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A monthly update of developments in critical care and intensive care medicine

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Perioperative Use of b-Blockers Reduces Neurologic Complications After Cardiac Surgery

ABSTRACT & COMMENTARY

This study was conducted to test the hypothesis that perioperative β-adrenergic receptor (βAR) antagonist administration provides protection against adverse cerebral complications during cardiac surgery. Subjects were 2575 patients, identified from the Duke Heart Center database, who underwent elective coronary artery bypass grafting surgery (CABG) over a 2.5-year period (June 1994 to December 1996). Patients were excluded if they underwent valvular surgery or noncardiac procedures that might increase neurologic complications, eg, carotid endarterectomy or abdominal aortic aneurysm repair. Perioperative drug administration was defined to include βAR antagonist administration within 48 hours of surgery or during surgery. Postoperative neurologic complications were defined as stroke, coma, transient ischemic attack (TIA), or clinical cognitive change (confusion or delirium) before hospital discharge. All complications were confirmed by retrospective chart review by a board-certified neurologist.

A total of 2296 (89%) patients received βAR antagonist therapy and 279 (11%) did not. There were 113 (4.4%) postoperative neurologic complications, including stroke (n = 44), coma (n = 12), TIA (n = 3), and confusion or delirium (n = 54). Adverse neurologic events occurred in 3.9% of patients who received perioperative βAR antagonist administration, compared to 8.2% of patients who did not receive these medications (odds ratio, 0.45; 95% confidence interval [CI], 0.28-0.73; $P = 0.003$). Severe neurologic outcomes (stroke and coma) occurred in 1.9% of patients who received βAR antagonists and 4.3% of patients who did not receive these medications (odds ratio, 0.43; 95% CI, 0.23-0.83; $P = 0.016$). The time of administration (preoperative, intraoperative, both) had no effect on neurologic outcome. Patients who did not receive βAR antagonists were older ($P = 0.0026$) and more likely to have a preoperative diagnosis of CHF ($P = 0002$) or COPD ($P < 0.0001$), but these patients did not differ in gender or incidence of hypertension, diabetes, prior stroke, or peripheral vascular disease. The incidence of atrial fibrillation was higher in the non-βAR

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antagonist group (25.1%), compared to the βAR antagonist group (20.5%), but the difference was not significant (P = 0.0863). (Amory DW, et al. Neuroprotection is associated with beta-adrenergic receptor antagonists during cardiac surgery: Evidence from 2575 patients. *J Cardiothorac Vasc Anesth*. 2002;16:270-277.)

■ COMMENT BY LESLIE A. HOFFMAN, RN, PhD

Despite advances in management, neurologic complications continue to be a major cause of morbidity and mortality after cardiac surgery. This study was conducted in response to anecdotal observations by the cardiac anesthesiology team suggesting that βAR antagonists might decrease adverse neurologic outcomes. Results of this retrospective analysis suggest that administration of βAR antagonists prior to or during cardiac surgery results in a substantial reduction in the total number of adverse neurologic events (3.9% with βAR antagonists; 8.2% without), an effect not previously reported.

Several actions of these medications might contribute to these positive outcomes. Propranolol has been found to shift the oxyhemoglobin dissociation curve to the right, which could increase availability of oxygen to ischemic tissue. Other βAR antagonists have been shown to improve neurologic outcomes in an animal model of transient cerebral ischemia. Atrial fibrillation is a common complication of CABG surgery, and the development of this complication significantly increases the risk of stroke. There was a tendency for patients who received βAR antagonists to have a lower incidence of atrial fibrillation, but the difference was not significant.

The study did not test a protocol for drug administration. Consequently, study results shed no light on optimal timing, dosing, or whether some drugs are more effective than others. Patients received βAR antagonists at the discretion of the anesthesiologist. General indications were continuation of β-blockade in patients already receiving βAR antagonists, initiation of new β-blockade to prevent or ameliorate heart rate responses to surgery, and prophylaxis to enhance myocardial protection during the entire operative period. The drugs used were atenolol, propranolol, and metoprolol. A strength of the study was the step of confirming all neurologic complications through retrospective chart review by a board-certified neurologist.

Although study findings are exciting, they were collected retrospectively and, therefore, do not provide confirmation of a neuroprotective effect of βAR antagonists during cardiac surgery. A prospective, randomized, blinded trial would be necessary to confirm study findings. If confirmed, use of βAR antagonists could represent an important and inexpensive method to decrease neurologic complications following cardiac surgery. ■

Critical Care Alert, ISSN 1067-9502, is published monthly by American Health Consultants, 3525 Piedmont Rd., NE, Bldg. 6, Suite 400, Atlanta, GA 30305.

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GST Registration Number: R128870672.

Periodicals postage paid at Atlanta, GA.

POSTMASTER: Send address changes to ***Critical Care Alert,*** P.O. Box 740059, Atlanta, GA 30374.

Back issues: $38.

Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issues date.

THOMSON
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World Wide Web: http://www.ahcpub.com

Subscription Prices

United States

1 year *with* free AMA Category 1 credits: $279

(Student/Resident rate: $140)

Multiple Copies

1-9 additional copies: $206 each; 10 or more copies: $183 each.

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In order to reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Ms. Ball serves as a consultant to Steris Corp, IC Medical, and AMT-Coherent (Canada), is a stockholder of Steris and SLT, and is on the speaker's bureau of AORN. Dr. Hoffman is involved in research with an NIH-funded grant that has no industry support. Dr. Pierson is a consultant to SeQual and Lynntech and is on the speaker's bureau of GlaxoSmithKline, Boehringer-Ingelheim, 3M, and Bayer. Dr. Rubenfeld is a paid consultant to Eli Lilly and has received educational grants from Byk-Gulden and Bayer in his name to the University of Washington. Drs. Baigorri, Durbin, Hess, Johnson, and O'Keefe report no consultant, stockholder, speaker's bureau, research, or other financial relationships with companies having ties to this field of study. Drs. Crawford, Gladwin, Nanavaty, and Takezawa did not return a 2002 financial disclosure form.

Central Venous and Bladder Pressures During Pressure Support Ventilation

ABSTRACT & COMMENTARY

Synopsis: Respiratory variation in central venous pressure and bladder pressure reflect respiratory muscle effort during reductions in pressure support ventilation.

Source: Chieveley-Williams S, et al. Central venous and bladder pressure reflect transdiaphragmatic pressure during pressure support ventilation. *Chest.* 2002;121:533-538.

The purpose of this study was to determine whether changes in central venous pressure and bladder pressure reflect changes in esophageal pressure and

gastric pressure during reductions of the level of pressure support ventilation. This was a prospective observational study. The study included 10 patients receiving pressure support ventilation through an endotracheal or tracheostomy tube. Central venous pressure was measured from the distal port of a triple-lumen catheter positioned in the superior vena cava. Bladder pressure was measured using the urinary drainage catheter after instilling 50 mL of saline. Esophageal and gastric pressures were measured from air-filled balloons placed into the esophagus and stomach, respectively. Airway pressure and flow were measured from the proximal airway. To minimize the effects of cardiac oscillations on the central venous pressure, the central venous pressure signal was averaged 30 or more breaths. After baseline measurements, the level of pressure support was reduced by 5 cm H_2O decrements until the pressure support level was 5 cm H_2O. At each level of pressure support, measurements were recorded after 10-20 minutes of equilibration.

Changes in central venous pressure correlated closely ($r = 0.95$) with changes in esophageal pressure, and changes in bladder pressure correlated closely ($r = 0.90$) with changes in gastric pressure. When the transdiaphragmatic pressure calculated using esophageal and gastric pressure was compared to that calculated using central venous and bladder pressures as the pressure support level was changed in individual patients, the correlation coefficients varied from 0.95 to 0.99. Chieveley-Williams and colleagues concluded that measurements of respiratory variations in central venous pressure and bladder pressure reflect respiratory muscle effort during reductions in the level of pressure support ventilation.

■ COMMENT BY DEAN R. HESS, PhD, RRT

Pressure, flow, and volume are commonly measured at the proximal airway during mechanical ventilation. During passive ventilation, these measurements, made under dynamic and static conditions, are used to calculate airways resistance and respiratory system compliance. Unless the pleural pressure is measured, however, it is not possible to separate lung and chest wall compliance. Even with a measure of pleural pressure, it is not possible to separate the effects of the rib cage and abdomen unless intra-abdominal pressure is measured. During spontaneous breathing efforts, pressure measurements from the proximal airway are affected little by respiratory muscle effort. For example, airway pressure changes during pressure support ventilation reflect nothing about respiratory muscle effort. Traditionally, air-filled balloons have been placed into the esophagus and stomach to estimate pleural and abdominal pressures, respectively. However, this technology is not commonly available and is seldom used except in research applications in academic centers. It has been known for many years that respiratory variation in central venous pressure reflects changes in pleural pressure.

This study demonstrates that measurements of respiratory variations in central venous pressure and bladder pressure can be used to estimate transdiaphragmatic pressure with reasonable, albeit not perfect, correlation during sequential reductions in the level of pressure support ventilation. Although this was a relatively small study of only 10 patients, it does provide promise for the use of relatively simple measurements of central venous and bladder pressures to quantify respiratory muscle effort during withdrawal of mechanical ventilation. It remains to be determined whether such measurements facilitate the ventilator weaning process.

In my practice, I have used respiratory variation in central venous pressure to titrate reductions in pressure support ventilation. I have reasoned that large negative deflections in central venous pressure during pressure support ventilation reflect an undesirably large load on the respiratory muscles, and I have titrated the level of support accordingly. The results published in this paper support that practice. There are other potential uses for the measurements of respiratory variation in central venous and bladder pressures. These include estimation of chest wall and abdominal compliance in passively ventilated patients, estimation of auto-PEEP in patients with airflow obstruction, and identification of diaphragmatic paralysis. It is interesting that the respiratory variations that make interpretation of central venous pressure problematic may be used to evaluate respiratory function. ■

Hospital Volume and Surgical Mortality

ABSTRACT & COMMENTARY

Synopsis: Mortality decreased as volume increased for all 14 procedures examined in this large study. Mortality differences between very-low and very-high volume hospitals were highest for pancreatic resection (16.3% vs 3.8%) and lowest for carotid endarterectomy (1.7% vs 1.5%).

Source: Birkmeyer JD, et al. *N Engl J Med*. 2002;346: 1128-1137.

Hospital volume (the number of procedures performed at a hospital) is known to affect surgical mor-

tality. This study was undertaken to clarify the relationship between hospital volume and surgical mortality for 6 different types of cardiovascular procedures and 8 types of major cancer resections. The procedures were selected because all are relatively complex, associated with a nontrivial risk of operative mortality, and most often are performed on an elective basis. The sample included 2.5 million Medicare patients who underwent surgery between 1994 and 1999, who were identified using the national Medicare claims database and the Nationwide Inpatient Sample. Patients who were younger than 65 years or older than 99 years were excluded, as were the approximately 10% of Medicare patients enrolled in risk-bearing HMOs. Mortality was defined as the rate of death before hospital discharge or within 30 days after the index procedures. Hospital volume was defined as the total number of procedures performed at the institution, not the number of procedures performed on Medicare patients. Five categories of volume were identified (very low, low, medium, high, very high).

Mortality decreased as volume increased for all 14 types of procedures ($P < 0.001$). The relative importance of volume varied markedly according to the type of procedure. Absolute differences in adjusted mortality rates between very-low-volume hospitals and very-high-volume hospitals ranged from more than 12% (for pancreatic resection, 16.3% vs 3.8%) to only 0.2% (for carotid endarterectomy, 1.7% vs 1.5%). The absolute difference in adjusted mortality rates between very-low-volume hospitals and very-high-volume hospitals was greater than 5% for esophagectomy and pneumonectomy, 2-5% for gastrectomy, cystectomy, repair of a nonruptured abdominal aneurysm, and replacement of an aortic or mitral valve, and less than 2% for coronary artery bypass grafting (CABG), lower extremity bypass, colectomy, lobectomy, and nephrectomy. Birkmeyer and associates concluded that, in the absence of other information about the quality of surgery at the hospitals near them, Medicare patients undergoing selected cardiovascular or cancer procedures can significantly reduce their risk for operative death by selecting a high-volume hospital.

COMMENT BY LESLIE A. HOFFMAN, RN, PhD

Findings of this study, likely the largest conducted to date, support a relationship that has been documented in several prior studies, namely that higher hospital volume translates into lower surgical mortality rates. The primary goal of this study was to examine hospital volume in regard to selected procedures, and marked variations were found in the absolute magnitude of difference between very-low-volume and very-high-volume hospitals. For all procedures, an inverse relationship was seen. Surgical mortality was significantly lower in very-high-volume hospitals compared to very-low-volume hospitals. However, there were marked differences among the types of procedures with the highest differences seen for procedures that are less frequently performed, eg, pancreatic resection (12.5%) and esophagectomy (11.9%).

Even small differences can be important. Considering procedure frequency, the researchers concluded that 314 deaths a year could have been avoided if the morality rate for CABG procedures (very common; volume had a moderate effect) at very-low-volume hospitals was reduced to that at very-high-volume hospitals. Conversely, 32 deaths a year would have been avoided for pancreatic resection (relatively uncommon; volume had a large effect).

It is notable that a recent review of 128 analyses involving 40 different procedures reported lower mortality at higher volume hospitals in 123 (96%) cases.[1] Therefore, it seems reasonable to promote use of high-volume centers where differences are the greatest and/or to establish some minimum standards for procedure frequency. The Leapfrog Group, a consortium that provides health insurance to more than 33 million people, has set volume thresholds for 5 procedures: CABG (500 per year), coronary angioplasty (400 per year), carotid endarterectomy (100 per year), elective abdominal aortic aneurysm repair (30 per year) and esophagectomy for cancer (6 per year).[2] Group members are promoting selective referral in urban centers through education and, in some cases, financial incentives.

Many object to such initiatives, noting that some low-volume hospitals have superior outcomes. However, it is difficult for the public to obtain such information. Notably, health care providers tend to follow similar guidelines. Few health care providers would knowingly suggest that a family member or friend undergo a high-risk, elective procedure at a hospital where such operations were rarely performed or refer to a physician who rarely performed the procedure. Absent reporting systems that provide appropriate information, it appears that the best way to improve one's odds of survival is to select a high-volume hospital. ■

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2. Birkmeyer JD, et al. Volume standards for high-risk surgical procedures: Potential benefits of the Leapfrog initiative. *Surgery*. 2001;130:415-422.

Special Feature

Optimal Ventilator Settings in Respiratory Failure From the Viewpoint of Pulmonary Mechanics

By Jun Takezawa, MD

Types of Respiratory Failure

Although respiratory failure is usually classified as insufficiency in pulmonary oxygenation and respiratory muscle pump performance, it also can be classified depending on lung mechanics—the lung with low compliance, high airway resistance, and a combination of the both. This classification is useful in determining optimal ventilator settings in patients with various types of respiratory failures independently of their pathogenesis.[1]

Respiratory Failure with Respect to Respiratory Mechanics

While lung compliance is decreased in pneumonia, the acute respiratory distress syndrome (ARDS), and atelectasis, airway resistance is increased in bronchial asthma. Increases in both compliance and airway resistance can be observed in patients with emphysema (usually with chronic obstructive pulmonary disease [COPD]) complicated with bronchial asthma, which requires the most sophisticated ventilator settings.

Optimal Settings for Full Ventilatory Support

Condition 1: The Lung with Decreased Compliance. The lung with low compliance requires high alveolar pressure (Palv) to provide the usual tidal volume (VT, 10-12 mL/kg). During controlled ventilation, this can be accomplished by allowing the development of high peak inspiratory airway pressure (PIP). Because pulmonary oxygenation is related to the mean airway pressure (mPaw), achieving the desired VT can be accomplished by adopting the following 3 methods; increasing PIP, positive end-expiratory pressure (PEEP), and inspiration-to-expiration (I/E) ratio.[2] No other methods, which do not result in an increase in mPaw, contribute to improve partial pressure alveolar oxygen (PaO_2).

Because plateau pressure (Pplat) is limited to be less than 30-35 cmH_2O for avoiding over-distension of the alveoli and ventilator-induced lung injury, and if sufficient PaO_2 cannot be obtained, the remaining options should be considered. Although the optimal PEEP, which is advocated to be set as equal to or more than lower inflection point (Pinf) on the relaxed pressure-volume curve, the measurement of Pinf is extremely difficult and time-consuming. Under usual clinical settings, optimal PEEP can be chosen on a trial-and-error basis. Another option is to increase the I/E ratio. This can be accomplished by prolonging the end-inspiratory period (Pplat or pause time). The I/E ratio can be increased until auto-PEEP develops. The decrease in expiratory time (TE) without development of auto-PEEP may result in hypercapnia, which can be allowed as permissive hypercapnia.

Condition 2: The Lung with High Airway Resistance. The target of optimal ventilator settings for the lung with high airway resistance is to provide maximal alveolar ventilation without the development of either auto-PEEP or high alveolar distending pressure. The highest alveolar pressure should be limited to be less than 30-35 cm H_2O, as in the lung with low compliance. Airway pressure during inspiration does not reflect alveolar pressure because of the presence of high airway resistance. The discrepancy between PIP and Pplat increases with an increase in airway resistance. Therefore, PIP can be increased until the alveolar pressure reaches 30-35 cm H_2O. The alveolar pressure is estimated by using end-inspiratory airway occlusion (Pplat).

In order to measure auto-PEEP, either the end-expiratory occlusion or the end-inspiratory occlusion method can be used. The end-expiratory airway occlusion method measures the airway pressure at the end of expiration after occlusion of airway opening, which subsequently becomes the same as alveolar pressure. The end-inspiratory occlusion method measures the difference between alveolar pressure (Pplat) at the given ventilator settings and that measured after prolonging the expiratory time (TE).[3] The presence of auto-PEEP can easily be demonstrated by disconnecting the ventilator from the patient or by stopping providing ventilator breaths and observing the reduction in central venous pressure (CVP), pulmonary capillary wedge pressure (Pcwp), and mean pulmonary artery pressure. The lowest value of these parameters should be used for evaluation of cardiac performance. Counter-PEEP can be used when a dynamic component of airway resistance is present; in other situations, provision of counter-PEEP results in an increase in alveolar pressure, and no changes in the level of auto-PEEP.

Auto-PEEP

Auto-PEEP is the alveolar pressure, which exceeds the airway opening pressure at the end of expiration. Because the clinician cannot detect or monitor auto-PEEP by means of the ventilator's routine pressure readouts, this phenomenon can cause patient harm without being recognized. Examples of this harm include 1) barotrauma (volutrauma); 2) a decrease in cardiac output due to decreased venous return, increased pulmonary resistance, and decreased ventricular filling pressure; 3) Increased ICP due to an increase in intravascular volume of intracranial capacitance vessels; and 4) Trigger failure of the ventilator during partial ventilatory assist.

The development of auto-PEEP can be understood by considering the time constant of the lung (τ), as shown in Figure 1. The lung can be passively inflated or deflated following the single exponential curve, where τ can be calculated, as compliance times resistance ($C \times R$). An expiratory time exceeding $4 \times \tau$ is required for the lung volume to return to the baseline level. Therefore, a subsequent ventilator breath that starts within 4 time constants ($\tau \times 4$) results in the development of auto-PEEP. Thus, auto-PEEP develops depending on VT, τ, and TE.

The optimal ventilator settings for full ventilatory support are shown in Fig 2. The ventilator strategies to improve oxygenation and to facilitate optimal CO_2 elimination are shown separately. When hypercapnia persists despite use of the algorithm shown, high-frequency oscillatory ventilation is one possibility for promotion of CO_2 elimination.

Figure 1

Single Exponential Curve and Time Constant

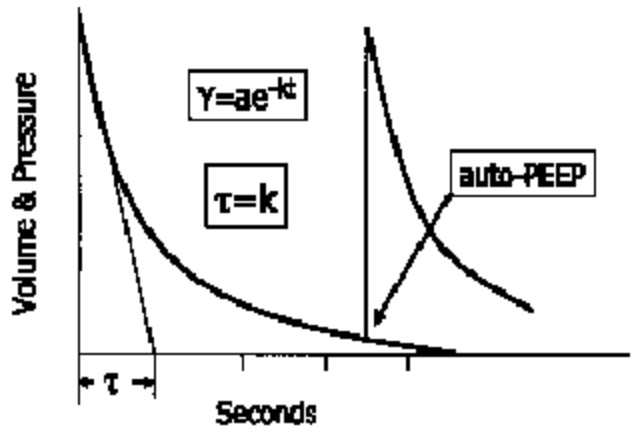
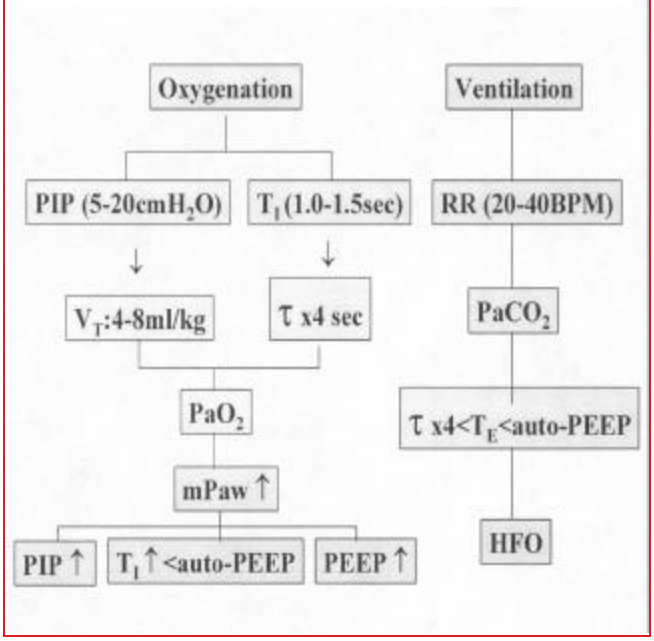


Figure 2

Optimal Ventilator Settings in Full Ventilatory Assist



Partial Ventilatory Support

Maximum Transalveolar Pressure (Ptalv)

During partial ventilatory support, with the patient doing a portion of the total work of breathing, because inspiratory effort is partially supported by the ventilator, the driving pressure to inflate the lung is a function of both alveolar pressure from the airways and pleural pressure, which is in the opposite direction: Ptalv = (Palv-Ppl), which can be approximated as trans-pulmonary pressure (Ptp). The maximum Palv, which is restricted to be less than 30-35 cm H_2O during full ventilatory support, can be used to restrict Ptp to be less than 30-35 cm H_2O during partial ventilatory support. However, because Ptp cannot be monitored routinely during partial ventilatory support, allowable PIP cannot be determined unless esophageal pressure is monitored.

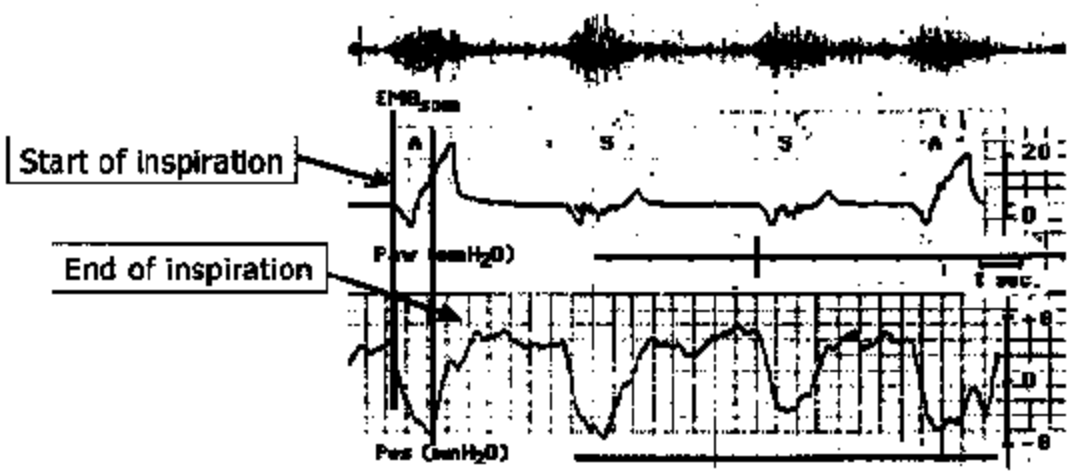
Patient-Ventilator Dyssynchrony

Another problem during partial ventilatory support is patient-ventilator dyssynchrony. Both inspiratory trigger delay and expiration cycling mismatch cause patient-ventilator dyssynchrony. Inspiratory trigger delay loads additional inspiratory work to the patient. Inspiratory trigger delay occurs in the lung with low compliance and high resistance, as well as in the lung with auto-PEEP.

As shown in Fig 3, a significant trigger delay (approximately 500 msec) is present in triggering the ventilator. Because no change in lung volume occurs in

Figure 3

Patient-Ventilator Dyssynchrony During Partial Ventilatory Support



this trigger phase, no inspiratory work can be calculated. However, patient places significant efforts to trigger the ventilator. Another finding in this figure is the expiration cycling delay. Approximately 500 msec is present from the end of inspiratory effort to the end of ventilatory breath.

In Figure 3, Diaphragmatic electromyographic (EMG) activity is shown at the top, indicating patient inspiratory effort; airway pressure is shown in the middle, and esophageal pressure (Pes, representing pleural pressure) is shown on the bottom. The start and end of inspiration are shown. (Adapted, with permission, from Imsand C, Feihl F, Perret C, Fitting JW. Regulation of inspiratory neuromuscular output during synchronized intermittent mechanical ventilation. *Anesthesiology*. 1994;80[1]:13-22.)

If alveolar rupture occurs it is likely a function of the maximum Ptp. Ptp may not always become greatest during the initial inspiratory phase in partial ventilatory support. It can become greatest even during the expiratory phase. Whichever phase produces the greatest Ptp, the chance of alveolar rapture increases during partial ventilatory support as compared to full ventilatory support, because of patient-ventilator dyssynchrony (between Ppl and Palv).

On the other hand, premature termination of inspiratory flow during pressure support ventilation (PSV) is observed in the lung with low compliance. This is mostly due to the incorporation of high inspiratory flow termination criteria in the ventilator's algorithm (eg, 5 L/min or 25% of the peak inspiratory flow), which allows the ventilator to stop delivering flow to the patient, even though the patient is still in the inspiratory phase. This problem can be overcome by adjusting the termination flow criterion to a lower value. Although pressure control ventilation (PCV) has been advocated to overcome this limitation of PSV, variation of both VT and TI becomes too large to permit synchronization by the set TI in PCV. Therefore, PCV should be only used as full ventilatory support and should not be used as partial ventilatory support.

Summary

In summary, ventilator strategy to improve pulmonary gas exchange in respiratory failure is established in FVS. Optimal ventilator settings can be easily understood, when pulmonary mechanics is taken into account. However, optimal ventilator settings for PVS are still unknown. Patient-ventilator dyssynchrony is still a remaining problem to be solved in terms of developing barotraumas and imposing respiratory work of breathing on patients with respiratory failure. ■

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CME/CE Questions

28. Use of bAR antagonists resulted in fewer adverse neurologic events when the drugs were given:
a. preoperatively only.
b. intraoperatively only.
c. both preoperatively and intraoperatively.
d. to hypertensive patients.
e. to male, but not female patients.

29. Complications found to be reduced in cardiac surgery patients who had received bAR antagonists perioperatively were:
a. stroke.
b. seizures.
c. focal paralysis.
d. All of the above
e. None of the above

30. The study by Chieveley-Williams et al demonstrated that respiratory variation in central venous pressure can be used to estimate:
a. fluid status.
b. cardiac output.
c. pleural pressure changes.
d. pulmonary vascular resistance.
e. None of the above

31. During pressure support ventilation, changes in proximally-measured airway pressure:
a. indicate corresponding changes in lung compliance.
b. reflect changes in airways resistance.
c. reflect patient inspiratory effort.
d. All of the above
e. None of the above

32. The difference in surgical mortality in very-low vs. very-high volume hospitals varied the most for:
a. cardiovascular procedures.
b. major cancer-related procedures.
c. patients aged 85-90 years.
d. patients enrolled in a HMO.
e. pancreatic resections.

33. Which of the following is *not* a procedure for which recommended hospital volume thresholds have been established by the Leapfrog Group?
a. Coronary artery bypass grafting
b. Cesarian section
c. Carotid endarterectomy
d. Coronary angioplasty
e. Esophagectomy

34. During spontaneous breathing with pressure support in a patient with reduced lung compliance, which of the following is the most important problem?
a. High peak airway pressure
b. Premature termination of inspiratory flow
c. Premature termination of expiratory flow
d. Excessive muscle effort to initiate inspiration
e. Insufficient peak inspiratory flow

35. In order to prevent dynamic hyperinflation and auto-PEEP in the presence of increased expiratory airway resistance, which of the following is required?
a. An I:E ratio of 0.50 or more
b. A corrected tidal volume of 6 mL/kg or more
c. Spontaneous termination of expiration by the patient
d. An expiratory time of at least 4 time constants
e. An arterial PCO_2 greater than 50 mm Hg

36. The expiratory time constant is calculated using which of the following?
a. Peak airway pressure and expiratory time
b. Inspiratory flow rate and expiratory time
c. Compliance and airway resistance
d. Inspiratory time and total respiratory rate
e. Plateau pressure and expiratory time

Readers are Invited. . .

Readers are invited to submit questions or comments on material seen in or relevant to *Critical Care Alert*. Send your questions to: Robin Mason, *Critical Care Alert*, c/o American Health Consultants, P.O. Box 740059, Atlanta, GA 30374. For subscription information, you can reach the editors and customer service personnel for *Critical Care Alert* via the internet by sending e-mail to robin.mason@ahcpub.com. ■

CME/CE Objectives

After reading each issue of *Critical Care Alert*, readers will be able to do the following:

- Identify the particular clinical, legal, or scientific issues related to critical care.
- Describe how those issues affect nurses, health care workers, hospitals, or the health care industry in general.
- Cite solutions to the problems associated with those issues.

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Increasing Respiratory Rate
to Improve $PACO_2$ in Patients with ALI and ARDS

CRITICAL CARE Plus

EXPANDING YOUR FOCUS IN INTENSIVE CARE

Re-Engineering Reimbursement Would Improve Outcomes, Experts Say

Lack of economic component seen as dysfunctional

By Julie Crawshaw, CRC Plus Editor

Physicians and hospitals are not receiving an economic incentive to produce outstanding patient health outcomes, according to Mark A. Cwiek, J.D., MHA, FACHE, associate professor in the School of Health Sciences, College of Health Professions at Central Michigan University in Mount Pleasant.

Not only are current reimbursement practices flawed, they're downright dysfunctional when viewed from a policy perspective, Cwiek says. "There is now no particular economic incentive for doing the right thing," Cwiek says. "You could go to three doctors with the same condition—one could be brilliant, one average and one below average—and they will all receive the same reimbursement."

Cwiek and fellow researcher Gerald R. Ledlow recommend that the American Medicare and Medicaid insurance programs take a page from the incentive-based German national system, a move they say would improve outcomes and achieve U.S. national health goals. "Providers cannot stay in business long when they aren't being reimbursed for the cost of services," says Cwiek. "What we propose is actually paying more in the short-term to providers and to be able to negotiate their reimbursement rates."

Cwiek isn't talking about cutting back on existing payments, but about building additional financial incentives. For example, he points out that state administrators could decide to bring down the rate of a particular illness, use a base reimbursement rate, and pay incrementally more for increased success, all of which would be negotiated on the front end. "We need to be on the very positive side of the spectrum, improving systems and rewarding excellent outcomes," Cwiek says.

Time for a Macro Approach

Such re-engineering requires both a macro approach and a certain degree of involvement and cooperative spirit to make it work, Cwiek acknowledges. However, the concept applies equally to a comparatively small system like an HMO and to the huge Medicare system. It's about setting goals and figuring out how to provide positive incentives for providers to do the right thing, Cwiek says, then measuring it and developing a report card to let people know how they're doing.

"You have to go to where payment comes from and then look at what stimulates policy," Cwiek says. "If the mandate comes from on high, wonderful things could start happening. Wouldn't it be wonderful to get the policy wonks in Washington to look at this?"

Testing how well the German system could work here could be as simple as policy-makers choosing three things to accomplish over the next three-to-five years, Cwiek says, and finding a way to pay providers for accomplishing them. He points out that this doesn't have to be as dramatic as demonstrating that one hospital's patients are 20% better off than patients at other facilities. "We could even pay providers just for developing new internal systems," Cwiek says, such as linking information from urgent care facilities to local hospitals so that when a patient enters the ICU a week after being seen in an urgent care facility, ICU clinicians have immediate access to the urgent care

records. "Develop those kinds of concepts at a macro level," Cwiek advises. "It can be done. The Germans are doing it now."

Community Agencies, Volunteer Spirit not Enough

Though well-defined national health care goals exist, implementing them is left to community health agencies and the volunteer spirit, Cwiek says, and this isn't getting the job done. "We're not purposely incentivizing physicians and hospitals to do better," Cwiek says, adding that the federal government must take a leading role. "If I could do just one thing, it would be to sit down with Tommy Thompson [secretary of the Department of Health and Human Services] and explain these principles."

A former hospital CEO, Cwiek worked in administration for 15 years before entering academia. He points to the success of the German system, in which providers, insurers, and government negotiate base rates for health services in 2-4 year cycles. Each 'coded' health care service has a base rate set and agreed upon by all parties. Base rates are published along with the weighted average (1.0 through 9.0) that the provider can charge. For example, with a base rate of 1.0 the provider can charge 2.5 or higher if the patient has private insurance, has documented/justified higher acuity, or is more time consuming.

Judging only by the percentage of gross domestic product paid for health care, the US medical system is the most expensive in the world, Cwiek says. A large part of the reason health care is so expensive here is that the U.S. leads the world in technology development, pharmaceuticals and surgical procedures. "That usually gets to the tertiary level of care, not primary care or wellness," Cwiek says.

In dollars that go for the kinds of public health initiatives that could reduce the eventual number of critical care patients, the U.S. is at the low end of the spectrum. "We pay more but our mortality and morbidity or death and quality of life are not as good as many other countries."

Cwiek and Ledlow say the new Medicare and Medicaid Incentive Payment Model they advocate[1] would:

- Be parsimonious;
- Be systematic and rational;
- Offer incentives for targeted areas based on economic, health measures, and forecasted dimensions;
- Allow for regional fluctuation using existing health referral regions and health service areas;
- Be inclusive. Providers, insurers, accrediting bodies, employers, patients, and the government should collaborate and problem solve in a systematic way to reach national healthcare and health related goals;
- Be measurable, 'forecastable,' and 'trendable.'In other words, measures must achieve a strong level of validity and reliability and must be anchored in simple yet reality-based terms;
- Be mutually beneficial to the patient, provider, insurer, and taxpayer;
- Assure a method to budget forecast and plan well into the future; and
- Be sensitive to state/regional jurisdictional issues and political pressures.

Cwiek and Ledlow say that what they propose can be done anywhere. Cwiek notes that a physician-hospital organization could easily discover an HMO's most important goals and negotiate for incentives to accomplish them beyond a capitated payment or discounted fee for service. "Doctors and hospitals are getting beaten up with discounting," he says. "Turn it around—what positive incentives can be built in when aspirations and goals are met?"

Cwiek and Ledlow suggest that stakeholders involved in negotiating base rates of payment and reimbursement every two or three years would be:

- Providers (including but not limited to representatives from the American Medical Association, American Osteopathic Association, American Hospital Association, long-term care, home care, medical laboratory, pharmaceuticals, etc. . .);
- Insurers (including but not limited to representatives from the Health Insurance Association of America);
- Employers (perhaps comprised of rotating membership from the top [non-self insured] 100 US employers where 7-9 member panel serves on rotating basis) and the National Chamber of Commerce;
- Patients (7- 9 member panel representing patients from advocacy groups, academia, etc. appointed by the President, approved by House of Representatives);
- Accreditation bodies (including but not limited to representatives from the National Committee for Quality Assurance, the Joint Commission on Accreditation of Healthcare Organizations and the Utilization Review Accreditation Committee); and
- Government (including but not limited to representatives from the National Institutes of Health, the U.S. Department of Health and Human Services, Medicare, the National Association of Insurance Commissioners, and the National Association of Attorneys General).
- For any incentive re-engineering to succeed, Cwiek

notes, physicians must apply pressure to the powers that keep the current system in place. "Physicians think primarily as advocates for their patients," he says. "Unfortunately, in this world of managed care they are being forced to think in terms of social justice, which is a different ethical model. Now, it's represent your patients and the system." (Contact Information: Mark Cwiek [989] 774-1338.) ■

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$350,000 Outbreak Hits Hospital NICU

Think prevention is expensive? Try infection

The cost of a single nosocomial outbreak was recently broken down into staggering detail by an economically minded clinician at Columbia University Hospital in New York City. The message to administrators: Think prevention is expensive? Try infection.

"A four-month outbreak—a third of a million dollars," said Patricia Stone, PhD, RN, a health services researcher at Columbia. "This outbreak costs the hospital $350,000, and that is a conservative estimate."

The expensive pathogen behind the outbreak was extended spectrum â–lactamase *Klebsiella pneumoniae*, which is often linked to hospital-acquired infections occurring in intensive care units. In this case, the setting was a neonatal intensive care unit (NICU), which had eight infected and 14 colonized neonates over a four-month period. The persistent outbreak ultimately resulted in closing the unit, bringing lost revenues into the economic picture on top of all the other expenses.[1]

"There were 14 infants that were not admitted to the unit during the outbreak period," she reported in Nashville at the annual meeting of the Association for Professionals in Infection Control and Epidemiology (APIC). "We looked at the lost revenue to the hospital in terms of those lost beds and the NICU closure."

The closed beds resulted in a revenue loss of $110,000. The infected babies who were admitted had a mean length of stay of 85.5 days, 48 days longer than a similarly risk-adjusted patient group using comparative data. At $590 a day, the attributable cost of increased stay for infected neonates totaled $229,000. Of course, costs associated with fighting the outbreak pushed the total higher, but the efforts revealed two NICU nurses with persistent hand carriage *K pneumoniae* due to long or artificial fingernails. The hospital has now banned fake and long nails.

"Economic evaluations like this increase understanding of the burden of outbreaks," Stone said. "Evidence such as this may encourage more effort and resources to be put in [infection] prevention. You start to be able to make evidence based arguments."

But will the cost figures be used to lobby administration for an enhanced investment in infection control resources?

"It's being done as we speak," she told APIC attendees at a special session on pediatric infection control.

HCW Linked to Outbreaks Years Apart

Another study presented at the same session featured a similar case of a colonized worker, but with an unusual twist. A nurse with recurrent carriage of a strain of methicillin-resistant *Staphylococcus aureus* was linked to separate outbreaks two years apart.

Between Dec. 17, 1999, and Jan. 5, 2000, 13 infants in an NICU at Virginia Commonwealth University (VCU) Health System in Richmond were infected or colonized with MRSA. The index case was an 800 g infant delivered in an ambulance.[2]

"The first four cases were within 10 days of one another," said Lynn Reynolds, RN, infection control professional at VCU. "There were two deaths; one related to the [MRSA] bacteremia."

Eight babies were asymptomatically colonized in the umbilicus and/or nares. Before the source was discovered the infection control team went to great efforts to prevent further spread.

"Our interventions initially for the first two cases included placing infected infants on contact precautions," she said. "The infected infants were moved to the back row of the unit away from the main traffic stream. We cohorted staff and infants; we reinforced the wearing of gowns and gloves for staff and visitors. And of course, [we did] education of staff. Unfortunately, despite our early interventions, we continued to have more cases of MRSA bacteremia."

Additional interventions included weekly surveillance cultures of the nares and umbilicus of all babies in the NICU. MRSA-positive infants were treated with intranasal mupiricin. Thorough cleaning of the unit was

done daily to prevent environmental contamination. Access to the unit was limited to one entrance, which was through the scrub area. Underscoring the severity of the situation, a security guard was placed at the entrance to ensure hand washing. Employees were cultured for the outbreak strain.

"We were able to identify more cases of infected and colonized babies even after our additional interventions were instituted," she said. "If nosocomial transmission continued, there was discussion that we would close the unit."

Nares swabs were obtained on 140 hospital workers and the two EMS workers who delivered the index case. All cultures were negative except for one taken from an NICU nurse. Her isolates were identical to the outbreak strain by molecular typing. Control strains from other units were different from the outbreak strain.

"Further investigation of the colonized health care worker revealed that she was also positive for MRSA [during] a 1997 NICU outbreak," Reynolds said. "At that time, she was treated with intranasal mupiricin and had a follow-up culture that was negative."

The colonized nurse directly cared for eight of the 13 infants who were infected or colonized in the outbreak. She had no underlying medical conditions. Interestingly enough, this health care worker's colonizing strains from the 1997 outbreak and the 2000 outbreak were genetically identical. She was removed from the unit, retreated with intranasal mupiricin, and successfully decolonized. She was reassigned to a non-NICU unit. "Once the heath care worker was removed, we had no new cases of MRSA infection or colonization the two months that followed," Reynolds said.

However, the story doesn't end there. The NICU nurse—one of the best on the unit—eventually left the hospital after the reassignment.

"[One] argument was that we should allow her to return to the unit because she was considered one of the best nurses in the unit," Reynolds said. "She was highly trained and skilled, and deeply committed."

But the risk of subsequent infections—and the morbidity, mortality, and costs they could entail—compelled the hospital to ban the nurse from the NICU.

"This outbreak illustrates the controversy that arises when health care workers are found colonized with multidrug-resistant organisms," she said.

Winning the RSV Season

In other research presented at the pediatric session, an infection control practitioner (ICP) detailed the development of a successful policy for preventing seasonal outbreaks of nosocomial respiratory syncytial virus (RSV).[3]

"There are various conditions that increase the risk of a person having severe or even fatal RSV infection," said Elizabeth Fuss, RN, MS, CIC, an ICP at Johns Hopkins Hospital in Baltimore. "[Those include] congenital heart disease, underlying pulmonary disease in children, and prematurity—and immunodeficiency and immune suppression at any age. Nosocomial transmission of RSV is very well described, and if you look in the literature among reported outbreaks, you can find mortality rates as high as 44%."

ICPs at Johns Hopkins started tracking nosocomial transmission of RSV in 1989.

"It's a good think we did, because in 1990-91, we had a terrible [rate]: 20.2% of all RSV cases were acquired nosocomially," she said.

The two-stage control measures that were originally put in place in 1991 have continued to evolve in the hospitals 140-bed pediatric unit. Stage 1 begins when the first case of RSV is admitted each fall. The protocol requires obtaining RSV antigen testing and viral cultures on all children under age 6 who have been diagnosed with bronchiolitis or pneumonia. Stage 2 begins when five patients have been admitted with RSV and expands RSV antigen testing and viral cultures to all children under 6 with any respiratory symptoms. They stay under RSV precautions until the antigen is negative, which sometimes is only a few hours with the rapid testing, she said. A nosocomial case is defined as a child who develops RSV at least four days after admission. Stage two stays in affect until 10 days have passed without admission of a community-acquired case or discovery of nosocomial transmission. ■

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