NEWSLETTER

ASCCA INTERCHANGE

AMERICAN SOCIETY OF CRITICAL CARE ANESTHESIOLOGISTS

Volume 21 Number 2 2010 www.ascca.org

President's Message

Time For Change: Society of Critical Care Anesthesiologists (SOCCA)

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ASCCA President

It's time to consider a change. I propose to you that our name no longer serves our purpose or mission. The ICUs are not what they were 24 years ago when this Society was formed, and essentially everything about how we live and how we provide care has changed dramatically. Our Society has been changing, too, and thus it is time to consider a name change as an opportunity to better position ourselves for the future.

Let me start by reminding you, at least for those of you not *copes mentis* in 1986, what the world was like 24 years ago. The Dow Jones closed at year end at 1,895, the average price of a new car was $9,255, gas averaged 85 cents a gallon, Spain and Portugal joined the European Union, the Chernobyl explosion occurs, Bovine spongiform encephalopathy is identified, the U.K. and France announce plans to build the Channel Tunnel, the Space Shuttle Challenger disintegrates, the 386 microprocessor is released by Intel, IBM releases the first laptop, the human genome project is started, "Crocodile Dundee" and "Top Gun" were at the theater, Debbie Harry and the Pet Shop Boys were on the radio, and "Magnum P.I." and "Cheers" were on the tube. Our President was Reagan, Russia's was Gorbachev, Thatcher was U.K. Prime Minister and Mitterrand was in charge in France.

Obviously, people, tastes and values change. The American Society of Critical Care Anesthesiologists (ASCCA) was formed during this mid- to late-80s time frame. Although it is true that some of things from 1986 are timeless – e.g., "Top Gun" (the movie), "Cheers" (the TV show) – the majority of things have changed, and most by leaps and bounds. Computer processing, for instance, is at least 1,000 times faster, and the Human Genome Project completed the major goals of mapping the human genome.

The past presidents of ASCCA read like a hall of fame of leaders in our field. The work they did to birth this Society and to guide it to its present place is simply phenomenal. In 1986, critical care medicine was relatively new, and certification was just beginning. The Society was formed to serve as a home for this "new" field of critical care within anesthesiology. Like the world around us, the world of anesthesiology and the world of critical care are evolving, and thus ASCCA has been maturing.

About eight years ago, a small task force was created to examine the organization construct of this Society and to address its focus. That group brought back many suggestions that have helped revitalize this organization. The Society decided to focus more on education than any other domain while growing its involvement on the politico-economic field. Committees were streamlined, some were dropped, and the bylaws were changed to mirror the values and direction of the Society. The focus on education has clearly helped us progressively grow membership back to the highest point in a decade and to grow the annual meeting to its largest meeting ever – over 250 attendees. Our involvement in politico-economic affairs has helped us to be seen as a "player" by the other critical care societies. We have been a vital member of the Critical Care Work Group (CCWG), and we have even chaired this group for a two-year term.

In order to create some consistency in leadership and programmatic design, the Society extended its executive leadership terms of office to two years. Recently, new approaches to planning of the Annual Meeting, the editorial board of this newsletter and the "Resident's Guide to the ICU" have reinvigorated these valuable member benefits while providing a forum for membership involvement and have also added succession planning to activities. Our journal has changed from *Anesthesiology* to *Anesthesia & Analgesia,* and we are now about to help appoint a new CCM section editor while also launching a joint membership with IARS for those interested.

At this past Annual Meeting in New Orleans, the bylaws were updated to ensure that they reflected all of these important upgrades to our Society. Additionally, the category of "international member" was removed as we began the process to embrace a broader membership in this more globally connected world. In fact, we are indeed experiencing growth in international members as increasingly they see us as a home.

What does all of this have to do with changing our name? This is not about getting a new emperor or the emperor getting a new pocket watch, but about an emperor better prepared

Continued on page 14

CONTENTS

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If you would like to contribute a review for a Fellowship Program at your institution in a future issue of the ASCCA Interchange, please contact Chris Dionne at **c.dionne@asahq.org**.

The ASCCA Interchange is published by the American Society of Critical Care Anesthesiologists, 520 N. Northwest Highway, Park Ridge, IL 60068-2573; (847) 825-5586.

PRO: Statins in the Critically Ill – Go, Statins, Go!

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The beneficial effects of statins are growing well beyond the cholesterol-lowering effects of inhibition of HMG CoA-reductase. The pleiotropic effects of statins have been documented in decreasing oxidative stress and inflammation, improving endothelial function, enhancing the stability of atherosclerotic plaques and inhibiting the thrombogenic response.[1] Whether in the treatment of sepsis or post-operative critically ill patient, an opportunity to treat lies before us.

In a subgroup analysis of healthy patients from the JUPITER trial, statin therapy significantly reduced the risk of venous thromboembolism compared to placebo.[2] In healthy patients with elevated CRP (and without dyslipidemia), statin therapy decreased major cardiovascular events, including stroke, MI or death.[3] What we have here is a physiologic link to an improved outcome. What critically ill patient needs more DVT/PE/MI/CVA?

Healthy patients are one thing, the critically ill may be another. For our SICU population, a recent study of patients undergoing vascular surgery found perioperative statin therapy to be associated with improved postoperative cardiac outcome as well as decreased levels of markers for inflammation.[4] This finding mirrors those from above, that of improved outcomes as well as physiologic pathways to define them.

In sepsis, statins decrease oxidative stress and inflammation, improve endothelial function, enhance the stability of atherosclerotic plaques, and inhibit the thrombogenic response.[1] In a case-matched series, statins seem to confer an immunomodulatory benefit for reducing pneumonia in diabetics.[6] Some venture to say that community – acquired pneumonia may well be an indication for statin therapy.[7]

One argument against statins is the risk of rebound.[5] Schouten et al. did not find this phenomenon in this patient population, in whom no increase in adverse events was noted after being started on statins pre-operatively, with a following interruption for two days.[4] Puccetti et al. suggest the risk window is as long as 14 days. They also agree that the platelet aggregability reverses with re-initiation of treatment.[8] If you believe rebound exists, you must also agree that it is reversible and treatable.

Although we are still elementary in our understanding of the effects of statins, we already are seeing evidence of outcome benefit. As much as statins may prevent patients from landing in our ICUs, they may help our patients launching out of them as well.

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CON: Statins in the Critically Ill – No, Statins, No!

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For the past 20 years, 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (statins) have been used as primary agents in both the primary and secondary prevention of coronary artery disease (CAD). Numerous trials have demonstrated their pleiotropic effects as well as their utility in reducing the risk of cardiovascular events.[1,2]

All of this is well and good to avoid a hospital admission, but in the event that a patient lands in your ICU, continuing that statin may be difficult, nay, impossible. NPO status and the non-existence of an I.V. form forces discontinuation. Perhaps your patient can take PO but cannot absorb it enterally; again, discontinuation. That critically ill patient may now be at risk for thromboembolic, hemorrhagic and myocardial complications.

Using a combination of brachial artery ultrasound and cell culture, Chen et al. found that abrupt withdrawal of statins leads to dysfunctional endothelial-dependent vasodilatation, worse than pre-treatment status. This effect lasted longer in high-risk patients compared to healthy subjects.[3] This fact raises the question: what other effects of statins are accentuated by critical illness? Statins + hepatotoxicity + rhabdomyolysis have been examined[4,5]; critical illness + statins have not.

Three studies highlight major adverse events upon statin discontinuation. **Perioperative**: In patients taking statins prior to major vascular surgery, an interruption of statin therapy for more than four days post-op is associated with a higher risk of acute myocardial infarction.[6] **Post-CVA**: An observational study one year after ischemic stroke; discontinuation of statin therapy was an independent factor in increasing mortality, even in those patients who did not have evidence of coronary artery disease.[7] **Pre-CVA**: In a case-control matched study involving more than 11,000 patients, the risk of subarachnoid hemorrhage doubled in patients who had discontinued their statin therapy.[8]

One possible explanation of these results is increased platelet aggregation. In a small study that compared patients who changed statin to patients who discontinued then reestablished statin therapy after 60 days, there was significant platelet aggregation 14 days after discontinuing therapy. This increased platelet aggregation decreased after reestablishing statin therapy.[9]

Statins have benefit – this much is clear. Unfortunately, their benefit is much less defined when shrouded with critical illness. Until a parenteral solution is available, and until their role in critical illness comes into focus, statins' place in the world will remain in the clinics, not the ICU.

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PRO: Hypertonic Saline Solutions for Resuscitation in the Critically Ill

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In 1980, the accidental infusion of 100 mL of 7.5 percent saline to an obtunded and hypotensive Brazilian patient with end-stage renal failure stimulated a renewed interest in hypertonic saline solutions (HSS) as a potential resuscitation fluid for the critically ill.[1] While it is true that there is no clear benefit of HHS in terms of survival, owing to its potential beneficial microcirculatory, cardiac, immunomodulatory and other physiological effects, HSS may be used safely as a resuscitation fluid in hypovolemic and septic shock.

HSS facilitates a nearly instantaneous mobilization of fluids from intracellular to extracellular compartments through an osmotic gradient. In various models of hypovolemic and septic shock, HSS infusions have been shown to redistribute fluid from the perivascular to the intravascular space by mobilizing water along an osmotic gradient, drawing on the vast reservoirs of water contained in cells and the interstitium.[1,2] With an infusion of 7.5 percent HSS and dextran, the intravascular volume can be increased by up to four times greater than the infused volume itself.[3] For every liter of HSS administered, a plasma volume expansion of approximately 750 mL is achieved versus a 300 mL expansion with 1 L of isotonic crystalloid.[4] HSS is often used in conjunction with colloids such as dextran or hydroxylethyl starch. Colloids help increase the oncotic pressure, but the volume effect is small and the principal benefit gained by co-administration of these colloids is an improvement in the duration of effect of HSS.[4]

HSS has beneficial microvascular and vascular effects. Endothelial cell edema occurs during the early phases of hypovolemia and shock and is caused by hypoxemia, progressive ATP deficiency and membrane ion exchange dysfunction. As a result, endothelial cells become edematous, leading to capillary lumen narrowing and reduced oxygen transport. Infusions of HSS may help reduce endothelial volume by up to 20 percent, increasing capillary diameter and reducing resistance to flow.[5,6] Whereas the mechanisms underlying the relaxant effects of HSS on vascular smooth muscle remain unclear, regional blood flow improvement with HSS has been demonstrated in virtually all areas of the microcirculation.[4,7]

HSS has been shown to increase preload, decrease afterload and increase myocardial contractility.[8] Boluses of HSS may precipitate a brief initial decrease in mean arterial blood pressure via a reduction in systemic vascular resistance, but in the early hyperdynamic stage of sepsis, HSS leads to improvements in myocardial performance that appear to be unrelated to changes in myocardial oxygen consumption or changes in coronary flow.[9] Improvements in myocardial performance may be related to decreased myocyte edema or other complex effects at the cellular level.

Resuscitation with HSS may have protective immunomodulatory effects for patients with hypovolemic or septic shock. HSS has been shown to attenuate lung injury after hemorrhagic shock through complex mechanisms that may include reduced neutrophil accumulation and activation, in addition to altered cytokine production.[10] HSS has the ability to enhance the function of normal T-cells by co-stimulating interleukin-2 production,[11] enhancing bactericidal pro-inflammatory cascades,[12] and altering the expression and release of elastase, cytokines, free radicals, tumor necrosis factor and adhesion molecules.[8] In one case-control study involving postoperative ICU care following major spinal surgery, a mortality benefit and decreased incidence of postoperative infections were attributed partially to the likely immunomodulatory effects of HSS.[13]

Several meta-analyses and randomized trials on the use of HSS have shown that boluses clearly improve blood pressure in both hypovolemic and septic shock, although there is insufficient data about the ideal concentration, dose or length of infusion time.[4,8] The anti-inflammatory and immunomodulatory properties of HSS may limit or abrogate some of the damaging ischemia and inflammation associated with sepsis. Even though there is a lack of evidence to support a survival benefit with use of HSS in shock states, an expanding body work in both animal and human subjects suggests numerous physiological benefits. In view of the fact that the majority of investigations have been complicated by heterogeneous data, different concentrations and the addition of colloids, further studies with strict protocols are warranted. In the meantime, current best evidence suggests that HSS, either alone or in combination with colloid solutions, may be safely used as an effective solution for resuscitation in patients with shock secondary to hypovolemia or sepsis.

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Continued on page 7

CON: Hypertonic Saline Solutions for Resuscitation in the Critically Ill – More Trouble Than It's Worth

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Hypertonic saline solutions (HSS) have been used as a resuscitative fluid for decades. To date, there has yet to be a consensus on the benefits of HSS over crystalloid solution. Moreover, the purported beneficial effects of HSS have yet to confer a mortality benefit.

Hypertonic saline is an attractive option for a resuscitative fluid given its theoretical benefit of expanding plasma volume by mobilizing water along an osmotic gradient. Ideally, hypertonic saline should remain intravascular and improve hemodynamics for a longer period of time than crystalloid. This would translate into less fluid required to meet hemodynamic goals and an overall smaller incidence of fluid overload and its related sequelae. Although there is some evidence that boluses of hypertonic saline solution increase blood pressure in hemorrhagic shock,[1] this effect is not consistent across similar studies and does not extend to other types of shock.[5] There has been some discussion about the potential immunomodulatory effects of hypertonic saline, but this has mostly been shown in animal studies. The human study by Rizoli and collegues[2] did show that HSS blunted neutrophil activation and altered the cytokine production profile; however, the patients were given hypertonic saline plus dextran solution, there were a small number of patients enrolled, and it was not designed or powered to identify clinical benefit. Also, one study suggests that perioperative use of hypertonic saline reduces postoperative infections and may lower mortality, but the mortality benefit was not statistically significant and the study was retrospective.[3]

Use of hypertonic saline as a resuscitative fluid has many potential associated risks. Predictably, HSS causes an increase in serum sodium. Hypernatremia is associated with increased morbidity and mortality in hospitalized patients, especially if the onset is acute.[7] Potential risks of hypernatremia include brain hemorrhage, dehydration and altered mental status.[6] Many of the studies using HSS involve burn victims. The burn literature has established the association of hypertonic sodium resuscitation with acute renal failure. In a study by Huang,[4] patients resuscitated with HSS had a four-fold increase in renal failure over lactated Ringer's. HSS patients also had two-fold increased mortality and the total resuscitation volume required was not significantly reduced. Additionally, resuscitation with higher-concentration saline solutions requires a large central vein for infusion due to the risks of extravasation.

Using hypertonic saline as a resuscitative fluid remains a controversial issue, primarily because of the lack of homogeneity in the currently available studies. The concentration of hypertonic saline used in present studies ranges from 1.8-7.5 percent or higher. Also, most of the studies use hypertonic saline as a bolus dose and in combination with dextran or hextend. Based on this, it is difficult to determine which is beneficial – the hypertonicity of saline, the hyperosmolarity of dextran/hextend or the combination of both. The few meta-analyses on the subject failed to establish the effectiveness of hypertonic saline as a resuscitative fluid over crystalloid.[8,9]

In summary, the benefits of hypertonic saline as a resuscitative fluid remain more theoretical than factual. There has been no convincing evidence to date demonstrating the superiority of hypertonic saline solution over crystalloid solution. Given the potential complications associated with hypertonic saline, it may be prudent to avoid it rather than expose patients to a therapy that has proven risk, but no proven benefit.

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PRO: Pulmonary Artery Catheter: More Than a Decade-Long Debate – Where Do We Stand With Its Use in the ICU?

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Forssmann, Cournand and Richards were first to pioneer work with right heart catheterization and enhance our understanding of physiology and pathological changes of the circulatory system. Dexter was the first to develop the pulmonary artery catheter (PAC). H.J.C. Swan and William Ganz incorporated the Stewart-Hamilton thermodilution method for measurement of cardiac output into the design of the right heart catheter PAC that remains in clinical use. The introduction of the PAC enabled the bedside clinician the ability to directly measure central venous, pulmonary capillary wedge and pulmonary pressures, cardiac output, mixed venous oxygen saturation, and further derive variables such as oxygen delivery, oxygen uptake and oxygen extraction ratio. This clinical information has provided the experienced clinician, for the most part, a reliable source of information in the management of patients during critical illness for at least the last three decades. However, clinical trials failed to demonstrate that PAC use improves patient outcomes such as mortality and morbidity in managing critically ill patients. Other alternative monitors such as pulse contour cardiac output technology has, to date, been equally disappointing. In a direct comparison of these two leading technologies, neither was found to be superior over the other in improving clinical outcomes.[1] One may argue that the placement of the PAC does not significantly increase the risk of central venous access. Antibiotic-coated central venous catheters may offer advantage over the PAC for the prevention of central line–associated bloodstream infections (CLABSI). However, with careful attention to placement, maintenance and assessment for the need of continued use, this "ICU bench mark" (CLABSI) can be maintained at a low level. Complications such aspulmonary artery rupture or pulmonary hemorrhage are extremely rare, and common arrhythmias associated with the PAC do not worsen patient outcome.[2] PAC is the only clinically available method for continuously measuring pulmonary artery pressures. Patients and conditions where acute rise in pulmonary pressure may lead to acute right failure may be best managed with a PAC. For that purpose, in cardiac surgery patients, the PAC remains to be widely used.[3]

Until we have data that clearly demonstrate a superior monitor for the management of critically ill patients, choice of monitoring may not be crucial. Understanding the use of the chosen technology, understanding of the underlying disease process and monitoring limitations may be of greater importance in caring for critically ill patients. Validation, risk and cost are additional factors that need to be taken into consideration when a choice of monitor is made.

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PRO: Hypertonic Saline Solutions for Resuscitation in the Critically Ill

Continued from page 5

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CON: Pulmonary Artery Catheter: More Than a Decade-Long Debate – Where Do We Stand With Its Use in the ICU?

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In the 1970s, David M. Eddy, M.D., Ph.D. initiated an era of awareness in recommending treatment modalities based on supported data from clinical trials. A new chapter in clinical practice was introduced at a time of a growing body of competing medical advances marketed for patient care. The pulmonary artery catheter (PAC) was introduced into clinical practice without evidence from outcome studies. With widespread clinical use in critically ill patients as a diagnostic and monitoring tool, validated indications for placement had become an area of great controversy. Classical teaching of the use of the PAC to assess the patient's volume status and end point of resuscitation, diagnose and manage patients with conditions such as sepsis, heart failure and hypovolemic shock have not shown to improve patient outcome. Furthermore, clinical use has been challenged in a number of studies, scientific papers and articles.[1] Sandam et al. in 2003 concluded no benefit to therapy directed by PAC for elderly high-risk surgical patients requiring intensive care. In acute lung injury, PAC-guided therapy did not improve survival compared to central venous catheter-guided therapy.[2] Complications from line placement have also contributed to the development of less- and non-invasive monitoring systems that provide validated clinical information. A number of monitors have been developed to measure cardiac output based on the "fick principle," changes in bio impedance as well as Echo technology, which also may serve to provide equivalent information.[3] However, despite validation in comparison to the PAC, no technology has shown to be superior in outcome studies. Early goal-directed resuscitation as a strategy, not as a specific monitoring modality in sepsis, has shown to improve patient mortality.[4] Outcomes such as shortened ICU and hospital length of stay have been reported in short series by utilizing similar management principles with non-invasive cardiac output monitors.[5] At present, the PAC is the only available monitor for continuously monitoring pulmonary pressures. The current accepted indications for the use of the PAC include surgical procedures or conditions associated with significant blood loss or fluid shifts in the setting of known significant pulmonary hypertension and or evidence of right heart strain. The use of the PAC may be justified in the management of patients with decompensated severe COPD and evidence of right heart compromise.[6] Besides goal-directed early resuscitation in septic shock as a strategy, the search continues to identify a monitor that will enable improved outcomes, morbidity and mortality with respect to end point of resuscitation while monitoring patients in critical illness.

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Literature Review I: ECMO for H1N1 Influenza ARDS

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Article:
ANZ ECMO. Extracorporeal Membrane Oxygenation for 2009 Influenza A (H1N1) Acute Respiratory Distress Syndrome. *JAMA.* 2009; 302(17):1888-1895.

The H1N1 influenza A pandemic emerged upon the international scene in Mexico and the United States in spring 2009. Primarily affecting children and adults under 60 years old, this new strain of influenza resulted in more intensive care unit (ICU) admissions, severe acute respiratory distress syndrome (ARDS) and increasingly refractory hypoxemia and hypercapnia despite optimal management. The increased morbidity among healthier patients has been attributed to invasion of the lower respiratory tract and higher titers of virus in lung tissue, as compared to seasonal influenza.[1]

During winter 2009 in the southern hemisphere, the Australia and New Zealand ECMO Influenza Investigators (ANZ ECMO) performed an observational study of their experiences and outcomes in 68 patients treated with ECMO for severe ARDS due to H1N1. In the ICUs employing ECMO support, nearly one-third of patients on mechanical ventilation required ECMO. The diagnosis of H1N1 was confirmed in 78 percent of patients by polymerase chain reaction or viral culture, or highly suspected in 12 percent due to serological data. The observed common co-morbidities for requiring ECMO for H1N1 were obesity (BMI > 30), asthma and diabetes mellitus (50 percent, 28 percent and 15 percent of patients, respectively). The calculated incidence of ECMO for H1N1 was 2.6 cases per million people in Australia and New Zealand.

The patients requiring ECMO demonstrated severe ARDS, oftentimes failing conventional mechanical ventilation and varied rescue therapies. The frequent rescue therapies included recruitment maneuvers, prone positioning, high-frequency oscillation, nitric oxide and prostacyclin. Despite rescue therapies, the median nadir $Pa0_2/Fi0_2$ ratio was 56, pH was 7.2 and highest $PaC0_2$ was 69 mm Hg. The median modified acute lung injury score, or Murray score, was 3.8 out of 4.

The median duration of ECMO support was 10 days; in fact, two of the 68 patients studied remained on ECMO at the end of data collection. The majority of patients received veno-venous cannulation via the femoral and/or jugular vessels. Hemorrhagic complications occurred in a little over half of patients – most commonly at ECMO cannulation sites, but also occurring in the gastrointestinal, respiratory, genitourinary and central nervous systems.

Although the data collection and follow-up times were brief, the authors describe that nearly 80 percent of patients were weaned from ECMO successfully. Of the 52 patients who transitioned from ECMO, 48 patients survived to ICU discharge, and 32 patients survived to hospital discharge. The median ICU and hospital lengths of stay were 27 days and 39 days, respectively. Among the attributed causes of death, unspecified hemorrhage, intracranial hemorrhage and intractable respiratory failure occurred most commonly.

Discussion: This quickly prepared and published observational study describes the complete experience of ECMO support for H1N1-related ARDS in Australia and New Zealand. However, the investigators curtailed data collection in order to prepare the northern hemisphere for an impending crisis. A follow-up study describing the long-term outcomes will be much anticipated.

The application of ECMO for severe ARDS due to H1N1 may rejuvenate an orphaned therapy. The early enthusiasm for ECMO support for refractory hypercapnia and hypoxemia in the 1970s was tempered by significantly disappointing results from the NIH-sponsored prospective, randomized trial in 1979.[2] However, the current H1N1 pandemic affecting previously healthy adults and children, and causing isolated respiratory failure, may generate more interest in ECMO therapy. Coincidentally, the much-anticipated CESAR trial from the U.K. was published recently as well, which demonstrated a 16-percent absolute risk reduction in death or severe disability at six months.[3] Although the ANZ ECMO study was only observational in design, all patients examined would have qualified for the CESAR trial, suggesting that ECMO support may prove beneficial for H1N1-related ARDS. Future randomized, controlled studies may provide evidence for an increasing role for ECMO therapy in selective patients with refractory ARDS.

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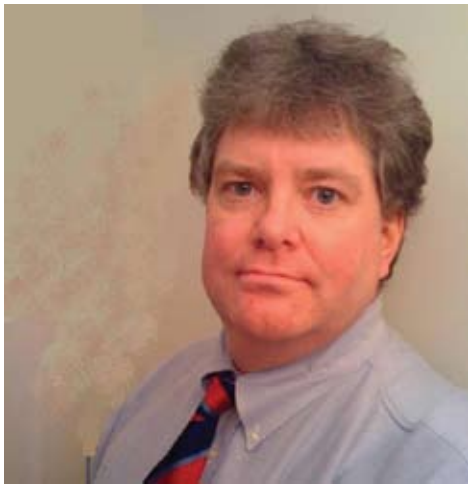
Literature Review II:
Efficacy and Safety of Quetiapine in Critically Ill Patients With Delirium: A Prospective, Multicenter, Randomized, Double-Blind, Placebo-Controlled Pilot Study



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Article:

Devlin JW, Roberts RJ, Fong JJ, Skrobik Y, Riker RR, Hill NS, Robbins T, Garpestad E. *Crit Care Med*. 2010; 38(2):1-9.

Introduction: Delirium is a syndrome of fluctuating mental status, including disordered thinking, hallucinations and disorientation, which is very common among ICU patients. A recent summary editorial by Caplan[2] notes that delirium is found in up to 62 percent of ICU patients and up to 82 percent of mechanically ventilated patients. It contributes to prolonged hospitalization, to increased six-month mortality, to increases in hospitalization costs by a third or more, to increased stress and demands on staff, and it even may predispose to long-term psychiatric morbidity, namely post-traumatic stress disorder.[2] These costs, added up even conservatively, are staggering. A recent poll of critical care workers showed that the scope and magnitude of the problem of ICU delirium is widely recognized, but that objective assessments, sedation-limiting protocols, standardized therapies and compliance with any of these things is suboptimal.[1]

There are no sound data on the best ways of treating ICU delirium, but the standard method is by limiting risks to patients, such as with sedatives, avoiding sleep irregularity among ICU patients and by the use of conventional antipsychotic tranquilizers such haloperidol. Recently, a class of drugs known as atypical antipsychotics have become widely used because they are more effective than conventional antipsychotic medications and have a better safety profile, especially with regard to serious side effects such as QT prolongation, *Toursades de pointes*, and extrapyramidal symptoms. So it is not surprising that the off-label use of the atypical antipsychotic drugs in the setting of ICU delirium would become common, which it has.[3] This paper represents a methodical initial approach to the application of one atypical antipsychotic, quetiapine, to the problem of ICU delirium.

Objectives: The purpose of this study was to assess the efficacy of quetiapine (Seroquel®, AstraZeneca, Wilmington, DE) given in an increasing, scheduled fashion, versus placebo, to a population of ICU patients who were already being treated with as-needed (*prn*) haloperidol for their symptoms of delirium.

Design: The small study was prospective, randomized, double-blind and placebo-controlled and was conducted at three academic medical centers: Maine Medical Center, Portland; Tufts Medical Center, Boston; and Maisonneuve-Rosemont Hospital, affiliated with the University of Montreal.

Methods: Patients were 36 adult ICU patients with delirium but without other complicating neurological diagnoses. They were screened for delirium using a screen developed and validated at the Maisonneuve-Rosemont Hospital called the Intensive Care Delirium Screening Checklist (ICDSC).[4] Delirium was diagnosed by an ICDSC score of 4 or greater. All patients before randomization were tolerating enteral nutrition and were being treated with a first-line agent, haloperidol, on an as-needed (i.e., *prn*) basis. They were randomized into two groups, one group receiving scheduled placebo (n = 18) and the other scheduled quetiapine (n = 18). The quetiapine (or placebo) was given per nasogastric tube as follows: first 50 mg q 12 hours then titrated upward on a daily basis in increments of 50 mg q 12 hours, up to a maximum of 200 mg q 12 hours. All the patients were allowed to continue receiving intravenous haloperidol on a *prn* basis up to 10 mg q 2 hours for symptoms of delirium. The study drug (or placebo) was given until 1) the attending intensivist felt the patient had no longer shown symptoms of delirium; 2) 10 days of therapy had taken place; 3) ICU discharge occurred; or 4) an adverse event potentially attributable to the use of the study drug occurred. The primary outcome was time to first resolution of delirium, denoted by an ICDSC score ≤ 3. Secondary outcomes included total hours spent delirious; total hours spent deeply sedated or agitated; length of mechanical ventilation, ICU stay and hospitalization; hospital mortality; use of haloperidol; episodes of subject-initiated device removal; and disposition of subjects after discharge from the hospital.

Results: Baseline characteristics were similar between the quetiapine and placebo groups. The most common diagnoses for admission to the ICU were severe sepsis/acute respiratory distress syndrome, in 42 percent of the patients.

Continued on page 11

Literature Review II:

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Continued from page 10

Those in the quetiapine group had a significantly shorter time to first resolution of delirium, 1.0 days (interquartile range, 0.5-3.0 days) versus 4.5 days (interquartile range, 2.0-7.0, $p = 0.001$). During the quetiapine administration, all patients had resolution of delirium at least once, but among placebo patients, resolution of delirium occurred once in only 78 percent of patients ($p < 0.05$). Subjects receiving quetiapine spent significantly fewer hours in delirium (36 hours versus 120 hours, $p = 0.006$) and required a shorter duration of study drug therapy. Those receiving quetiapine also spent fewer hours of agitation (6 hours versus 36 hours, $p = 0.02$) than those receiving placebo. The proportion of patients who were "deeply sedated" or who removed devices was the same in both groups.

Duration of mechanical ventilation, ICU and hospital stay were the same in both groups, as was mortality. But patients receiving quetiapine were more likely to be discharged from hospital to either home or a rehabilitation facility as opposed to a chronic care facility (89 versus 56 percent, $p = 0.06$).

In terms of safety, the patients receiving quetiapine had no significantly increased adverse events than did those treated with placebo. No extrapyramidal symptoms were exhibited by any patient. *Toursades de pointes* was not observed in any patient. There was no difference in the fraction of patients who had significant QT prolongation between the quetiapine and placebo groups (39 versus 44 percent, $p = 1.0$).

Discussion: The use of quetiapine in a scheduled fashion as in this small but well-designed study appears to be efficacious at reducing the burden of ICU delirium in this set of patients. Although the basis of ICU delirium is not clear, it is possible that it represents derangements in several nuclei in the CNS and in function of many different classes of neurons. The pharmacology of therapy of ICU delirium may involve more than one class of drug, as it may also involve alterations of more than one class of neurotransmitter. But as monotherapy, or combined with haloperidol, quetiapine deserves a larger clinical study to prove its efficacy.

The struggle against delirium is reminiscent of the struggle against postoperative or post-chemotherapy nausea and vomiting. Although incremental advances have been made in recent years by the use of a combined approach of several classes of antiemetics and reduction of several anesthetic risk factors, ultimately, significant progress has been made in combating nausea after anesthesia and chemotherapy.[5] That doesn't mean that the advent 20 years ago of serotonin antagonists[6] was not also huge progress against nausea. Our thinking about ICU delirium should involve the search for more effective drugs as well as protocols of combining several drugs along with other interventions to reduce the morbidity of delirium. The first drugs to consider are the other atypical antipsychotic drugs, such as risperidone or olanzapine; but, as noted by the authors of this study, their side effect profile is not as favorable as quetiapine's.

Quetiapine is largely antihistaminic in its effect. But we know that the drug physostigmine, a central procholinergic agent, is effective against postoperative or emergence delirium. We also know that haloperidol is antidopaminergic in its action and it is effective against delirium. Should quetiapine be used as a first-line therapy when delirium occurs, rather than haloperidol? Is the addition of a third class of drug, such as physostigmine, conceivable in combating delirium? Should the other atypical antipsychotic drugs (olanzapine and risperidone) be tried is this setting as well, even though their use presents greater risk? This excellent paper is of smaller scope but it leads one to consider these other approaches.

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Literature Review III: Tissue Oxygen Saturation Predicts the Development of Organ Dysfunction During Traumatic Shock Resuscitation

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Article:

Cohn SM, Nathens AB, Moore FA, Rhee P, Puyan JC, Moore EE, Beilman GJ. *J Trauma.* 2007; 62:44-55.

Study objective: The primary objective of the study was to determine if StO2 (near infrared spectroscopy) could reliably identify clinically relevant hypoperfusion and predict the development of multi-organ dysfunction syndrome (MODS). The secondary goal was to assess the utility of StO2 in predicting 28-day mortality in hemorrhagic shock. In addition, the area under the receiver operating characteristic (ROC) curve, sensitivity, specificity, positive and negative predictive value for base deficit (BD) and systolic blood pressure (SBP) were calculated for qualitative comparison to StO2. This was a multicenter, prospective, non-randomized cohort study. Patients were enrolled after admission through the ED within six hours of a high-risk torso trauma and evidence of hemorrhagic shock. (SBP < 90 mmHg or BD ≥ 6mEq/l).

Study protocol: A thenar StO2 sensor was placed on study patients within 30 minutes of ED arrival and data collected for 24 hours or until death. Patients met criteria for MOD syndrome when MOD score was ≥ 6. Blood pressure, base deficit, serum lactate, hemoglobin and vitals were closely monitored.

Methods: StO2 (in spectra spectrometer, Hutchinson Technology, Inc.), permits continuous and non-invasive measurement of capillary oxygen saturation on the thenar muscle. This site has shown to be reliable and practical for clinical use. The physiologic background is based on the notion that "peripheral hypoperfusion" as a result of "blood flow redistribution" (shunting toward vital organs) in hypovolemic shock is a marker of worsening patient condition, and if it persists, leads to MODS. StO2 threshold values were assessed in parallel to routinely used measurements, such as blood pressure and base deficit.

Results: In this study, minimum StO2 and maximum BD performed similarly in the discrimination of MODS patients. Minimum StO2 value of 75 percent collected within first hour of ED arrival is the best cut-off value that provided the best trade off value between sensitivity and specificity. Both measures had a similar negative predictive value of approximately 90 percent. This has an important clinical implication — namely, a patient who is able to maintain StO2 above 75 percent (or BD below 6 mEq/L) has a high probability of not developing MODS. Unfortunately, both measures have a high false-positive rate. Minimum StO2 performed similarly to BD and SBP in its ability to predict the likelihood of MODS and death.

In summary, based on the data presented in this study, StO2 seems to be a sensitive but less specific measure of poor clinical outcomes seen in hypoperfusion states. This is similar to currently used markers such as BD and SBP. The advantage of using StO2 comes from the ability to have a non-invasive and continuous measure for monitoring resuscitation efforts in hemorrhagic shock.

Comments: Enhancing our understanding of the "microcirculation" has been an area of great interest. Mechanisms that are responsible for the development of "vasomotor paralysis" in advanced stages of shock have been extensively studied. A number of different monitors are being developed with a goal to improve patient outcome in shock.

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Case Report: A 12-Month-Old Girl With Partial Airway Obstruction Caused by an Esophageal Coin

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Case Report

A 12-month-old girl presented with drooling and retractions after placing an object in her mouth. We kept her seated in her mother's lap for comfort. She improved significantly while awaiting ENT consultation. After she improved, we felt it was safe to obtain a portable radiograph. The first image (Panel A) shows a round metallic object overlying the cervical esophagus with associated partial left lung collapse. The mother's hand is seen holding the child. The child subsequently vomited a coin, and her remaining symptoms resolved. A follow-up radiograph (Panel B) demonstrates absence of the foreign body and resolution of the partial lung collapse.

Discussion

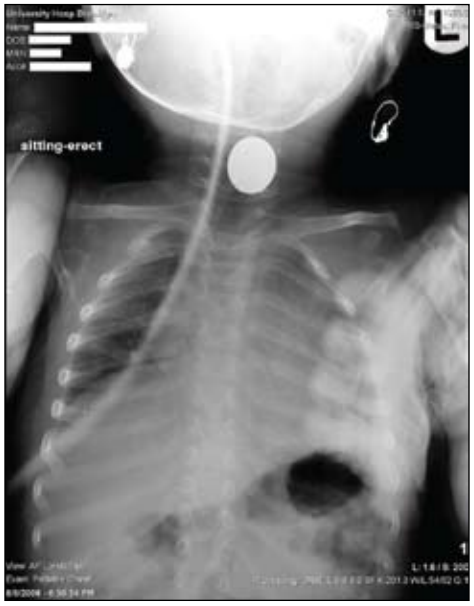
Children most commonly aspirate or swallow foreign bodies between the ages of 1 and 3 years because they can grasp objects, tend to explore their surroundings using oral sensory input and have immature upper-airway reflexes.[1] Symptoms of supraglottic, glottic and subglottic airway foreign bodies can be mild or dramatic. Large upper-airway foreign bodies can cause choking and death, and small foreign bodies that lodge distal to the carina can produce wheezing, atelectasis or pnuemonia. Swallowed foreign bodies can be asymptomatic, cause gastrointestinal obstruction or perforation, or present with respiratory symptoms if the object deforms the trachea from behind. Because infants' and toddlers' tracheal cartilages are less calcified and compress more readily than adult cartilage, children in this age group are more likely to experience respiratory compromise.

Aerodigestive foreign bodies in children often resolve spontaneously. Children may expel them by coughing or vomiting, or they may pass through the gastrointestinal tract without symptoms. As a result, it is impossible to determine the true incidence of these events.[1] One series evaluated 53 children with foreign body ingestions. Three quarters of the children were aged 0-4 years; boys presented three times more often than girls, and about 60 percent of the foreign bodies lodged in the airway. The majority of airway foreign bodies are small food items such as nuts; although a significant number are non-food items. Esophageal foreign bodies are less likely to be food, and most are coins, perhaps because coins tend to lodge in the esophagus at the cricopharyngeal muscle or at C-6.[1]

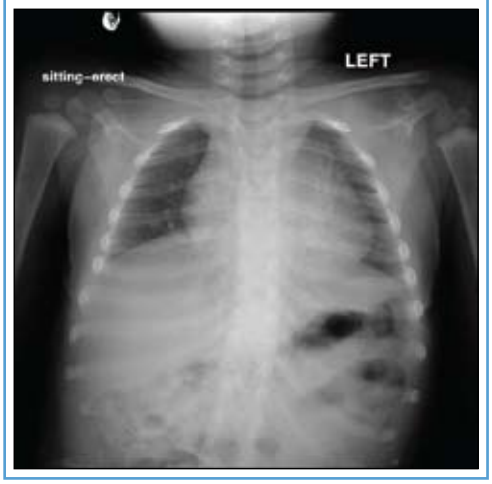
Clinicians can often determine the nature and position of aerodigestive foreign bodies by using plain radiography. For example, the opposition of tongue and soft palate and esophageal musculature tend to orient swallowed coins so that they enter and proceed through the esophagus *en face*. In addition, the esophagus sits in the posterior mediastinum between the spine and anterior mediastinal structures, and the compressed lumen is widest in the coronal plane. As a result, the coins appear as a circular disc on film. In contrast, tracheal coins tend to lie in the sagital position because the C-shaped cartilaginous rings open posteriorly to the distensible membranous trachea. Tracheal coins typically appear as a thick line extending cranio-caudally.[2]

Esophageal coins often pass through the GI tract without causing harm, and removing them may subject the child to iatrogenic injury, so children with ingested coins may be given a trial of observation.[3,4] Ingested disc batteries also tend to lodge *en face* in the esophagus. The risk that they will leak and cause luminal perforation justifies a more aggressive approach.[5] One can sometimes distinguish disc batteries from coins because the raised inner portion of a

Panel A



Panel B



Continued on page 14

Case Report: A 12-Month-Old Girl With Partial Airway Obstruction Caused by an Esophageal Coin

Continued from page 13

battery may be visible on lateral view and a slight haziness of the outer part of the circle may appear on the antero-posterior projection. Because batteries may still be mistaken for coins, the clinician must take a detailed history from the caregiver.

As has been reported elsewhere, esophageal coins occasionally lodge in the sagital plane.[2] The presentation and subsequent clinical and radiological course in this case suggests that the coin initially lodged on edge, causing tracheal compression. The coin then shifted within the esophagus to the *en face* position (Panel A), minimizing pressure on the trachea and mitigating the respiratory distress. Because the child was able to take larger breaths following the coin's shift, she re-expanded her left lung. The radiograph that we obtained immediately after she vomited the coin demonstrates this (Panel B).

In this case, an esophageal coin caused life-threatening symptoms by partially occluding the airway. Surgical control of the infant or toddler airway is extremely difficult, and intervention may worsen a partial obstruction. Non-specialists should intervene as little as possible unless the child develops complete obstruction or impending respiratory arrest. These cases are best managed in the operating room by a team of expert clinicians prepared to perform bronchoscopy and tracheostomy.[6,7]

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Time For Change: Society of Critical Care Anesthesiologists (SOCCA)

Continued from page 1

for the world of the future. To mirror the removal of a special category for international members, it is time to consider removal of "American" from our name. Let's face it, the label limits us to a certain extent; it is not required by our parent, the ASA, the name does not roll off your tongue (no one says I'm going to *"ASKA"),* and folks are always getting the letters confused and mixed up.

I propose to you that we become the Society of Critical Care Anesthesiologists, or SOCCA. This simple change carries with it all the attributes that our present name lacks. It's simple, it indeed rolls off your tongue (even sounds a little like an international sport that is fairly famous in most of the world), and the letters are unlikely to get confused. It represents who we are becoming – an international society of critical care anesthesiologists, and it mirrors the international focus of our journal. Finally, in ASCCA, folks are never quite sure whether we are a society "of" or a society "for" critical care anesthesiologists. This new name clarifies that as well.

In case you are concerned, the name has been discussed with some of the ASA leadership, who were incredibly supportive. The Web domain SOCCAhq.org has been reserved to match the other ASA Web sites. We would keep the ASCCA.org domain name as well, and this would merely redirect folks automatically and transparently. These steps help protect the move to a new name if we choose to finalize this decision.

What's left to do? Only the most important component of this entire process: engage you, the membership, and gain your perspective. I hope to trigger some discourse over this topic between now and our Annual Meeting in San Diego in 2010. At the business meeting on Friday, October 15, 2010, this item will come up for a vote. If passed, a matching set of updated bylaws will need to be introduced for a vote as well.

Please feel free to e-mail me at **tdorman@jhmi.edu** with your thoughts or concerns regarding this proposed change.

Anesthesiology Critical Care Medicine Program at University of Texas Health Science Center San Antonio

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The Anesthesiology Critical Care Medicine Program at the University of Texas Health Science Center San Antonio (UTHSCSA) provides trainees with a broad-based exposure to a primarily adult critically ill population with the goal of developing outstanding clinicians and leaders in the dynamic specialty of critical care medicine. Our 12-month, 13-rotation program meets all certification requirements of the American Board of Anesthesiology, and it is accredited by the Accreditation Council for Graduate Medical Education (ACGME).

Diverse Clinical Rotations:

Although the program is based at UTHSCSA, fellows experience rotations at multiple centers within San Antonio, including the adjoining University Hospital (UH), the adjacent Audie L. Murphy Memorial Veteran's Hospital, the San Antonio Military Medical Center (SAMMC) at Fort Sam Houston, and Christus Santa Rosa Children's Hospital. The clinical program provides experience in the care of surgical, trauma, burn, cardiothoracic, pediatric, and medical intensive care unit patients. Both SAMMC and UH are level one trauma centers and large referral hospitals. SAMMC is home to the regional burn referral center, where the fellows will rotate for a period of four weeks. The VA hospital offers exposure to a high-acuity patient population and a significant number of cardiothoracic surgical patients. A rotation in the medical intensive care unit at the VA hospital provides exposure to pathophysiology distinct to that typically seen in the surgical patient population, with teaching from outstanding pulmonary critical care medicine specialists. Each setting is routinely involved in the education of residents and critical care fellows from different specialties, providing multidisciplinary environments of care. An elective experience in transesophageal echocardiography (TEE) is also available. Participation in at least one quality-improvement project highlights the program's emphasis on evidence-based practice and health care improvement.

Multidisciplinary Integration:

A foundational element of our program is its integration into the Trauma Institute of San Antonio (TRISAT) Educational Consortium, which is composed of four adult critical care fellowship programs **www.surgery.uthscsa.edu/trisat/education.asp**. The Consortium develops and administers components of the integrated schedules and curricula across the four critical care fellowship programs. Affiliation with this outstanding organization provides a broad and diverse clinical experience and access to teaching faculty from multiple disciplines who are all keenly focused on education. The consortium also provides an environment to participate in and to perform research efforts.

Solid Didactics:

At the core of the program is a solid didactic schedule composed of a structured, twice-per-week conference series, which is broadcasted to SAMMC and UH, a defined reading compendium, journal clubs, and formal instruction in research and appraisal of the literature. Fellows are asked to prepare occasional lectures for the teleconference series. Fellows also participate in daily ICU team lectures. As an adjunct to program didactics, the fellows are afforded time and resources to attend the Society of Critical Care Medicine's Annual Congress.

Opportunities for Research:

The research portion of the fellowship involves dedicated research didactics, and it encourages the fellow to complete a project worthy of peer-reviewed journal submission. Integral to the curriculum, fellows complete a two-week course titled "Introduction to Clinical Investigation," which is presented by the Institute for the Integration of Medicine and Science/Clinical-Translational Research Education Office at UTHSCSA. This course introduces fellows to basic principles in study design, research methods, biostatistical techniques, the ethical principles underlying the conduct of research, and granstmanship pertinent to clinical and translational science research.

In summary, the Anesthesiology Critical Care Medicine Fellowship at UTHSCSA offers an outstanding opportunity for fellows to train and excel within this exciting and growing specialty. Further, San Antonio is a wonderful city in which to live, work and raise a family. I strongly encourage interested physicians to inquire about our exciting program.

For additional information, please contact the fellowship Program Director, Steven Venticinque, M.D. **venticinque@uthscsa.edu** or our Program Coordinator, Candy Beckingham **beckinghamr@uthscsa.edu**. We can also be reached by telephone at (210) 567-6137.

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American Society of Critical Care Anesthesiologists

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