NEWSLETTER

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AMERICAN SOCIETY OF CRITICAL CARE ANESTHESIOLOGISTS

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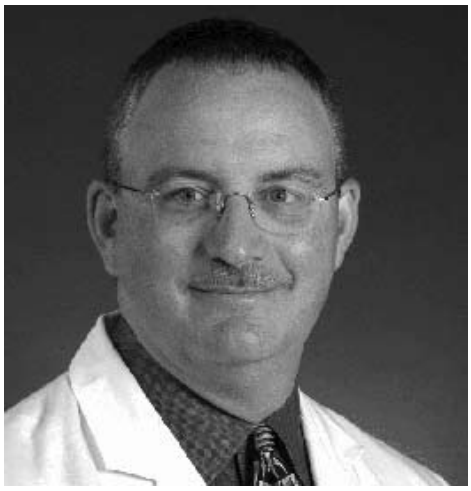
President's Message

# Advancing Science to Advance Care



Todd Dorman, M.D., F.C.C.M.
ASCCA President

Those simple words in the title of this piece capture a significant aspect of our mission as a professional society. We strive to accomplish this lofty goal through our annual meeting, its presentations, abstracts, poster sessions and awards. Our commitment to fellowship training is evidenced not only by a long-standing focus on specialty education, but also supported by the recent creation of a new Fellowship Committee. We have partnered with the Foundation for Anesthesia Education and Research to offer the combined research grant that has been so generously funded by Hospira; and to date, five recipients have benefited from this award. Thus it is with great pleasure that I have the privilege of announcing what we hope will be a new award. As I write this message, we are in the final days of negotiation with an anonymous donor. By the time this edition of the *Interchange* is actually published, it is highly likely that we will have signed the agreement.

This award is for innovation in devices. The idea is to promote the advancement of medical technologies, stimulate innovative ideas that solve clinical problems and enhance safe and effective uses, cultivate next-generation medical innovators and leadership, encourage and foster translational research (bench, applied and clinical), and facilitate academic promotion of new anesthesiologists/critical care intensivists.

> "The Society believes that true innovation can result from a device idea that has an immediate impact on health care delivery and introduces a new device or procedural technique, changes clinical practice or could demonstrate improvement in patient outcomes."

The new award, the Innovator Award, will be given on an annual basis to a medical resident, fellow or clinical faculty member at an accredited North American training program who has a device idea addressing the practice of anesthesiology or critical care medicine that qualifies as a "true innovation." The Society believes that true innovation can result from a device idea that has an immediate impact on health care delivery and introduces a new device or procedural technique, changes clinical practice or could demonstrate improvement in patient outcomes.

The award recipient will receive $10,000 toward development of the innovative idea. A formal grant mechanism with an awards committee will be established. Once the infrastructure is in place, we will announce an RFP to our membership. Grant submissions will be accepted by the deadline established in the RFP. We hope to award the first recipient of this new Innovator Award at the 2011 ASCCA Annual Meeting (well, if things go well at this upcoming annual meeting, it will be the 2011 "SOCCA" Annual Meeting), which is scheduled to be held on October 14, 2011 in Chicago.

This award truly represents another step in this Society's commitment to advancing science to advance care.

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# EDITORIAL NOTES

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# **PRO:** ARDSnet Low Tidal Volume Ventilation – The Safe Initial Default Mode for 'ALL' Patients

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The NIH-sponsored ARDS-net trial, conducted at 10 major centers between 1996 and 1999, enrolled 861 patients and showed that low tidal volume ventilation (6 cc/kg predicted body weight compared with 12cc/kg) reduced mortality in patients with acute respiratory distress syndrome (ARDS) and acute lung injury (ALI) from 40 percent to 31 percent.[1] Ten years later, the question for many intensivists is no longer whether the ARDSnet protocol should be applied to patients with ARDS and ALI, but whether the protocol should be applied to almost all critically ill patients requiring mechanical ventilation.[2,3]

The physiologic argument for "default mode" ARDSnet ventilation ("low Vt") is that positive pressure ventilation, when coupled with critical illness, causes or aggravates pulmonary injury (ventilator-associated lung injury, or VILI) and that low Vt mitigates this. Mechanical ventilation can lead to VILI by causing volutrauma, barotrauma and atelctrauma (the shear stress caused as alveoli open and close during the respiratory cycle).[4] A fourth process, chemotrauma, or inflammatory cytokine synthesis, results from the others and leads to edema, parenchymal immunocyte infiltration, and ultimately, fibrosis.[3]

Volutrauma and barotrauma independently cause harm, and there is debate about the relative importance of each.[4,5] Separating them for analysis is difficult in humans[6] because increased transpulmonary pressure causes increased alveolar distension[4] and because higher set volumes raise plateau pressures.[6,7] Clinically, their interdependence means that reducing tidal volume also decreases inspiratory airway pressures.[7]

Although low Vt addresses atelectrauma through graduated positive end-expiratory pressure (PEEP) settings,[1] two prospective, randomized studies found no mortality difference when low Vt was employed with either relatively lower or higher PEEP (e.g., 8-10 versus 12-15 cm/$H_2O$).[8,9] A recent metanalysis found that higher PEEP settings were beneficial for patients who met ARDS criteria, but not for those with ALI.[10] The current ARDSnet protocol does not mandate use of the PEEP/$FiO_2$ pairings published in the original study.[11] Although data now at hand suggest that PEEP is important for oxygenation[9] and lung protection, specific levels probably matter less than does restricting tidal volume.

The empiric case for making low Vt the default mode for almost all patients includes one recent prospective, randomized trial[2] and multiple observations that incidence of VILI – including ALI, ARDS and frank radiographic barotrauma – declines as clinicians adopt low Vt.[3,7,12-14] Dettermen et al. randomized patients without ALI/ARDS to 6 versus 10cc/kilo and found that patients in the high Vt group were more likely to develop lung injury.[2] This trial cannot stand alone in support of prophylactic low Vt: the strongest predictor of ALI/ARDS was not tidal volume, but initial bronchalveolar lavage IL-6 levels, the ventilation protocol differed significantly from the ARDSnet protocol, and there was no difference in the primary study outcome -- cytokine differences between 6 and 10cc/kg Vt.

In a retrospective, multivariate regression model derived from 3,261 patients without baseline ARDS, Gajic et al. found that Vt > 700cc was the variable most strongly associated with subsequent development of ARDS (OR 2.6).[12] In a later study, Gajic and a different group of collaborators found that the odds ratio for developing ALI was 1.3 for each additional milliliter of Vt above 6 cc/kg predicted body weight.[13] The investigators observed that there was a trend toward women developing ALI more often then men (29 versus 20 percent, $p = 0.07$). Women received higher tidal volumes, an observation that adds explanatory power to the link between higher Vt and ALI – higher Vt in women may be rooted in clinicians overestimating women's lung volumes, as opposed to the confounding clinical events that often undermine retrospective findings.

One cannot responsibly extend ARDSnet ventilation from its best-studied application in ALI/ARDS without addressing its critics. Clinicians, ethicists and others[15] have expressed concern that the original trial was ethically and methodologically[5] flawed. Eichacker et al. argued in 2002[5] that 12 cc/kg Vt and other ARDSnet control group limits (e.g., $P_{plat}$ 50cc $H_2O$) exceeded the then-current standard of care. This argument is based on the pre-randomization standard tidal volume of 10cc/kg for the ARDSnet participants, surveys and control volumes used in other low Vt studies. From this follows that as control group Vt was inappropriately raised, $P_{plat}$ increased, and this increase worsened control group outcomes such that 6cc/kg appeared to be beneficial. Eichacker et al. note that three trials published within two years of ARDSnet comparing 7 to 10cc/kg found no mortality difference.

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Because the ARDSnet investigators' research hypothesis was that low Vt reduces mortality,[1] it's reasonable to posit that the study design was ethically flawed. A fair question to ask the ARDSnet researchers is whether they would accept for themselves or their loved ones, as patients with ALI or ARDS between 1996 and 1999, that their tidal volumes be raised from 10 to 12cc/kg and their $P_{plat}$ be allowed to rise to 50cm$H_2O$. If the answer to this question is not unequivocally affirmative, the trial would fail the "golden rule" test.[16]

> "One cannot responsibly extend ARDSnet ventilation from its best-studied application in ALI/ARDS without addressing its critics. Clinicians, ethicists and others[15] have expressed concern that the original trial was ethically and methodologically[5] flawed."

If we grant that valid ethical concerns do not rise to a level sufficient to disregard ARDSnet, what is left is a positive study that numerically swamps the three negative ones. In addition:

1. ARDSnet control group mortality was approximately equal to or lower than the roughly contemporaneous 7 versus 10cc/kg studies.
2. Treatment group mortality in ARDSnet was lower than in any other low Vt study.[5]

Although differences in tidal volume targets and ventilation-body weight calculation complicate inter-trial comparisons, these findings seriously weaken the argument by Eichbacker et al. that the mortality benefit of low Vt emerged only in comparison to an overdistended control group.

The physiologic argument by Eichbacker et al. that $P_{plat}$ and not low tidal volumes drove the mortality difference between groups fails to undermine the ARDSnet conclusions because Vt and $P_{plat}$ are closely coupled in multiple studies. In a re-analysis of the ARDSnet data, Hager et al. found no lower limit for safer $P_{plat}$ and no $P_{plat}$ level at which an ARDSnet control patient would not have benefited from receiving lower tidal volumes.[6]

Concerns that low Vt leads to air hunger, asynchrony and agitation are challenged by findings that low Vt patients did not require additional sedation compared to control patients at two centers during the trial.[17,18] A study that described a significant incidence of breath stacking during low Vt found that stacked breaths were not significantly associated with sedation score or sedation interruptions, that $P_{plat}$ remained within ARDSnet protocol limits on 86 percent of measurements (mean 24-25 cm$H_2O$), and that asynchronous stacked breaths could be reduced by 60 percent for each cc/kg tidal volume increase above 5.9. As a result, the investigators were able to keep Vt at baseline or within the low Vt protocol-specified volume step-ups of 1-2 cc/kg during 66 percent of recorded measurements.[1, 9,11]

Another challenge to "default" low VT is made by proponents of airway pressure release ventilation (APRV). Habashi claims to have acheived ARDS mortality outcomes comparable to or better than low Vt.[20] He argues that APRV has physiologic advantages compared to other positive pressure modes that benefit a range of critically ill patients with respiratory failure. The long $P_{high}$ phase is intended to recruit atelectatic and long inspiratory time constant lung units more effectively than other modalities. This re-establishes physiologic FRC, which in turn decreases shunt and atelectrauma. Critically, restoring FRC also increases the mechanical advantage of the diaphragm so that efficient spontaneous breathing can occur. With this comes decreased sedation requirements and improved systemic perfusion that occurs because of negative relative pressure fluctuations around $P_{high}$. Improved perfusion in turn decreases the incidence of organ failure that is often fatal to ARDS patients. The argument for APRV in ARDS/ALI and other causes of respiratory failure is compelling, but no large multicenter, prospective trials compare it to low Vt. As a result, we don't know how APRV would perform outside of centers that have developed the most expertise with the modality.

Compared to other ventilatory strategies, low Vt is unique in having support from several prospective and multiple retrospective studies in a variety of centers over many years.[1-3,7,12-14] It has been tested as a treatment for ARDS and as a prophylactic measure against VILI.[1,2] Taken as a whole, the data suggest that low Vt is less likely to cause harm to a range of patients than modalities in widespread use before it. Because the goal of mechanical ventilation is to support patients in respiratory failure while minimizing harm, the question about low Vt cannot be "who should get it," but "who should not."

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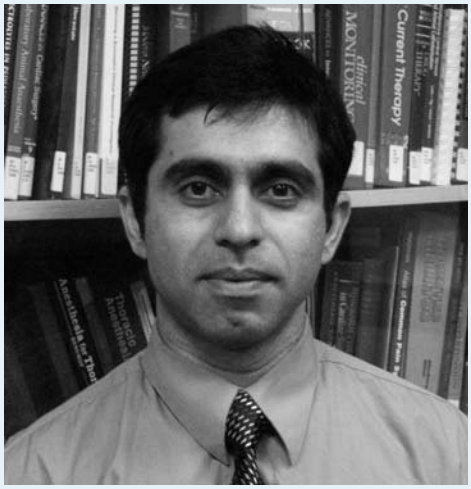
# **CON:** Application of the ARDSnet Protocol for Ventilator Management of Patients With Normal Lung



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The ARDSnet trial showed that in patients with ARDS, mechanical ventilation using tidal volume of 6 ml/kg was associated with decreased mortality in comparison to ventilation using tidal volume of 12 ml/kg.[1] One criticism of this trial was that the tidal volumes in the control group were much higher than what would be routinely used in these patients based on airway pressures. Critics stated that clinicians would have decreased tidal volumes in ARDS patients to avoid plateau pressures that were generated in patients in the control group (34-37 cm $H_2O$); these are high enough to have caused barotrauma and increased mortality.[2]

A metanalysis showed that based on three other studies prior to the ARDSnet trial,[3-5] low tidal volume (7.2 ml/kg) was not associated with improved outcome in comparison to high tidal volume (10.2 ml/kg) as long as plateau airway pressure was kept at 28-32 $cmH_2O$ in the control group.[2] Indeed, the low tidal volume group showed a trend toward increased mortality even though the difference did not reach statistical significance. Hence the question arises whether low tidal volumes in the ARDSnet trial decreased mortality or high tidal volume and airway pressure in the control group increased mortality, due to ventilator-associated barotrauma. As seen in the ARDS net trial, adjustments in tidal volumes were not done in the control group until the airway pressures were greater than 50 cm $H_2O$. In fact, a survey showed increased mortality rate with both low and high tidal volumes.[6] It might have been more informative if the ARDS net trial included an intermediate volume group of 8-9 ml/kg with separation of volume and pressure parameters to better understand the role of tidal volume in mortality.

Disadvantages of using low tidal volume (6 ml/kg) in mechanically ventilated patients include:

1. A sensation of dyspnea (air hunger) leading to tachypnea and asynchrony with the ventilator, which might lead to increased use of sedatives and neuromuscular blockers.
2. Higher carbon dioxide ($CO_2$) levels, which may lead to increased sympathetic discharge causing pulmonary vasoconstriction, arrhythmias and cerebral vasodilation leading to increased intracranial pressure. This is particularly harmful in patients with heart disease and increased intracranial pressure (ICP).
3. Atelectasis, which can worsen gas exchange and increase risk of infection.
4. Low tidal volume might require compensation with high respiratory rate leading to stacking of breaths. The resulting air entrapment leads to auto-PEEP, which may lead to barotrauma.

"A metanalysis showed that based on three other studies prior to the ARDSnet trial,[3-5] low tidal volume (7.2 ml/kg) was not associated with improved outcome in comparison to high tidal volume (10.2 ml/kg) as long as plateau airway pressure was kept at 28-32 $cmH_2O$ in the control group.[2]"

There is insufficient evidence to support the use of the low tidal volume (6 ml/kg) protocol in patients with normal lungs. The most important factor in managing mechanically ventilated patients is limiting the airway pressure. Both low and high tidal volumes may be associated with worse outcomes compared with intermediate tidal volumes.[6] Animal studies in which there were increases and decreases in tidal volume have also correlated with worsening of lung function.[7] There is more evidence in support of intermediate tidal volumes of 8 ml/kg, which is more physiologic.[8] There is sufficient evidence that the most important factor in limiting iatrogenic lung injury during mechanical ventilation is limiting plateau airway pressure to 35 $cmH_2O$.[4] Advantages of intermediate tidal volumes (8 ml/kg) include avoiding high airway pressure that could be associated with high tidal volumes (12 ml/kg). A recent study in pigs showed no benefit on the systemic and pulmonary inflammatory response when using low tidal volume (6 ml/kg) ventilation. The use of low tidal volume (6 ml/kg) and high PEEP of 10 cm $H_2O$ showed increased production of inflammatory markers.[9]

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# **PRO:** The PAC Should Be Abandoned for Hemodynamic Monitoring in Septic Shock!

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Central venous oxygen saturation ($ScvO_2$) monitoring requires only a central venous catheter (usually placed in the superior vena cava) and should therefore be less invasive than mixed venous oxygen saturation ($SvO_2$) monitoring. However, is $SvcO_2$ as sensitive and reliable in monitoring efficiency of oxygen utilization as mixed venous oxygen saturation?

The absolute values of $ScvO_2$ and SvO2 appear to vary only slightly in critically ill patients and, in fact, this has been verified in a wide variety of clinical circumstances — with correlation coefficients closely matched (r=0.97).[1,3,6] Even in patients with neurological conditions, continuous measurements of $ScvO_2$ and $SvO_2$ suggested an acceptable correlation (r=0.755).[2] As long as absolute values are not needed for the clinical decision-making, $ScvO_2$ is equivalent to $SvO_2$ and trends are interchangeable.[3,6] Guidelines for management of severe sepsis and septic shock indicated that both $SvO_2$ and $ScvO_2$ are acceptable with $ScvO_2$ above 70 percent as therapy target.[4] The recommendation is based on a study from Rivers showing that patients with severe sepsis or septic shock have a higher change of survival if $ScvO_2$ is above 70 percent in addition to central venous pressure (CVP) 8-12mmHg and maintained urine output.[5]

In contrast to $SvO_2$, $ScvO_2$ is easier to obtain and does not expose the critically ill patient to additional invasive techniques. Patients with advanced sepsis will require placement of central venous catheter for CVP monitoring and vasoactive medications anyway, so using a central venous catheter with oximetry instead of a conventional triple lumen catheter will not expose the patient to additional risks. Since some complications of the pulmonary artery catheter (PAC) are related to indwelling time, physicians are advised to remove PACs as soon as clinically possible – typically earlier than for the central venous access. Therefore, using $ScvO_2$ might allow for a longer monitoring of venous saturation than using $SvO_2$ per PAC accompanied by a potentially decreased risk of catheter-associated bloodstream infection.

The ultimate goal for monitoring venous saturation is to assess the balance between oxygen delivery and oxygen consumption. $ScvO_2$ monitoring allows implementation and adjustments of an appropriate treatment plan in septic patients to avoid tissue hypoxia.

"Since some complications of the pulmonary artery catheter (PAC) are related to indwelling time, physicians are advised to remove PACs as soon as clinically possible – typically earlier than for the central venous access."

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# **CON:** The PAC Should NOT Be Abandoned for Hemodynamic Monitoring in Septic Shock!

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Septic shock is a condition that requires, among many things, a rapid restoration of appropriate systemic hemodynamics to prevent end-organ injury. To date, several algorithms for resuscitation have been described, validated and incorporated into multispecialty guidelines.[1] Unfortunately, these algorithms and protocols cannot take into account individual patient variability, and the appropriate use of these tools is not always possible given specific patient populations.

The use of central venous catheters over the more invasive pulmonary artery catheter has gained significant recent attention due to the use of central venous oxygen saturation ($ScvO_2$) monitoring which has been shown (in selected, but not all, populations) to be a surrogate for mixed venous saturation and is therefore widely cited as an acceptable (and even safer) alternative to the mixed venous saturation. In fact, in most septic shock resuscitation algorithms, it is now one of the three targeted hemodynamic end-points, which typically include the mean arterial pressure, the central venous pressure and the $ScvO_2$. In the majority of these protocols, $ScvO_2$ is used as a diagnostic tool for diagnosing reduced tissue perfusion and as an endpoint for goal-directed therapy. In this way, "normal" $ScvO_2$ is targeted by providing specific therapies, including additional fluid resuscitation, red blood cell transfusion or inotropic support, as appropriate. For this reason, many people argue that the pulmonary artery catheter can be abandoned, in favor of a central venous catheter alone, in patients with septic shock.

There are specific limitations, however, to the use of $ScvO_2$ monitoring in these patients, including an unreliable correlation with mixed venous oxygen saturation ($SvO_2$) and an incomplete understanding of the underlying cardiovascular abnormalities that may be reflected in the decreased oxygen delivery defect. In fact, recent data suggest that $ScvO_2$ and $SvO_2$ have poor correlation at both "high" and "low" values and that the use of $ScvO_2$ alone may underestimate the oxygen delivery balance when used for diagnosis and overestimate the oxygen delivery balance when targeting for therapy — neither condition would be optimal![2] It should also be noted that a few specific patient populations are routinely excluded from studies evaluating the use of $ScvO_2$ in septic shock resuscitation and that there are specific populations where $ScvO_2$ has been shown to be of decreased utility.[3,4,5]

The use of a central venous catheter to monitor $ScvO_2$ may be preferable over the pulmonary artery catheter, particularly in some patient populations, and part of a treatment protocol that has been designed to continuously re-evaluate the effectiveness of each individual therapy and to use other diagnostic modes and tests as appropriate. However, clinicians should always consider the use of a pulmonary artery catheter, perhaps not to direct fluid management but to use true mixed venous oxygen saturation monitoring for a better (more reliable) understanding of the inherent pathophysiologic consequences of their disease states. For this reason, I think, we should *never* fully abandon the use of the pulmonary artery catheter in septic shock!!

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# **Literature Review I:** Just Say No… to Continuous Sedation

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Strom T, Martinussen T, Toft P. A protocol of no sedation for critically ill patients receiving mechanical ventilation : a randomised trial. *Lancet*. 2010; published online January 29. DOI: 10.1016/S0140-6736(09)62072-9.

This single-center study describes dangers of sedating the critically ill. While the semantics of the title garner objection ("less" rather than "no"), the protocol is inspiring, the outcomes possibly alarming.

The paper begins with references to a continuum of sedation studies. If less sedation is good, none must be ideal. We find that the Danes have been "going green" with ICU sedatives for over 10 years. This means their "standard treatment of no sedation" predates the publication of Kress' "Daily interruption of sedative infusion…"

**The place:** A mixed med/surg ICU, 18 beds and 18 nurses. In addition to 1:1 staffing, "if needed, a person was assigned to verbally comfort and reassure the patient. Physical restraints were never used." At this point, I wonder if nurses in Denmark have experience in hand-to-hand combat.

Of 428 patients assessed, 288 were excluded. Exclusion criteria include patients who were too healthy (expected extubation within 24h, fulfilled weaning criteria) or too sick (moribund or treatment limited, status epilepticus, death within 48 hours of starting the protocol). Fifty eight patients (13.5 percent) were excluded because they (or their proxy) refused. Would 85 percent of Americans agree to randomize themselves to no sedation?

"Fifty eight patients (13.5 percent) were excluded because they (or their proxy) refused. Would 85 percent of Americans agree to randomize themselves to no sedation? "

One hundred forty patients were randomized to intervention ("no sedation" protocol) or control (continuous propofol or midazolam, intermittent morphine with daily interruption). The "no sedation" protocol (intervention group) included intermittent morphine. Agitation was addressed with the following sequence:

1. M.D. evaluation for readily reversible cases of agitation (ETT obstruction)
2. Additional staffing for verbal reassurance
3. Haloperidol if delirious
4. Interruption of "no sedation" with continuous propofol for 6 hours
5. Redo sequence until third failure, then proceed with control group sedation protocol.

Intervention patients who endured until they received control protocol were analyzed as intervention patients. That is, crossover was not allowed statistically. That having been said, 10 patients (18 percent) in the intervention group received the control protocol. The number of patients who received propofol "naps" but did not endure to the control protocol is not listed. One intervention patient received the control protocol at the behest of family.

The control protocol was continuous infusion of propofol for 48 hours, followed by midazolam by continuous infusion. Daily interruptions of sedation were concluded when patients awoke, as determined by RN and M.D. Investigators resolved dissenting opinions. The study does not list how often this occurred.

Despite mechanical ventilation, both groups were mobilized daily. Once a control patient improved to FiO2 40 percent on PEEP 5, continuous sedatives were discontinued.

The primary outcome measure was days without mechanical ventilation in a 28-day period. Twenty five percent of patients remained in the ICU over 28 days. Reintubation within 24 hours of extubation counted as a day of ventilation. Other outcome measures included ICU/hospital stay, ICU/hospital mortality, need for brain imaging, accidental extubations, VAP and delirium (per DSM IV criteria).

## Results

"No sedation" was associated with more days without ventilation (13 vs. 9), shorter ICU stay (13 vs. 22 days), shorter hospital stays (34 vs. 58 days) and less mortality in the ICU (22 percent vs. 38 percent). Both groups consisted of patients with tracheostomy (29 percent). Except for delirium, complication rates were comparable between groups, including VAP (11 percent vs. 12 percent), accidental extubations, need for brain scans and reintubation rate. Delirium was higher in the "No sedation" group (20 percent vs. 7 percent).

Table 1 on the next page is adapted to represent familiar doses of the medications using

the average patient weight of 80 kg. "Comforting persons" were utilized in 20 percent of "no sedation" patients and 5 percent of control patients.

## Discussion

The reduction in ventilator days, ICU days, hospital days and ICU mortality is unnerving. The large percent of patients with prolonged ICU stays (>28 days) and tracheostomy may amplify this signal. Causes of death were not listed.

The drug doses, while statistically significant, do not implicate overdose as a source of the mortality or length of stay differences. Propofol of 12 mcg/kg/min hardly allows a patient to tolerate peripheral I.V. placement, but perhaps it is keeping that patient in-house for an extra three weeks. The implication that intermittent haloperidol and morphine is superior to propofol/morphine or midazolam/morphine is novel. Would you trade propofol infusion syndrome for tardive dyskinesia?

The lack of complications, such as unplanned extubations, is impressive. Mitigating factors include 1:1 staffing, reserve staffing with a comforting person, and the significant incidence of tracheostomy.

The increased use of haloperidol in the "No sedation" group may be explained with a need for alternative sedation or that sedation masks delirium in the ventilated patient. Similar to the use of propofol and midazolam, the haloperidol doses appear low. The writers acknowledge that hypoactive delirium was likely underdiagnosed by study criteria. They are planning a one-year follow-up to assess chronic psychiatric effects of "No sedation" during critical illness.

Strom et al. theorize that their daily mobilization of intubated/ventilated patients may have contributed to comparable incidence of VAP in each group. They acknowledge that their staffing capabilities are unique, including their ability to deploy "comforters" prn. In addition to their one-year follow-up, the authors look forward to a multicenter trial.

## Commentary

The protocol is daring. In an era of drug shortages and cost limitations, the confluence of outcome benefit to withholding a medication is rare and poignant. Could a multicenter trial include American sites? The staffing requirements alone restrict candidacy. Daily mobilization of the ventilated patient is an exciting arena. From a practitioner perspective, deferring sedatives for verbal reassurance would be a challenge, but not as much of a challenge as the consent/IRB process for patients or their families.

## Table 1

| Drug | "No sedation" | Sedation/Interrupted |
|---|---|---|
| Propofol (mcg/k/m)<br>(max dose during 48h) | 0 (0-8.6) | 12.8 (2.6-27.5) |
| Midazolam (mg/h) | 0 | 0.3 (0-1.9) |
| Morphine (mg/h of ventilation) | 0.4 (0.1-0.9) | 0.4 (0.2-0.5) |
| Haloperidol (mg/d of ventilation) | 0 (0-1.2) | 0 |

# **Literature Review II:** Is Ketamine a Reasonable Option for Rapid Sequence Induction in Critically Ill Patients?

Heather D. McFarland, D.O.
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**Article:**

Jabre P, et al. Etomidate versus ketamine for rapid sequence intubation in acutely ill patients: a multicentre randomised controlled trial. *Lancet*. 2009; 374:293-300.

Intubation is often required emergently in critically ill patients. This single-blinded, prospective RCT compared early and 28-day mortality after a single dose of etomidate versus ketamine for RSI. 689 patients were assessed and final analysis included 234 and 235 for etomidate versus ketamine, respectively. Patients were sedated and protocol outlined for paralytic and postintubation sedation. Safety values, SOFA and GCS scores, baseline characteristics and intubation difficulty between the two groups did not differ significantly. There were no statistical differences found when looking at changes in blood pressure, SpO2, 28-day mortality, catecholamine support and ICU-free days. Adrenal axis function was assessed in 232 patients. There were lower baseline cortisol levels, more nonresponders and adrenally insufficient patients in the etomidate group.

As we analyze the two primary endpoints for this study, we can see that the absolute risk reduction in 28-day mortality was 4 percent, the relative risk reduction 11 percent and number needed to treat 25 in the ketamine group. In accordance with the adrenal insufficiency, the ARR, RRR, and NNT were 38 percent, 44 percent, and 3, respectively, in the ketamine group.

In most critically ill patients, ketamine appears to be a safe and effective means for RSI and intubation. There are certain situations where ketamine is not appropriate, as with any anesthesia-inducing drug. The small NNT to reduce adrenal insufficiency by using ketamine seems to be a feasible option. The questions of cost, access and familiarity with ketamine make it harder to recommend across the board for RSI in critically ill patients.

# Fellowship Review:
## Anesthesia Critical Care Fellowship at the University of Maryland

Caron Hong, M.D., M.Sc.
Instructor
Department of Anesthesiology
University of Maryland School of Medicine

The anesthesia critical care fellowship at the University of Maryland is a 12-month, ACGME-accredited, in-depth program. It is established in partnership with the R. Adams Cowley Shock Trauma Center at the University of Maryland and offers a distinctive experience incorporating cutting-edge critical care medical practices with patient care, research and education.

### Clinical Training

At the University of Maryland, the comprehensive training of critical care fellows integrates the trauma intensive care units (ICU), surgical ICU, cardiac surgical ICU, neurosurgical ICU and medical ICU. Under the guidance of strong faculty within these units, fellows master progressive techniques of critical care medicine, including new modes and methods of mechanical ventilation, extracorporeal support of lungs, kidneys and hearts, and management of severe brain injury. Fellows coordinate the full spectrum of patient care in these units and become proficient in the management of critically ill patients.

### Education and Research

The fellowship is a one-year program involving patient care, research and education. Nine months are dedicated to multidisciplinary ICUs. Electives are available in trauma radiology, trauma anesthesiology, trauma infectious diseases, transesophageal echocardiography, pediatric, medical and neurosurgical ICU as well as aspects and experiences in the emergency medical services. All electives are arranged and approved with the fellow's future career goals in mind.

The curriculum includes weekly didactic sessions and case conferences incorporating the core competencies as well as advanced techniques and research in critical care. Fellows participate in monthly journal clubs and morbidity and mortality conferences as well as research seminars. They become proficient in the evaluation of critically ill patients and diagnose and implement management of care for these patients. They also serve as facilitators, leading and educating the team of caregivers for these patients and families.

This program is enhanced by its prospective clinical, translational and basic science research endeavors. Clinical research opportunities are available within each multidisciplinary unit, and laboratory research is available in the department's basic science research laboratories and at the National Institutes of Health (NIH).

### Overview

The anesthesia critical care fellowship at the University of Maryland offers a renowned experience that provides a unique and multidisciplinary approach to critical care medicine. It encompasses a solid basis of knowledge, incorporating education and research experiences which produce academic leaders in this field.

### Contact

For further information about this program, please visit our website **medschool.umaryland.edu/anesthesiology** or contact the director of critical care: Dr. Vadivelu Sivaraman. 22 S. Greene Street, S11C, Baltimore Maryland, 21201. Telephone: (410) 328-6120. E-mail: **vsivaraman@anes.umm.edu.**

# ASCCA 23RD ANNUAL MEETING SCHEDULE
## OCTOBER 15, 2010

| | | |
|---|---|---|
| **6:30 a.m. – 5:30 p.m.** | **Registration** | |
| **7:00 – 7:25 a.m.** | **Continental Breakfast** | |
| **7:25 – 7:30 a.m.** | **Welcome and Introduction**<br>**Laureen L. Hill, M.D.; Andrew L. Rosenberg, M.D.** | |
| **7:30 – 9:00 a.m.** | **SESSION I**<br>**Moderator: Ross Blank, M.D.** | |
| | **7:30 – 8:00 a.m.** | **Obstetrical Critical Care Update**<br>Arvind Palanisamy, M.B.B.S., M.D. |
| | **8:00 – 8:30 a.m.** | **Obesity in the ICU and Post Operative Care for the Bariatric Patient**<br>Charles Weissman , M.D. |
| | **8:30 – 9:00 a.m.** | **ICU Pharmacology: What's New in 2010?**<br>Andrew Patterson, M.D. |
| **9:00 – 9:20 a.m.** | **Break and Exhibits** | |
| **9:20 – 10:50 a.m.** | **CELEBRATING SCIENCE** | |
| | **9:20 - 10:00 a.m.** | **Facilitated Poster Session** |
| | **10:00 – 10:25 a.m.** | **ASCCA-FAER-Hospira Physician Scientist Award Lecture "Molecular Mechanisms of Regional Lung Dysfunction in Ventilator- Associated Lung Injury"**<br>R. Blaine Easley, M.D. |
| | **10:25 – 10:50 a.m.** | **Young Investigator Award and Abstract Presentation**<br>Presenter: Michael F. O'Connor, M.D. |
| **10:50 – 11:00 a.m.** | **Burchardi Award**<br>Recipient: Heidi B. Kummer, M.D., M.P.H.<br>Presenter: Todd Dorman, M.D., FCCM; William E. Hurford, M.D. | |
| **11:00 – 11:30 a.m.** | **Lifetime Achievement Award**<br>Recipient: M. Christine Stock, M.D., FCCP, FCCM<br>Presenter: William Peruzzi, M.D. | |
| **11:30 – 11:40 a.m.** | **Introduction of ASA President-Elect**<br>Todd Dorman, M.D., FCCM | |
| **11:40 am - Noon** | **ASA President-Elect Address**<br>Mark A. Warner, M.D. | |
| **Noon – 1:00 p.m.** | **Lunch and Lecture: "Regional Anesthesia in Austere Environments and Battlefields. Lessons Learned From Iraq and Afghanistan"**<br>Chester C. Buckenmaier, III, M.D. | |
| **1:00 – 2:00 p.m.** | **Ethics Debate: Interactive Session on Donation After Cardiac Death (DCD)**<br>Moderator: Robert N, Sladen, M.D.<br>Nicholas Sadovnikoff, M.D.; Michael F. O'Connor, M.D. | |

| | | |
|---|---|---|
| **2:00 – 3:00 p.m.** | **SESSION II**<br>Moderator: Steven A. Deem, M.D. | |
| | **2:00 – 2:30 p.m.** | **Infectious Disease Update**<br>Sylvia Y. Dolinski, M.D. |
| | **2:30 – 3:00 p.m.** | **Perioperative Care of the Patient for Pulmonary Thromboembolism**<br>William C. Wilson, M.D. |
| **3:00 – 3:30 p.m.** | **Break and Exhibits** | |
| **3:30 – 4:10 p.m.** | **Education and Competencies in the ICU**<br>Neal H. Cohen, M.D., M.P.H., M.S. | |
| **4:10 – 5:15 p.m.** | **Interactive ICU "Rounds" with Junior Faculty**<br>Moderator: Douglas B. Coursin, M.D. | |
| **5:15 – 6:00 p.m.** | **ASCCA Annual Business Meeting** | |
| **6:00 - 7:45 p.m.** | **Wine and Cheese Reception** | |

For complete program information and to register online, visit **www.ASCCA.org**

# ASCCA Breakfast Panel at ANESTHESIOLOGY 2010

"Management of the Septic Patient in the Operating Room: State of the Art"
Tuesday, October 19, 2010, 7:00 – 8:15 a.m.
San Diego Convention Center, Upper 10

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**Objectives:** Upon completion of this learning activity, participants should be able to: 1) Review altered physiology present in sepsis patients; 2) Discuss practical approaches to assessing perfusion pressure and organ function in septic patients; 3) Describe currently recommended management of the septic patient and implications for intraoperative care.

**Clinical Trials in Critical Ill Patients:**
**How Should They Impact Intraoperative Care?**
Daniel R. Brown, M.D., Ph.D., FCCM
Mayo Clinic, Rochester, Minnesota

**Practical Approaches to Assessing Perfusion Pressure and Organ Function in Septic Patients**
Andrea Gabrielli, M.D.
University of Florida, Gainesville, Florida

**Sepsis Pathophysiology and Anesthesia:**
**Why The Septic Patient is Different**
Stephen D. Surgenor, M.D.
Dartmouth Hitchcock Medical Center, Lebanon, New Hampshire

# ASCCA Mentorship Program

Michael S. Avidan, M.B.B.Ch., F.C.A., S.A.
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Washington University School of Medicine
Departments of Anesthesiology & Surgery
Division Chief, CT Anesthesiology &
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The 2009 ASCCA Annual Meeting in New Orleans concluded with a lively and provocative town hall-style meeting involving residents, fellows and a group of critical care anesthesiologists. This meeting was attended by a large number of trainees. In general, there was tremendous enthusiasm among residents about critical care anesthesiology. However, several of the participants felt that they did not have a good sense of the opportunities that were afforded to anesthesiologists who chose to pursue careers in critical care. It was suggested that this should be addressed at the 2010 meeting in San Diego.

At previous ASCCA meetings, the mentorship program has received resoundingly positive feedback. As in previous years, residents will be actively encouraged and sponsored to attend the ASCCA Annual Meeting. Each resident will be paired with a critical care anesthesiologist who will engage him/her in discussion about the opportunities in critical care and the exciting and diverse lifestyle options open to critical care anesthesiologists. With exposure to inspirational mentors, many talented and enthusiastic residents will subspecialize in critical care. This will be to the benefit of patients in general, and to the subspecialty of anesthesiology critical care, as well as its parent specialty, anesthesiology. We encourage ASCCA members and residents interested in critical care to sign up for the mentorship program at this year's annual meeting.

Residents and fellows are also encouraged to submit scientific abstracts for presentation at this year's ASCCA Annual Meeting in San Diego. Any basic science or clinical science topic that is of relevance to critical care medicine will be welcome. Presenting at the ASCCA does not preclude presenting your work at other scientific meetings. As the ASSCA is a focused subspecialty meeting, all attendees will view presentations. You will receive constructive and targeted feedback in a nurturing setting from your mentor and from other experts in intensive care medicine. The ASCCA promotes scientific research and awards grants to talented trainees pursuing research that is relevant to intensive care medicine.

## **PRO:** ARDSnet Low Tidal Volume Ventilation – The Safe Initial Default Mode for "ALL" Patients

*Continued from page 4*


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## **CON:** Application of the ARDSnet Protocol for Ventilator Management of Patients With Normal Lung

*Continued from page 5*

Lung protective ventilation may be useful in ARDS patients who have heterogeneous disease by avoiding the generation of shear forces and breakdown of aerated units. However, this same strategy does not seem suitable and might even be harmful in patients with normal lungs.

Low-tidal volume with ARDS still leaves room for debate; it's an even bigger leap to say almost all patients should get it based on retrospective data and a single weak prospective trial. Subjecting well patients to the known adverse effects of extreme low Vt on the basis of weak data is not justified. This therapy has potential to do harm, so even if these harms are balanced out for the disease state ARDS, that does not mean they will be balanced out if there is no lung disease to be treated. Cytokine studies have been inconsistent, and theory plus retrospective data has historically been a bad recipe for universal recommendations. The low Vt strategy still needs a multicenter trial by disinterested investigators comparing it to mid-range (8 ml/kg) tidal volumes for ARDS. Then it needs one for patients without lung disease. If those are both positive, then it would be reasonable to recommend it as a general "one-size-fits-all" ventilation strategy.

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