Critical Care CME Program

Module 5

CSA's Critical Care CME Program will consist of eight modules. The fifth module appears in this issue of the *Bulletin,* and Modules 6 through 8 will appear in upcoming consecutive issues of the *Bulletin.* The test questions and evaluation for this module are at the end of this article. Submit answers to the ten questions to the CSA office with the registration page to receive the CME credit. Your CME certificate will be mailed from the CSA office. Alternatively, the full text of each module of this CME program will be accessible through the CSA Web Site, **www.csahq.org**, in the Online CME Program section, and as part of the online *CSA Bulletin.* Instructions to complete Module 5 online are given in the Information pages. After completing the assessment, print your CME certificate. Members will need their usernames and passwords to do the modules online.

Important Information about Critical Care Module 5

The following information must be read and acknowledged before proceeding to the rest of the module. Check the acknowledgement box on the registration page.

Faculty/Disclosures

All faculty participating in continuing medical education activities sponsored by the CSA are required to disclose any real or apparent conflict(s) of interest related to the content of their presentation(s) or any of the industry sponsors of the meeting. In addition, speakers must disclose when a product is not labeled for the use under discussion or when a product is still investigational.

Matthew Harris, M.D. (Co-author of Module 5)
CA-3 Resident
University of California, Irvine School of Medicine, Orange, California

Dr. Harris discloses that he owns stock in Masimo Corporation. He has no other relevant financial relationships with any commercial interests.

Maria E.F. Shier, M.D. (Co-author of Module 5)
Health Science Clinical Professor
Department of Anesthesiology and Perioperative Care
Division Director, Resident Education, ICU Director,
Medical Student Education, ICU
Core Course, Department of Anesthesiology and Perioperative Care
University of California, Irvine School of Medicine, Orange, California
Staff Anesthesiologist and Intensivist
VA Long Beach Healthcare System
Long Beach, CA

Dr. Shier has no relevant financial relationships with any commercial interests.

Nitin Shah, M.D. (Editor and Chair of the Critical Care CME Program)
Clinical Professor of Anesthesiology, Director, Neuroanesthesia
University of California, Irvine, Medical Center, Orange, California
Chief, Combined ICU, Long Beach Veterans Healthcare System
Long Beach, California

Dr. Shah has received honoraria from Masimo, Abbott, and Baxter for his role as speaker. He owns stock in Masimo Corporation.

Registration/Instructions

Method of Participation: The physician will read and study the materials and complete a quiz and evaluation of the module. Some modules may have slides available online. To register for and complete this module: Read and study all of the module pages, complete the registration page, go to the test questions that can be found after the article, complete the quiz and the evaluation that follows, submit your quiz to the CSA office by mail or fax (650-345-3269). Your CME certificate will be mailed to you.

Estimated Time to Complete the Module: One hour

Availability

Module 5: Newer Ventilatory Modes and Strategies including Low Tidal Volume for ARDS and non-ARDS Patients

Release Date: March 31, 2009
Expiration Date: March 31, 2012

CME Sponsor/Accreditation

The California Society of Anesthesiologists is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

The California Society of Anesthesiologists Educational Programs Division designates this critical care program for a maximum of 8 *AMA PRA Category 1 Credit(s)*™. The program consists of eight modules with 1 credit per module. Physicians should claim credits commensurate with the extent of their participation in the activity.

Fees

The modules are free to CSA members. Nonmembers pay $30 for each module. Each module is worth one *AMA PRA Category 1 Credit*™.

Target Audience

This program is intended for all licensed physicians, including residents.

Evaluation

An evaluation of each module of this series is offered after the test questions.

Privacy Policy

CSA has a privacy policy that is a general policy for information obtained regarding all online interactive pages, including online CME activities. To review this policy, please go to www.csahq.org/privacy.vp.html.

Acknowledgement

To proceed with this module, please acknowledge that you have read everything on these introductory pages by checking the box on the registration page.

Objectives

Upon completion of this CME activity, participants will be able to:

- Describe factors contributing to ventilator-associated lung injury
- Define criteria for ARDS
- Describe the role of and the reasons behind low tidal volume
- Mechanical ventilation in ARDS and Non-ARDS patients
- Describe the lung recruitment strategies in patients with ARDS and controversies surrounding these strategies
- Describe other new alternative strategies of mechanical ventilation in patients with and without ARDS

Newer Ventilatory Modes and Strategies, including Low Tidal Volume for ARDS and non-ARDS Patients

By Matthew Harris, M.D., and Maria E. F. Shier, M.D.

Matthew Harris, M.D., is a CA-3 anesthesiology resident at the University of California, Irvine Medical Center, Orange, California, and is chief resident for the 2008-2009 year.

Maria E. F. Shier, M.D., trained in Anesthesia at Albany Medical Center, College of Medicine and University of California, Irvine, School of Medicine and Critical Care Medicine at VA Long Beach Healthcare System. She has been practicing Anesthesia and CCM at the University of California, Irvine and the VA Long Beach since 1996, and is currently Health Science Clinical Professor, Department of Anesthesiology and Perioperative Care, Division Director, Resident Education, ICU, Medical Student Education, ICU Core Course. Dr. Shier's main areas of interest include difficult airway management techniques in the OR and ICU, Regional Anesthesia and continuing education at all levels.

Ventilator-Associated Lung Injury (VALI)

There are four major types of ventilator-associated lung injury: barotrauma, volutrauma, atelectrauma, and biotrauma.[1] The mechanism of barotrauma is still largely debated, but the hypothesis is that there is overdistention of lung causing air to gain access to the interstitial tissues, which then tracks along the bronchovesicular sheath toward the mediastinum, causing pneumomediastinum, subcutaneous emphysema, pneumothorax, pneumopericardium, pneumoretroperitoneum, pulmonary interstitial emphysema, and systemic air embolism.[2]

Volutrauma occurs when excessive tidal volume is used during mechanical ventilation. It is postulated that the increased volume causes pulmonary capillary leak leading to edema. Others have postulated that microvascular capillary permeability is modulated by a cellular response to mechanical injury. The main determinant of volutrauma appears to be the end inspiratory volume rather than tidal volume or functional residual capacity (FRC). Overdistention lung injury is also associated with a variety of pathophysiologic abnormalities, including an increase in endothelial permeability, rupture of the alveolar-capillary barrier, and exacerbation of the local inflammatory cascade. Given the heterogeneity of the infiltrates found in radiographic studies, it is thought that certain lung regions may be more sensitive, such as those in the anterior thorax and those areas bordering areas of alveolar collapse.[2,3,4]

Atelectrauma occurs when there is ventilation at low lung volumes that is related to opening and closing to lung units. This injury pattern is attributed to the excessive shear force applied to lung tissue immediately adjacent to the atelectatic regions during tidal ventilation. Although there is experimental data to support this form of VALI, there is no definitive evidence that altering ventilatory strategy to reduce atelectasis improved clinical outcomes.[2]

Biotrauma occurs from mechanical factors exerted on lung, leading to injury that is cell and inflammatory mediator based. There is upregulation of the inflammatory response in injured and overdistended lung. This has been demonstrated in normal lung that has been exposed to high volume/pressure ventilation resulting in high concentrations of neutrophils in the alveolar lavage. These inflammatory mediators may enter the systemic circulation leading to multi-organ system failure, as frequently seen in ARDS patients.[2,3,5]

The Acute Respiratory Distress Syndrome (ARDS): Background and Definition

The acute respiratory distress syndrome is a common and severe lung disease characterized by inflammation of lung parenchyma leading to impaired gas exchange and, frequently, multiple organ system failure. The long term effects can be devastating, and the disease itself carries a mortality rate of approximately 40 percent to 50 percent. The first description of ARDS appeared in 1967 when Ashbaugh described 12 patients with acute respiratory distress, cyanosis unresponsive to oxygen therapy, decreased lung compliance, and diffuse infiltrates on CXR.[6] Because his original definition lacked specific criteria for inclusion of patients in ARDS, controversy arose over incidence and natural course of the disease.[5]

In 1988, there was an expanded definition proposed that used a four-point lung injury scoring system based on the level of positive end expiratory pressure (PEEP), ratio of partial pressure of arterial oxygen to fraction of inspired oxygen, static lung compliance and the degree of infiltration on CXR. This lung scoring system was useful in quantifying severity of disease after 72 hours.[7] In 1994, a new definition was recommended by the American-European Consensus Conference Committee. This new definition separates those with acute lung injury and those with ARDS defined by a ratio of arterial partial pressure of oxygen to fraction of inspired oxygen (P:F ratio). Those with P:F ratios of 200-300 were categorized as having acute lung injury and those with P:F ratios lower than 200 were categorized as having ARDS. There must also be clinical features of acute onset, bilateral infiltrates on CXR, and pulmonary artery wedge pressure less than 18 mm Hg or absence of clinical evidence of left atrial hypertension. The mainstay of treatment for ARDS has been mechanical ventilation in an intensive care unit, treatment of the underlying cause of ARDS, appropriate supportive care, and antibiotic treatment when appropriate.[8]

Low Tidal Volume Ventilation in Patients with ARDS

The rationale behind low tidal volume ventilation in ARDS is to minimize the amount of VALI while attempting to maintain adequate ventilation in order to allow the lung to heal. There have been numerous studies looking at various forms of mechanical ventilation in ARDS dating back to 1975.[9] The definitive article was published in the *New England Journal of Medicine* May 4, 2000. This study was powered to detect a difference between standard ventilation using 12 cc per kilogram tidal volume and that using 6 cc per kilogram while maintaining plateau pressures lower than 30 cm H_2O. The study consisted of 861 patients and was terminated early as mortality was decreased by 22 percent in patients receiving 6 cc per kilogram tidal volume.[10] This study differed from two previous studies of low tidal volumes, which showed no mortality benefit. There are several possible explanations for this. First, the NIH study used the lowest tidal volume when compared to previous studies. Second, the NIH study treated respiratory acidosis associated with alveolar hypoventilation and hypercapnia by allowing the respiratory rate to increase to 35 breaths per minute and by giving $NaHCO_3$. Untreated respiratory acidosis may have contributed to the untoward effects found in the treatment groups of previous studies.[5]

The use of low tidal volume mechanical ventilation in ARDS patients reduces lung stretch. This would decrease the amount of barotrauma and volutrauma. Decreases in atelectrauma are somewhat treated using PEEP, and by decreasing these three forms of VALI, biotrauma would also be reduced. The ARDSnet study in 2000 led to further discussion of optimal lung recruitment strategies and optimizing positive end expiratory pressure.[10-11]

Lung Recruitment Strategies

After this initial breakthrough study, another was released comparing low positive end expiratory pressures to high positive end expiratory pressures. As the clinical outcomes of each group did not vary significantly, this led to controversy about the usefulness of PEEP.[11]

The lung protective strategy has led to the universal use of lower tidal volumes while using PEEP to prevent regional stress and strain on lung parenchyma. The addition of higher levels of PEEP, however, has not been shown to increase survival, as had the utilization of lower tidal volume ventilation. In fact, the addition of high levels of PEEP on lung that does not have large areas in need of recruitment may be more harmful than beneficial, because this would serve to only increase inflation of lung that is otherwise already open, increasing stress and strain on these regions.[11] It would follow then that it would be beneficial to know before instituting PEEP how much lung is available for recruitment. Use of computed tomography (CT) lends itself to helping determine the areas of potentially recruitable lung. The amount of potentially recruitable lung varied widely in patients with ARDS, varying from 2 percent to 28 percent. Improvement in aeration is highly correlated to the amount of recruitable lung on CT scan.[12] There is also a very high correlation between potentially recruitable lung and mortality. Those with high amounts of recruitable lung had higher mortality, lower P:F ratio, lower respiratory system compliance, higher $PaCO_2$, higher dead space, and greater shunt fraction. The best bedside test of potentially recruitable lung was determined to be a P:F ratio less than 150 at a PEEP of 5, any increased alveolar dead space, and an increase in respiratory system compliance when PEEP was increased from 5 to 15 cm H2O.[13]

It has also been shown that ventilating patients with ARDS using the ARDSnet protocol can lead to alveolar hyperinflation when high levels of PEEP are used. This can lead to further damage of lung parenchyma.[14] Using a physiologic approach to PEEP on an individual basis can be accomplished using something called the stress index. The stress index is a monitoring tool based on a complex computer program intended to quantify tidal alveolar hyperinflation and recruiting/derecruiting that occurs during constant flow ventilation. This was shown to decrease the amount of alveolar hyperinflation in patients with ARDS, but may not be of clinical use thus far as many institutions are not equipped with the needed software.[14]

There has been recent development in the issue of derecruitment during tasks such as suctioning. This becomes problematic when areas of lung that have been recruited successfully become derecruited. Two new recruitment maneuvers have been explored, continuous positive airway pressure (CPAP) with 40 cm H_2O for 40 seconds and/or an extended sigh consisting of PEEP maintained at 10 cm H_2O above the lower inflection point of the pressure volume curve for 15 minutes. Both maneuvers showed increased oxygenation, but only the extended sigh increased recruited volume. Outcome studies would be warranted to prove that these recruitment maneuvers do not increase parenchymal damage and lead to worsened VALI.[15,16,17,18]

Newer Modes of Ventilation in ARDS and Non-ARDS Patients

Newer modes of ventilation have recently been studied, along with their effects on ARDS and non-ARDS patients. The modes are high-frequency oscillatory ventilation, neurally adjusted ventilatory assist ventilation, proportional assist ventilation, airway pressure release ventilation, and biphasic intermittent positive airway pressure ventilation.

High-frequency oscillatory ventilation (HFOV) is characterized by the hasty delivery of small tidal volumes and the application of high mean airway pressures. It is these characteristics that make HFOV attractive for patients with ARDS. The high mean airway pressures applied could potentially prevent the cyclical derecruitment of the lung while the small tidal volumes can limit alveolar hyperdistention. When used as a rescue mode of ventilation, HFOV has been shown to have a sustainable increase in oxygenation in patients with ARDS, but randomized controlled trials have failed to show a mortality benefit. This may be due to improper patient selection, technique, actual tidal volume delivered, and the use of HFOV with other interventions such as prone positioning, recruitment, and nitric oxide.[19,20,21]

Neurally adjusted ventilatory assist ventilation is an experimental mode of ventilation that was designed to improve patient ventilator interaction by matching the ventilator with neural output from the respiratory centers. In this mode, assistance from the ventilator is delivered based on the electrical activity of the diaphragm, as determined by an esophageal electrode. This mode is dependent on the respiratory centers, phrenic nerves, and neuromuscular junctions remaining intact.[22]

Proportional assist ventilation (PAV) amplifies patient effort and improves patient ventilator synchrony. In this mode, the ventilator pressure is proportional to instantaneous flow and volume and to the pressure generated by the respiratory muscles. When compared to pressure support ventilation in patients with ARDS for 30 minutes, there are equivocal short-term effects on gas exchange and hemodynamics. There is, however, wide variability in desired tidal volume among patients ventilated on proportional assist ventilation, which may be tolerated by patients with no lung comorbidities, but problematic in those with ALI or ARDS. At low tidal volumes oxygenation would be compromised and at higher tidal volumes there would be damage to the lung caused by overdistention. The long-term effects in patients with ARDS have not been studied.[23]

Airway pressure release ventilation (APRV) is a pressure limited, time cycled mode of mechanical ventilation that permits spontaneous breathing throughout the ventilatory cycle while delivering high levels of continuous positive airway pressure (CPAP). This mode of ventilation consists of a high and low pressure setting. Recruitment and oxygenation occur during the high pressure setting and ventilation occurs by brief controlled release to a low pressure setting. The ability to safely and adequately ventilate patients with ARDS can be very challenging with low tidal volume ventilation. Low tidal volume ventilation can lead to decreases in the P:F ratio, increase in shunt, and decreased lung compliance. The ideal mode of lung recruitment remains to be found. When APRV was used to improve recruitment in patients with ARDS, there was an improvement in P:F ratio, a decrease in airway pressure, and release tidal volumes improved up to 13 percent. In patients who spontaneously breathe, airway pressure release ventilation resulted in less sedation, improved cardiopulmonary function, and decreased duration of ventilatory support, which is promising in those with and without ARDS.[24-25]

Biphasic positive airway pressure ventilation is similar to airway pressure release ventilation in allowing spontaneous breathing, but there are no restrictions on the timing of the pressure release. It is not to be confused with bi-level positive airway pressure (BIPAP). Spontaneous breathing efforts may be present during the longer release phase. BIPAP and APRV have gained increasing popularity because of their ability to reduce sedation and neuromuscular blockade in patients with ARDS. Both involve spontaneous breathing and tidal ventilation to accomplish CO_2 clearance in reverse sequence of other conventional modes. When mechanically delivered breaths are a minimal proportion of total minute ventilation, patients may be transitioned to CPAP or pressure support ventilation (PSV). With both BIPAP and APRV, and spontaneous breathing, there is improved matching of ventilation and perfusion in the lung, reduced physiologic dead space, airway pressures can be lowered, cardiac index increases, renal blood flow increases, GFR is higher, and limitation of sedation and neuromuscular blockade. There are also challenges using BIPAP and APRV. Since the ventilation is pressure limited, tidal volume delivered may be variable depending on lung and chest wall compliance. Whether larger tidal volumes in patients with peak airway pressures less than 30 cm H_2O remains controversial, but the possibility of overdistention is present. It has also been demonstrated that patients use more energy when breathing on BIPAP vs. PSV, and this may also be a consideration. Even with these limited side effects, the benefits appear to be wide ranging, but early randomization of patients to APRV and BIPAP or conventional ventilation still need to be performed in order to demonstrate a benefit in clinical outcome.[25]

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Critical Care CME Program

In this issue of the *Bulletin*, Module 5 of the Critical Care CME Program is available. There will be eight modules for this program. After each module is published in the *CSA Bulletin* (one per season), it is posted on the CSA Web Site at www.csahq.org. Each online module uses a self-assessment and evaluation; once these are completed, you may print your CME certificate. You may also contact the CSA office at 800-345-3691 to obtain the materials by fax or mail.

Questions

1. Which is not one of the four major types of ventilator-associated lung injury?
   a. Barotrauma
   b. Volutrauma
   c. Atelectrauma
   d. Biotrauma
   e. Mechanitrauma

2. Which statement is not in the definition of ARDS?
   a. P:F ratio greater than 200
   b. Clinical features of acute onset
   c. Bilateral infiltrates on CXR
   d. Pulmonary artery wedge pressure less than 18 mm Hg or absence of clinical evidence of left atrial hypertension

3. What is the optimal cc/kg tidal volume in the management of a patient with ARDS?
   a. 4 cc/kg
   b. 6 cc/kg
   c. 8 cc/kg
   d. 10 cc/kg
   e. 12 cc/kg

4. What is the best bedside test to quantify potentially recruitable lung?
   a. A P:F ratio less than 150 at a PEEP of 5
   b. A P:F ratio less than 150 at a PEEP of 15
   c. Worsening bilateral ground glass infiltrates on CXR
   d. Traumatic lung injury as the source for ARDS vs. biologically mediated source of ARDS

5. Which newer mode of ventilation shows the most promise for use in patients with ARDS?
   a. High Frequency Oscillatory Ventilation
   b. Neurally Adjusted Ventilatory Assist Ventilation
   c. Proportional Assist Ventilation
   d. Airway Pressure Release Ventilation
   e. Biphasic Positive Airway Pressure Ventilation

6. Which type of ventilator associated lung injury consists of injury that is cell and inflammatory mediator based?
   a. Barotrauma
   b. Volutrauma
   c. Atelectrauma
   d. Biotrauma

7. What is the mortality rate of patients with ARDS?
    a. 10 percent to 20 percent
    b. 20 percent to 30 percent
    c. 30 percent to 40 percent
    d. 40 percent to 50 percent
    e. 50 percent to 60 percent

8. Which mode of ventilation was designed to improve patient ventilator interaction by matching the ventilator with neural output from the respiratory centers, but is still in experimental stages?
    a. High-frequency oscillatory ventilation
    b. Neurally adjusted ventilatory assist ventilation
    c. Proportional assist ventilation
    d. Airway pressure release ventilation
    e. Biphasic positive airway pressure ventilation

9. There is a universally accepted method for recruiting lungs in patients with ARDS.
    a. True
    b. False

10. What two factors have increased BIPAP and APRV popularity in mechanically ventilated patients with and without ARDS?
    a. Reduction in sedation and neuromuscular blockade
    b. Fewer ventilated days as compared to other modes of ventilation
    c. Fewer cases of multiorgan system dysfunction
    d. Ease in weaning to extubate

Evaluation of Module 5

As part of the CSA Educational Programs Division's ongoing efforts to offer continuing medical education, the following evaluation of this program is requested. This is a useful tool for the EPD in preparing future CME programs.

1. How well were the learning objectives of this program met?

Very Well	5	Above Average	4
Average	3	Below Average	2
Not Well at All	1		

2. How relevant was the information in this program to your clinical practice?

Very Relevant	5	Above Average	4
Average	3	Below Average	2
Not Relevant	1		

3. How would you rate this program overall?

Excellent	5	Above Average	4
Average	3	Below Average	2
Poor	1		

4. Did you detect any commercial bias in this module? Yes No

Registration

Complete this form, the test, and the evaluation, and **mail or fax** all three to the CSA office at 951 Mariner's Island Boulevard #270, San Mateo, CA 94404 or FAX to 650-345-3269. The CSA CME journal courses are also available on the CSA Web Site at www.csahq.org.

Critical Care CME Course, Module 5
Available March 31, 2009, to March 31, 2012

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❑ **I acknowledge that I have read the Important Information about Module 5.**