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INSIDE

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Is Clot Burden on CT Angiogram Predictive of Mortality in Pulmonary Thromboembolism?

ABSTRACT & COMMENTARY

By David J. Pierson MD, Editor

Synopsis: *A prospective study comparing angiographic clot burden score and ECG score in 105 patients with PE found no correlation between the two, and neither predictor correlated with 12-month mortality. In a second, retrospective study of 33 consecutive patients with massive PE by conventional clinical criteria, there was also no correlation between findings on CT angiography and mortality.*

Sources: Subramaniam RM, et al. Pulmonary embolism outcome: A prospective evaluation of CT pulmonary angiographic clot burden score and ECG score. *AJR Am J Roentgenol* 2008;190:1599-1604; Findik S, et al. Massive pulmonary emboli and CT pulmonary angiography. *Respiration* 2008 Jul 22; Epub ahead of print.

WITH PULMONARY COMPUTED TOMOGRAPHIC (CT) ANGIOGRAPHY increasingly used to diagnose acute pulmonary thromboembolism (PE), it has become commonplace to report not only the presence of clot when the study is positive, but also an estimate of the clot burden. In at least some institutions, the CT angiograms of patients with large quantities of visualized thrombus are read out as "massive PE." By examining the assumed relationship between the CT angiographic findings and clinical outcomes in patients with PE, two recent articles shed light on the clinical appropriateness of using such terminology.

Subramaniam and associates at Waikato Hospital in Hamilton, New Zealand, carried out a prospective study to examine the claimed predictive value of each of two published scoring schemes for patients with PE: the CT pulmonary angiographic burden score of Qanadli et al[1] and an electrocardiographic (ECG) score correlated to the extent of pulmonary perfusion impairment.[2] The CT score weighted different assessments of the site and degree of pulmonary arterial obstruction, the latter used to calculate the percentage of obstruction. The ECG score assigned varying weights to several measures related to right ventricular strain, such as right bundle

branch block, precordial T wave inversion, and the S1Q3T3 pattern. Two CT angiographers independently determined the CT scores, and two clinicians independently determined the ECG score, for the 105 patients with positive CT angiograms of 523 consecutive patients who underwent evaluation. Correlations were sought between the two indices, and also with the patients' clinical outcomes as determined at 12 months after diagnosis.

The mean (SD) clot burden score percentage was 23.7% (16.8%) and the mean (SD) ECG score was 2.4 (2.8) out of a possible 21. There was no significant correlation between the two indices at the time of diagnosis ($r = 0.09$; $P = 0.39$). At one year, 13 patients had died, and neither the CT clot burden score nor the ECG score correlated with whether they were alive or dead (all-cause mortality).

Findik and colleagues at Ondokuz Mayis University, Samsun, Turkey, carried out a retrospective analysis of a different index of the extent of pulmonary arterial obstruction, along with clinical data and mortality, in 33 consecutive patients with massive PE. The latter was diagnosed by the presence of a systolic blood pressure < 90 mm Hg, syncope, and/or shock. All the patients had CT angiography and an assessment of right ventricular function. Hemodynamic severity was assessed by the extent of right ventricular dysfunction, the diameter of the main pulmonary artery, the shape of the interventricular septum, and the extent of obstruction to the pulmonary arterial circulation using a CT obstruction index.

All 33 patients had emboli in the central pulmonary arteries. All of them also had right ventricular dysfunction, which was judged to be severe in 94%. The shape of the interventricular septum was abnormal in all the patients, and the diameter of the main pulmonary artery was increased in 76% of them. The CT obstruction index was 50% or more in 85% of the patients. Twenty-eight (84%) of the patients survived, and the authors found no correlation between the CT angiographic findings and survival.

COMMENTARY

For decades, studies of the epidemiology and therapy of PE have used clinical criteria, not the estimated quantity of clot in the pulmonary arterial tree, to define "massive PE." The principal criteria are arterial hypotension and cardiogenic shock.[3] Arterial hypotension is defined as a systolic blood pressure < 90 mm Hg, or a drop in systolic arterial pressure of at least 40 mm Hg for at least 15 minutes. The definition of shock is less quantitative, but includes evidence of tissue hypoperfusion and hypoxia, such as altered level of consciousness, oliguria, and/or cool, clammy extremities. Patients with massive PE defined in this way have an early mortality of at least 15%, with the degree and persistence of hemodynamic compromise generally the most powerful predictors of in-hospital death. Although PE is commonly encountered among hospitalized patients, in one multicenter study of 2454 patients admitted with this diagnosis, only 4.2% of them met criteria for massive PE.[4]

Although both the Subramaniam and Findik studies raise issues of patient selection and other design features (and the Findik study was grossly underpowered for differences in mortality), they both emphasize that massive PE is a clinical diagnosis, and not determined by the angiographic extent of visualized thrombus. This is important because of the implications of this diagnosis for thrombolysis and other therapy. The new American College of Physicians Evidence-Based Clinical Practice Guidelines (8th edition) emphasize the importance of clinical risk stratification, and recommend thrombolytic therapy in PE for patients with evidence of hemodynamic compromise.[5] The guidelines do not include the clot burden as visualized by CT angiography in either defining massive PE or in selecting appropriate therapy. ■

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Noninvasive Ventilation in Myasthenic Crisis

ABSTRACT & COMMENTARY

By David J. Pierson, MD, Editor

Synopsis: *In a retrospective study of acute respiratory failure complicating myasthenic crisis, 14 of 24 patients who were managed initially with noninvasive ventilation successfully avoided intubation, but pneumonia occurred in 80% of the instances in which this approach was unsuccessful.*

Source: Seneviratne J, et al. Noninvasive ventilation in myasthenic crisis. *Arch Neurol* 2008;65:54-58.

Investigators in the department of neurology at the Mayo Clinic in Rochester, MN, reviewed the charts of patients admitted with myasthenic crisis (MC) between 1987 and 2006 who received either invasive or noninvasive mechanical ventilation. Only patients with de novo MC were included, and postoperative cases plus those associated with underlying cardiopulmonary disease were excluded. Fifty-two patients qualified, with a total of 60 episodes of MC. In 24 (40%) of these episodes, noninvasive ventilation (NIV) was attempted; NIV was the only form of ventilator support used in 14 of the 24, and in 10 instances endotracheal intubation and invasive mechanical ventilation (ET-MV) was subsequently used. In the other 36 episodes (60%), ET-MV was the initial procedure. The authors examined the clinical, physiologic, and outcome information available in the patients' charts for these 3 groups (i.e., NIV alone, NIV followed by ET-MV, and ET-MV alone).

Statistical analysis revealed no significant differences among the 3 groups with respect to age, precipitating event, initial arterial blood gases, initial vital capacity, or initial maximal inspiratory or expiratory pressures, although the numbers of patients who had these different variables measured prior to initiation of ventilatory support are not provided. Among patients initially treated with NIV, those who subsequently were intubated were more likely to have initial arterial PCO_2 values exceeding 45 mm Hg. Patients who were intubated spent longer on the ventilator than those in whom NIV was successful. Pneumonia occurred in 3 of the 14 patients successfully treated with NIV, and in 25 of the 46 patients who required intubation—including 8 of the 10 in whom NIV was tried unsuccessfully.

COMMENTARY

Admission of a patient in MC is a very uncommon event in most ICUs. However, such patients share a number of clinical features and management challenges in common with a larger population of acutely ill patients cared for by intensivists, nurses, and respiratory therapists. The most important of these features are respiratory muscle weakness and impaired airway protection, which are encountered in patients with Guillain-Barré syndrome, spinal cord injury, amyotrophic lateral sclerosis, multiple sclerosis, stroke, and a variety of other disorders affecting neuromuscular function. The most important life-threatening complications in patients with these problems are acute ventilatory failure and pneumonia. Thus, this study of patients with MC, which found very high rates of both of these complications, may be more broadly applicable to ICU practice than just in the context of myasthenia gravis.

The study by Seneviratne and colleagues comes from an acknowledged center of excellence in managing patients with myasthenia gravis, and has the advantage of access to the records of a larger number of such patients than would be the case at most institutions. This large cohort permitted comparisons of patient characteristics and outcomes in patients receiving NIV with those in patients who were intubated during an episode of MC. However, a retrospective chart review has serious inherent limitations when it comes to detecting differences between patients with the same diagnosis, and I think the fact that the patients appeared similar in most aspects examined by the authors illustrates some of these limitations. In this center of excellence, where patients were managed by clinicians

experienced in the management of MC, some patients received trials of NIV and some were intubated as the initial form of ventilator support. Why? There were no statistically significant differences in initial arterial blood gases or maximal respiratory muscle pressures. In fact, the mean arterial pH and PCO_2 values in the patients receiving NIV were within normal limits. Because this was not a prospective, randomized trial, and instead describes actual practice based on individual patient presentation and clinician judgment, there must have been other findings that led to the decision to try NIV (in 40% of the patients) or to intubate (in 60%). Initial respiratory rates and other vital signs are not provided, nor are descriptions of the patients' levels of alertness or cooperation.

Noninvasive ventilation did not come into wide use in treating acute respiratory failure until the second half of the 19-year period of this review. Thus, it is likely that most of the NIV patients were managed relatively recently, and possible that other important temporal trends in patient assessment and management could have occurred. These things illustrate some of the inherent limitations of a retrospective chart review for telling us why patients were managed in a particular way. Because of such limitations and other factors, studies like this one are useful in generating hypotheses as to the reasons for differences observed among the patients—hypotheses appropriate for testing in prospective clinical trials—but much less reliable when it comes to drawing conclusions about how patients should be managed.

A very high proportion of the patients in this study developed pneumonia—including 80% of those initially treated with NIV who subsequently were intubated. This finding supports the notion that many of these patients were unable to protect their lower airways, and raises concern about the advisability of ventilator support without intubation. Patients managed with NIV who did not have initial hypercapnia tended to do better in this series, but the numbers are fairly small. In fact, bulbar weakness and other circumstances of impaired airway protection are generally listed among the contraindications to NIV in acute respiratory failure. Thus, although this study's findings may be used to support the authors' conclusion that NIV "is effective in the treatment of acute respiratory failure in patients with myasthenia gravis," they could also be cited in support of the opposite conclusion. Prospective studies of NIV vs ET-MV in patients with acute respiratory failure and neuromuscular weakness could tell us with much greater confidence whether the former is an advisable approach. In the meantime, I think great caution should be exercised whenever the use of NIV is contemplated in such patients—particularly if they have hypercapnia or obvious bulbar weakness on initial presentation. ■

What Is the Optimal Time to Perform Tracheostomy?

ABSTRACT & COMMENTARY

By Andrew M. Luks, MD

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Dr. Luks reports no financial relationship to this field of study.

Synopsis: *This retrospective cohort analysis demonstrates that patients who receive tracheostomy within the first 10 days of critical illness have a small, yet statistically significant, mortality benefit compared to those who undergo tracheostomy at a later point in time, but provides no insight into factors governing patient selection for early timing of the procedure.*

Source: Scales DC, et al. The effect of tracheostomy timing during critical illness on long-term survival. *Crit Care Med* 2008;36:2547-2557.

TRACHEOSTOMY CONFERS PATIENT BENEFITS SUCH AS decreasing laryngeal irritation, improving patient communication, and decreasing sedation requirements, but the optimal timing of this procedure in critically ill patients remains a subject of considerable debate. Scales and colleagues conducted a retrospective cohort study to determine whether performing tracheostomy earlier in the course of critical illness was associated with improvements in patient survival.

The authors examined the Ontario Health Databases to identify all critically ill patients who underwent tracheostomy across multiple centers over a 12-year period (1992 to 2004). Patients were excluded from the analysis if they underwent tracheostomy within 48 hours or after 28 days of initiating mechanical ventilation. The major independent variable in the analysis was the timing of tracheostomy. This was treated as a continuous variable, but they also conducted analyses in which it was treated as a dichotomous variable in which tracheostomy was defined as being "early" if it occurred between 3 and 10 days after initiation of mechanical ventilation and "late" if it occurred between 11 and 28 days following mechanical ventilation. The 10-day cutoff was selected

based on the results of prior observational studies, which showed that the median time to tracheostomy is 9-14 days. The primary outcome measure was the hazard of dying after initiation of mechanical ventilation. Secondary outcomes included 90-day and 1-year mortality, time from tracheostomy to discontinuation of mechanical ventilation, ICU length of stay, and ventilator-free days at 28 at 180 days. All survivors were followed for between 1 and 13 years (median, 6.4 years).

A total of 10,927 patients were included in the analysis; 3758 (34%) of the patients underwent "early" tracheostomy while 7169 (66%) underwent a "late" procedure. There were important differences between the two groups as the early tracheostomy patients were older, had more cardiac disease, and more physician visits prior to their ICU admission, while the late tracheostomy group was less likely to have a neurologic disorder or traumatic injury. Overall, 7219 (66%) of patients died during the study period. Cumulative mortality was lower in the early tracheostomy group at 90 days (34.8% vs 36.9%; $P = 0.03$), one year (46.5% vs 49.8%; $P = 0.001$), and during the entire study period (63.9% vs 67.2%; $P < 0.001$). Multivariable analyses in which tracheostomy was treated as a time-dependent variable demonstrated that each additional 1-day delay in tracheostomy was associated with increased mortality (hazard ratio, 1.008; 95% confidence interval, 1.004-1.012). Patients receiving early tracheostomy had more ventilator-free days than the late tracheostomy patients at both 28 (mean number, 9.7 vs 3.0; $P < 0.001$) and 90 (mean, 94.2 vs 81.2; $P < 0.001$) days and faster weaning from mechanical ventilation (7 vs 13 days).

COMMENTARY

The results of this study are intriguing but should not leave anyone running for the percutaneous tracheostomy kit and bronchoscope just yet. Although there was a mortality benefit to early tracheostomy at 90 days, at one year, and over the entire study period, the absolute reductions were very small and likely only reached statistical significance because of the very large sample size. More importantly, it must be remembered that this was a retrospective study and, as a result, the authors were unable to control for important variables such as the reasons patients underwent tracheostomy at particular times. In fact, the Ontario Health Database that served as the foundation of this study did not include such information. To their credit, they did use multiple analyses to adjust for unmeasured confounding variables and these analyses consistently showed a small mortality benefit to early tracheostomy. However, as they admit themselves in the discussion and as was revealed in some of the demographic data about the early and late groups (e.g., the percentage of people with cardiac disease or neurologic injury), it is likely that there are systematic differences between the two groups that limit the applicability of these results. Rather than interpreting their results as indicative of a mortality benefit to early tracheostomy, I would argue that the very small differences observed in this study suggest we are probably not doing our patients any harm by waiting longer to see if they can get off the ventilator and then doing tracheostomy if they fail attempts at weaning.

Beyond the issue of whether the timing of tracheostomy affects mortality, there was one other particularly striking feature of the data; more than 30% of patients, regardless of the timing of their tracheostomy, died within 90 days of the procedure, while up to 67% of patients died within the study period, which lasted in some cases up to 13 years. These are surprisingly high numbers and suggest that the need for tracheostomy in critical illness portends an overall poor long-term prognosis for the patient. ■

Special Feature

Tidal Volume Limitation for All Intubated Mechanically Ventilated Patients: Less Is More

By Dean R Hess, PhD, RRT

Respiratory Care, Massachusetts General Hospital, Department of Anesthesiology, Harvard Medical School

Dr. Hess reports no financial relationship to this field of study.

THE LUNG-PROTECTIVE EFFECTS OF LOW TIDAL VOLumes, as demonstrated by the ARDSnet study, are well accepted in patients with acute lung injury (ALI) or acute respiratory distress syndrome (ARDS). In the original ARDSnet study, 861 patients were assigned to receive a tidal volume of 6 mL/kg predicted body weight (PBW) or a tidal volume of 12 mL/kg PBW.[1] In the lower tidal volume group, tidal volume was reduced further to a minimum of 4 mL/kg PBW if the end-inspiratory plateau pressure was greater than 30 cm H_2O. In cases of severe acidosis or ventilator dyssynchrony, the tidal volume was increased to a maximum of 8 mL/kg,

provided plateau pressure was ≤ 30 cm H_2O. The lower tidal volume strategy was associated with a 31% patient mortality, whereas the conventional tidal volume strategy was associated with 40% mortality (relative risk reduction of 22%).

The results of the ARDSnet trial, combined with other supporting data, have resulted in a reduction in tidal volumes in patients with ALI and ARDS. Some argue that tidal volume limitation is less important than maintaining plateau pressures < 30 cm H_2O.[2] However, results from a secondary analysis of the ARDSnet trial suggest that there is benefit for tidal volume reduction from 12 mL/kg to 6 mL/kg PBW even when plateau pressure is ≤ 30 cm H_2O.[3] What is more controversial is whether low tidal volumes should be used even in patients who do not have ALI or ARDS at the time of intubation.

It is interesting to note that a tidal volume of 6 mL/kg is typically referred to as a low tidal volume in mechanically ventilated patients. However, normal tidal volume is 6 mL/kg. So it might make more sense to refer to normal tidal volumes vs large tidal volumes. Normal lung volumes are predicted from sex and height, as follows:

Males: PBW = 50 + 2.3 × (inches of height - 60)

Females: PBW = 45.5 + 2.3 × (inches of height - 60)

Traditionally, tidal volume settings have been recommended without regard to sex or height, exposing women and shorter patients to potentially injurious tidal volumes.

Low-Tidal-Volume Ventilation in Patients without ALI or ARDS

Gajic et al conducted a retrospective study of patients who received invasive mechanical ventilation for > 48 hours between January and December of 2001 in 4 ICUs.[4] Of 332 patients who did not have ALI at the time of intubation, 80 patients (24%) developed ALI within the first 5 days of mechanical ventilation. Women were ventilated with larger tidal volumes than men (mean, 11.4 mL/kg vs 10.4 mL/kg PBW; $P < 0.001$) and tended to develop ALI more often (29% vs 20%; $P = 0.068$). In a multivariate analysis, the main risk factors associated with development of ALI were the use of large tidal volumes (odds ratio [OR], 1.3 for each mL above 6 mL/kg PBW; $P < 0.001$), transfusion of blood products (OR, 3.0; $P < 0.001$), pH < 7.35 (OR, 2.0; $P = 0.032$), and a history of restrictive lung disease (OR, 3.6; $P = 0.044$).

In another study by Gajic and colleagues, patients were identified who required mechanical ventilation for 48 hours but did not have ARDS at the onset of mechanical ventilation.[5] Of 3261 mechanically ventilated patients who did not have ARDS at the time of intubation, 205 (6.2%) developed ARDS 48 hours or more after the onset of mechanical ventilation. Multivariate logistic regression analysis found the development of ARDS was associated with the initial ventilator settings: high tidal volume (OR, 2.6 for > 700 mL), high peak airway pressure (OR, 1.6 for > 30 cm H_2O), and high PEEP (OR, 1.7 for > 5 cm H_2O).

Fernández-Pérez et al evaluated whether intraoperative mechanical ventilation with large tidal volumes was associated with increased risk of post-pneumonectomy respiratory failure.[6] They studied patients undergoing elective pneumonectomy from January 1999 to January 2003. Of 170 pneumonectomy patients who met inclusion criteria, 30 (18%) developed postoperative respiratory failure. Patients who developed respiratory failure were ventilated with larger intraoperative tidal volumes than those who did not (median, 8.3 mL/kg vs 6.7 mL/kg PBW; $P < 0.001$). In a multivariate regression analysis, larger intraoperative tidal volume was associated with development of postoperative respiratory failure (OR, 1.56 for each mL/kg increase).

In a prospective multicenter observational study (in 4 European ICUs), Mascia et al investigated the role of ventilatory management as a predictor of ALI in brain-injured patients.[7] The study enrolled 86 severely brain-injured patients (Glasgow Coma Scale score < 9). Of these, 18 patients (22%) developed ALI. Those who developed ALI were initially ventilated with significantly higher tidal volume (9.5 ± 1 mL/kg vs 10.4 ± 1.1 mL/kg PBW), respiratory rate, and minute ventilation, and more often required vasoactive drugs ($P < 0.05$). The use of high tidal volume (OR, 5.4) and relatively high respiratory rate (OR, 1.8) were independent predictors of acute lung injury ($P < 0.01$).

Jia et al conducted a retrospective analysis of patients who received mechanical ventilation for > 48 hours between 2001 and 2005.[8] Of 789 patients who did not have ARDS at hospital admission, ARDS developed in 152 patients (19%). Multivariable logistic regression showed that peak pressure, high net fluid balance, plasma transfusion, sepsis, and tidal volume were significantly associated with the development of ARDS. The authors concluded that these findings suggest that ARDS may be a preventable complication in some cases.

Based on their review of the literature, Shultz et al recommend avoidance of high plateau pressures and high tidal volumes in patients who do not have ALI or ARDS at the onset of mechanical ventilation.[9] They acknowledge that this recommendation is based on expert opinion and low-level evidence. In the absence of higher levels of evidence, this recommendation appears prudent. Many mechanically ventilated and critically ill patients are at risk of developing ALI. These patients may have lung injury, but do not meet the criteria for

ALI or ARDS. One or more subsequent hits can result in the development of ALI. Inappropriately high tidal volume may initiate or exacerbate pulmonary inflammation, which may induce the primary hit or form a second or third hit.

Yilmaz et al evaluated the effect of low-tidal-volume ventilation and a restrictive transfusion policy on the development of acute lung injury in mechanically ventilated patients.[10] A multidisciplinary team of intensivists and respiratory therapists designed a protocol to limit tidal volume to ≤ 10 mL/kg PBW in all patients receiving invasive ventilation, and a recommendation to use 6-8 mL/kg PBW for patients at any risk of ALI or ARDS. A chart with calculated values of PBW was attached to each ventilator and provided on-line. Comprehensive didactic and web-based teaching was provided to physicians, nurses, and respiratory therapists. An interdisciplinary team of intensivists, surgeons, nurses, and transfusion specialists designed an algorithm for evidence-based transfusion. The frequency of ALI/ARDS was calculated per number of patients ventilated > 48 hours. Data were collected prospectively from 3 adult ICUs from June 2005 to May 2006 and compared with a historical cohort of patients who were treated in the same ICUs before protocol introduction (January to December 2001).

Of 375 patients who met the inclusion and exclusion criteria, 212 were ventilated before and 163 after the interventions. There was a decrease in tidal volume (10.6 mL/kg to 7.7 mL/kg PBW; $P < 0.001$), in peak airway pressure (31 cm H_2O to 25 cm H_2O; $P < 0.001$), and in the percentage of transfused patients (63% to 38%; $P < 0.001$) after the intervention. The frequency of ALI decreased from 28% to 10% ($P < 0.001$). The duration of mechanical ventilation decreased from a median of 5 to 4 days ($P = 0.03$). When adjusted for baseline characteristics in a multivariate logistic regression analysis, protocol intervention was associated with a reduction in the frequency of new ALI (OR, 0.21). The results of this study suggest that many cases of ALI may be iatrogenic and preventable.

Chronically ventilated patients with spinal cord injury are commonly ventilated with very large tidal volumes. The motivation for this strategy seems to be for dyspnea relief and avoidance of atelectasis. However, this strategy has not been rigorously evaluated against a low tidal volume strategy. Although harm related to this approach has not been reported, it is prudent to lower the tidal volume when these patients are admitted to the hospital for an acute illness, as an acute illness may be a second hit (in addition to the larger tidal volume), which could increase the risk for ALI.

Is the Use of Low Tidal Volumes Harmful?

There are several potentially adverse effects of low-tidal-volume ventilation. Lower tidal volumes may increase the likelihood of atelectasis.[11] However, atelectasis may be avoided by use of PEEP, which can decrease not only the risk of atelectasis, but also the risk for ventilator-associated pneumonia.[12] There is also concern for increased ventilator dyssynchrony with tidal volume limitation. When lower tidal volumes are used, it is important to increase the respiratory rate to avoid respiratory acidosis, which increases respiratory drive. Manipulations of ventilator settings, such as inspiratory flow, can also improve patient-ventilator interactions.[13] In some cases, additional sedation may be necessary. One might speculate that the pain and anxiety associated with invasive mechanical ventilation might stimulate the respiratory drive and the sensation of air hunger when tidal volume is limited. It is interesting to note that use of lower tidal volumes in the ARDSnet study was not associated with a greater sedative requirement.[14,15]

Conclusion

High-level evidence strongly supports tidal volume limitation in patients with ALI and ARDS. Although the strength of the evidence is lower for patients who do not have ALI, it seems prudent to limit tidal volume to < 10 mL/kg in all intubated and mechanically ventilated patients. Although no randomized controlled trials have addressed the issue, accumulating lower levels of evidence suggest that lower tidal volumes than those used in the past should be adopted in most patients requiring mechanical ventilation. ■

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CME / CNE 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.

CME / CNE Questions

31. Which of the following is correlated with mortality among patients with pulmonary thromboembolism, according to the studies abstracted in this issue?

a. Both CT angiographic clot burden and ECG score
b. CT angiographic clot burden but not ECG score
c. ECG score but not angiographic clot burden
d. Neither CT angiographic clot burden nor ECG score

32. Which of the following complications occurred in 80% of patients with myasthenic crisis and acute respiratory failure who failed a trial of noninvasive ventilation?

a. Acute respiratory distress syndrome
b. Pulmonary thromboembolism
c. Pneumonia
d. Upper gastrointestinal hemorrhage
e. *Clostridium difficile* colitis

33. Which of the following outcomes is associated with performing tracheostomy within 10 days of initiating mechanical ventilation?

a. A small increase in 90-day mortality
b. A small increase in 28-day mortality
c. A decrease in the number of ventilator free days at 28 days
d. Faster weaning from mechanical ventilation

34. Which of the following statements is correct?

a. High-level evidence supports the use of tidal volumes of 4-8 mL/kg in patients with ALI/ARDS.
b. Lower levels of evidence support the use of tidal volumes on < 10 mL/kg in all invasively mechanically ventilated patients.
c. Lower tidal volumes decrease the risk for acute lung injury.
d. Tidal volume should be based on predicted body weight.
e. All of the above

35. In which of the following settings has the routine use of large tidal volumes been associated with an increased incidence of ALI /ARDS?

a. Patients with known risk factors for ALI/ARDS
b. Patients undergoing pneumonectomy
c. Brain-injured patients
d. All of the above
e. None of the above

Answers: 31. (d); 32. (c), 33. (d), 34. (e), 35. (d).