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SPECIAL FEATURE

Swallowing Dysfunction in Critical Illness

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Dr. Guttendorf reports no financial relationships relevant to this field of study.

Impaired swallowing in patients with critical illness is estimated to occur in about 10-93% of patients.[1-6] The sequela of impaired swallowing is aspiration, either overt or silent, with resulting risks for pneumonia, pneumonitis, acute lung injury, reintubation, malnutrition, and dehydration. Swallowing dysfunction is associated with increased length of stay and poor patient outcomes.[5,6] Patients documented to have swallowing dysfunction incur additional risks related to placement of feeding tubes (nasogastric, nasoduodenal, percutaneous endoscopic gastrostomy) and prolonged enteral nutrition, which may further contribute to aspiration risk.

Swallowing dysfunction after critical illness is in part due to conditions present prior to ICU admission, including neuromuscular disorders (e.g., amyotrophic lateral sclerosis, cerebral palsy, multiple sclerosis, myasthenia gravis, muscular dystrophy, and Parkinson's disease), cognitive dysfunction (e.g., Alzheimer's disease and other dementias, psychiatric diagnoses), and preexisting physical conditions (e.g., head and neck cancer, esophageal disorders).

The elderly are more likely to experience swallowing difficulty. In one study of critically ill elderly patients intubated for ≥ 48 hours, researchers detected aspiration in 52% of patients (age > 65 years) as compared to 36% of patient controls (age < 65 years), and the elderly continued to exhibit persistent swallowing deficits at 2 weeks. In a multivariate analysis, only preadmission functional status was a determinant of delayed resolution of swallowing deficit (hazard ratio [HR], 1.68; 95% confidence interval [CI], 1.26-3.97).[3]

Additional factors related to the etiology for ICU admission and the course of critical illness may contribute to the development of subsequent swallowing dysfunction. For example, patients suffering stroke, facial burns or inhalation injury, trauma, acute alterations of mental status (such as delirium, weakness, and deconditioning), survivors of multiple system organ failure, sepsis, and particularly those experiencing prolonged intubation (usually defined as ≥ 7 days) are at

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higher risk of developing swallowing dysfunction.[1,2,7]

In one study of acute and long-term dysphagia in sepsis patients at 14 days, sepsis patients showed significantly more aspiration than non-sepsis patients (P = 0.002). Both severe sepsis and tracheostomy were independent risk factors for severe dysphagia with aspiration at 14 days, and mortality at 4 months was significantly higher in the sepsis group (P = 0.006).[7]

The duration of intubation contributes significantly to the risk of developing swallowing dysfunction. Brodsky et al evaluated the association between patient-reported aspiration and the duration of endotracheal intubation in acute lung injury patients. In a multivariate regression analysis, duration of oral intubation was associated with dysphagia symptoms (HR, 1.79; 95% CI, 1.15-2.79) for the first 6 days, but additional days did not contribute additionally to dysphagia risk.[8] Kim et al demonstrated similar findings in patients with non-neurologic critical illness. In a multivariate regression analysis, duration of endotracheal intubation was associated significantly with post-extubation aspiration (HR, 1.09; 95% CI, 1.01-1.18; P = 0.04).[9]

The number of patients treated with mechanical ventilation is significant and continues to increase each year. A retrospective cohort study to determine the incidence of mechanically ventilated patients in 1 year across six states demonstrated more than 180,000 patients received mechanical ventilation and more than 40% of those had intubations of > 96 hours.[10] Given this high incidence of acute respiratory failure requiring intubation and mechanical ventilation, the associated burden of swallowing dysfunction in these patients also is expected to be proportionally high.

MECHANICS OF SWALLOWING

Swallowing is a complex function, requiring the coordinated effort of more than 30 muscles, innervated by several peripheral and cranial nerves, and interfaced with the swallowing center in the medulla of the brainstem.[11] Swallowing is divided into four sequential phases: oral preparatory phase, oral transport phase, pharyngeal phase, and esophageal phase. A key portion of the swallowing process is to functionally protect the airway. During the pharyngeal phase, which is timed with the start of exhalation, respiration briefly ceases, the vocal folds close, and the epiglottis mobilizes to deflect the food bolus toward the esophagus.[11] Aspiration of food or fluids into the airway can lead to pneumonia, pneumonitis, and acute lung injury, extensive morbidity, and even aspiration-associated mortality. When clinically apparent, aspiration is associated with coughing, sputtering, or choking while drinking or swallowing. However, often aspiration is not clinically apparent and presents as occult or "silent aspiration" with the same adverse consequences.

TESTS FOR THE EVALUATION OF SWALLOWING

Tests commonly used in the clinical setting to evaluate swallowing function include:

Provider-observed Water Swallow Test: Nurse or other bedside care provider observes the patient swallowing a small amount of water, and makes note of any evidence of aspiration (coughing, choking, sputtering).[12]

Bedside Swallow Evaluation (BSE): A speech language pathologist (SLP) performs a clinical bedside screening and evaluation, which involves a patient interview, directed physical exam, and evaluation for aspiration during bedside swallowing trials of different consistency liquids.[12]

One primary limitation to clinical BSE is that it can fail to identify silent aspiration, as this still relies on a patient developing a cough, gag, or gurgling sound upon aspiration. This prompts some clinicians to consider either a fiberoptic endoscopic evaluation of swallowing (FEES) or a modified barium swallow study (MBSS) in all patients considered to be at high risk of aspiration based on preexisting conditions or clinical conditions in the ICU (duration of intubation, mental status, etc.).
While there can be some variability in the BSE reliability, in one study of 16 burn patients, the clinical BSE was predictive

of a subsequent abnormal modified barium swallow study.[4] Eleven patients had an abnormal clinical swallow exam and underwent subsequent MBSS, which revealed abnormal swallowing in 10 of the 11 patients, identifying either oral dysphagia, pharyngeal dysphagia, esophageal dysphagia, or a combination.[4]

Fiberoptic Endoscopic Evaluation of Swallowing: Passage of a small caliber videoscope through the nares to visualize in real time the passage of food through the mouth, pharynx, larynx, and upper esophagus.[12,13] FEES provides a view of the anatomic structures, secretion burden, secretion management, and the sensory function. Two primary advantages of the FEES over a MBSS are that the FEES exam can be performed at the bedside, and that it offers the additional benefit of evaluating vocal fold mobility, which is important for prevention of aspiration.

Modified Barium Swallow Study (also referred to as Videofluoroscopic Swallow Study [VFSS]): Under fluoroscopy, the patient swallows barium-containing foods/liquids per an established protocol for progression, with guidance by a SLP, and a radiologist reviews the videography. The MBSS provides better evaluation of the oral phase of swallowing and upper esophageal dysfunction than FEES, but is limited because it requires patient transport from the ICU to the radiology suite and exposes the patient to radiation.[12,13]

SCREENING AND EVALUATION OF SWALLOWING

Evaluation of swallowing usually proceeds in a tiered fashion. Soon after extubation and before beginning oral intake, patients are first screened for high-risk features for potential aspiration (e.g., preexisting conditions, prolonged intubation/mechanical ventilation, altered mental status). If deemed safe to do so, begin with a provider-observed water swallow test. If the patient fails the provider-observed swallow, a repeat evaluation can be performed within the next 12-24 hours. Significantly high-risk patients can bypass the water swallow test.

In patients deemed significantly high risk for aspiration based on initial screening, or in those failing the water swallow test, the next tier would be a BSE. Patients who pass the BSE without overt evidence of aspiration may be trialed with an oral diet. Patients who fail the BSE should be further evaluated with either a FEES or MBSS. A change in level of alertness should prompt reevaluation. Evaluation of swallowing should be a multi-disciplinary approach, involving nurses, advanced practice providers, physicians, and SLPs. Should swallowing difficulties occur, physicians may try other interventions, such as modification of dietary texture, position changes (chin tuck, head rotation), and other compensatory mechanisms to reduce risk during swallowing (employing multiple swallows, breath holding, etc.).[12,13] An SLP should direct reevaluation for improvement and progression.

Given the growing numbers of mechanically ventilated patients and the high incidence of aspiration associated with swallowing dysfunction, maintaining a high index of suspicion for the presence of possible swallowing dysfunction is key for the clinician. Since there is wide variability in reports of the incidence of swallowing dysfunction, consider screening all patients who have been intubated, regardless of duration of intubation. Formally evaluate swallowing in all patients with high risk for dysfunction based on baseline clinical features as well as those with prolonged intubation, altered mental status, and significant risk for morbidity and mortality should aspiration ensue. Failure of the water swallow test and the BSE to detect silent aspiration should prompt a more definitive evaluation with either FEES or MBSS. ■

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ABSTRACT & COMMENTARY

New Insights Into Alveolar Mechanics and Gas Exchange in Experimental Acute Respiratory Distress Syndrome

By Richard Kallet, MS, RRT, FAARC, FCCM

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Mr. Kallet reports no financial relationships relevant to this field of study.

SYNOPSIS: Experimental acute respiratory distress syndrome was induced in mice to study local alveolar gas dynamics using advanced microimaging techniques. Heretofore unrecognized disturbances in alveolar ventilation will alter our understanding of alveolar mechanics and gas exchange dysfunction as well as promote the use of recruitment maneuvers.

SOURCE: Tabuchi A, et al. Acute lung injury causes asynchronous alveolar ventilation which can be corrected by individual sighs. *Am J Respir Crit Care Med* 2015;Oct 29 [Epub ahead of print].

Researchers induced experimental acute respiratory distress syndrome (ARDS) mimicking aspiration, transfusion reaction, and surfactant inactivation in mice and observed the short-term effects on subpleural alveolar clusters using intravital and dark field microscopy and multi-spectral O_2 saturation imaging. Within 10 minutes of inducing ARDS, pendelluft motion (PM) replaced synchronous alveolar inflation/deflation. This was characterized by alveoli that slowly inflated during expiration and deflated during inspiration. This behavior occurred both dynamically as well as during imposed end-expiratory and end-inspiratory pauses. Alveoli immediately adjacent to alveoli with PM tended to show larger volume changes compared to nearby alveoli, and both continued to ventilate normally. PM resolved spontaneously in the majority of cases within 60 minutes.

The magnitude of PM reflected injury severity. PM occurred with similar frequency regardless of injury mechanism and, in general, did not adversely affect alveolar gas exchange or local O_2 saturation. However, severe dysfunction occurred more commonly in acid-induced ARDS (affecting approximately 50% of alveoli) and resulted in marked gas exchange dysfunction. Recruitment maneuvers (RMs) of 30 cm H_2O for 10 seconds reversed alveolar synchrony and improved gas exchange in acid-induced ARDS but had no effect on the surfactant-deficient model.

■ COMMENTARY

This study enriches our understanding of pulmonary micromechanics and gas exchange dysfunction in ARDS, supports the hypothesis of Otis et al regarding the existence of PM,[1] and advances our understanding of how RMs stabilize alveoli and improve oxygenation. In particular, the authors provided a detailed description of heretofore unrecognized, nuanced variations in alveolar filling and emptying.

Sixty years ago, Otis et al reasoned that PM occurs between proximal alveolar units when differences develop in their respective time constants (the product of resistance and compliance) such that injured units with a relatively brief time constant (e.g., low compliance the predominant disturbance) reach equilibration faster. As a result, these "fast units" empty into nearby, relatively normal alveoli (which have higher compliance) still undergoing inflation. The Tabuchi et al study confirms the existence of PM at the alveolar level and provides more detailed insight into alveolar gas dynamics occurring throughout the ventilatory cycle as well as persisting during end-inspiratory and end-expiratory pauses.

A striking finding was the apparent progression from PM to alveolar stunning in severe ARDS induced by acid-injury. Authors described this as cessation of ventilation in alveoli that intriguingly maintained a fixed volume. However, the authors operationally defined alveolar stunning as alveolar volume change < 25% of its individual baseline. Therefore, some degree of ventilation or bidirectional gas diffusion may persist in these units. Both the cause and gas exchange implications of alveolar stunning remain speculative at best. Severe injury progressing to alveolar flooding is the most obvious explanation, save for the fact that discreet alveoli are involved without causing severe injury to neighboring alveoli. Other possible explanations are alveolar obstruction by liquid bridge formation, interstitial edema, tethering by adjacent alveoli, or some combination of these.

Whereas PM is readily apparent as a source of severe ventilation-perfusion mismatch (and hence increased dead-space ventilation when CO_2 is the tracer gas),

the effect of alveolar stunning on gas exchange remains less obvious. This would partly depend on the intensity of hypoxic pulmonary vasoconstriction, the strength of which is heterogeneously distributed throughout the lungs and appears to fail once local alveolar PO_2 is < 50 mmHg.[2] That would invite speculation of a shunt-like effect. Moreover, the corrective effect of RM in this study suggests that obstruction of alveolar units and/or pulmonary edema might explain the source of alveolar stunning. Finally, the fact that only subpleural alveoli could be examined in this study limits its generalization to overall mechanical and gas exchange dysfunction in ARDS. The heterogeneous nature of clinical lung injury and its distribution and the effects of chest wall compliance, gravitational forces, pulmonary vascular injury, and other numerous factors make definitive pronouncements regarding the pathophysiologic mechanisms of ARDS elusive. Nonetheless, the emerging picture has become ever more fascinating. ■

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ABSTRACT & COMMENTARY

Effects of a Rapid Response System Driven by Real-time Automated Clinical Alerts on Hospital Mortality and Length of Stay

By Samuel Nadler, MD, PhD

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Dr. Nadler reports no financial relationships relevant to this field of study.

SYNOPSIS: The addition of an automated real-time clinical deterioration alert system to a rapid response system had marginal effects.

SOURCE: Kollef MH, et al. Mortality and length of stay trends following implementation of a rapid response system and real-time automated clinical deterioration alerts. *Am J Med Qual* 2015 Nov 13 [Epub ahead of print].

Triage of medical admissions is imprecise, and patients on general medical wards may deteriorate, requiring higher levels of care. Early detection and treatment of these individuals should improve outcomes, but current staffing models preclude clinical staff from continuously monitoring every patient. Development of an automated, real-time system could assist in identifying those high-risk patients. Kollef et al previously published a prospective study over a 6-month period that demonstrated rapid response system (RRS) activations did not reduce ICU transfers, mortality, or the need for long-term placement, but did decrease hospital length of stay (LOS).[1]

This study retrospectively examined trends in hospital mortality, rates of cardiopulmonary arrests (CPAs), hospital LOS, and RRS activations from 2003-2014, before, during, and after the implementation of automated real-time clinical deterioration alerts (RTCDAs). The RRS was implemented between 2006 and 2008 and RTCDAs started in 2009. The RTCDAs monitored 36 input variables with heaviest weighting of respiratory rates, oxygen (O_2) saturation, shock index, systolic and diastolic blood pressure, heart rate, and coagulation modifiers. During this period, researchers monitored 163,311 consecutive patients. Linear regressions identified study year as an independent determinant of hospital mortality (r = - 0.794, P = 0.002), CPAs (r = - 0.792, P = 0.006), and LOS (r = - 0.841, P = 0.001). Accordingly, RRS activations increased (r = 0.997, P < 0.001).

■ COMMENTARY

At first glance, this study seems to demonstrate improvements in important clinical outcomes with the implementation of an automated alert system. However, closer examination of the data calls this conclusion into question. The linear regression models examined the 11-year study period as a whole. During this period, there does appear to be a reduction in mortality and CPAs with an increase in RRS activations. This effect is most pronounced with the development of the RRS system from 2005-2008. After the automated RTCDAs started in 2009, although the rates of RRS activations increase dramatically (~170 to > 400), if anything, there is a mild increase in hospital mortality. The rates of CPAs increased

from 2010-2011 before declining once again 2012-2014. The hospital LOS is lowest in 2009 and increased through 2011 before once again decreasing. If the linear regression were calculated during the period of time when the RTCDA system was active from 2009-2014, it is not clear that there would be a significant positive association.

How does this study alter our knowledge of RRSs and RTCDAs to improve clinical outcomes? It further supports the notion that RRSs improve clinical outcomes. The most recent meta-analysis demonstrated that RRSs reduce hospital mortality and rates of CPAs.[2] But the automated system in this study did not seem to improve outcomes beyond that observed with RRSs alone. An additional 200 RRS activations did not seem to affect outcomes. The RRS itself may have functioned well enough that most instances of real clinical deterioration were detected. It leaves open the question whether the automated system alone may have worked as well. Further, the automated system itself may not be sensitive enough to improve clinical outcomes. Bailey et al separately published the operating characteristics a real-time alert system within the same system.[3] That study reported good sensitivity for ICU transfers and mortality (89.6% and 89.2%, respectively), but the positive predictive values were poor (15.2% and 10.4%, respectively), as these events were relatively rare. Thus, many alerts occurred when a clinical deterioration may not have happened.

Increasing evidence shows RRSs improve outcomes. The addition of RTCDAs in this study did not seem to add to this pre-existing system. Considering the diversion of time and resources these additional alerts caused, they do not seem to confer an advantage. Only with an improved RTCDA would this system improve clinical outcomes. ■

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ABSTRACT & COMMENTARY

Does the Use of Saline vs Buffered Crystalloid Reduce Risk of Acute Kidney Injury in ICU?

By Kathryn Radigan, MD

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Dr. Radigan reports no financial relationships relevant to this field of study.

SYNOPSIS: The use of a buffered crystalloid compared with saline did not reduce the risk of acute kidney injury (AKI) in patients receiving crystalloid fluid therapy in the ICU.

SOURCE: Young P et al. Effect of a buffered crystalloid solution vs saline on acute kidney injury among patients in the intensive care unit. *JAMA* 2015;314:1701-1710.

The use of saline (0.9% sodium chloride) in critically ill patients is a common intervention to increase intravascular volume or maintain hydration. Although the use of saline in the critically ill is widespread, there is concern that the high chloride content of saline contributes to the development of acute kidney injury (AKI), and its use may be associated with an increased risk of mortality. It is unclear whether a buffered crystalloid solution with an electrolyte composition that more closely resembles plasma would lead to better outcomes.

Since the relationship between the use of saline in critically ill patients and renal failure is unclear, Young et al pursued a double-blind, cluster randomized, double-crossover trial from April 2014 through October 2014 to determine the effect of a buffered crystalloid compared with saline on renal complications in ICU patients. The trial took place in four New Zealand ICUs and included all patients admitted to the ICU who required crystalloid fluid therapy. The trial excluded patients with established AKI requiring renal replacement therapy (RRT) or expected to require RRT within 6 hours. Most patients were admitted to the ICU following elective surgery, most commonly cardiovascular surgery, and few had

comorbidities. Of the 2278 eligible patients enrolled in the study, researchers analyzed 1152 of the 1162 patients (99.1%) receiving buffered crystalloid and 1110 of the 1116 patients (99.5%) receiving saline. Researchers randomized two of four ICUs to saline intervention and the other two to buffered crystalloid for alternating treatment blocks of 7 weeks. Two crossovers occurred so that each ICU used one of two study fluids twice over the 28-week period. The treating physician determined the rate and frequency of fluid administration. The primary outcome was proportion of patients with AKI (defined as a rise in serum creatinine level of at least two-fold or a serum creatinine level of ≥ 3.96 mg/dL with an increase of ≥ 0.5 mg/dL). The incidence of RRT and in-hospital mortality were the main secondary outcomes.

For the patients who received buffered crystalloid, 102 developed AKI compared with 94 in the saline group (absolute difference [AD], 0.4%; 95% confidence interval [CI], -2.1% to 2.9%; relative risk [RR], 1.04; 95% CI, 0.80-1.36; $P = 0.77$). Similarly, for patients who received buffered crystalloid, RRT was required in 38 (3.3%) compared with 38 (3.4%) in the saline group (AD, -0.1%; 95% CI, -1.6% to 1.4%; RR, 0.96; 95% CI, 0.62-1.50; $P = 0.91$). Additionally, there was no difference in mortality between the buffered crystalloid and saline groups (7.6% vs 8.6%; AD, -1.0%; 95% CI, -3.3% to 1.2%; RR, 0.88; 95% CI, 0.67-1.17; $P = 0.40$). In summary, for patients who received crystalloid fluid therapy in the ICU, the use of a buffered crystalloid compared with saline did not reduce the risk of AKI, RRT, or in-hospital mortality.

■ COMMENTARY

The administration of IV fluids for hydration and resuscitation is common in critically ill patients. Many different types of IV fluid, including normal saline, contain supraphysiological concentrations of chloride. Excessive chloride administration is associated with hyperchloremic metabolic acidosis and may lead to renal vasoconstriction along with a reduced GFR.[1]

Since renal failure in the ICU is associated with increased hospital mortality,[2] there has been substantial interest in examining the relationship between the high chloride content of saline and the development of AKI. In a single-center, open-label study of chloride-rich (n = 760) vs chloride-restrictive (n = 773) fluids in the ICU, Yunos et al found that implementation of a chloride-restrictive strategy in a tertiary ICU was associated with a significant decrease in the incidence of AKI and use of RRT. Unfortunately, physicians should interpret cautiously the outcomes of this trial, as one of the alternative IV fluids used was a synthetic gelatin-based colloid that has been associated with increased risk of AKI in patients with sepsis.[1] Other recently conducted studies, including a retrospective study and a meta-analysis, favored balanced fluids, but results were tempered in light of the innate design of the trials.[3,4] In light of the remaining question, it was expected that this double-blind, cluster randomized, double-crossover trial might finally achieve some clarity on the subject.

Unfortunately, the trial revealed that there was no difference in AKI and mortality between patients who received 0.9% saline vs buffered crystalloid. Although results of the trial appeared to be definitive, there were aspects of the trial design worth noting. First, 90% of patients received fluids prior to admission to the ICU; the majority of this IV fluid was buffered crystalloid with 1.2 L administered to the buffered crystalloid group and 1 L of buffered crystalloid administered to the saline group. Although 1 L of fluid does not appear to be significant, the average fluid administration between groups over the entire study period was only 2 L. This low volume of fluid may be insufficient to demonstrate a hazard, especially in study groups that are lower risk and may not be representative of a typical ICU patient population since the majority of patients were surgical. Second, the design of the trial may require more consideration. Although this trial anticipated examining the effect of IV fluids on AKI, RRT, and mortality, the design of the study was not based on this particular intervention. For instance, the treating physician determined the rate and frequency of fluid administration but the reason for the fluid administration was unknown. Therefore, the effectiveness of the fluid provided for each indication could not be measured, and researchers measured the overall adverse events instead.

Although this study fails to reveal an ideal fluid management strategy for our high-risk, severely septic patients who will need aggressive fluid administration, it does emphasize that neither 0.9% saline nor a low-chloride electrolyte IV fluid is particularly hazardous in a lower-risk patient population receiving on average 2 L of fluid. Until further studies examine a higher-risk patient population, there is insufficient evidence to support the use of one type of IV crystalloid fluid vs another. Therefore, it is our responsibility to continue to be thoughtful regarding the administration of fluids in our patients on a case-by-case basis. ■

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analysis. *Crit Care* 2006;10:R73.
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CME/CE QUESTIONS

1. **Which of the following is associated with swallowing dysfunction after critical illness?**
   a. Baseline neuromuscular disorder
   b. Alteration in mental status
   c. Prolonged orotracheal intubation
   d. Elderly age
   e. All of the above

2. **Which of the following is true regarding the study on alveolar ventilation dynamics?**
   a. Otis' theory of alveolar Pendelluft motion was disproven.
   b. Pendelluft motion was a common response to experimentally induced acute lung injury.
   c. Pendelluft motion persisted for hours following injury.
   d. The mere presence of Pendelluft motion did not necessarily cause gas exchange dysfunction.
   e. Both b and d

3. **In the study of a rapid response system augmented by real-time clinical deterioration alerts, implementation of a rapid response system and automated alerts:**
   a. were causally linked to improved outcomes.
   b. were temporally associated with improved outcomes.
   c. had no effect on clinical outcomes.
   d. were not evaluated.
   e. None of the above

4. **Neither 0.9% saline nor a low-chloride electrolyte IV fluid is particularly hazardous in a lower-risk patient population receiving on average how much fluid?**
   a. 2 L
   b. 6 L
   c. 8 L
   d. 10 L
   e. None of the above

CME/CE OBJECTIVES

Upon completion of this educational activity, participants should be able to:

- identify the particular clinical, legal, or scientific issues related to critical care;
- describe how those issues affect physicians, nurses, health care workers, hospitals, or the health care industry; and
- cite solutions to the problems associated with those issues.

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