew rapidly in impact. The highly respected *APSF Newsletter* was first published in March 1986 with John H. Eichhorn, M.D. as the editor. The newsletter became and remains the most effective vehicle for rapid dissemination of anesthesia patient safety information, with a current circulation in excess of 94,000 recipients (Table 1).[1] The APSF research grant program has funded many projects that provided insight into and suggested solutions for safety problems. At the end of 2010, APSF had funded 92 grants and awarded more than $6.77 million since its initiation in 1987. Prior to APSF, there was no centralized effort to fund research directed specifically to patient safety. Over the years, a cadre of patient safety investigators has evolved as a result of the APSF research awards program.

Continued on page 14

Continued from page 13

Technology Advances

In the early 1980s, important advances in technology became available. Electronic monitoring that extended the human senses (inspired oxygen measurement, pulse oximetry, capnography) allowed genuine, real-time continuous monitoring of oxygen delivery and patient ventilation and oxygenation. Other engineering advances made anesthesia delivery systems safer, such as gas ratio protection that prevented accidental shut-off of oxygen flow. The FDA anesthesia machine checkout protocol was developed and widely adopted. Improvements in anesthesia medications afforded more specific and controllable pharmacological actions and fewer dangerous side effects.

Pulse oximeter

In the mid 1980s, medical liability concerns continued. ASA inaugurated the Closed Claims Study, which continues today and has yielded new understandings of adverse events through study of anesthesia mishaps. Also, a committee was formed at Harvard to study the causes of anesthesia accidents there. The analysis led to the first standards of practice for minimum intraoperative monitoring. The intention was to codify and institutionalize specific behaviors that constituted "safety monitoring," a strategy for preventing anesthesia accidents. In 1986, the ASA adopted an expanded form as a national standard, a landmark step for a medical professional society and which epitomized the lead role taken by anesthesiology in the nascent patient safety movement. Additional ASA standards, guidelines and practice parameters followed, including the widely respected "difficult airway" guideline.

Improving Education

Human factor and resource issues also played key roles in improving anesthesia patient safety. In 1990, the APSF and the FDA convened an unprecedented expert workshop on human error in anesthesia practice that helped stimulate later advances. The improved quality both of trainees entering the field and anesthesia training programs are certainly important elements of the anesthesia patient safety story. The extension of the residency to three years and the explosion of anesthesia textbooks, journals and meetings contributed via the knowledge base. The incorporation of sessions on safety topics in the scientific program of the ASA Annual Meeting also raised awareness while disseminating research and information.

In the late 1980s, supported by APSF grant funding, realistic patient simulators were introduced into anesthesiology. Further publicity and advocacy from APSF have led to anesthesiology becoming the leader in the application and adoption of simulators, with strong patient safety implications through education (residents attempting new skills for the first time on a mannequin), training (teamwork, critical event management) and research (human performance). Use of realistic simulators has now become common in several other specialties.


Resident team training exercise with patient simulator.

Others Adopt APSF Model

The success of the anesthesia patient safety movement was recognized significantly in 1996 when the American Medical Association and corporate partners founded the National Patient Safety Foundation, based on the APSF model. Further recognition for safety efforts and leadership came to the APSF in the landmark 1999 report from the Institute of Medicine on errors in medical care. A June 21, 2005 front page article in *The Wall Street Journal* singled out anesthesiology, ASA and APSF for their roles in making anesthesia safer, resulting in dramatic decreases in professional liability insurance premiums paid by anesthesiologists.

A "culture of safety" has developed in anesthesia practice, highlighted by the hard work of the APSF and the ASA, as well as by the adoption of a more systems-based approach by many anesthesia departments and groups interested in optimizing outcome of anesthesia care. Overall, the combined impact of all the initiatives has been a 10- to 20-fold reduction in mortality and catastrophic morbidity for healthy patients undergoing routine anesthetics, an evolution of which the entire profession can be justifiably proud. By the mid 1990s, liability payouts

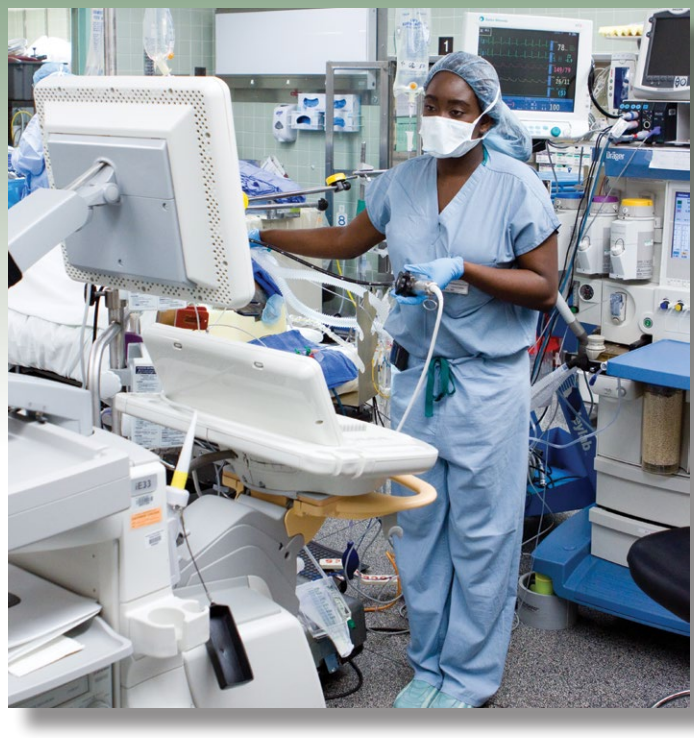
had decreased to a proportionate percentage, and the insurance "risk relativity rating" for anesthesiology compared to other specialties had been dramatically reduced.

Future Challenges and Opportunities

The work of improving anesthesia patient safety continues. Equipment and systems still at times fail and, also, basic preventable human errors still do sometimes occur. Increasing "production pressure" and expanding clinical demands in the face of diminishing resources may threaten previously won safety gains.

APSF continues to work diligently both on established tenets and new safety principles. Recent emphasis has been on integrating electronic anesthesia information management systems and audible alarms on physiologic monitors into safety strategies. This stimulated major projects to standardize terminology for anesthesia records and definitions for a proposed widespread anesthesia outcome reporting system that is being developed by the APSF Data Dictionary Task Force/ International Organization for Terminology in Anesthesia committee, with support from clinicians and vendors of automated information systems. Medication safety in the operating room and electronic monitoring of patients at risk for drug-induced respiratory depression in the postoperative period represent opportunities to improve patient safety.

APSF is urging anesthesia professionals to view oxygen as a drug with indications and risks when confronted with operations on the upper body and a concomitant risk of fire. In this regard, APSF has developed an operating room fire safety video for anesthesia professionals. APSF is the principal financial supporter for the creation of a pediatric anesthesia adverse events registry being developed by the Society for Pediatric Anesthesia. An adverse events registry for cognitive dysfunction after surgery in the head-up position has been



established by APSF, and a research grant to study the effects of the head above heart surgical position on the adequacy of cerebral perfusion has been funded by APSF.

Changes designed to improve patient safety often parallel the experience in aviation. Anesthesia safety, like aviation safety, was achieved by applying a host of changes that made sense (seemed like the right thing to do) and were based on an understanding of human factor principles. Improved anesthesia patient safety reflects doing a number of "little things" that, in the aggregate, make a big difference. Insisting on evidence-based data to justify patient safety changes may be counterproductive and delay adoption of important safety technology (pulse oximetry, capnography, audible physiologic alarms, technology training, electronic medical records).

The APSF persists in pursuit of its mission of zero tolerance for injury to patients. It serves as a model for the pioneering collaboration and commitment of the entire constellation of anesthesia-related professions to the common goal of patient safety.

Much remains to be accomplished, but the past 25 years and the contributions of APSF to anesthesia patient safety can be proudly viewed by all as a success story unique to the medical specialty of anesthesiology.

References are available at the back of the online version of this NEWSLETTER *at* ***www.asahq.org****, or by request by e-mailing* ***communications@asahq.org****.*

Anesthesia Patient Safety Foundation Turns 25,

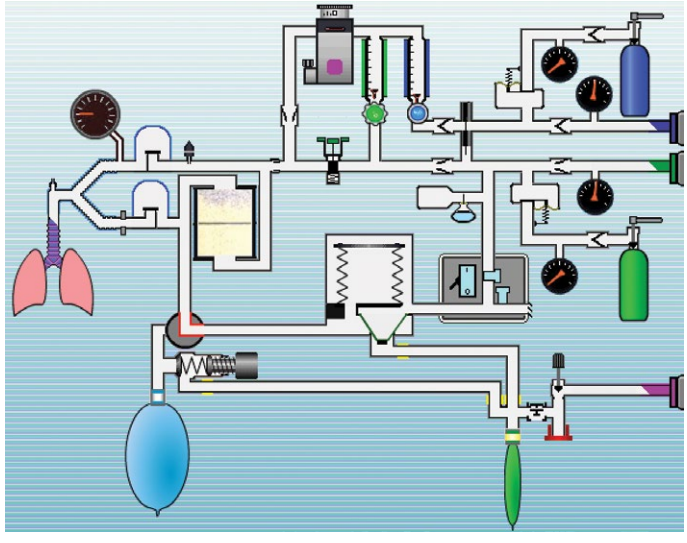
John H. Eichhorn, M.D.

The Anesthesia Patient Safety Foundation (APSF) in 1985 established "patient safety" as a specific concept and a discipline. At its 25th anniversary celebration, the APSF reflected on its innovative contributions and accomplishments and also on the many challenges yet ahead.

Since its inception as the very first patient safety organization, the APSF has been driven by the vision "that no patient shall be harmed by anesthesia." While rarely recognized as the true pioneer it is, the APSF (where the term "patient safety" originated) and the profession of anesthesia can legitimately be credited with igniting the entire "patient safety movement" that has blossomed into one of the major forces in modern health care everywhere. The APSF was, and remains, a driving force and catalyst.

While inspiring so many others outside anesthesiology, the APSF has worked tirelessly to accomplish its mission: "to improve continually the safety of patients during anesthesia care by encouraging and conducting: safety research and education, patient safety programs and campaigns, along with national and international exchange of information and ideas."

The *APSF Newsletter* has been a main vehicle for communication and education on issues related to anesthesia and patient safety since its first issue in 1986. This highly respected publication became, and remains, the largest-circulation anesthesia publication in the world and serves to transmit safety-related news, ideas and opinions. The APSF research grant program supports pioneering work that, for example, helped validate high-fidelity simulation as an education and research tool and, beyond that, has funded many projects that suggested solutions for safety problems. Safety advocacy and educational efforts have included publication of books, co-sponsorship of a video series and organization of the heavily-trafficked "patient safety booth" among the exhibits at the ASA Annual Meeting. More recently, APSF has sponsored targeted workshops and consensus conferences resulting in recommendations for definitive action. It hosts the popular APSF website **www.apsf.org** that includes a wealth of safety-related resource material and attractive interactive features such as the monthly poll on a current anesthesia question and also the "Virtual Anesthesia Machine," which the APSF helped to support over the years.



The Virtual Anesthesia Machine can be programmed to provide basic education about how an anesthesia machine works, and it can also be programmed to simulate failures at various parts of the machines, or mistakes in use.

John H. Eichhorn, M.D., is Professor of Anesthesiology, University of Kentucky College of Medicine, Medical Center, and Provost's Distinguished Service Professor, University of Kentucky, Lexington.

Success and Recognition

The success of the anesthesia patient safety movement was recognized significantly in 1996 when the American Medical Association and partners founded the National Patient Safety Foundation (NPSF), based directly on the APSF as their model. Further recognition came in the landmark 1999 report *To Err Is Human* from the Institute of Medicine on injuries from errors in medical care. APSF was the only organization cited as making a demonstrable positive impact on patient safety. Further, a June 21, 2005, front page article in the *Wall Street Journal* singled out the anesthesia profession, ASA and APSF for their roles in making anesthesia safer,

Savors Success, Targets Future

which resulted in dramatic decreases in professional liability insurance premiums for anesthesiologists. A "culture of safety" has developed in anesthesia practice, contributing significantly to a 10- to 20-fold reduction in mortality and catastrophic morbidity from anesthesia errors for healthy patients undergoing routine anesthetics.

Original Coincidences

Coincidence played a role in the creation of the APSF. As he related in his 1995 ASA Rovenstine Lecture,[1] Ellison C. ("Jeep") Pierce, Jr., M.D. (1928-2011), of Harvard, had been interested in "anesthesia accidents," particularly unrecognized esophageal intubations. Then, in April 1982, the ABC television program "20/20" aired a segment titled "The Deep Sleep: 6,000 Will Die or Suffer Brain Damage." It opened with: "If you are going to go into anesthesia, you are going on a long trip and you should not do it, if you can avoid it in any way. General anesthesia is safe most of the time, but there are dangers from human error, carelessness and a critical shortage of anesthesiologists ..." This watershed presentation provoked public concern about the safety of anesthesia. Taking advantage of his impending year as ASA President, Dr. Pierce convinced the ASA leaders to create the Committee on Patient Safety and Risk Management.

At the same time, groundbreaking research led by Jeffrey B. Cooper, Ph.D., a bioengineer in the Department of Anesthesia at the Massachusetts General Hospital, had focused on human errors causing preventable anesthesia accidents, adapting the techniques of "critical incident analysis" (used to study aviation accidents) to anesthesia events.[2] An international meeting was organized and hosted in Boston in 1984. At the closing session, Dr. Pierce reflected on the obvious great interest in the topic, the lively debate, the need for action and the potential to raise funds to support safety efforts. Dr. Pierce outlined his proposal to build on the idea and create an independent foundation dedicated solely to improving the safety of anesthesia care. Enthusiastic agreement was unanimous. Dr. Pierce asked, "What should we call it?" Dr. Cooper suggested that it simply be called what it would be, the "Anesthesia Patient Safety Foundation." And so it was.

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DR. EICHHORN WINS 2010 NQF PATIENT SAFETY AWARD

The 2010 John M. Eisenberg Patient Safety and Quality Award in the category of "Individual Achievement" was given to Dr. Eichhorn in February 2011. Dr. Eichhorn, University of Kentucky, Lexington, was recognized for his contributions that have led to dramatic and sustained reductions in catastrophic intra-operative anesthesia accidents.

Past recipients of the "Individual Achievement" award have been taken by such anesthesiology/patient safety luminaries as Jeffrey B. Cooper, Ph.D., a founding member of APSF and a current Executive Committee member, in 2003; Lucian Leape, M.D. in 2004; and Peter J. Pronovost, M.D., Ph.D., who received an award in a "Research" category in 2004.

The patient safety awards program, launched in 2002 by the National Quality Forum (NQF) and The Joint Commission, honors John M. Eisenberg, M.D., M.B.A., former administrator of the Agency for Healthcare Research and Quality (AHRQ). Dr. Eisenberg was one of the founding leaders of NQF and sat on its Board of Directors. In his roles both as AHRQ administrator and chair of the federal government's Quality Inter-Agency Coordination Task Force, he was a passionate advocate for patient safety and health care quality and personally led AHRQ's grant program to support patient safety research.

The two other award categories are "Innovation in Patient Safety and Quality at the National Level" and "Innovation in Patient Safety and Quality at the Local Level."

The awards were presented last February at the NQF's Annual Conference in Washington, D.C. An early 2011 issue of "The Joint Commission Journal on Quality and Patient Safety" also featured the achievements of each of the award recipients. Further information on Dr. Eichhorn's award can be found here: **http://www.qualityforum.org/Events/Awards/Eisenberg_Award/John_M__Eisenberg_Patient_Safety_and_Quality_Awards.aspx**.

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APSF Organized

Dr. Pierce, having just finished his term as ASA President, envisioned a relatively small, dedicated core group driving an independent foundation that was not directly controlled by any large organization. This would facilitate nimble, rapid, targeted action unfettered by a slow bureaucratic approval process and also open engagement on the politically sensitive topic of anesthesia accidents. Importantly, this would allow a very broad base of participants, including all possible interested groups of constituents: anesthesiologists, nurse anesthetists, nurses, bioengineers, epidemiologists, equipment and pharmaceutical manufacturers, government regulators, risk managers and insurance industry executives, and even surgeons.

Goals were: sponsor research, create programs to reduce injuries, promote communication, and establish an information newsletter to be delivered free of charge to all anesthesia providers. An initial contribution of $100,000 from ASA had been matched by both an offshoot of the Puritan Bennett Corporation and from Ohmeda, Inc. Accordingly, the viability of the foundation was clearly established, and the official incorporation was October 2, 1985.

Original APSF Executive Committee, 1986: Left to right: J.S. Gravenstein, M.D.; Jeffrey. B. Cooper, Ph.D., E.S.; (Rick) Siker, M.D. (Secretary); Mr. James E. Holzer; Ellison C. (Jeep) Pierce, Jr., M.D. (President); Mr. Burton A. Dole (Treasurer); and Mr. W. Dekle Rountree (Vice President).

Committees were addressed. The proposed quarterly newsletter was considered the top priority. Dr. Pierce tapped the prior newspaper editing experience of John H. Eichhorn, M.D. and persuaded him to create and edit the *APSF Newsletter*. The research grant program was the other priority. Arthur S. Keats, M.D. was named chair of the Scientific Advisory Committee, and he structured this effort like an NIH study section. The Committee on Education and Training was formed by J.S. Gravenstein, M.D. The Committee on Technology was also created. The initial Board of Directors did achieve the great diversity intended. It was agreed that the first APSF research grants would be up to $35,000. Also, Dr. Pierce started a lively discussion about intraoperative monitoring standards for three related reasons: the work of the Harvard Risk Management Committee, chaired by Dr. Eichhorn, and the recent adoption of formal anesthesia monitoring standards at Harvard; a meeting of an industry-sponsored "Anesthesia Safety Consortium" that supported standards; and the focus on monitoring by the newly formed ASA Committee on Standards of Care. An important consensus was achieved that the APSF would not present itself as a standards-setting organization, but rather would focus on education and advocacy to help improve safety.

Early Action

Organizational efforts came to fruition. Creation of the initial *APSF Newsletter* was a formidable task for Dr. Eichhorn, but was helped by a generous further donation to the APSF by Mr. Dole and the Puritan Bennett Corp. of free use of that company's graphics and printing facilities. (Likewise, later, another contribution from The Hewlett-Packard Company of what was then novel technology, a computer that did word processing, also helped the publishing process.) The first issue of the *Newsletter* was mailed to 45,000 recipients (ASA, AANA, risk managers, corporate and international supporters) in March 1986 and was well received. Beyond the lead story about the creation of APSF, there was an article questioning "minimal monitoring" and also a report of the initiation of the ASA Closed Claims Study. Later that year, there was announcement of the first FDA anesthesia machine check-out protocol. Strong support for the universal use of intraoperative pulse oximetry, and later capnography, was a major early APSF theme, and the concept of special safety risks outside traditional hospital operating rooms was introduced in the *Newsletter*.

The APSF partnered in the beginning with the ASA in co-producing a series of educational videotapes about a wide variety of anesthesia patient safety topics and, just recently, such efforts were revived when the APSF partnered with ECRI Institute to produce and distribute a DVD on

ANESTHESIA PATIENT SAFETY FOUNDATION
NEWSLETTER
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Safety Foundation Organized

Statement of Purpose

This is the first Newsletter of the Anesthesia Patient Safety Foundation, which was incorporated on September 30, 1985. The mission of the APSF is clear and simple—to encourage activities that will prevent patients from being harmed by the effects of anesthesia. Why such a foundation? What activities shall it promote to fulfill its mission? What resources will support those activities? What can you do to help?

It is generally agreed that anesthesia is safer than it has ever been, but that it still isn't safe enough. In the United States, annually some several thousand patients die or are seriously injured at least in part by their anesthetic experience. There is strong evidence that more than half of these adverse outcomes are preventable by applying known precepts of anesthesia management. Yet, the causes of preventable deaths and injuries are diverse and complicated. There is no one evil and no simple cure.

The first step toward improvement is creating awareness that a problem exists. Education, training, application of current and developing technologies and acquiring new knowledge about the causes and prevention of mishaps are components of a solution matrix.

Anesthesia mortality is everybody's problem. Most people will be exposed to the risk several times in their life. When a bad outcome occurs, it affects not only the patient, but has a lasting impact on the family, the anesthetist, and the anesthetist's colleagues as well. It is also a problem for many other constituencies—the manufacturer and designer of equipment that is involved or implicated in an accident and the hospital administrator in whose operating room an accident occurred. For the companies that provide liability insurance, there is the clear and present danger that the malpractice crisis, caused at least in part by preventable injuries, may severely damage or cripple the viability of their organizations. That this crisis puts the entire health care system in jeopardy makes this a problem for the federal government also.

Because there has been no place that these constituencies can join forces to promote change, the Anesthesia Patient Safety Foundation was formed. Its goals are:

*To foster investigations that will provide a better understanding of preventable anesthetic injuries;

*To encourage programs that will reduce the number of anesthetic injuries; and

*To promote national and international communication of information and ideas about the causes and prevention of anesthetic injuries.

During the first year, the Foundation's aims are to start a communication vehicle (this newsletter) and to establish a research fund, awarding several grants. Committees have been created to implement these activities.

Who is the APSF? Its 30-member Board of Directors includes representatives from anesthesiology, nurse anesthesia, device and pharmaceutical manufacturing, the insurance industry, hospitals, biomedical engineering, and the FDA (see page 5 of this newsletter for a complete list of the Board and committees). Membership in the APSF is open to any individual contributing at least $25 and any corporation contributing at least $500. Contributions will go toward funding the cost of producing and distributing this newsletter to the approximately 45,000 people who have a stake in preventing anesthesia injuries and toward the support of safety-related research activities.

You won't have to be a member of the APSF to benefit from its efforts but, yes, you will receive a certificate of membership if you join. The real reason to contribute $25 or more is because you want to make anesthesia safer. Because it can be. Because it should be. We think that some improvements, through increasing awareness and through implementing some new technologies, can be had in the short term—a few years. But, the ultimate goal of near-absolutely injury-free anesthesia will take longer because the impact of training, of education, and of innovative ideas derived from research take time to percolate through a culture. But, it can be done. We need your help.

Jeffrey B. Cooper, Ph.D.
Ellison C. Pierce, M.D.
For the Executive Committee

THE APSF EXECUTIVE COMMITTEE recently met in Atlanta, GA. Left to right: Dr. J.S. Gravenstein, Dr. J.B. Cooper, Dr. E.S. Siker (Secretary), Mr. J.F. Holzer, Dr. E.C. Pierce (President), Mr. B.A. Dole (Treasurer), and Mr. W.D. Rountree (Vice President).

Grant Applications Sought—Pg. 3

"Prevention and Management of Operating Room Fires."

The APSF tradition of education and advocacy through targeted workshops, seminars and task forces began in 1987 with the convening of a meeting, "Safety and Cost Containment in Anesthesia." In 1990, the APSF, partnering with the FDA, convened an unprecedented expert workshop on human error in anesthesia practice that helped stimulate later advances.

Patient safety research was (and remains) a core goal of APSF. In the first year, 27 research grant applications were received and four grants were awarded concerning risk, outcomes, safer machines and, to David M. Gaba, M.D., of Stanford, for "Evaluation of Anesthesiologist Problem Solving Using Realistic Simulations." Dr. Gaba went on to become one of the founders and leaders of using high-fidelity mannequin simulation in patient safety, human factors/safety research and medical training, as well as a fixture in the APSF leadership. The APSF helped organize in 1988 and 1989 what well may have been the first meetings on medical simulation. Overall, the APSF extensively supported (advocacy and grant funding) the development and implementation of simulation in anesthesia with its extensive implications regarding human performance, teamwork, crisis management and resident training. Again, while little recognized over the years, the APSF appropriately deserves credit for a truly pivotal role in the development and popularization of medical simulation, which is now a hugely successful, universal and an integral component of health care education. As was true with the original concept of patient safety and with formal standards of care, with simulation, the profession of anesthesiology was there first and should be proud of its leadership, a theme captured in a book summarizing the formative years of the APSF.[3] Others researching human factors, such as Matthew B. Weinger, M.D., have also had grant support, and they have made significant contributions to safety. Each year, the research grant recipients appear in the winter issue of the *APSF Newsletter* (the complete catalogue of which is readily available on the website **www.apsf.org**).

Spreading the Word

The APSF documented the decrease in morbidity and mortality from catastrophic anesthesia accidents – the beginning of the remarkable improvement in anesthesia patient safety that persists today. Also, each year, the *APSF Newsletter* carries an account of the patient safety-related presentations at the ASA Annual Meeting, showing the increase in the emphasis on safety. First were a handful in the late 1980s that were added to the end of "more traditional" abstract sessions, and this has grown consistently until now when there are multiple abstract and poster sessions over the entire meeting

Continued on page 20

David M. Gaba, M.D., a pioneer in anesthesia simulation and APSF Executive Committee member, was instrumental in popularizing anesthesia simulation for education, training and recertification evaluation and research.

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involving well over 100 safety-related presentations.

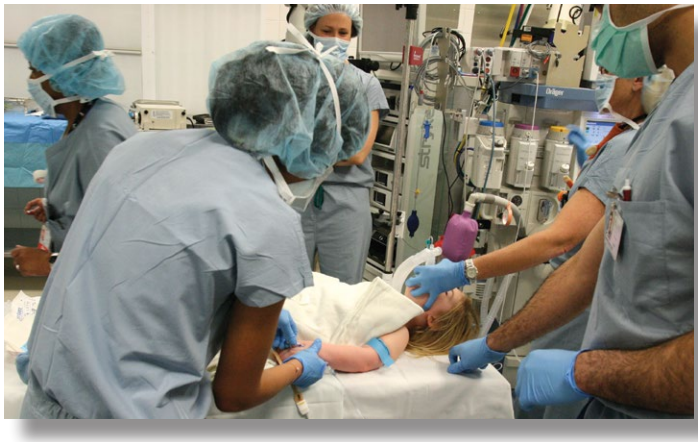
With its core goal of rapid communication of safety-related clinical information, over its history the APSF has consistently received, researched and publicized alerts on previously unknown issues. Examples number many more than can be listed here. In 1991, the *APSF Newsletter* published the first report of "Monday morning carbon monoxide poisoning" from the unusual interaction of desiccated CO_2 absorbent material with halogenated anesthetics. The question of danger to allergic patients from adding sulfite as a preservative to a new formulation of propofol was first raised by an alarming communication to the newsletter from a prominent academic department chair who, himself, was at risk. More recently, the danger of infectious complications, particularly hepatitis, from syringes used on more than one patient were broadcast by APSF. One prominent alert example concerned an unfortunate death when I.V. tubing was connected to a tracheostomy tube cuff inflation port instead of the adjacent central venous catheter. Raising awareness occurs each October at the ASA Annual Meeting where the APSF "patient safety booth" sits prominently in the exhibit hall and draws in passers-by with bold displays of relevant, current patient safety news as well as on-screen presentations of safety videos and the Virtual Anesthesia Machine program.

Advantageous Alliances

The APSF partnered with *Anesthesia & Analgesia* in 2007, creating a new patient safety section in that journal. The chair of the APSF Committee on Scientific Evaluation, Sorin J. Brull, M.D., was named section editor. Also, the APSF has sponsored panel discussions at the annual meeting of the International Anesthesia Research Society, ably organized and conducted by Richard C. Prielipp, M.D., chair of the APSF Committee on Education and Training.

Another major initiative arose from collaboration (and co-funding) with the manufacturers of anesthesia information management systems. This led APSF to organize the Data Dictionary Task Force to develop a common standardized terminology in clinical anesthesia practice that allows computerized records to generate consistent and compatible (and comparable) data. This is another instance of very few people being aware of the key, seminal role of APSF. Further, an alliance with the Society for Pediatric Anesthesia (SPA), along with special APSF funding, helped launch "Wake Up Safe," a network of pediatric hospitals and SPA, with the mission of creating an incident reporting system and event analysis paradigm. Recently, the APSF was an endorser of the World Health Organization global campaign "Safe Surgery Saves Lives," which features the "Surgical Safety Checklist," and the APSF conducted an extensive informational campaign to help facilitate its implementation.

APSF Booth at ANESTHESIOLOGY 2010, the ASA Annual Meeting in San Diego.



An alliance with the Society for Pediatric Anesthesia (SPA), along with special APSF funding, helped launch "Wake Up Safe."

New News

The *APSF Newsletter* has evolved also. Founding Editor Dr. Eichhorn turned over the editorial pen and scissors in 2002 to Robert C. Morell, M.D., who was joined in 2009 by Co-Editor Lorri Lee, M.D. Spirited, stimulating "pro-con" debates have played out on its pages, covering topics such as the safety implications of anticoagulation and regional anesthesia, routine succinylcholine in children, reading (and now surfing or texting) in the O.R. during cases (many times), and risks of PCA narcotic infusion pumps. Later, the "Dear SIRS" column ("Safety Information Response System") was added, initiated by Michael A. Olympio, M.D., then chair of the Committee on Technology. Technical issues, often involving problems with equipment, are discussed by panels of experts, including usually the manufacturer of any equipment in question. Also, while "Letters to the Editor" always was and is a popular feature, the volume and intensity of submissions have increased in recent years. Various landmark articles have had widespread impact on perceptions in anesthesia patient safety over the years. Examples include "How Safe Is Safe?" by Dr. Gravenstein in 1995, and "Patient Perspectives Personalize Patient Safety" by Dr. Eichhorn in 2005 (a report on Dr. Cooper's dramatic APSF workshop in which survivors or family members detailed the impact of catastrophic anesthesia accidents, including on the involved providers).

Other recent APSF workshops covered long-term patient outcome and the potential for deleterious effects of anesthetics, safety of postoperative opioid medication, medication safety in the O.R., technology training (or the lack of it) for anesthesia professionals, and danger of decreased cerebral perfusion pressure in "head-up" cases such as shoulder surgery in the "beach-chair" position (billed as "How low can you go?").

Beyond those mentioned, the list of other initiatives and projects undertaken by the APSF (mostly successful, some not so much) during its quarter century is long and varied (and outlined in the Summer 2010 *APSF Newsletter* **www.apsf.org**).

Research Renaissance

Prior to APSF, there was no research funding for patient safety. Sponsorship and promotion of safety research remain top APSF priorities, consuming the large majority of the APSF budget. In recent years, significant increases in research grant funding have been made possible by donor contributions of funds targeted for research support. Today, the maximum possible grant award is $150,000. For each of 2008 (nine grants) and 2009 (six grants), research support by the APSF peaked with total awards of approximately $1 million. The downturn in the economy did reduce contributions, and for 2010 there were five grants totaling $670,000, and for 2011, four grants for $660,000. The spectrum of topics remains vast and fascinating.

Reflection and Conclusion

APSF efforts to improve safety involve sound principles, technical theory, experience and pursuit of some real-life problems that have not been subjected to controlled experiments. This does not mean that evidence-based medicine should be ignored. Rather, this recognizes that logical safety changes that impact extremely rare events may not lend themselves to traditional *"randomized double blind studies with $p<.05$"* to determine validity or efficacy.

The profession of anesthesia has been recognized as "the only system in health care that begins to approach the vaunted 'six sigma' level of perfection."[4] Achievement of improved anesthesia patient safety was not attributed to any single practice, but rather to application of a broad array of changes in process, equipment and technology, resources, organizations, supervision, training, teamwork and even practitioner personalities. The APSF has led the way in this regard at many junctures over the last quarter century.

The work of improving anesthesia patient safety is by no means done. Systems, organizations and equipment still at times fail, and basic preventable human errors still do sometimes occur. Further, increasing "production pressure" in anesthesia practice from expanding clinical demands in the face of diminishing resources may threaten previously won gains. The anesthesia profession must consider and address this danger. The APSF serves as a model of the pioneering successful collaboration and commitment of the entire constellation of anesthesia-related professions and groups to the common goal of optimal patient safety. Very proud of its precedent-setting 25-year history of contributions, the APSF persists vigorously in pursuit of its mission "that no patient shall be harmed by anesthesia."

References are available at the back of the online version of this NEWSLETTER *at* ***www.asahq.org****, or by request by e-mailing* ***communications@asahq.org****.*

The Harvard Standards: 25th Anniversary of APSF

SILVER ANNIVERSARY: SILVER STANDARDS

Susan A. Vassallo, M.D.

In July 1985, the original "Harvard monitoring standards" were implemented in nine Harvard anesthesia departments. Although almost at the same time, this was quite separate from the founding of the Anesthesia Patient Safety Foundation (APSF). The APSF mission was "that no patient shall be harmed from the effects of anesthesia,"[1] and the APSF lobbied extensively for the adoption of monitoring standards after the ASA "Standards for Basic Intraoperative Monitoring" (modeled after the Harvard standards) were adopted in October 1986. Today, "safety monitoring," including the continuous use of the sophisticated electronic devices capnographs and pulse oximeters, is automatic and required behavior for anesthesiologists. However, there was a time not so long ago when those devices did not exist and monitoring was intermittent, subjective and sometimes fairly random. The protocol for monitoring the patient and the anesthesia delivery system described in the Harvard standards initiated the evolution of a permanent major change in the practice of our profession and, thus, deserves review on the occasion of the 25th anniversary.

Surgical theater, circa 1900: the anesthetist holds the mask, probably an ether inhaler.

Susan A. Vassallo, M.D., is Anesthetist, Massachusetts General Hospital; Assistant Professor of Anaesthesia, Harvard Medical School, Boston. She is Vice President, Wood Library-Museum of Anesthesiology.

Who Established and Communicated "The Harvard Standards?"

In 1986, the *Journal of the American Medical Association* (JAMA) published a Special Communications titled "Standards for Patient Monitoring During Anesthesia at Harvard Medical School."[2] These monitoring standards became known as "The Harvard Standards" and they represent the first instance whereby standards for minimal patient monitoring were required throughout a medical school's anesthesia department: The development, introduction and acceptance of these standards were significant milestones in our specialty. It was the first time ever any medical group had published formal "standards" with all the attendant significant medical-legal implications. The historical aspects of the process are part of a wonderful story of collaboration and progress directed toward the goal of safe anesthesia.

Why Was It Necessary to Implement Monitoring Standards Within the Department of Anaesthesia at Harvard Medical School?

Harvard's medical malpractice company, the Controlled Risk Insurance Company, Ltd. (CRICO), was created in 1976. It insured all Harvard hospitals, physicians and anesthesia providers. CRICO created the Risk Management Foundation (RMF) as a subdivision, and its purpose was to investigate malpractice losses. In 1984, RMF recognized that there were an alarming number of incidents, claims and deaths involving anesthesia care. Mr. James Holzer, the Director of Loss Prevention for CRICO, approached the chairs of the Harvard anesthesia departments and asked that they consider what could be done about the rising claims experience. The chairs appointed six faculty members to form the Harvard Anesthesia Risk Management Committee. They were charged with the task of analyzing the critical events that occurred between 1976 and 1984. The members were John H. Eichhorn, M.D., chairman, Jeffrey B. Cooper, Ph.D., David J. Cullen, M.D., Ward R. Maier, M.D., James H. Philip, M.D. and Robert G. Seeman, M.D. In 1985, the Harvard Medical School Department of Anaesthesia consisted of nine teaching hospitals and included four academic centers with anesthesia residency and fellowship programs, specialty hospitals and community hospitals. Together, the total annual number of anesthetics was approximately 100,000. The Anesthesia Risk Management Committee reviewed the events from the RMF files. The accidents and deaths were usually related to failure to ventilate or, sometimes, oxygenate a patient during anesthesia. The committee concluded that a protocol for genuinely continuous monitoring during an anesthetic was needed. To stress the importance of the protocol and show that it was required, not optional, behavior, formal "standards" for minimal monitoring were developed. They approached the Harvard chairs with this recommendation and included examples of disastrous events.

What Types of Critical Events Drew the Attention of CRICO?

Between 1976 and 1984, approximately 1 million anesthetics were administered in the nine hospitals. During this period, 58 cases were identified as serious events; 11 were classified as major intra-operative accidents and described by Dr. Eichhorn in a later paper published in *Anesthesiology* titled "Prevention of Intra-operative Anesthesia Accidents and Related Severe Injury through Safety Monitoring."[3] Failure to ventilate the patient was implicated in seven cases, and failure to provide oxygen was cited in one case. How did these horrific errors happen? For the most part, anesthesia was administered safely in a reliable operating room environment. However, unfamiliar situations posed unforeseen risks: a 27-year-old man was transferred to the angiography suite with massive gastrointestinal bleeding. A spare anesthesia machine retrieved from the closet was used during the embolization. The relief anesthesiologist did not appreciate that the anesthesia machine was of a British design – the oxygen flow meter was on the left side. This resident turned down the left flow meter and turned up the right flow meter – the patient received 100-percent nitrous oxide. There was no oxygen concentration monitor, and in a dimly lit room the error went unrecognized. Cardiac arrest, irreversible brain damage and death resulted.

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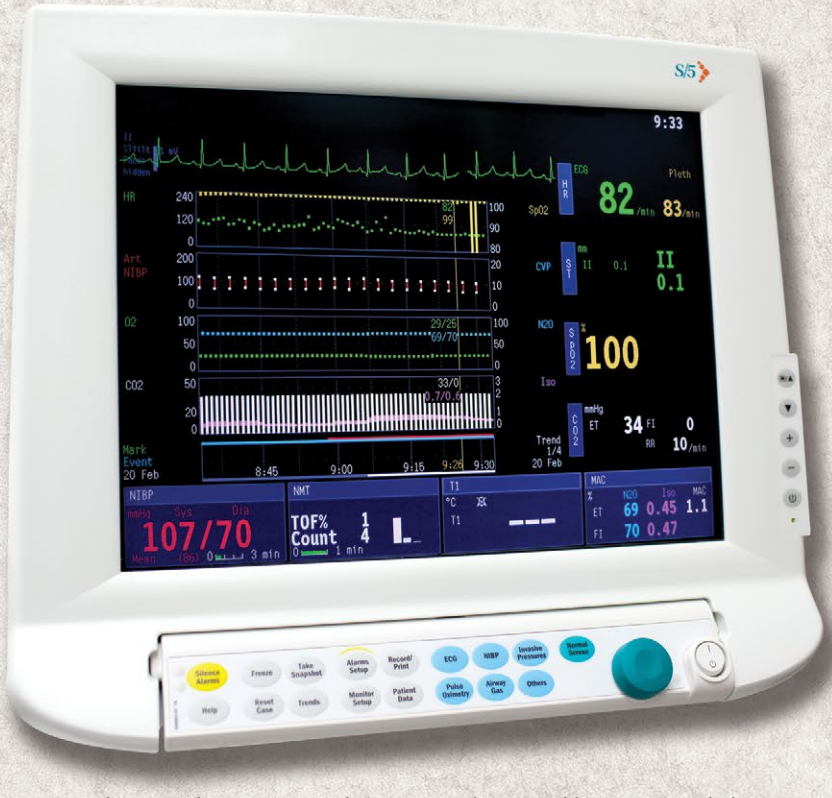

Current anesthesia machines continuously monitor vital signs and have integrated alarms.

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What Were the Objectives of the Harvard Minimal Monitoring Standards?

The four goals stated in the original *JAMA* article were:

1. To improve patient care.
2. To enhance detection of relatively low-frequency events.
3. To provide a means for objective evaluation: a benchmark; the standards were observed or were not.
4. To establish a precedent so that standards for other aspects of anesthesia care could be set.

What Factors Did the Risk Management Committee Consider When Establishing the Harvard Monitoring Standards?

Always aware that the anesthesia professional is the best monitor of all, the continuous presence of a qualified person was the first and foremost point. Beyond that, the committee considered each monitor's availability, cost and simplicity of use, intra-operative distracting effect, relative sensitivity, relative specificity and durability. The group wisely recognized that monitors can help but occasionally can overwhelm one's senses; they were also mindful of whether the standards were in the realm of current "reasonable care." The committee looked to the daily practices within the individual Harvard Medical School Anaesthesia Departments and asked, "What monitors are desirable?" and "What monitors should be mandatory?" Prior CRICO claims involving anesthesia were reviewed, and the committee analyzed the role of monitoring or lack thereof. Intermittent lapses of attention were unacceptable, leading to the requirement for truly continuous monitoring.

What Were the Harvard Minimal Monitoring Standards During Anesthesia?

The standards applied to any anesthetic "involving department of anaesthesia personnel and are specifically referable to preplanned anesthetics administered in designated anesthetizing locations (specific exclusion: administration of epidural analgesia for labor or pain management)." Abridged version follows:

- **Anesthesiologist's or Nurse Anesthetist's Presence in Operating Room throughout the conduct of general anesthesia, regional anesthesia or monitored intravenous anesthesia.**
- **Blood Pressure and Heart Rate measured at least every five minutes.**
- **Electrocardiogram should be continuously displayed.**
- **Continuous Monitoring during every anesthetic:**

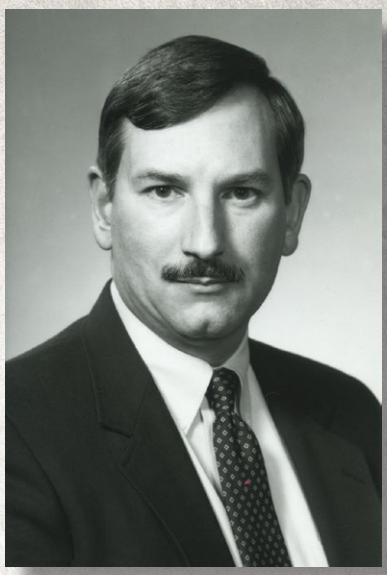
  For Ventilation: Auscultation of breath sounds or measurement of end-tidal carbon dioxide.

  For Circulation: Palpation of a pulse, auscultation of heart sounds or intra-arterial pressure.
- **Breathing System Disconnection Monitoring with an audible alarm.**
- **Oxygen Analyzer with a low concentration limit alarm.**
- **Ability to Measure Temperature.**

Standards adopted March 25,1985, Revised July 3, 1985

How Was the Introduction of Monitoring Standards Received?

The anesthesia chairs of the Harvard hospitals accepted the committee's recommended set of standards as mandatory for practice within the Harvard Medical School Department of Anaesthesia. The committee and the chairs anticipated resistance from the faculty, perhaps because there was lack of "scientific" evidence. Dr. Eichhorn, Chair of the Risk Management Committee, visited each department and shared examples of anesthesia catastrophes that could have been prevented with appropriate use of standard monitoring behaviors and equipment. The crux of his position was, "These are accidents in young, healthy ASA I and ASA II patients, not ASA III and ASA IV patients."[4] Pulse oximetry and capnography existed in the 1980s, but the committee astutely refrained from deeming these new monitors mandatory. This was not without controversy within the committee. Yet a consensus was reached that it was wise not to enable resistance from naysayers because this might delay implementation.[5] Alas, the standards were accepted and embraced, and many anesthesiologists were using these monitors already; there were relatively few holdouts.



John H. Eichhorn, M.D.

What Were the Immediate and Long-term Ramifications of Monitoring?

CRICO genuinely was concerned about the tragic anesthesia accidents occurring in the 1976-1984 time span. The loss of life or function despite modern medical interventions was hard to reconcile. It was counterintuitive: Anesthesia was supposed to aid surgery, not lead to injury and death. To give some meaning to this observation, in 1984 the Harvard Medical School Department of Anaesthesia accounted for 3 percent of CRICO's physicians, but the severity of their accidents represented 11 percent of insurance claims.[6] In October 1986, ASA adopted a similar set of monitoring standards. Over time, the standards were expanded to include required pulse oximetry and capnography. Their efficacy as highly sensitive extensions of the human senses was accepted and eventually embraced. The international community followed suit when Great Britain and Australia adopted monitoring standards as the earliest of many countries to do so.

Summary

Anesthesia was the vanguard of the patient safety movement. No other medical specialty had ever specifically, intensely and publically created a campaign to eliminate harmful medical errors. The very term "patient safety" was a concept cultivated by people committed to improving the care of patients during anesthesia. *The APSF was the first patient safety organization*. Long before checklists, time-outs and pre-operative huddles, before proclaiming, "To Err is Human," anesthesia recognized the potential for eliminating accidents. By analyzing rare, catastrophic events and identifying those accidents solely attributable to anesthesia, the Harvard Anesthesia Risk Management Committee recommended changing human behavior and establishing a minimal standard for monitoring. The tireless methodical efforts of this group serve as a superb example of collaboration and persistence. We thank CRICO and the Risk Management Committee, along with the Harvard Department of Anaesthesia chairs and faculty, for their dedication to patient safety throughout the years.

The author would like to acknowledge John H. Eichhorn, M.D. and Jeffrey B. Cooper, Ph.D., for sharing their thoughts and the staff of the Wood Library-Museum of Anesthesiology for their research help.

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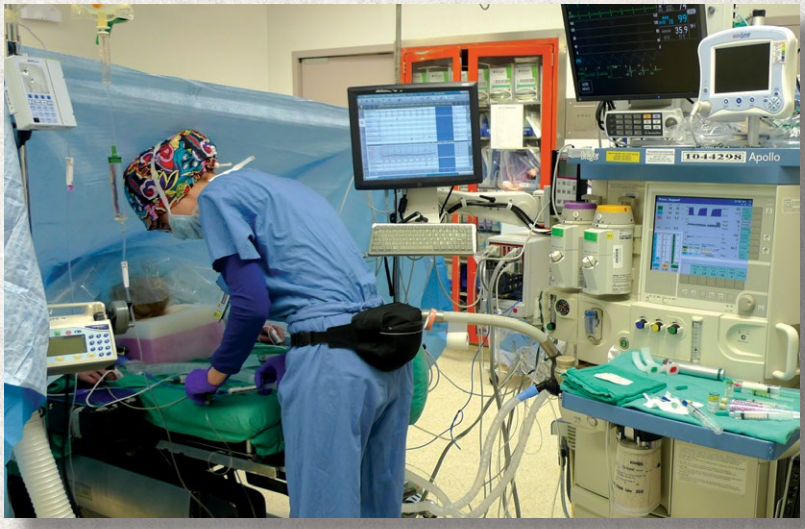

History of Anesthesia Records

Gerald L. Zeitlin, M.D., F.R.C.A.

Historians believe that the first consistent recording of physiological variables during anesthesia was the work of E.A. Codman and Harvey Cushing when they were "Junior House Pupils" at Massachusetts General Hospital in 1895. Codman later developed the modern outcomes assessment movement in medicine, and Cushing is considered one of the founders of modern neurosurgery.

Years later, Dr. Cushing described how they came to keep records when they gave ether.[1] *"Dr. Codman and I having entered the hospital together … we gave the anesthesia. I hesitate to recall what an awful business it was and how many fatalities there were.*

I was called down from the seats (of the surgical amphitheater) and told to put the patient to sleep. I proceeded as best I could under the orderly's directions. The operation was started … there was a sudden great gush of fluid from the patient's mouth, most of which he inhaled and he died."

Cushing then described how he slunk out of the hospital guilty and ashamed, only to be told later that these things were frequent and inevitable. He continues, *"Codman and I resolved that we would improve our technique of giving ether. We made a wager of a dinner as to who could give the best anesthesia. We both became very much more skillful … than we otherwise would have become but it was particularly due to the detailed attention which we had to put upon the patient by the careful recording of the pulse rate throughout the operation. On going abroad and getting interested in blood pressure, I discovered in Padua a simple recording instrument in Riva-Rocci's clinic.* On returning home I came to utilize this always during the course of my neurological operations."* Cushing concludes:

"A much more elaborate ether chart was thereupon prepared, on which not only pulse rate and respiration but the systolic blood pressure was recorded."

It remained until 1905 for Korotkov to describe the sounds he heard with a stethoscope as the cuff was deflated, for the diastolic to become measurable. Inspection of one of Cushing's records (Figure 1, next page) shows only the systolic as felt at the radial pulse. Riva-Rocci's method was by no means the first attempt to measure blood pressure; it was just the simplest and most reliable to that date.

In a fascinating letter, A.J. Wright describes how record-keeping of vital signs gradually spread into everyday anesthesia practice.[2] A Dr. Rogan used charting in Selma, Alabama as early as 1901. Wright has also published a meticulous chronology for the serious student of anesthesia records.[3] Two important histories of anesthesia were published just after World War II. They also reflect the gradual adoption of record-keeping. The American book, Thomas Keys' *History of Surgical Anesthesia*[4], gives us a full description of the later developments in anesthesia record-keeping, whereas the British author Barbara Duncum (*Development of Inhalation Anaesthesia*),[5] who ends her story in 1900, makes no reference to it.

Looking at early textbooks about anesthesia might be another way to elicit whether record-keeping became universal in a way analogous to the rapid worldwide spread of the use of ether within a year of Morton's demonstration.

Four books published in the United Kingdom make *no* mention of routine blood pressure recording. Please note their dates of publication. They are *Practical Anaesthetics* by J. Edmund Boyle (of Boyle Machine fame) in 1907, *Handbook of Anaesthetics* (1912) by J. Stuart Ross, a proponent of the dry-cleaning agent ethyl chloride as a general anesthetic in 1924, and *Anaesthesia and Anaesthetics* by Rood and Webber in 1929. This last book was also sold in the U.S.

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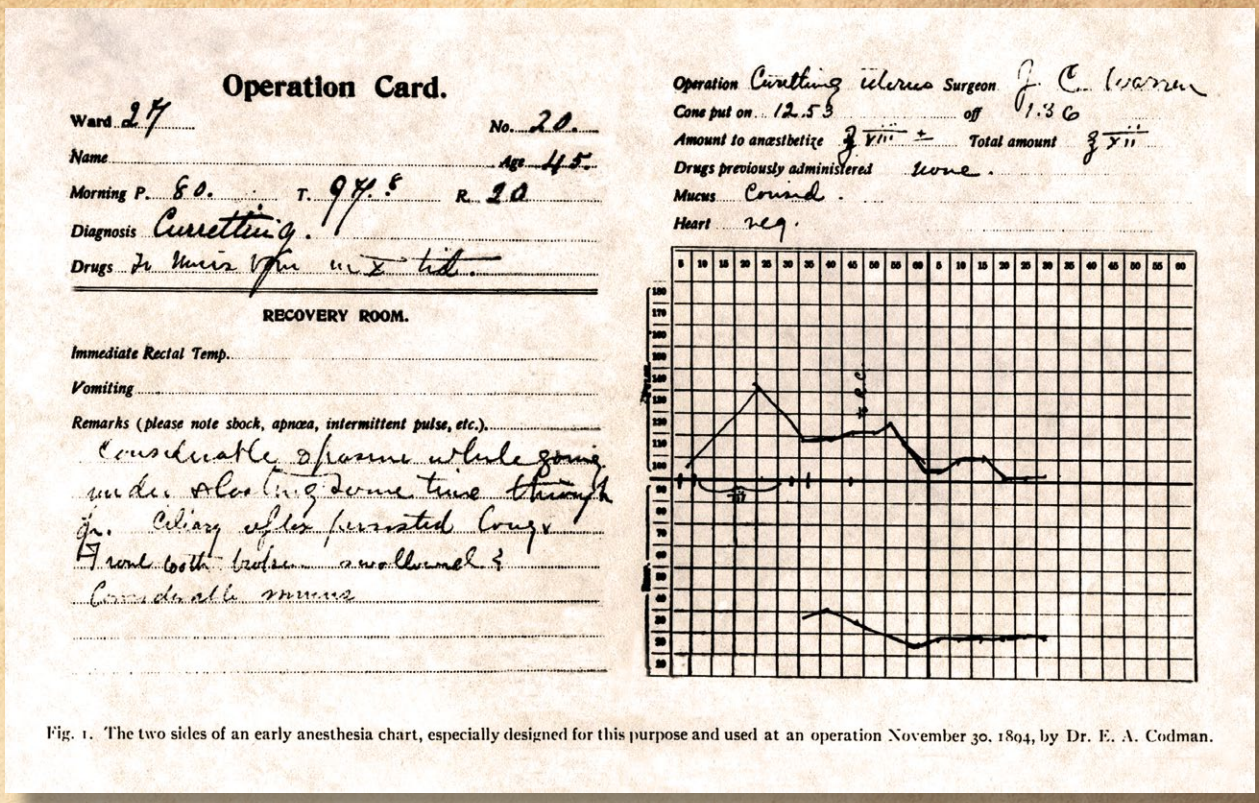
**Cushing is in error here. Riva-Rocci practiced medicine in Pavia.*



Operation Card.

Ward 27 No. 20.
Name Age 45.
Morning P. 80. T. 97.5 R. 20
Diagnosis Curetting.
Drugs Tr Nucis Vom m x tid.

RECOVERY ROOM.

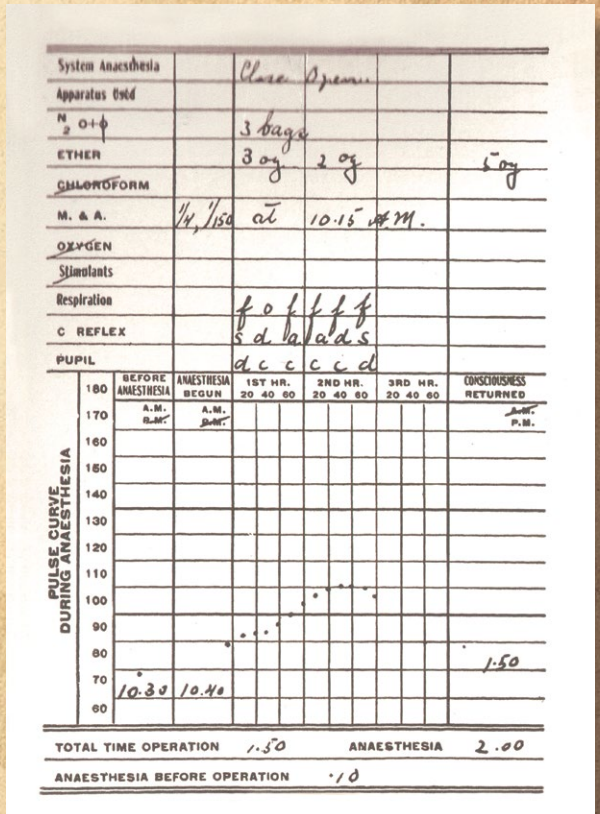
Immediate Rectal Temp.
Vomiting
Remarks (please note shock, apnœa, intermittent pulse, etc.).
Considerable dyspnœa while going under Ether & some time through it. Ciliary reflex persisted Cough & vomit both broken up allowed 3 Considerable mucus

Operation Curetting Uterus Surgeon J. C. Cushing
Cone put on 12.53 off 1.36
Amount to anæsthetize ℥ viii ± Total amount ℥ xii
Drugs previously administered None.
Mucus Consid.
Heart neg.

Fig. 1. The two sides of an early anesthesia chart, especially designed for this purpose and used at an operation November 30, 1894, by Dr. E. A. Codman.

Figure 1 *(above): Two sides of an anesthesia chart kept by E.A. Codman, M.D., November 30, 1894. From: Beecher HK. The first anesthesia records (Codman, Cushing). 1940;* Surg Gyn & Obs. *71:689.*

Figure 2 *(at right): Anesthesia record from Flagg, PJ* The Art of Anesthesia, *5th Edition. Philadelphia, J.B. Lippincott and Co; 1932.*



In 1920, J.F.W. Silk in London in his *Modern Anaesthetics* wrote the following:

"The importance of observing the variations in blood pressure of a patient while under an anaesthetic has been suggested. In fact it is insisted upon in some quarters that such observation should be made as a matter of routine … and that the necessary apparatus should form part of the equipment of the anaesthetist."

What about the U.S? In the first edition of Gwathmey's tome *Anesthesia* (1914), he displays many blood pressure diagrams from laboratory studies but does not mention recording during clinical anesthesia; nor does he in his discussion of the medicolegal difficulties of anesthesiologists. On the other hand, nearly two decades later, Dr. Paluel Flagg of New York in the 5th edition (1932) of his *The Art of Anaesthesia* devotes a short but complete chapter to charting (Figure 2).

There were some exceptions. In 1903, Crile, the Cleveland surgeon who conceived the idea of blocking noxious surgical stimuli in addition to the use of general anesthesia (anoci-association), quickly adopted Cushing's records. In 1907, Elmer McKesson in Toledo, Ohio began to

Continued on page 28

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keep accurate blood pressure records during anesthesia. In the next 25 years, leaders in the specialty such as Brown in Adelaide, Australia, Lundy in 1923 in the Pacific Northwest, and later, Ralph Waters and E.A. Rovenstine, followed suit.

McKesson was an inventive genius who developed the first piece of equipment that automatically recorded intraoperative blood pressures. He called this device a Nargraf (Figure 3).

By the late 1930s, custom-made charts were developed on both sides of the Atlantic. The example in Figure 4 (page 29) by the British anesthesiologist Nosworthy is striking both for its completeness and for the use of the explosive agent cyclopropane. In the U.S., conventional anesthesia records were transferred to adaptations of Hollerith punched cards at the Doctors Mayo's Clinic. These had been brought into industrial use by IBM in the 1920s. Anesthetists used them for later analysis of outcomes in groups of patients. The Committee of Records and Statistics of the American Association of Anesthetists lent its authority to this.

Nowadays, observing and recording vital signs each five minutes have become routine in addition to the notation of drugs and their dosages and all other intraoperative events. Developments in electronics have allowed all this to become increasingly automated, supposedly allowing the anesthesiologist to concentrate on the patient's condition by not having to write something every five minutes. One of the assumptions here is that the machine is objective and neutral. It is interesting that although automatic recording devices first appeared about 20 years ago, recent estimates reveal only one in three anesthesiologists uses them.

The question remains: why do we continue this ritual? One answer is that it is fundamental to teaching our residents that close and precise observation of the patient is vital. That is inarguable.

Does an experienced Board-certified anesthesiologist need to continue doing something that *was once* central to the scientific development of our specialty? I wonder.

A patient suffers a myocardial infarction and for several hours is much more unstable than are most patients we anesthetize these days. His cardiologist pays close attention, and with precise therapeutic maneuvers helped his patient to survive. But he does *not* keep a five-minute handwritten record of my many variables during those early frightening hours. Later that evening, she goes to the dictation machine and gives a literate and comprehensible description of the evening's drama.

I believe we anesthesiologists should abandon our "squiggle" or "railroad track" charts and learn to dictate what happened during each anesthetic we give. Those pieces of literature in the patient's hospital chart would illustrate the *reasons* for each of the drugs given and the moves made in response to both the patient's vital signs and our surgical colleagues' maneuvers. The ultimate question is: why do we act differently from all other physicians practicing acute medicine? Are we not as well qualified to express our observations as the average cardiologist? The current anesthesia record, whether handwritten or automatic, is mindless.

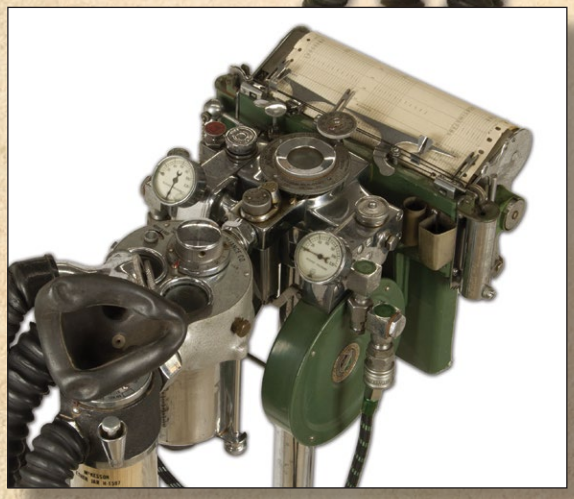


Figure 3: *McKesson's Nargraf Recording Machine. Image courtesy Wood Library-Museum of Anesthesiology, Park Ridge, Illinois.*

The other reason given for keeping five-minute records is that they could act as a defensive shield in the event we become defendants in a malpractice suit. Is this true?

Karen L. Posner, Ph.D., who is Laura Cheney Professor in Anesthesia Patient Safety, kindly researched this question from the database of the ASA Closed Claims Project.[7] In part she wrote:

"While our data do not allow us to easily assess the role of inadequate, changed and multiple records in these claims, we did observe a significant correlation between inadequate records and appropriateness of care. In general, 59 percent of claims with inadequate records were assessed as evidencing substandard *anesthesia care, while 63 percent of claims with adequate records were assessed as evidencing* appropriate *anesthesia care."*

Later in her report, Professor Posner makes the following comment:

"We were unable to assess the specific role of the records in these payment outcomes beyond the observed correlations." And further, *"However, many of these claims revealed multiple problems with the care provided and the records were one of many issues in the claim resolution process."*

Despite their fascinating history, has the time not come for anesthesiologists to rethink the place of the current recording system and substitute more intelligent reporting of perioperative care?

This article was written to honor the late Ellison C. Pierce, M.D.

References are available at the back of the online version of this NEWSLETTER *at* ***www.asahq.org****, or by request by e-mailing* ***communications@asahq.org****.*

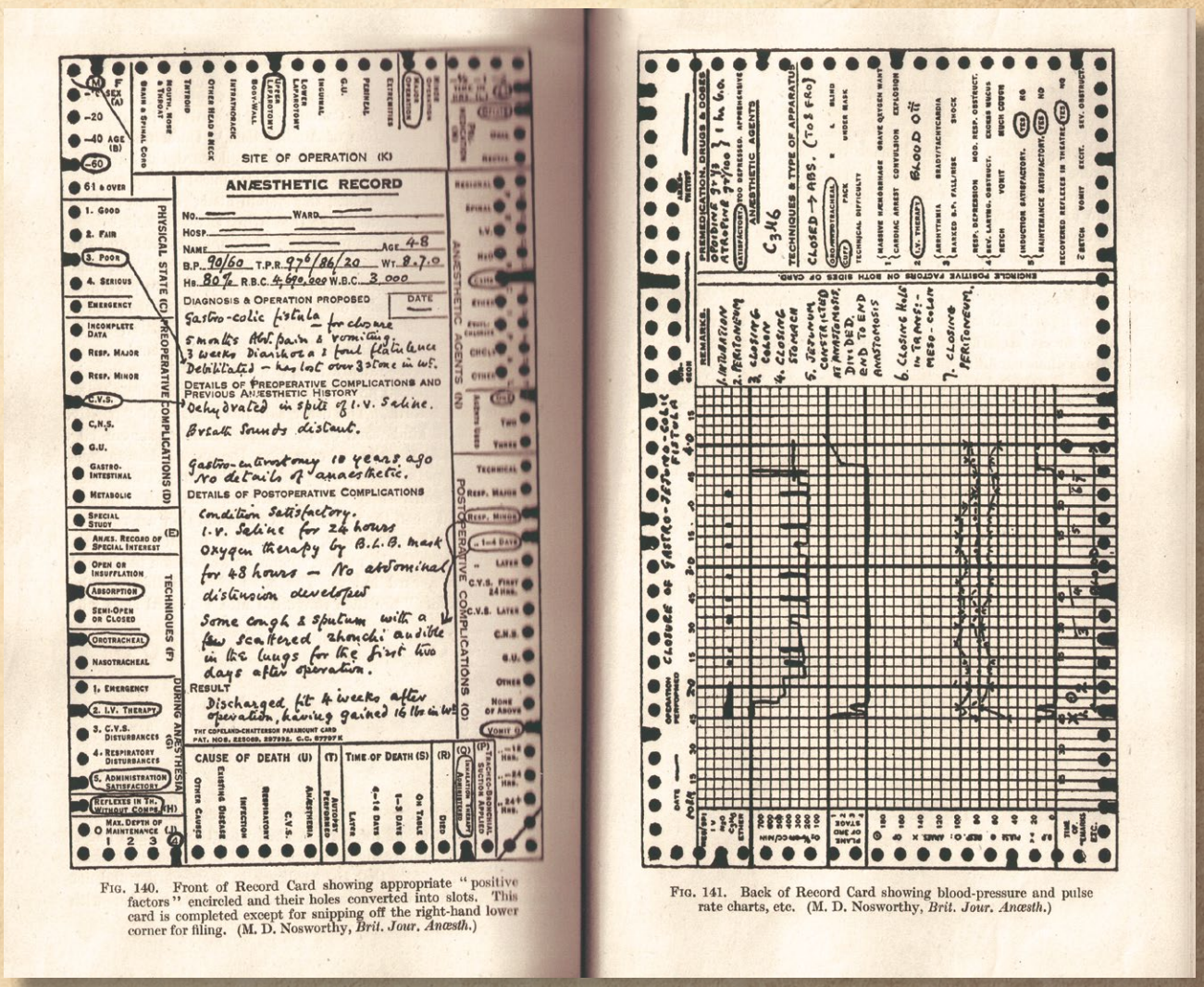


FIG. 140. Front of Record Card showing appropriate "positive factors" encircled and their holes converted into slots. This card is completed except for snipping off the right-hand lower corner for filing. (M. D. Nosworthy, *Brit. Jour. Anæsth.*)

FIG. 141. Back of Record Card showing blood-pressure and pulse rate charts, etc. (M. D. Nosworthy, *Brit. Jour. Anæsth.*)

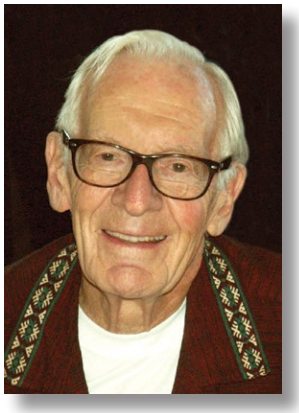
Figure 4: *Anesthesia record from Nosworthy M.D. A method of keeping anaesthetic records and assessing results.* Brit J Anaesth. *1943; 18(4):160-179.*

Monitoring **Oxygenation**

John W. Severinghaus, M.D., Dr Med HC, F.R.C.A.

On December 11, 1844, in Hartford, Connecticut, Horace Wells, as a test of possible painless surgery, decided to breathe 100-percent N_2O prepared by Gardner Quincy Colton for the extraction by his associate John Mankey Riggs of his painful wisdom tooth. Wells showed no sign of any reaction to the extraction under the world's first deliberate inhalational surgical anesthetic. "There was no doubt in their keen Yankee minds that they were in the presence of something of transcending significance."[1] For the following century, dentists repeated this procedure millions of times with a noteworthy safety record. Cyanosis was expected and not feared. Although recovery was rapid, if the patients turned very blue, recovery took longer. This led to a method about 1900 called "secondary saturation," doing brief non-dental surgery during the delayed awakening after severe cyanosis. The dangers of chloroform were cardiac arrest (or fibrillation, unrecognized), not anoxia, while only very deep ether depressed ventilation. Anesthetists "monitored" only skin color and chest movement until in the 1930s thiopental, cyclopropane and curare impaired ventilation and deaths under anesthesia followed.

In 1947, Comroe and Botelho subjected 20 white volunteers to low arterial oxygen saturation (SaO_2) measured with a Millikan ear oximeter.[2] Definite cyanosis was not detected by over half of 127 physician staff and medical students until SaO_2 was below 80 percent. One fourth couldn't identify cyanosis until SaO_2 <75 percent. The authors considered this too low to be used safely in anesthesia despite the century of mostly safe brief use of 100-percent N_2O in dentistry. In 1954, Beecher and Todd[3] reported on deaths under anesthesia, implicating curare and hypoxia. The impact of this paper was to enormously boost a demand for better monitoring of oxygenation.



John W. Severinghaus, M.D., Dr Med HC, F.R.C.A., is Professor Emeritus of Anesthesia and Cardiovascular Research Institute, University of California, San Francisco.

The science of measuring and monitoring SaO_2 is now called oximetry. It is based on spectroscopy, invented by G.R. Kirchhoff and Robert Bunsen in Heidelberg in 1860 to analyze solar light.[4] Hemoglobin was identified as oxygen's carrier, and its spectrum was studied in the 1860s by Gabriel Stokes[5] and Felix Hoppe-Seyler.[6] Karl von Vierordt measured the change of red light transmission through his tourniqueted finger in 1874.[7] But no further studies were done for half a century until three German physiologists, Ludwig Nicolai[8] and his student Karl Kramer[9] in Göttingen and Karl Matthes in Leipzig,[10] developed instruments to optically measure SaO_2 in vivo. In 1936, Matthes developed the first ear oximeter, balancing light transmission of red and green (actually infrared) wavelengths.

In 1939, the chief of surgery in Detroit's Henry Ford Hospital, Roy D. McClure,[11] was the first to warn the American Surgical Association that, because anoxia was a source of possible complications in surgical anesthesia, SaO_2 should be continuously monitored. He and his associate Frank W. Hartman[12] introduced oximetry with their "oxyhemoglobinograph" (Figure 1) and tried to promote its use, but it was never commercially developed.

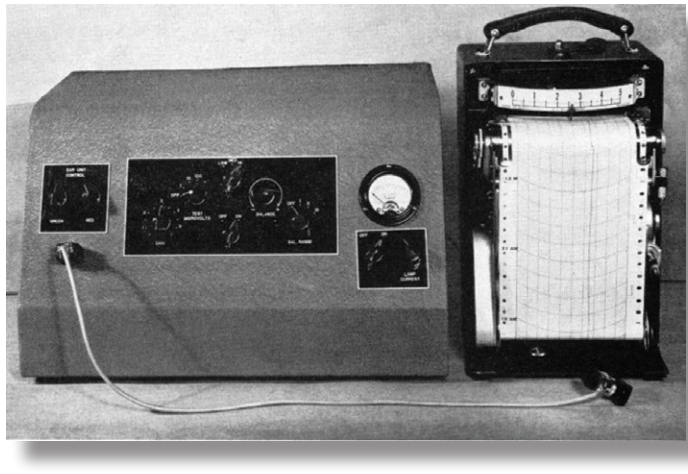


Figure 1: Oxyhemoglobinograph

Development of oximetry in the United States and Britain was stimulated by the needs of World War II fighter aircraft, most of which lacked pressurized cabins but carried O_2 and masks. In 1941, requested by physiologist Lord Adrian for the British military, Glen Millikan developed a lightweight ear oxygen meter for which he coined the term **oximeter** (Figure 2).[13] But because measurement required a large, heavy,

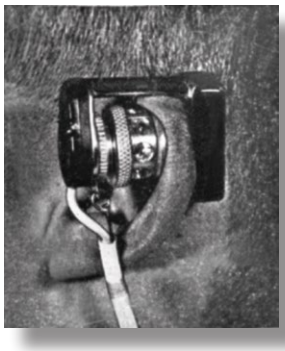


Figure 2: Millikan Oximeter on Ear

motion-sensitive galvanometer, his oximeter could not be used in aviation. The oximeter was first used in 1949 during anesthesia at the Mayo Clinic to demonstrate maintenance of 100-percent SaO_2 during 2 Atm hyperbaric 50 percent N_2O.[14] During the resulting surgical anesthesia, EEG showed high-voltage delta waves at 4 to 6 cps.

Although several oximeters were made available after 1950, almost none were routinely used for anesthesia oxygenation monitoring. Earl Wood's addition of a tissue compression membrane to the Millikan earpiece[15] generated 4 data (red and IR signals with bloodless and perfused ear) from which SaO_2 could be computed without in vivo calibration. Most others found it needed calibration with each subject. It used the same bulky galvanometer and it sometimes burned the ear. Brinkman and Zijlstra's forehead light reflection oximeter, named "Cyclops," required apparatus that was too hard to use in anesthesia, and it was never commercially developed.[16]

An accurate multi-wavelength ear oximeter developed by surgeon Robert Shaw and eventually marketed by Hewlett Packard (Figure 3) was widely used in physiology experiments, but its heavy earpiece, 25-pound cabinet and >$10,000 cost kept it out of routine anesthetic use.

Figure 3: Hewlett Packard multi-wavelength ear oximeter

Blood Gas Analysis

In 1954, Richard Stow at Ohio State University reported invention of a membrane-covered PCO_2 electrode, but he could not make it stable.[17] Severinghaus had stabilized it by the crucial addition of bicarbonate to the electrolyte. In 1956, Leland Clark at Antioch University in Ohio publically revealed his 1954 invention of a membrane-covered polarographic oxygen electrode.[18] Because of its large cathode and high O_2 consumption, it required stirring if used for blood or other liquid. Severinghaus combined the PO_2 and PCO_2 electrodes in a 37°C water bath in 1957-58[19] and added a pH electrode in 1959 (Figure 4). Beginning in 1960, blood gas analyzers became commercially available. Within a decade they were widely used in anesthesia and recovery rooms, neonatal and adult intensive care, and emergency medicine. After 1967, internal computers performed automatic calibration and calculation of bicarbonate, base excess and later standard base excess and SaO_2. But blood gas analysis was not monitoring in the sense of a continuous source of useful information.

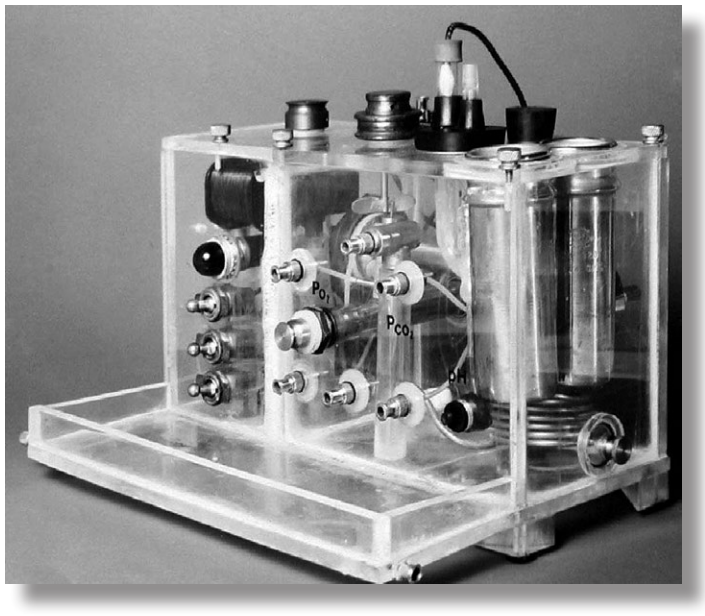


Figure 4: First three-function blood gas analyzer

Introduction of Transcutaneous Oxygen Tension Monitoring

Skin surface polarographic PO_2 was the first widely used continuous monitor of oxygenation. In the early 1960s, when airway positive pressure ventilation with oxygen was introduced to inflate the collapsed lungs in very premature infants, many of the saved infants became blind. This blindness was shown to be due to hyperoxia causing RLF (retrolental fibroplasia) or ROP (retinopathy of prematurity). This created an urgent need for continuous non-invasive monitoring of premature infant blood oxygen. Physiologists studying skin respiration had shown that human skin breathes, taking up oxygen and giving off CO_2 to the air. If skin is covered (as by a flat, unheated PO_2 electrode), the surface PO_2 falls to zero in a few minutes. In 1951, Baumberger and Goodfriend showed that if skin blood flow is greatly increased by the highest tolerable heat (45°C), the surface PO_2 rises to approximately PaO_2 (arterial blood).[20] Within a year after Clark announced his invention of the membrane-covered platinum polarographic electrode (1956), studies with polarographic electrodes confirmed the Baumberger report.[21] Nothing came of this due to the need for skin vasodilation until 1972,

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when two groups reported use of electrically heated O_2 skin surface electrodes[22,23] (Figure 5). The resulting "transcutaneous" $PtcO_2$ was remarkably similar to arterial blood PO_2 in infants. In the late 1970s, transcutaneous oxygen monitoring was made commercially available, becoming universally used in premature nurseries to facilitate adjustment of inspired O_2 concentration between the dangers of too high (>100 torr) and too low (<50 torr) (Figure 6). Since 1980, transcutaneous combined PO_2 PCO_2 electrodes (Figure 7) have been used in neonatology, anesthesia, recovery and critical care. $PtcO_2$ is less accurate in older children and adults, especially at high PO_2.[24]

Figure 5: $PtcO_2$ electrodes, 1971

However, neither transcutaneous blood gas monitoring nor oximetry became "essential" in routine anesthesia, primarily because, in the mid 1980s, an easier to use and more accurate invention captured the market. Pulse oximetry was precalibrated, inexpensive, easy to use, and saturation rather than PO_2 was recognized as the more important index to be monitored.

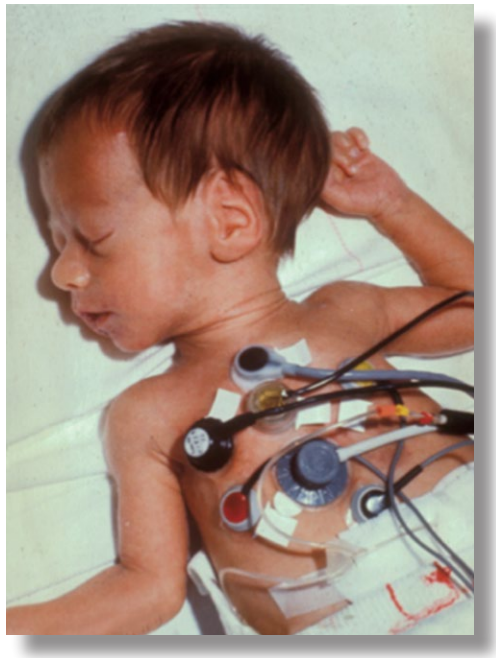

Figure 6: Baby with tc Electrodes

Figure 7: Combined PO_2 $PC0_2$ electrodes

Invention of Pulse Oximetry

The concept of pulse oximetry may be considered a development of the techniques of Squire,[25] Goldie[26] and Wood.[27] Each had realized that the ratio of ratios of absorption of red and IR light from perfused and bloodless tissues provided the 4 values needed to compute arterial oxygen saturation.

The Japanese physiological bioengineer Takuo Aoyagi (Figure 8) was the first to realize that the variations of light transmitted through the ear or finger caused by arterial pulsation could be used to compute SaO_2.[28] Aoyagi graduated in 1958 from the Faculty of Engineering at Niigata University with a degree in electrical engineering. Stimulated by a summer course in 1971 at Baylor University on dye dilution cardiac output methods, Aoyagi joined the Research Division of Nihon Kohden Co. He planned to use an ear oximeter like that of Millikan as a densitometer to measure cardiac output by dye dilution without continuous arterial blood sampling. He found that the light signal transmitted through the ear contained pulsatile variations that prevented the accurate extrapolation of dye concentration during washout. He eliminated the pulses by subtracting the earpiece IR signal from the red signal, but noted that the cancellation failed during breath-holding hypoxemia. He then wondered if he could use the pulse variations to measure oxygen saturation (a classic example of the adage that "one man's noise is another man's signal").

In 1971, after studying Earl Wood's work, he suddenly realized that the pulse-induced variations in red and infrared light should provide the 4 values needed to compute the ratio of ratios equivalent to Earl Wood's use of the pneumatic cuff. Takuo Aoyagi's insight gave birth to pulse oximetry.

He developed the first prototype of a pulse oximeter using a copy of the Millikan earpiece. On April 26, 1974, Aoyagi presented an abstract titled "Improvement of the Earpiece Oximeter," to the Japanese Society of Medical Electronics and Biological Engineering. Clinical tests of his first instrument, the OLV-5100, were reported by Nakajima, Hirai and Takase, originally as "New pulse type earpiece oximeter" in Japanese: *Kokyo To Junkan 1975;23:709-713*.[29]

Figure 8: Takuo Aoyagi

However, the Nihon Kohden management failed to appreciate the potential of Aoyagi's discovery and transferred him to a desk job in 1975. Ten years later, Aoyagi was permitted to resume research. He then was able to resume his original 1971 plan to invent and develop a dye dilution method to measure cardiac output non-invasively. He called it a "pulse spectrophotometer." From a single dye injection, it also determined plasma volume and liver blood flow.

After seeing Aoyagi's 1974 abstract (probably before the presentation), the Minolta Co. applied for a U.S. patent and developed a competing pulse oximeter, the Oximet MET-1471. In the late 1970s, the renamed Minolta 101 pulse oximeter was tested at low saturation by Frank Sarnquist, Christine Todd and Charles Whitcher at Stanford University Medical School's Anesthesia Department.[30] The correlation was excellent and linear but at SaO_2=50 percent the pulse oximeter read 70 percent. Minolta then used Sarnquist's data to correct the slope error in their software.[28] The excellent response led Stanford anesthesiologist William New to recognize the enormous potential of pulse oximetry. New and engineer Jack Lloyd developed a finger pulse oximeter and started the Nellcor company in 1983.[31]

Also noting the Sarnquist study, Ohmeda, of the Life Support Equipment Division of BOC Health Care, rapidly developed and marketed an ear pulse oximeter, Biox III. In 1985, in collaboration with Professor J.P. Payne of the Research Department of Anaesthetics at the Royal College of Surgeons of England, Ohmeda sponsored an international conference of the findings of 24 groups using pulse oximetry at Chartridge, Buckinghamshire.[32,33]

By the early 1990s, pulse oximetry had become almost universally used in anesthesia. A self-contained, battery-operated finger tip model (Nonin Onyx) was employed at the summit of Mt. Everest by climber David Brashears during his IMAX filming in 1997 (Figure 9). It is arguably the most important technological advance ever made in monitoring the well-being and safety of patients during anesthesia, recovery and critical care.

Figure 9: Nonin Onyx Pulse Oximeter

The recent more than 10-fold reduction in deaths attributed to anesthesia in the U.S. coincided with the widespread use of pulse oximetry.[34] In an effort to link these events, a Danish anesthesia group initiated multi-institutional double-blind studies of anesthesia in which the anesthesiologist was unaware of the pulse oximeter readings in half the patients.[35] Four groups have repeated this for a total of 23,000 study patients. None of these studies succeeded in finding a statistically significant beneficial role of pulse oximetry.

No conclusive explanation has been offered for this anomaly. Perhaps even a brief experience with a pulse oximeter and the resulting better understanding of the importance of cardio-respiratory physiology provoked anesthesiologists to pay more careful attention to their patients.

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Perioperative Thermoregulation

Daniel I. Sessler, M.D.

This work was supported by internal funds only. The Department of Outcomes Research is supported by numerous companies with interests in temperature monitoring and thermal management. For example, the unpublished result presented in Figure 4 is based on a study supported by Arizant Healthcare (Eden Prairie, Minnesota). However, Dr. Sessler has no personal financial interest related to this review. He is an ASA representative to the Physician Consortium for Performance Improvement.

General anesthesia profoundly impairs normal tight control of core-body temperature; to a lesser extent, neuraxial anesthesia does as well. Consequently, unwarmed surgical patients nearly all become 1-2°C hypothermic. Randomized trials show that even mild hypothermia increases the risk of morbid myocardial outcomes, bleeding and transfusion requirement, and surgical wound infection. Hypothermia also slows drug metabolism, prolongs recovery, and provokes shivering and thermal discomfort. Various warming methods are available and provide differing combinations of safety, efficacy, cost and ease of use; forced-air remains by far the most common approach.

Normal Thermoregulation

Body temperature is normally tightly regulated to near 37°C. Temperature is sensed at the skin surface and throughout the body; very roughly, the skin, deep tissues, spinal cord, hypothalamus, and remainder of the brain each contribute about 20 percent to central control. Control of body temperature is hierarchical, with the hypothalamus being the dominant center in mammals (it is the spinal cord in birds). Thermal receptors are phenomenally sensitive; for example, humans can detect localized increases in skin temperature of only 3 thousandths of a °C. Precision of thermoregulatory control is thus not limited by receptor sensitivity.

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Regulation can be divided into behavioral and autonomic responses. Behavioral defenses are triggered by individuals' perceptions of their thermal environment and include dressing warmly, adjusting a room thermostat and building shelter. Generally, behavior is by far the more powerful thermoregulatory defense and is the reasons humans can live in such diverse environments. The difficulty, of course, is that anesthetized patients have little or no access to behavioral responses – leaving them dependent on less powerful autonomic defenses.

The major autonomic defenses against heat in humans are active pre-capillary vasodilation and sweating. Sweating can dissipate a remarkable amount of heat in a dry, convective environment. One investigator, for example, spent a full hour in an industrial oven at 250°F without adverse effects. The major autonomic defenses against cold are arterio-venous shunt vasoconstriction and shivering. Instead of shivering, all infant mammals (except pigs) use non-shivering thermogenesis to directly convert chemical energy into heat in brown fat and muscle. For example, human infants – even somewhat premature ones – will double metabolic heat production in a cold environment, which is roughly comparable to heat production during sustained shivering. Small mammals generally maintain their preference for non-shivering thermogenesis as adults.

Various species use a remarkable range of thermoregulatory strategies and defenses. For example, penguins cannot sweat and have limited ability to dissipate heat (which they presumably rarely require); but if necessary, they will urinate on their feet and take advantage of the subsequent evaporative cooling. Flamingos stand on one foot to reduce heat loss into the water they stand in and use counter-current mechanisms to further reduce heat loss. Butterflies vary the thickness of wing scales to alter absorption of radiant heat. Camels and just a few other desert mammals take a different approach; instead of sweating, which would waste precious water, they let their core temperature vary up to 10°C during the circadian cycle. Interestingly, most animals regulate core temperature to approximately 37°C. But there are distinct exceptions, such as worms that live near deep-sea thermal vents; they tolerate temperatures as high as 80°C and can have gradients exceeding 60°C across their three-inch-long bodies.

Poikilothermia is defined by lack of autonomic thermoregulatory defenses. But poikilothermic species very much use behavioral thermoregulation. Given warm and cold

environmental options, for example, nearly all poikilothermic species will choose a location that gives them a core temperature near 37°C. Goldfish – which have no intrinsic way to modify their temperature – can be trained to press a button to adjust the temperature of water in their tank. Even bacteria will line up on a thermal gradient at about 37°C. And finally, not all poikilotherms are completely at the mercy of their thermal environments: sharks, for example, heat their eyes to (slightly) improve their vision.

The core temperature that triggers each defense defines its threshold. Human body temperature is normally maintained between the sweating and the vasoconstriction thresholds – defined as the interthreshold range – which usually spans only a few tenths of a °C. The shivering threshold is typically a full °C below the vasoconstriction threshold. People are thus already fairly hypothermic by the time they start shivering.

Anesthetic-Induced Thermoregulatory Impairment

General anesthetics only slightly increase the sweating threshold and thus minimally impair thermoregulatory defenses against heat. In contrast, all general anesthetics profoundly reduce the thresholds for vasoconstriction and shivering. Intravenous anesthetics – such as propofol, opioids and central alpha-2 agonists – reduce the vasoconstriction and shivering thresholds as a linear function of plasma concentration. The volatile anesthetics differ in disproportionately reducing the vasoconstriction and shivering thresholds at higher doses.

The consequence of anesthetic-induced thermoregulatory impairment is a 10-to-20-fold increase in the interthreshold, usually to between 2 and 4°C. Since, by definition, no autonomic regulation occurs within the interthreshold range, anesthetized patients are effectively poikilothermic over a broad range of temperatures extending from slightly above normal to well below normal (Figure 1).

With rare exceptions, such as meperidine,[1] anesthetics synchronously reduce the vasoconstriction and shivering thresholds; so while both decrease, the normal 1°C difference between the two thresholds is maintained at any dose.[2-5] The vasoconstriction threshold during anesthesia is similar in infants, children and adults. However, it is reduced by about a °C in the elderly. Neuraxial anesthesia also increases the interthreshold range,[6] although the magnitude of the effect is considerably smaller than with general anesthesia.

Heat Balance

Heat distribution in humans can roughly be divided into peripheral and core thermal compartments. Deep tissues of trunk and head constitute the core and represent about half the adult body mass. Temperature of these well-perfused tissues is nearly homogenous and is thus well represented by any single core temperature.

The arms, legs and skin constitute peripheral tissues. Peripheral tissue temperatures are distinctly inhomogeneous and there are usually substantial differences within this mass. Many temperatures must therefore be integrated to accurately characterize peripheral compartment temperature. Peripheral tissues serve as a thermal buffer for the core. Thus by allowing peripheral tissue temperature to vary considerably, the thermoregulatory system can maintain core temperature while rarely invoking metabolically expensive defenses such as sweating or shivering.

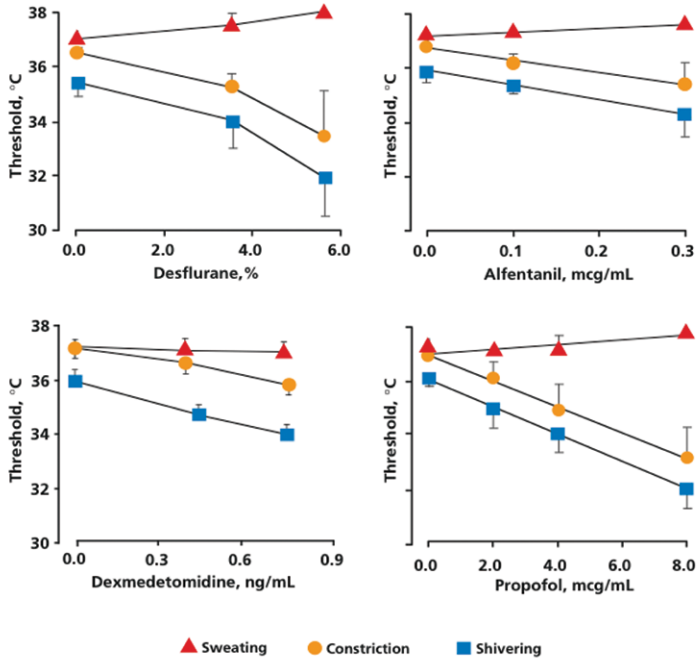

Figure 1: *The major autonomic thermoregulatory response thresholds in volunteers given desflurane, alfentanil, isoflurane or propofol. All the anesthetics slightly increase the sweating threshold (triggering core temperature), while markedly and synchronously decreasing the vasoconstriction and shivering thresholds. Used with permission.*[2-5]

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The Second Law of Thermodynamics specifies that heat can only flow down a thermal gradient. On average, over time, there must thus be a core-to-peripheral tissue temperature gradient to allow heat generated in the core to dissipate into the environment. The magnitude of the core-to-peripheral temperature gradient is determined by vasomotor tone and by past and present thermal environments, but is usually 2-4°C in hospital environments.[7]

In a typical (cool) operating suite, patients vasoconstrict to constrain metabolic heat to the core and maintain core temperature. But as noted above, general anesthesia profoundly impairs thermoregulatory control. After induction of anesthesia, the vasoconstriction threshold thus decreases to well below body temperature and thermoregulatory arteri-venous shunts open. The result is a large flow of heat from the core to peripheral thermal compartment and consequent rapid 1-1.5°C reduction in core temperature.[7] This internal redistribution of body heat – rather than net loss of heat to the environment – is the primary cause of hypothermia during the initial hour after induction of anesthesia (Figure 2). Redistribution is also the primary initial cause of hypothermia during neuraxial anesthesia, although vasodilation results largely from block-induced sympathectomy rather than impairment of central thermoregulatory control.[8]

During the next few hours of anesthesia, changes in core body temperature are mostly determined by systemic heat balance; that is, the difference between metabolic heat production and heat loss to the environment. Eventually, patients may reach a thermal equilibrium where heat production and loss are equal. But patients who become sufficiently hypothermic will trigger arteri-venous shunt constriction, which constrains metabolic heat to the thermal core and prevents further core hypothermia.[9] As discussed above, the vasoconstriction threshold during anesthesia depends on the type of anesthesia and its dose; but typically, vasoconstriction during general anesthesia re-emerges at about 34.5°C. Perioperative heat balance has been reviewed in detail.[10]

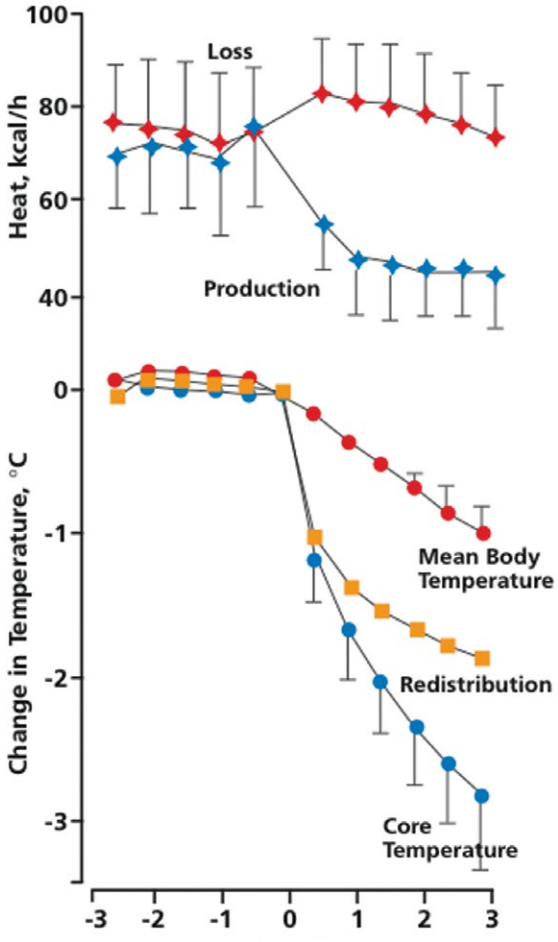

Figure 2: *To separate the contributions of decreased overall heat balance and internal redistribution of body heat to the decrease in core temperature, we multiplied the change in overall heat balance by body weight and the specific heat of humans. The resulting change in mean body temperature ("heat balance") was subtracted from the change in core temperature ("measured"), leaving the core hypothermia specifically resulting from redistribution ("redistribution"). After one hour of anesthesia, core temperature had decreased 1.6 ± 0.3°C, with redistribution contributing 81 percent to the decrease. During the subsequent two hours of anesthesia, core temperature decreased an additional 1.1 ± 0.3°C, with redistribution contributing only 43 percent. Redistribution thus contributed 65 percent to the entire 2.8 ± 0.5°C decrease in core temperature during the three hours of anesthesia. Induction of general anesthesia is identified as elapsed time zero; all values after elapsed time zero differ significantly from those preceding induction of anesthesia. Adapted with permission.*[7]

Temperature Monitoring

There are four consistently reliable core-temperature monitoring sites: pulmonary artery, distal esophagus, nasopharynx and tympanic membrane (as measured with a thermocouple). Except during the most extreme thermal perturbations – for example, cardiopulmonary bypass – these sites rarely differ by more than a few tenths of a °C and can be used interchangeably. During general endotracheal anesthesia, the esophagus is the most obvious place to measure core temperature since esophageal measurements are easy to obtain and resistant to artifact.

The difficulty is that in many patients, none of the core-temperature sites may be readily available or convenient. Core temperature can nonetheless be reasonably estimated from other sites, including the bladder, axilla, rectum and mouth. Reliability will be enhanced by thoughtful selection of the measurement site in a given patient and use of good technique. Postoperatively, electronic oral temperatures are generally reliable; in contrast, infrared aural canal ("tympanic") monitors perform poorly.[11]

Skin temperature is well below core temperature; furthermore, the core-to-skin gradient varies among patients and over

time within patients. Infrared aural canal ("tympanic") and forehead thermometers are not sufficiently accurate for clinical use. Anesthetic-induced thermoregulatory impairment and perioperative temperature monitoring have been reviewed in detail.[12]

Consequences of Hypothermia

Randomized trials have consistently demonstrated that mild hypothermia (i.e., 1.5-2°C) causes substantial morbidity (Table 1, page 39). The three most serious complications caused by hypothermia are morbid myocardial events, wound infections and coagulopathy.

The only randomized trial specific to cardiovascular events was conducted in 300 vascular surgery patients whose core temperatures differed by 1.3°C at the end of surgery. The primary outcome, a composite of serious cardiac complications, was reduced by a factor-of-three in patients assigned to normothermia.[13] Adverse cardiac events most likely result from autonomic stimulation, which increases circulating catecholamines and blood pressure.

All surgical wounds become contaminated; whether contamination progresses to clinical infection is largely determined by adequacy of host defense, especially oxidative killing by neutrophils. Hypothermia may increase wound infection risk by provoking vasoconstriction, which reduces delivery of oxygen and immune cells to surgical incisions, by directly impairing function of macrophages and other immune cells, and by reducing scar formation and wound healing.

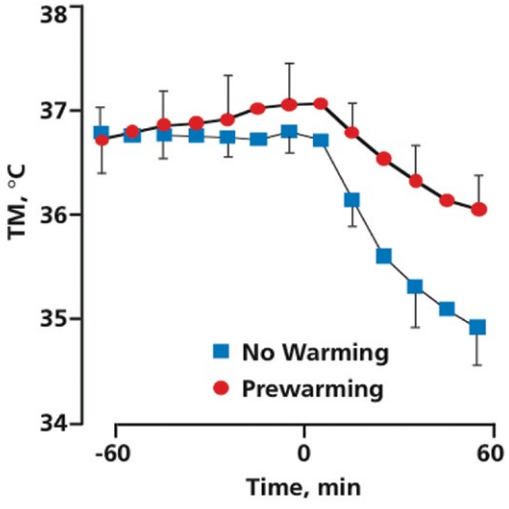

Figure 3: *During the preinduction period (–60 to 0 min), volunteers were either actively warmed or passively cooled (no warming). At induction of anesthesia (0 min), active warming was discontinued and volunteers were exposed to the ambient environment. Initial tympanic membrane temperatures were similar before each preinduction treatment. During the 60 minutes following induction of anesthesia, core temperature decreased less when volunteers had been prewarmed (Delta T = -1.1±0.3°C) than when the same volunteers had not been prewarmed (Delta T= -1.9±0.3°C). Data are presented as means ± standard deviations. Adapted with permission.*[19]

Two randomized trials evaluated surgical site infection in 200 patients undergoing colon resection[14] and in 421 general surgical patients.[15] Both found a three-fold reduction in wound infection risk. The temperature difference between the randomized groups was 1.9°C in Kurz et al.[14] and – amazingly – was not reported in Melling et al.[15] The findings are notable in that hypothermia was maintained only intraoperatively, and returned to normal within a couple of postoperative hours, whereas wound infections are typically identified one to two weeks after surgery. That hypothermia augments the risk of wound infection was possibly the first demonstration that intraoperative anesthetic management has long-term consequences – a general topic that remains under active investigation.[16]

Local tissue temperature – including temperature inside surgical incisions – is in equilibrium between core and ambient temperature and decreases roughly in proportion to core hypothermia. Platelet function (via release of thromboxane A_2) is a function of local tissue temperature. It is thus unsurprising that mild hypothermia increases blood loss and, consequently, transfusion requirement. The effect of hypothermia on each has been reviewed in a recent meta-analysis.[17]

Hypothermia also provokes a host of lesser complications, including slowed drug metabolism, prolonged recovery, thermal discomfort and shivering.

Maintaining Normothermia

The major initial cause of core hypothermia in most patients is core-to-peripheral redistribution of body heat. The amount of heat that redistributes is a strong function of the core-to-peripheral tissue-temperature gradient. Interventions that reduce the gradient thus reduce redistribution hypothermia. The easiest way to reduce the gradient is simply to warm patients before induction of anesthesia. Warming must be intense enough to provoke thermoregulatory vasodilation and sufficiently prolonged to transfer 50 or more kcal into the body (i.e., 30 minutes of forced-air warming[18]). Pre-warming has little effect on core temperature, which remains regulated, but does increase peripheral tissue temperature and body heat content. Subsequent induction of general or regional anesthesia thus provokes little redistribution hypothermia because core and peripheral tissue temperatures are already similar (Figure 3). At least five randomized trials have demonstrated the efficacy of pre-warming,[19,20] and the strategy probably deserves more use than it currently gets.

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Forced-air is by far the most common intraoperative warming approach because it is inexpensive, easy to use, effective and remarkably safe. Recently, some investigators have proposed that forced-air warming might disperse bacteria within operating rooms. This is a curious assertion since six studies demonstrate that properly used forced-air systems do not increase bacterial counts.[21,22] Furthermore, activation of forced-air warming does not reduce operating room air quality, even during laminar-flow ventilation (Figure 4). Finally, according to the Centers for Disease Control and Prevention (CDC), "... for most surgical site infections, the source of pathogens is the endogenous flora of the patient's skin, mucous membranes, or hollow viscera ..."[23] Forced-air is thus not only a perfectly appropriate way to keep surgical patients normothermic, but remains the only perioperative warming system that has been shown in randomized trials to significantly reduce surgical site infection risk.[15,24]

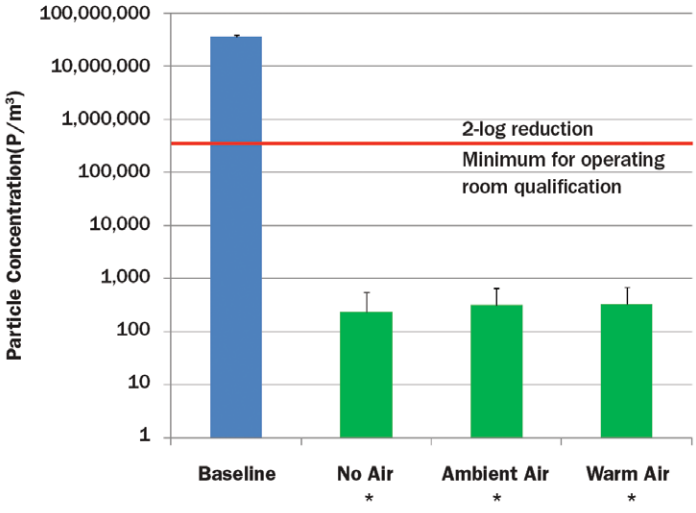

Figure 4: *Mean particle concentration at a putative surgical site (green) versus the background (baseline) particle load (blue) in operating rooms tested with an Arizant 522 upper-body forced-air blanket (Eden Prairie, Minnesota). Three different test conditions are shown: forced-air warming system set to off ("No Air"), ambient ("Ambient Air") or high ("Warm Air"). Each of the measurements at the surgical site was highly statistically significantly less than the baseline concentration (P<0.001); however, there were no statistically significant differences among the three surgical site measurements (P=0.39). Error bars are 95 percent confidence intervals. The horizontal red line shows the 2-log reduction in background particles that defines adequate laminar flow performance. Forced-air warming did not worsen the ability of the laminar flow environment to shield wounds from ambient particles.*

Only a tiny amount of heat is lost via ventilation, even with high fresh-gas flows; airway heating and humidification is thus an ineffective approach to maintaining perioperative normothermia. Similarly, only a small amount of heat is lost via conduction from the posterior surface. It is thus unsurprising that circulating-water mattresses are only marginally effective; they also occasionally cause burns. Nonetheless, newer systems that combine posterior heating with pressure relief materials (i.e., PerfecTemp, Medline) appear to maintain normothermia as well as forced-air, even in patients having major open abdominal surgery. Electric blankets also appear to maintain normothermia as well as forced-air. At least some circulating-water garments and anterior-surface pads transfer even more heat than forced-air. And finally, a novel system that combines a very small vacuum with circulating water, restricted to the hand and forearm (vitalHEAT vH_2, DynaTherm Medical), also seems to be as effective as forced-air.

It is impossible to transfer substantial amounts of heat into patients by warming intravenous fluids because the fluids cannot be heated to much above core temperature. However, it is very much possible to cool patients by giving large amounts of unwarmed crystalloids, colloids or blood. A liter of crystalloid or colloid at ambient temperature reduces mean-body temperature in a 70-kg adult by 0.25°C; a unit of blood reduces mean-body temperature by the same amount (it is twice as cold, but half the volume). It is thus unnecessary to warm fluids when small amounts (i.e., ≤1 liter/hour) are given; but it may be appropriate to warm larger volumes, especially if core temperature is drifting. Fluid warming, though, should never be the first-line defense against hypothermia because it cannot compensate for substantial heat loss from the skin surface and from within surgical incisions.

Consequences of perioperative hypothermia and warming strategies have been reviewed in detail.[25]

PQRS and SCIP

The Physician Quality Reporting System (PQRS) and Surgical Care Improvement Project (SCIP) are national attempts to improve various aspects of surgical care. Each includes provisions related to perioperative normothermia; fortunately, the provisions are harmonized and thus essentially identical for the two organizations. The incentives for participation are that compliance with SCIP provisions is publically reported and that reports to PQRS are linked to Medicare payments.

The PQRS and SCIP measures combine process and an intermediate outcome. The process component is use of warming techniques deemed effective and the outcome component is core temperature. The measures apply to surgical patients having

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Table 1: Major in Vivo Consequences of Mild Perioperative Hypothermia in Humans.

Potential Complications	First author	Year	N	ΔT_{core} (°C)	Normothermic	Hypothermic	P
Surgical wound infection	Kurz[14]	1996	200	1.9	6%	19%	<0.01
Surgical wound infection	Melling[15]	2001	421	?	5%	14%	0.001
Duration of hospitalization	Kurz[14]	1996	200	1.9	12.1 ± 4.4 days	14.7 ± 6.5 days	<0.01
Duration of hospitalization	Frank[13]	1997	300	1.3	8 (5-13, range)	8 (5-11)	N.S.
Blood loss	Schmied[26]	1996	60	1.6	1.7 ± 0.3 L	2.2 ± 0.5 L	<0.001
Blood loss	Winkler[27]	2000	150	0.4	0.5 L	0.6 L	0.002
Blood loss	Widman[28]	2002	46	0.5	0.5 ± 0.3 L	0.7 ± 0.3 L	<0.05
Blood loss	Persson[29]	2001	59	1.0	0.29 ± 0.03 L	0.30 ± 0.05 L	<0.05
Blood loss	Hofer[30]	2005	60	1.8	1.5 ± 0.5 L	2.7 ± 1.0 L	0.01
Blood loss	Johansson[31]	1999	50	0.8	1.0 ± 0.4 L	1.0 ± 0.4 L	N.S.
Blood loss	Vassiliades[32]	2003	94	0.9	0.6 ± 0.5 L	0.8 ± 0.5 L	0.015
Blood loss	Wong[33]	2007	103	?	Median 200 ml	Median 400 ml	0.01
Transfusion requirement	Nesher[34]	2003	60	1.0	1.7 units	3.5 units	<0.01
Transfusion requirement	Schmied[26]	1996	60	1.6	10 ± 55 ml	80 ± 154 ml	<0.05
Transfusion requirement	Kurz[14]	1996	200	1.9	0.4 ± 0.4 L	1.1 ± 0.9 L	0.013
Transfusion requirement	Hofer[30]	2005	60	1.8	0.4 ± 0.4 L	1.1 ± 0.9 L	0.013
Postoperative troponin 1	Nesher[34]	2003	60	1.0	22 ± 9 ng/ml	8 ± 5 ng/ml	
Morbid cardiac events	Frank[13]	1997	300	1.3	1%	6%	<0.05
Myocardial damage	Nesher[34]	2003	60	1.0	8 ± 5 ng/ml	22 ± 9 ng/ml	<0.01
Urinary excretion of nitrogen	Carli, et al [35]	1989	12	1.5	982 mmol/day	1,798 mmol/day	<0.05
Duration of vecuronium	Heier, et al [36]	1991	20	2.0	28 ± 4 min	62 ± 8 min	<0.001
Duration of atracurium	Leslie[37]	1995	6	3.0	44 ± 4 min	68 ± 7 min	<0.05
Postoperative shivering (oxygen consumption)	Just[38]	1992	14	2.3	141 ± 9 $ml \cdot min^{-1} \cdot m^{-2}$	269 ± 60 $ml \cdot min^{-1} \cdot m^{-2}$	<0.001
Duration of postanesthetic recovery	Lenhardt[39]	1997	150	1.9	53 ± 36 min	94 ± 65 min	<0.001
Adrenergic activation	Frank[40]	1995	74	1.5	330 ± 30 pg/ml	480 ± 70 pg/ml	<0.05
Thermal discomfort	Kurz [41]	1995	74	2.6	50 ± 10 mm VAS	18 ± 9 mm VAS	<0.001
Composite complications	Wong[33]	2007	103	?	32%	54%	0.027
Mortality after major trauma	Gentillo[42]	1997	57	?	2 / 29 (7%)	12 / 28 (43%)	<0.05

Only prospective, randomized human trials are included; subjective responses were evaluated by observers blinded to treatment group and core temperature. "N" = total number of subjects. ΔTcore = difference in core temperature between the treatment groups. Different outcomes of some studies are shown in separate rows. Table is restricted to hypothermia-related complications. VAS is a 100 mm-long visual analog scale (0 mm = intense cold, 100 mm = intense heat). Just et al. is but one of dozens of studies showing that hypothermia provokes shivering. Results presented as means ± SDs or median [interquartile range] unless otherwise specified. N.S. = not significant. Adopted from Sessler DI, Kurz A, *Anesthesiology News*, 2008.

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general or neuraxial anesthesia lasting at least 60 minutes who do not have documented intentional hypothermia. They can be met by fulfilling any one of the three following conditions: 1) active intraoperative over-body warming; 2) body temperature ≥36°C within 30 minutes before the end of anesthesia; or 3) body temperature ≥36°C within 15 minutes after anesthesia.

Limitations of the PQRS and SCIP measures include the fact that "active over-body" warming is deemed acceptable, although some over-body systems may not be especially effective and at least several other warming systems are also effective. This aspect of the measures remains controversial and may change as additional evidence becomes available. It is also possible that the measure may, at some point, become a pure outcome measure that will only be met by a body temperature ≥36°C. "Body temperature" is not currently defined; a variety of systems and measurement sites can thus be used, although many measurement sites are sub-optimal and some temperature monitoring systems are clearly inaccurate. And finally, neither measure includes "hard" outcomes such as cardiovascular complications, blood loss, transfusion requirement or surgical site infection.

Both measures are designed for simplicity and to be rigorous and easily auditable. Consequently, the provisions do not recognize the subtleties of clinical practice. But the basic message, that surgical patients should be kept normothermic, is well supported by many randomized trials. Maintaining normothermia is already the community standard of care, irrespective of PQRS and SCIP specifics. That said, how normothermia is maintained is entirely at the clinician's discretion. There is absolutely no requirement to use any particular warming approach for any particular patient in any particular environment. Whatever works is perfectly acceptable!

References are available at the back of the online version of this NEWSLETTER *at* ***www.asahq.org****, or by request by e-mailing* ***communications@asahq.org****.*

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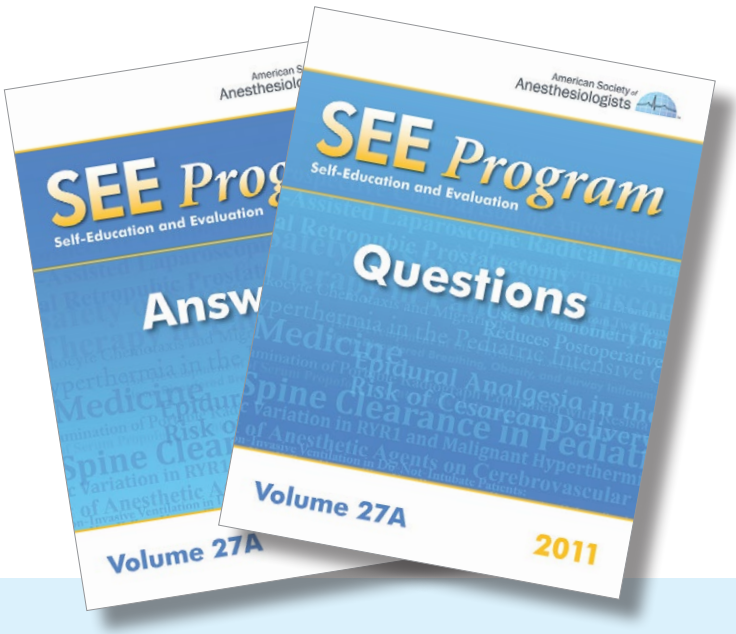

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The Development of Halogenated Agents

David J. Wilkinson, M.B., B.S.

Following the successful use of ether anesthesia in a public demonstration on October 16, 1846 in Boston, Massachusetts and the subsequent spreading of that news to Europe, there was interest in finding further agents that might produce a similar or better result.

James Mathews Duncan was employed by James Young Simpson in June 1847 as his assistant with a remit to research the possibility of other anaesthetic agents being available for obstetric use. Duncan had been working in Paris and had already translated Simpson's paper on the use of ether into French. There were no ethics committees at this time and Duncan's preferred method of investigation was to inhale any and all volatile substances that he could find. Many of these were quite toxic and made him ill, but on one occasion he awoke pleasantly from a state of deep unconsciousness. He took the bottle, containing chloroform, to Simpson and that evening the famous dinner table inhalation of chloroform took place, which convinced Simpson that he had found a significant new anesthetic agent.[1] The first halogenated anesthetic agent ($CHCl_3$) was in use the next day.

Although chloroform was retained in regular use in some U.K. centers well into the 1960s, its popularity began to decline once Levy had demonstrated its ability to sensitize the myocardium to circulating catecholamines, thus initiating severe ventricular arrhythmias. Its frequent association with delayed liver failure was another considerable concern, but when used in skilled hands its rapid onset, smooth anesthesia and potent and prolonged analgesia were very useful properties as was its lack of flammability.

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Methylene (CH_2Cl_2), also known as bichloride of methylene, was introduced by Benjamin Ward Richardson in 1867. Although used extensively by him and others like Spencer Wells, it was dismissed by Hewitt in his textbook of 1901 as a mechanical mixture of chloroform and methylic alcohol. However, the drug was very effective when used in London and in Munich where Nussbaum reported a series of 15,000 cases.[2]

Other halogenated agents were tried by various practitioners around that time. Bromide of ethyl (CH_3CH_2Br) was used extensively in the U.S., Germany and the U.K. between 1870 and the late 1880s. It was thought to be useful for short cases with an onset time of 60 seconds and duration of 46 seconds when 1 drachm (3.7mls) was placed on a towel applied to the patient's face. Prolonged use led to respiratory and circulatory failure, and deaths were frequently reported following its use. Ethidene dichloride ($CHCl_2.CH_3$) was tried by John Snow and assessed by the 1879 Glasgow Committee of the British Medical Journal, who stated that it had good efficacy and a safety point somewhere between ether and chloroform. It was never popular. Snow also used bromoform ($HCBr_3$) in animal studies but did not take the drug into clinical use. He also used "Dutch liquid" ($C_2H_4Cl_2$), which was very similar to ethidene dichloride but eventually rejected it in 1849 as having no advantage over chloroform.

The most effective of these new agents that followed the discovery of chloroform was ethyl chloride (C_2H_5Cl). Known since its synthesis in 1759 by Rouelle in France, its anesthetic properties were described by Flourens in 1847.[3] It was used by Heyfelder in Germany in 1848 but not widely adopted except for its surface cooling effects until its "rediscovery" by the dentist Carlson in Gothenburg, Sweden in 1894. Popularized by McCardie of Birmingham, U.K. in 1901 and Ware in the U.S. in 1902, it became widely used for short dental procedures and as a rapid induction agent particularly in pediatric practice. Its danger lay in its extreme potency, and overdosage with associated cardiac failure resulted in many deaths until this was appreciated. Like ether, it was very flammable.

Somnoform was introduced by George Rolland of Bordeaux. This was a mixture of 60 parts ethyl chloride, 35 parts methyl chloride and 5 parts ethyl bromide. While popular for a while, it was essentially ethyl chloride in another form.

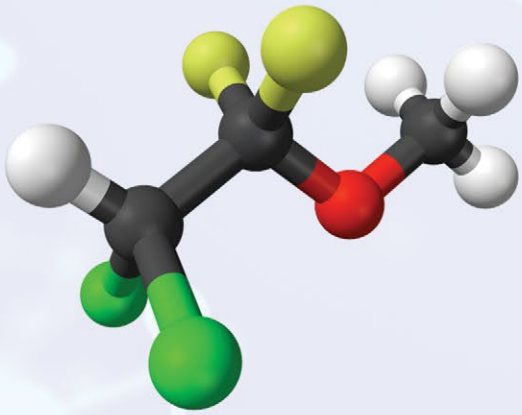
Trichloroethylene (Trilene/Trimar) (C_2HCl_3) was introduced into anesthetic practice in 1933 by Jackson of

Cincinnati[4] after some initial comments on its efficacy by Lehman in Wurtzburg in 1911. Despite further clinical work by Striker in the U.S., it was not until 1941 that the drug achieved any significant popularity. In that year a mysterious chemist by the name of Chambers in North London wrote to Charles Hadfield suggesting that trichlorethylene be investigated as a potential analgesic and anesthetic agent. Hewer wrote up their experience with its use in 133 patients[5] and the drug immediately became very popular in the U.K. Its powerful analgesic properties ensured its place in obstetric practice; a myriad of draw-over vaporizers creating a mixture of trichlorethylene in air for use by women in labour were created. A weak anesthetic agent, it was found to decompose in the presence of warm soda lime to create phosgene, and thus its popularity in the U.S. was very limited. C. Ron Stephens introduced the Duke Inhaler for analgesia for minor surgery and obstetric practice. Its advantage in general anesthesia was that its analgesic properties remained long after the anesthesia had worn off, but recovery from prolonged exposure often was very slow.

The late 1940s and early 1950s were associated with an intense search for new anesthetic agents that would be non-flammable and have low toxicity. Chemists had begun to experiment with fluoride, and after intense work in the 1930s by Booth and Bixby they were able to fluorinate organic compounds. Further work by Henne in 1937 created fluoroform, which had three flurorine atoms on a methane nucleus.[6] This work reached the anesthesia fraternity when Abreu from Oklahoma published his paper in *Anesthesiology* on unsaturated monohalogenated hydrocarbons as general anesthetic agents. Further impetus in relation to fluoride chemistry was given around this time by the Manhattan project, which used fluorination of uranium to create uranium-235; further fluoride chemistry was elucidated by its use in high-octane aviation fuels.[7] In 1946, Robbins reported the canine experiments on 46 halogenated anesthetic agents and demonstrated that a higher boiling point was associated with greater anesthetic potency and that this potency was enhanced by multiple halogenation. When bromine was used rather than chlorine atoms, there was a marked rise in potency. However, he noted that many of his compounds produced severe cardiac arrhythmias.

In 1953, working in Maryland along similar lines, Krantz reported his work in *Anesthesiology*.[8] The fluorinated hydrocarbons had been developed as refrigerants and had severe side effects such as respiratory irritation and muscle rigidity. However, the fluorinated ethers were more promising, and one trifluroethylvinylether entered clinical practice as Fluroxene or Fluoromar ($CF_3CH_2OC_2H_3$). The first recipient of the drug was Dr. Max Sadove, and the drug proved to be very effective with rapid onset and recovery, residual analgesia and no cardiac sensitization.[9] It is unclear (at least to this writer) why it did not receive more widespread acceptance and use.

However, the face of anesthesia was about to change radically with the introduction of halothane in 1956. C.W. Suckling, working at ICI's research center near Manchester, was working in parallel to these U.S. researchers, looking at fluorinated hydrocarbons. One of them was halothane ($CF_3CHClBr$), first synthesised in 1951. Its pharmacology was further investigated in animals by Raventos who found it had a rapid onset and offset, a very smooth induction, was non-inflammable and did not degrade in soda lime. It was extremely potent with a high saturated vapour pressure.[10] Michael Johnstone used the agent clinically for the first time in Manchester and confirmed its pharmacological properties in clinical use. With its general release soon associated with a whole series of temperature compensated vaporizers, the drug's future was assured. Not all were in favor of its use as its initial expense was quite considerable compared to its very cheap predecessors. The drug soon became widely used in the U.S., however, and then around the world.



Methoxyflurane ($CHCl_2.CF_2.OCH_3$)

Its propensity to cause severe bradycardia in a dose-dependent manner and the occurrence of severe hepatitis-like symptoms in some patients, particularly after multiple exposure to the drug, meant that the search for better agents continued. Methoxyflurane ($CHCl_2.CF_2.OCH_3$) was the next agent to be introduced. Used clinically by Artusio and Van Poznak in New York in 1960[11], after a series of animal experiments it was released by Abbott Laboratories to a further 30 centers in the U.S. It had a very slow onset of action and was not a potent drug but had the major advantage of producing profound analgesia that lasted well after recovery of consciousness. As it was not degradated by warm soda lime, it became the "trilene" for those countries that used soda lime extensively at that time, e.g., U.S. and Australia. Like trilene, it also became popular for use as an analgesic for short procedures and obstetric practice

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where its use in low concentrations did not result in anesthesia. It was found to be metabolized to a significant extent with the release of fluoride ions, which then resulted in nephrotoxicity with high output renal failure if the exposure to the drug was for more than 2MAC hours. It is still available for self-administered analgesia in Australia where it is used extensively in the pre-hospital setting by ambulance personnel.

While methoxyflurane was being developed and introduced, in the Ohio Medical Products Laboratories (now Baxter Healthcare Corp.), Ross C. Terrell, M.D. was synthesizing over 700 fluorinated compounds looking for a "better" anesthetic agent. Enflurane (CHF_2-O-CF_2CHFCl) was the 347th one of this series, and its clinical introduction was reported in 1966.[12] It rapidly gained popularity and started to supplant halothane as it had less effect on the cardiovascular system and contained no preservative. Concern was expressed about muscle twitching seen when used in high doses associated with hyperventilation, and there was also concern about the potential release of fluoride ions during metabolism; this latter property never caused the problems seen with methoxyflurane.

Isoflurane (CHF_2.OCHCL.CF_3) was #469 of Terrell's series and was an isomer of enflurane. This drug had minimal side effects and was metabolized significantly less than all previous anesthetic agents.[13] Otherwise its clinical profile was very similar to enflurane, and after careful pricing strategies the drug soon became one of the mainstays of inhalational anesthesia, and enflurane usage declined spectacularly. It was the first agent to really become close to the ideal anesthetic agent.

Desflurane (CHF_2-O-CHF-CF_3) was the 653rd compound investigated by Terrell. This compound was initially rejected for commercial development despite its excellent profile as an anesthetic agent, with low blood solubility and thus very fast onset and recovery, minimal metabolism and high potency because of its high vapour pressure and thus inability to administer from a standard vaporizer. A renewed interest in the 1980s on increasing throughput in ambulatory care, and thus the need for a drug with a particularly rapid recovery profile, caused a re-evaluation of the agent. In 1988 in London, Professor Ron Jones administered the drug for the first time to a human volunteer, his colleague Mike Cashman.[14] This event was filmed, perhaps another first for a new drug evaluation. The drug has established a firm place in most anesthetists' armamentarium.

Sevoflurane (CH_2F-O-$CH(CF_3)_2$) was synthesized by Bernard Regan working with Wallin and Napoli at the Travenol Laboratories, and its anesthetic properties were evaluated as early as 1960s. However, like desflurane, it was not immediately developed as there were concerns about its degradation in soda lime, and it was difficult and expensive to manufacture. However, for the same reasons as the renewed interest in desflurane, sevoflurane was re-evaluated in the early 1970s.[15] Major clinical trials were initiated in Japan in the 1990s, and its ease of use when performing inhalational induction and its high potency and very rapid recovery due to its low blood solubility made it a very attractive agent.

"Throughout the history of the use of these agents, our profession has constantly sought to improve the lot of our patients by searching for better and less toxic drugs."

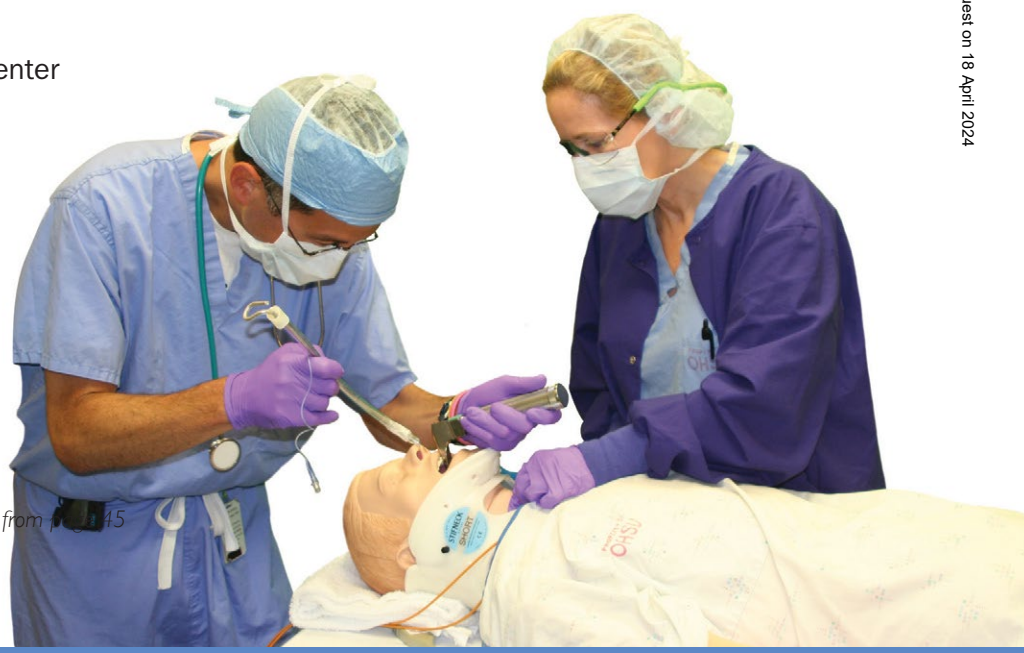
As we can see from this brief outline of the development of halogenated anesthetic inhalational agents, our profession has used a huge variety of these agents over the years. We have moved from the casual introduction of agents – just because they were volatile and without much concern about any deleterious effects to our patients – to a careful development of specific agents based on knowledge of structure and potency that have only been used clinically after extensive laboratory and further ethically controlled trials. Throughout the history of the use of these agents, our profession has constantly sought to improve the lot of our patients by searching for better and less toxic drugs. It is unlikely that we will see the introduction of further agents until the exact molecular mechanisms of anesthetic action are clearly defined, when we may see specific receptor binding agents, which may or may not be halogenated drugs.

References are available at the back of the online version of this NEWSLETTER *at* ***www.asahq.org****, or by request by e-mailing* ***communications@asahq.org****.*

ASA Simulation Education Network

The American Society of Anesthesiologists has endorsed simulation centers located throughout the United States who offer courses that help to fulfill one requirement of the American Board of Anesthesiology's (ABA) Maintenance of Certification in Anesthesiology Program (MOCA®) Part IV. The simulation education network includes:

- Beth Israel Deaconess Medical Center
- Center for Medical Simulation
- Cooper Simulation Laboratory
- Duke University Human Simulation and Patient Safety Center
- HELPS Center Mount Sinai School of Medicine
- ISIS, University of Washington
- Mayo Clinic – Rochester
- Medical University of South Carolina Healthcare Simulation Center
- Northwestern University
- Oregon Health & Science University
- Pennsylvania State University College of Medicine
- Stanford School of Medicine
- Stony Brook University Medical Center Clinical Skills Center
- Texas Tech University Health Sciences Center
- UCLA Simulation Center
- University of California, San Francisco
- University of Chicago
- University of Kentucky
- University of Miami – Jackson Memorial Hospital Center for Patient Safety
- University of New Mexico School of Medicine
- University of Texas Medical Branch at Galveston
- University of Utah Anesthesiology Department
- University of Virginia Health System
- Vanderbilt University
- Wake Forest University School of Medicine Baptist Medical Center
- Washington University in St. Louis School of Medicine
- WISER, University of Pittsburgh



*Please consult the ABA Booklet of Information, available on the ABA website at **www.theABA.org**, for a list of all MOCA® requirements.

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Visit **www.asahq.org/For-Members/Education-and-Events/Simulation-Education.aspx** for contact information and updates about new simulation programs added to the network.

Anesthesia Machines Proceed to Greater Safety

Jerry A. Dorsch, M.D.

Equipment misuse has always been more frequent than equipment failure. Many of the advances in anesthesia machine safety have been mechanisms to prevent operator error. One of the earliest devices was the Pin Index Safety System that was mandated by the Joint Commission on Accreditation of Hospitals around 1960. This prevented the installation of an incorrect gas cylinder on the anesthesia machine.

In the 1960s, the oxygen failure safety valve (commonly referred to as the fail-safe device) became available as an option on anesthesia machines. This device interrupts the flow of other gases if the oxygen pressure in the machine falls below a certain level. This addressed the problem of an oxygen cylinder becoming empty or the oxygen pipeline system failing, which could result in only nitrous oxide being administered. Unfortunately, many hospitals did not spend the additional money to purchase this device. Later it became standard on all anesthesia machines.

A major problem with early anesthesia machines was inaccurate gas flow measurement. One early attempt at determining gas flow was to bubble the gas through a water-filled chamber and measure the frequency of the bubbles. Another approach was to pass the gas through a tube with holes in the side inside a water-filled container. There was a water depression flowmeter in which the water level in a tube was depressed as the flow increased. The water depression was measured on a scale that indicated the gas flow. High flows could blow the water out of the tank. A major advance was to use a tube whose diameter increased gradually from bottom to top and an indicator that was pushed upward by the gas flow.

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After World War II, the rotameter, which had been in use in Europe for some time, became the standard flowmeter indicator in the United States, allowing accurate gas flow.

The first monitor to be added to anesthesia machines was for breathing system pressure. The Drager Pressure Monitor (DPM) was at first an add-on device. Later it was incorporated into the anesthesia machine. This was designed to prevent barotrauma and negative pressure pulmonary edema and detect some breathing circuit disconnections. The next monitor to be added to the anesthesia machine was the oxygen analyzer, which measured the oxygen concentration in the breathing system. Oxygen monitors were initially add-on devices but were later incorporated into the machine.

A major step in making anesthesia machines safer was adoption of the first anesthesia machine standard in 1979. This was a joint effort between clinicians and industry. It mandated an oxygen flow control knob that was fluted and larger than the other knobs to help the user identify it in a darkened room. Another change was to make the oxygen failure safety device standard on all new machines. Color-coded flowmeters were mandated by the standard. All flowmeters were to be in series rather than in parallel. The most commonly used fresh gas flow in those days was five liters/min. It was felt that the parallel arrangement was dangerous because sometimes the operator would turn on a high flow of nitrous oxide (e.g., 3 l/min) and a low flow of oxygen (e.g., 200 cc/min) since the fine flowmeter indicator would be at about the same height as the indicator in the high-flow oxygen flowmeter with a 2 l/min flow. Flowmeter sequence was mandated so that the oxygen flowmeter was placed on the right side of the flowmeter block and near the outlet from the gas manifold where all the gases joined before flowing to the machine outlet. This prevented a hypoxic mixture from being delivered if there was a crack or leak in the other flowmeter tubes.

In the 1970s, oxygen proportioning systems that prevented the operator from giving less than 25 percent oxygen began to appear. While the oxygen failure safety valve prevented a hypoxic mixture from being dispensed if the oxygen source failed, it did not prevent the operator from turning off the oxygen flow at the control valve. If the oxygen flow was decreased, the nitrous oxide was also decreased to maintain the minimum oxygen percentage. Proportioning systems are now standard on anesthesia machines.

Vaporizers on early machines were bubble-through or flow-over devices that gave no indication of the agent concentration being dispensed. They had no means of thermal compensation, so as the agent temperature decreased, the flow through the vaporizing chamber needed to be increased. The Copper Kettle (and later the Vernitrol) vaporizer was the first that delivered a precise output. This became more important when potent anesthetic agents such as halothane were introduced. Unfortunately, there were associated problems. The valve that directed the carrier gas flow through the vaporizer was also used for the oxygen flush. If the vaporizer was in use and the oxygen flush was activated, the valve needed to be returned to the vaporizer "On" position or no volatile agent would be delivered. Since any volatile agent could be used in these vaporizers, often one contained more than one agent. Earlier models of these vaporizers could be overfilled. Finally, to determine the vapor output it was necessary to make calculations that depended on the agent being dispensed. Since these calculations varied with the agent, the operator might use incorrect calculations and deliver an overdosage or underdosage of the agent.

In the 1950s, agent-specific vaporizers were developed. The first was the Fluotec Mark II, which was used for halothane. This was only accurate at high fresh flows and had an attached chart showing the output at lower flows. At that time, most anesthesia machines did not have a place to mount this type of vaporizer so it was often connected in the fresh gas delivery hose between the anesthesia machine and the breathing system. Unfortunately, these vaporizers were not able to handle the high gas flows produced when the oxygen flush was activated. This led to a different-style anesthesia machine where the vaporizer was mounted on a back bar and the flow from the oxygen flush was delivered downstream of the vaporizer.

Foregger Copper Kettle Anesthesia Machine
(Photo courtesy of Wood Library-Museum of Anesthesiology)

Anesthesia ventilators have also evolved. Early ventilators were pneumatically controlled and separate from the anesthesia machine. They were powered by oxygen pressure and connected to the bag mount when in use. The APL valve needed to be closed when the ventilator was in use. Electronically controlled ventilators became a part of the anesthesia machine in the 1970s. Using electronic controls has allowed more ventilatory modes, and the modern anesthesia ventilator has capabilities close to those of intensive care ventilators.

Recent years have seen the anesthesia machine evolve from a pneumatically controlled device with added-on vaporizers, ventilator and monitors to a computer-controlled anesthesia workstation with all these devices integrated and all information, including settings, presented on a single central display. Flowmeters have been replaced by flow sensors in many new machines. The flow control valve may be manual or electronic. The workstation monitors its functions to detect potentially unsafe conditions. Another significant change is increased automaticity of the pre-use checkout procedures. These changes have made the anesthesia workstation more flexible and user-friendly.

Unfortunately, this makes the anesthesia workstation dependent on electrical power. Failure of power in the operating room can cause major problems.[1,2] It is essential that each anesthesia provider know how to prevent and deal with power failures. This will vary with the make and model of the machine.

Another disadvantage is that there are significant differences in design among manufacturers and even among models from the same manufacturer. As a result, a single pre-use checkout procedure cannot be used for every machine. In 2007, new guidelines for Pre-Anesthesia Checkout were developed and approved.[3] All readers are encouraged to read the new checkout guidelines and learn what procedures are performed automatically and which require additional effort to reduce the risk of patient injury due to anesthesia equipment.

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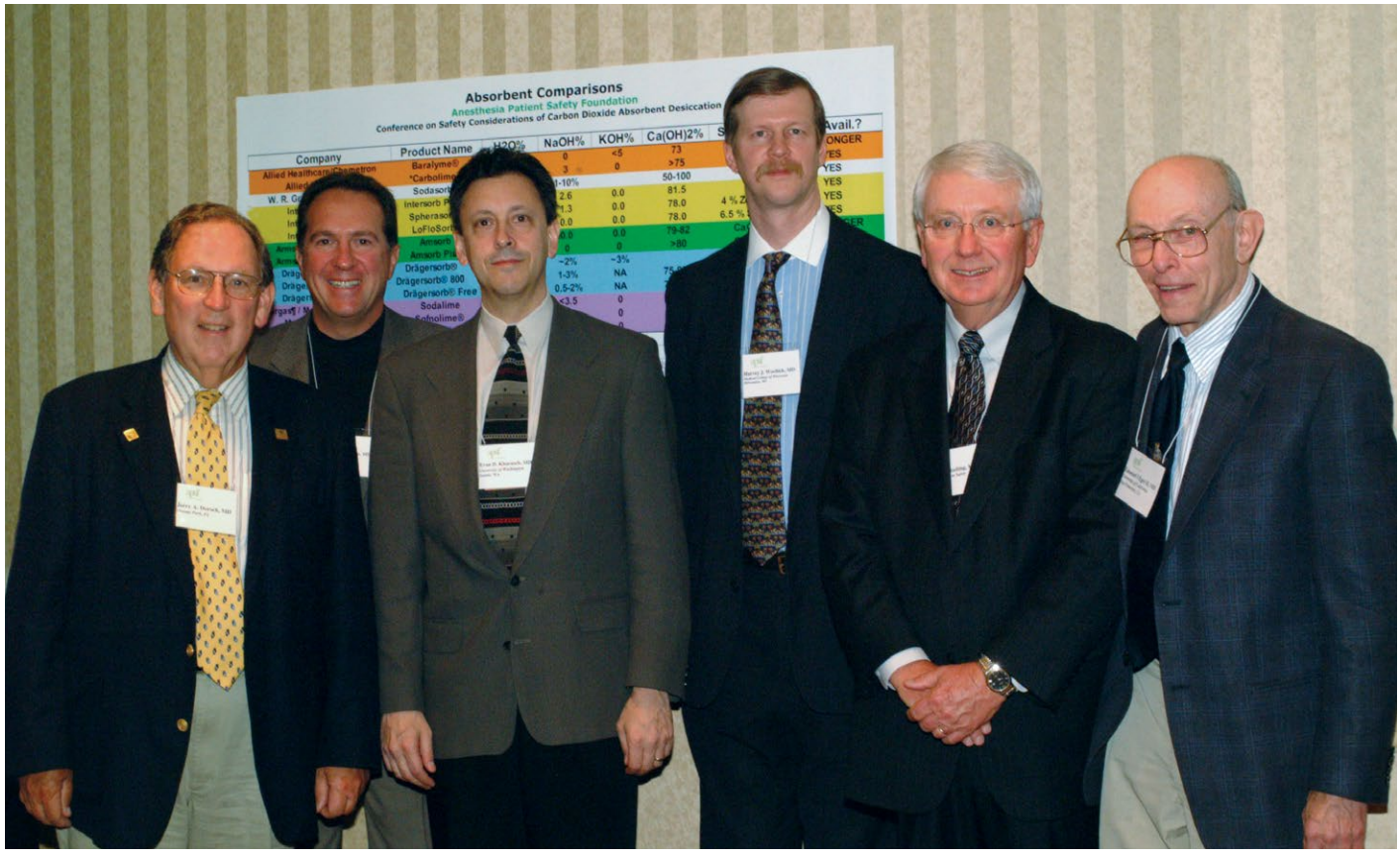

Jerry A. Dorsch, M.D., Michael A. Olympio, M.D., Evan D. Kharasch, M.D., Ph.D., Harvey J. Woehlck, M.D., Robert K. Stoelting, M.D., and Edmond I. Eger II, M.D. speak at the APSF Conference on Safety Considerations of Carbon Dioxide Absorbents in April 2005.