

Critical Care [ALERT]

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ABSTRACT & COMMENTARY

A Novel Prediction Tool for Hypoxemia During ICU Intubation

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Drs. Veerabattini and Niven report no financial relationships relevant to this field of study.

SYNOPSIS: The authors of this retrospective analysis of data from two prospective randomized trials of tracheal intubation developed the AT RISK (Age, Trainee, Race, Indication, SpO₂, Kg/m²) score to identify patients who may develop severe hypoxemia during this procedure. An AT RISK score of < 2 carried a high negative predictive value of procedural hypoxemia, while nearly half of patients with a score > 3 experienced an SpO₂ < 80%.

SOURCE: McKown AC, Casey JD, Russell DW, et al. Risk factors for and prediction of hypoxemia during tracheal intubation of critically ill adults. *Ann Am Thorac Soc* 2018; Aug 15. doi: 10.1513/AnnalsATS.201802-118OC. [Epub ahead of print].

Complications from endotracheal intubation in the critically ill remain unacceptably high. Well-designed clinical trials over the past decade have revealed hypoxemia rates of 20% despite optimal patient positioning and preoxygenation.^{1,2} Severe hypoxemia has been associated with the need for emergent surgical airway, anoxic brain injury, prolonged ICU stay, and death. Poor prospective identification of at-risk patients, inadequate preprocedural planning, and lack of communication have been identified as risk factors for adverse airway events.³ The MACOCHA score offers the best validated tool to identify critically ill patients at risk for difficult intubation, but clinical adoption has

been limited by its length and complexity.⁴ Investigators conducted a retrospective analysis of 442 tracheal intubations from two previously published prospective randomized trials to identify critically ill patients at risk for hypoxemia during this procedure. The first was a single-center trial of 150 intubations randomized to direct or video laryngoscopy, with further factorialized randomization to apneic vs. no apneic oxygenation. The second was a multicenter trial of 292 intubations randomized to a sniffing or modified ramped position, co-enrolled with randomization to use of usual care or a written preprocedural checklist. No intervention demonstrated a treatment effect. The database for this retrospective

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study included all patients > 18 years of age requiring endotracheal intubation in the ICU, with a supervised pulmonary and critical care fellow or anesthesiology trainee performing almost all initial procedures. The primary outcome was the lowest arterial oxygen saturation (SpO₂) from induction medication administration until two minutes after induction, which independent observers collected in real time in all cases.

Only nine patients were excluded from the original study databases because of incomplete pulse oximetry data, leaving 433 intubation events for analysis. Potential predictors of hypoxemia were selected *a priori*. Investigators developed a linear regression model. To create the model, investigators used covariates of race, gender, body mass index (BMI), age, use of noninvasive ventilation (NIV) or maximum fraction of inspired O₂ in the preceding six hours, APACHE II scores, indication for intubation (hypoxemic or hypercarbic respiratory failure or other), presence of sepsis, and operator experience. Then, the authors created a logistic regression model to identify risk factors for severe hypoxemia (defined as SpO₂ < 80% or a decrease in SpO₂ of > 10% for patients with an SpO₂ at induction < 90%) with the same covariates (except for recent FiO₂ or NIV use, sepsis, or APACHE II scores). The investigators performed multiple sensitivity analyses to assess for confounding variables. Finally, the authors compiled all significant variables from the severe hypoxemia logistic regression model into a point system for bedside risk-stratification using a penalized maximum likelihood estimation to improve generalizability.

Seventy-five percent of patients in the cohort had sepsis or septic shock, and 36% were intubated for hypoxemic respiratory failure. The median APACHE II score was 22 (interquartile range, 17-26). Critical care and pulmonary fellows performed almost all intubations. The authors used a linear multiple regression model to compare the 75th percentile to 25th percentile for each continuous variable. They found higher BMI, ethnicity other than black, younger age, hypoxemic respiratory failure as an indication for intubation, lower SpO₂ at induction, and more limited operator intubating experience to be independently associated with a lower SpO₂ during intubation. These factors persisted as independent predictors of severe hypoxemia

in the simplified logistic multiple regression model, with septic shock the only other independent predictor identified in the sensitivity analysis. Of these risk factors, the presence of hypoxemic respiratory failure and SpO₂ at induction were the strongest predictors of severe hypoxemia during intubation.

Simplification of a penalized maximum likelihood estimation algorithm resulted in the creation of the six-point AT RISK score. The authors assigned one point each to **A**ge < 50 years, **T**rainee (operator with < 100 prior intubations), **R**ace other than black, **I**ndication (hypoxemic respiratory failure), **S**pO₂ at induction < 94%, and **K**g/m² (BMI) > 35. Based on this predictive model, less than 2% of patients with an AT RISK score < 2 will develop significant desaturation during intubation (negative predictive value, 98.1%; 95% confidence interval [CI], 93.2-100). Nearly half of patients with a score > 3 will experience severe hypoxemia (positive predictive value, 47.4%; 95% CI, 37.1-59.1).

■ COMMENTARY

Benjamin Franklin's adage that "an ounce of prevention is worth a pound of cure" is especially true of tracheal intubation in the critically ill. Often, intubation in the ICU is emergent, limiting opportunities for systematic airway assessment and using anatomic features with limited predictive power to identify a difficult airway.⁵ Patients often present with significant cardiopulmonary disease, hemodynamic instability, and a full stomach, which makes preoxygenation difficult and the risk of desaturation and aspiration during intubation attempts high. Unfortunately, these factors are not under the practicing intensivist's control. The 4th National Audit Project of the Royal College of Anaesthetists and The Difficult Airway Society was the largest of a series of studies that have consistently identified an association between airway management complications and the lack of systematic preoxygenation, airway equipment and training, inconsistent communication and teamwork, and the absence of a clearly articulated backup plan if initial intubation attempts fail.³ The intubation team can control these factors and offer an important opportunity to improve outcomes from ICU intubation.

The McKown et al study is an important step toward more effective, early identification of patients at increased risk of severe

hypoxemia who can benefit most from deliberate pre-oxygenation efforts, teamwork, and planning to prevent this potentially life-threatening complication. Strengths of the AT RISK score include the high-quality methods used to collect the data employed for this study, the systematic data analysis, and the simplicity and performance characteristics of this tool. The similarity between many elements of the AT RISK and MACOCHA scores also strengthens the external validity of these results.⁴

As with any retrospective study, implementation of the AT RISK tool without prospective validation using a new patient cohort should be undertaken with caution. The reasons that younger patients were at greater risk for severe hypoxemia are difficult to discern from this data analysis and underline the importance of a systematic and deliberate approach to intubation planning and preparation, regardless of the patient and perceived physiologic reserve. The authors went to great lengths to discuss the effect of intubator experience on severe hypoxemia. It is difficult to draw meaningful conclusions regarding training or practice based on these data alone. There is significant literature suggesting that even experienced airway managers receive limited systematic training in emergent airway management in the critically ill. The difference in lowest oxygen saturation seen between intubators with 50 and 85 prior procedures in the McKown et al study was clinically insignificant. Efforts to use a systematic curriculum that employed a blend of didactics, simulation, and supervised clinical experience in pulmonary and critical care fellows with lower intubation volume have yielded equivalent outcomes and complication rates. This suggests procedure numbers alone should not be used as a sole criterion for competence, credentialing, and procedural scope of practice.⁶

These findings underline the fact that obese patients with hypoxemic respiratory failure are perhaps the greatest at-risk population of critically ill patients for airway management. Deliberate preoxygenation, preprocedural planning, and preparation in this setting also are vital. Unfortunately, the incidence of severe hypoxemia seen in even these high-quality clinical trials using best practices to minimize patient hypoxemia during intubation remains unacceptably high. Given the conflicting data with

previous commonly held practices of apneic oxygenation, the “ramped” position, and the use of intubation checklists in ICU patients, we look forward to other researchers using the AT RISK tool in future studies to identify better strategies to reduce the risk of intubation in the critically ill.⁷⁻¹² ■

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Should We Use Early Physical Therapy for Respiratory Muscles?

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SYNOPSIS: Mechanically ventilated patients undergoing inspiratory muscle training demonstrated significant increases in both maximum inspiratory and expiratory muscle pressures; the training was associated with reduced weaning duration of 2.3 days.

SOURCE: Vorona S, Sabatini U, Al-Maqbali S, et al. Inspiratory muscle rehabilitation in critically ill adults. A systematic review and meta-analysis. *Ann Am Thorac Soc* 2018;15:735-744.

This systematic review included 28 studies, of which 71% were randomized, controlled trials, and which included 1,185 adult patients. At baseline, patients exhibited substantial loss of inspiratory muscle strength (IMT), with a mean maximum inspiratory pressure (MIP) of 15-54 cm H₂O, which is approximately 10-35% of normal laboratory values.¹ Numerous IMT techniques were used, including strength vs. endurance conditioning (i.e., < 200 vs. > 200 repetitions/session) or physical therapy aimed at increasing minute ventilation demand and, therefore, respiratory muscle power output (work per minute). Most studies used threshold loading rather than resistive loading techniques. Commencement of IMT varied widely (ranging from 24 hours after intubation to only after initial weaning trial failure), as did the duration of therapy (ranging from three days to six weeks). However, these differences appeared to not affect therapeutic efficacy.

In the randomized, controlled trials, mean MIP increased by 40% in the IMT arm vs. 18% in controls. Mean maximum expiratory pressure (MEP), which represents abdominal muscle strength, increased by 63% vs. 17% in controls. MEP is a signifier for cough strength and is an indirect accessory muscle of inspiration during loaded breathing. However, both findings represented only a modest increase in strength at 6 cm H₂O (95% confidence interval [CI], 5-8 cm H₂O) and 9 cm H₂O (95% CI, 5-14 cm H₂O), respectively. Nevertheless, after excluding studies with serious risk for bias, there was a clinically significant reduction in weaning duration by 2.3 days. All studies concluded that IMT in critically ill patients is feasible with only a very rare incidence of serious adverse effects (e.g., bradycardia).

■ COMMENTARY

The authors are to be commended for drawing our attention to a peculiar paradox in pulmonary and critical care medicine. For decades, researchers have been preoccupied with weaning strategies and ventilator

dependence. However, we largely have ignored a crucial aspect of skeletal muscle functionality — namely, improving strength and endurance in debilitated patients requires systematic retraining. Although we reflexively accept this in terms of physical therapy for other skeletal muscle groups, the critical care community has never embraced this concept seriously.

Interestingly, this perplexing situation has coincided with decades-long misperceptions regarding respiratory muscle function in the critically ill, despite the revolution in our understanding of respiratory muscle physiology that began in the mid-1970s. Perhaps our greatest advancement in clinical practice was the return to spontaneous breathing trials, which irrefutably confirmed what previously was evident, but generally has not translated into clinical practice: Most mechanically ventilated patients (approximately 70%) exhibit little if any problem resuming unassisted breathing.² However, this still leaves a substantial percentage of patients requiring a structured program of respiratory muscle rehabilitation to improve patient-centered clinical outcomes.

In essence, most patients with substantial abnormalities in chest mechanics should be able to resume unassisted breathing when their minute ventilation demand comes down to a threshold whereby their respiratory muscles can handle imposed resistive and elastic loading. In other words, when the minute ventilation approaches or falls below 10 L/minute (suggesting reasonable respiratory muscle power output demands), failure to resume unassisted breathing represents an imbalance between work-per-breath (load) and muscle strength (capacity). When other correctable factors (positive fluid balance, pleural effusion, ascites, nutritional status, etc.) are addressed, then improving strength becomes the most obvious focus for treatment. Most mechanically ventilated patients suffer not only from slowly resolving airway and parenchymal inflammation, but also from respiratory muscle dysfunction.

The most salient finding by Vorona et al is that despite the diverse approaches to IMT, the mean reductions in weaning duration approximates those found in mechanical ventilation duration in randomized, controlled trials whose authors examined spontaneous breathing studies and daily sedation interruptions (i.e., 1.5- to two-day reductions). In addition, the Vorona et al study underscores the fact that no consensus exists on how respiratory muscle rehabilitation should be approached.

Areas that need to be explored include: the timing of IMT initiation, frequency and duration of treatments, how quickly to accelerate muscle loading, and whether and what combinations of strength vs. endurance conditioning should be used. Of paramount importance in both the acute and recovery phases of respiratory failure is what constitutes an appropriate balance between maintaining and improving respiratory muscle strength and providing adequate respiratory muscle rest and

recovery? This becomes particularly important during rehabilitation as weakened muscles are highly susceptible to fatigue. Chronic exposure to excessive workloads produces respiratory muscle inflammation and damage, contributing to ventilator dependence.¹ This last point is something that often eludes clinicians' consideration and a point Vorona et al emphasized. All these issues should be addressed in a systematic fashion to continue the progress already made in weaning and sedation practices that have produced substantial reductions in both the duration of mechanical ventilation and length of stay in the intensive care setting. ■

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ABSTRACT & COMMENTARY

Severe Sepsis and Septic Shock Early Management Bundle

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

SYNOPSIS: When the Severe Sepsis and Septic Shock Early Management Bundle was used to identify patients with severe sepsis or patients in septic shock, delays in lactate measurements for patients with abnormal lactate levels were associated with delayed initiation of antibiotic therapy and increased mortality.

SOURCE: Han X, Edelson DP, Snyder A, et al. Implications of Centers for Medicare & Medicaid Services Severe Sepsis and Septic Shock Early Management Bundle and initial lactate measurement on the management of sepsis. *Chest* 2018;154:302-308.

Sepsis remains the top cause of in-hospital death. Early recognition and timely treatment of sepsis through care bundles has become a priority. The Centers for Medicare & Medicaid Services (CMS) introduced the Severe Sepsis and Septic Shock Early Management Bundle (SEP-1) in October 2015. SEP-1 pinpoints patients with “severe sepsis” by selecting patients who meet two of four systemic inflammatory response syndrome (SIRS) criteria, demonstrate a documented suspicion of infection within a six-hour period, and exhibit one new organ dysfunction. Compliance with the bundle is measured by obtaining blood cultures, initiating antibiotics, and checking serum lactate between six hours before and three hours after presentation with a repeat lactate six hours later if elevated on initial draw. To characterize those who were affected and to analyze the implications of SEP-1 on patient care and outcomes, Han et al conducted a retrospective trial including 5,762 adult patients admitted to the University of Chicago from

November 2008 through January 2016. All patients met one of the International Classification of Diseases, Ninth Revision (ICD-9) codes specified by SEP-1. Time to lactate draw, along with antibiotic and IV fluid administration, were measured. Additionally, researchers assessed in-hospital mortality.

Lactates were checked within the appropriate period 32% of the time on the ward (n = 505), 55% in the ICU (n = 818), and 79% in the ED (n = 2,144). Mortality increased with higher initial lactate levels across all locations. If lactate measurement was delayed, these patients exhibited the highest in-hospital mortality (29%) and were associated with an increased time to antibiotic administration (median time, 3.9 vs. 2.0 hours). The odds of death increased with every additional hour delay in lactate measurement for patients with initial lactate > 2.0 mmol/L (odds ratio [OR], 1.02; 95% confidence interval [CI], 1.0003-1.05; P = 0.04). Delays in initial

lactate measurement that were found to be elevated were associated with delayed fluid administration, delayed antibiotics, and increased mortality.

■ COMMENTARY

Severe sepsis accounts for almost 10% of all deaths.¹ Sepsis bundles have been implemented to decrease mortality. Despite the SEP-1 criteria, little is known regarding the consequences of delayed lactate measurement, especially in areas of the hospital outside the ED.

Interestingly, the Han et al study revealed that lactate measurement may make a significant difference for patients with sepsis and septic shock. This is not surprising, as lactate clearance has remained a fundamental goal in sepsis management. Patients with elevated values often receive more aggressive and timely resuscitation. Delayed lactate measurement not only affects length of stay (LOS), but more importantly it affects overall mortality.

Patients who underwent a lactate draw within the SEP-1 window experienced the shortest LOS (median, 11 days; interquartile range [IQR], 7-19 days; $P < 0.01$). LOS was longest for patients who never received a lactate measurement (median, 18 days; IQR, 11-32 days), followed by those patients who experienced a delay in lactate measurement (median, 15 days; IQR, 9-26 days). As for mortality, patients with delayed lactates demonstrated the highest in-hospital mortality (29%), followed by those with lactate samples drawn within the CMS window (27%) and those without a lactate sample (23%; $P < 0.01$).

Although the authors did not address it specifically, it is interesting to note that there was a significant decrease in mortality and longer LOS for patients who never underwent a lactate draw. As opposed to the patients who underwent a lactate draw early and those with delayed lactate, one may infer that the severity of illness for patients who never underwent a lactate draw was less. Since these patients experienced decreased mortality, they are not dying early and, therefore, are in the hospital longer. The longer LOS also may be related to the fact that most of these patients were identified on the wards. Many of the events could have occurred later in the hospital stay.

Although the importance of early and appropriate antibiotics is known, many often forget that every hour delay in antimicrobial administration is associated with a 7.6% average decrease in survival.² Within this study, there was a significant increase in mortality for every hour of delay in initial lactate draw > 2.0 mmol/L, which was associated with a 2% increase in the odds of death in an adjusted analysis (OR, 1.02; 95% CI, 1.0003-1.05; $P = 0.04$). When these numbers

were adjusted for time to antibiotics and IV fluids, the association no longer was significant ($P = 0.51$). There was a two-hour delay in receiving antibiotics and 1.3-hour delay for IV fluid bolus for those who received lactate measurement within the time frame. This, compared to 3.9 hours to antibiotics and 4.8 hours for IV fluid bolus for those who were measured later. Based on these data, the difference in mortality was associated with earlier interventions that included antibiotics, fluids, and source control.

Critically, clinicians need to give special attention to patients already admitted to the hospital. This study demonstrates that many patients develop sepsis on the wards. It is time to broaden our focus outside the ED. Although not addressed in this study, the identification of sepsis is paramount. Often, doctors and nurses on general medicine floors and in the ICU miss sepsis. Even though 60% of patients with severe sepsis underwent serum lactate measurements within the mandated period, timelines varied based on location of patient. For instance, 32% of patients met the standard SEP-1 timeline on the wards, compared with 55% in the ICU and 79% in the ED.

Regarding the patients who did not receive lactate within the mandated time frame, 14% received delayed lactates (between three and 24 hours after the time of first suspicion of sepsis). More than one-quarter had no lactate measurements at all. Since patients with initial lactate levels > 2.0 mmol/L were at an increased odds of death by 2% for each hour in lactate delay, it is important that recognition of sepsis and lactate measurements improve.

Systematic, timely lactate measurements in sepsis patients may be useful in prompting earlier, potentially life-saving interventions, including IV fluids and antibiotics. Clinicians also should focus on early resuscitation efforts that include lactate measurements in those populations in which there often is delay, especially those on the wards and ICU.

Despite the obvious benefits, researchers also were concerned that mandating lactate measurements for all sepsis patients may lead to many unwarranted lactate measurements and excessive resource use. More studies are needed to further improve patient outcomes. ■

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Metabolic Acidosis in the ICU

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Dr. Shah reports he receives grant/research support from Actelion Pharmaceuticals, Liquidia Technologies, and Bayer Pharmaceuticals.

SYNOPSIS: Sodium bicarbonate therapy for severe metabolic acidemia did not affect a primary composite outcome of all-cause mortality at 28 days and at least one organ failure at day 7. However, in an *a priori*-defined stratum of patients with acute kidney injury, sodium bicarbonate therapy decreased 28-day mortality and the primary outcome.

SOURCE: Jaber S, Paugam C, Futier E, et al. Sodium bicarbonate therapy for patients with severe metabolic acidemia in the intensive care unit (BICAR-ICU): A multicentre, open-label, randomized controlled, phase 3 trial. *Lancet* 2018;392:31-40.

Sodium bicarbonate therapy for metabolic acidosis in the ICU setting remains controversial and understudied.¹ As such, Jaber et al hoped to address this controversy. BICAR-ICU was a multicenter, open-label, randomized, controlled, Phase III trial that included an intention-to-treat analysis of sodium bicarbonate therapy for severe metabolic acidosis in patients across 26 ICUs in France. Adult patients admitted < 48 hours to the ICU with severe acidemia (pH ≤ 7.2, PaCO₂ ≤ 45, and sodium bicarbonate ≤ 20 mmol/L) and with a Sequential Organ Failure Assessment (SOFA) score ≥ 4 or serum lactate ≥ 2 mmol/L were included. Notable exclusion criteria were: stage four chronic kidney disease, ketoacidosis, respiratory acidosis (PaCO₂ ≥ 45), proven digestive or urinary tract loss of sodium bicarbonate, and sodium bicarbonate infusion (including renal replacement therapy [RRT]) within 24 hours of screening. Eligible patients were randomized to sodium bicarbonate infusion vs. usual care, stratified according to study site and three pre-specified factors: presence or absence of suspected sepsis, age with cutoff of 65 years, and presence or absence of Acute Kidney Injury Network (AKIN) score of 2 or 3.

In the intervention group, 4.2% sodium bicarbonate was infused, with an aim of achieving an arterial pH ≥ 7.30 during 28-day ICU admission or until ICU discharge. The volume of each sodium bicarbonate infusion was restricted to 125-250 mL in 30 minutes, with a maximum of 1,000 mL within 24 hours. Arterial blood gas was performed one to four hours after the end of the infusion. Indications for RRT were standardized in both groups and at 24 hours after inclusion. RRT was recommended when two of three criteria were met: hyperkalemia > 6.5 mmol/L, urine output < 0.3 mL/kg/hour for at least 24 hours, and arterial pH < 7.2 despite resuscitation. The primary outcome was a composite of death from any cause by day 28 and the presence of at least one organ failure by day 7. Most patients received invasive mechanical ventilation (84% of bicarbonate group vs. 82% of controls), vasopressor support (79% of bicarbonate group vs. 80% of controls), had severe

acidosis (mean pH of 7.15 in both groups), and lactic acidosis (86% of bicarbonate group and 78% of controls had a serum lactate ≥ 2 mmol/L). In the overall population, there was no difference in the primary outcome between the bicarbonate intervention group vs. control (66% vs. 71%; *P* = 0.24). However, the need for renal replacement therapy was lower in the bicarbonate group vs. controls (35% vs. 52%; *P* = 0.0009). In patients with acute kidney injury (AKIN score, 2-3), both mortality and the composite endpoint were significantly lower in the bicarbonate group (46% vs. 63%; *P* = 0.0166; 70% vs. 82%; *P* = 0.0462, respectively). However, patients who received bicarbonate therapy experienced more hypocalcemia, hypernatremia, and metabolic alkalosis.

■ COMMENTARY

Metabolic acidosis is detrimental to the survival of ICU patients.² While this is common knowledge, current data evaluating sodium bicarbonate therapy for metabolic acidosis in the ICU setting largely have been negative for any beneficial effect.^{3,4} Bicarbonate therapy is associated with hypocalcemia and intracellular accumulation of carbon dioxide, both of which cause decreased cardiac contractility and increased arrhythmogenicity.^{1,5} Currently, sodium bicarbonate therapy has been used as a last resort in severe metabolic acidosis without much data to support its use.

Despite efforts by Jaber et al to clarify this issue, the use of sodium bicarbonate in mixed and anion gap metabolic acidosis remains controversial. In BICAR-ICU, most patients had lactic acidosis; hence, it is not surprising that bicarbonate therapy did not affect mortality or the primary composite outcome between groups. However, bicarbonate therapy decreased the need for renal replacement therapy compared to controls. In patients with acute kidney injury, bicarbonate therapy decreased mortality and the composite outcome in patients with severe metabolic acidosis, possibly by decreasing need for vasopressor and renal replacement therapies. Based on this trial, sodium bicarbonate therapy could be considered in patients with acute kidney injury and severe

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metabolic acidosis. Overall, the utility of sodium bicarbonate therapy for lactic acidosis in the absence of acute kidney injury remains unanswered. Efforts should focus on reversing the primary cause of lactic acidosis in these patients. ■

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CME/CE QUESTIONS

1. Based on the study by McKown et al, which of the following factors is associated with an increased risk of severe hypoxemia during intubation of the critically ill?
 - a. Advanced age
 - b. Body mass index
 - c. APACHE II score
 - d. Aspiration
2. Which of the following statements is false regarding inspiratory muscle training (IMT) therapy?
 - a. Improvement in maximum inspiratory pressure (MIP) and maximum expiratory pressure (MEP) did not differ based on the technique used.
 - b. Only studies incorporating threshold loading found improvements in MIP and MEP.
 - c. Improvement in MIP and MEP did not differ based on when IMT therapy was initiated.
 - d. Patients in the IMT treatment arm experienced a 2.3-day reduction in weaning duration.
3. In the study by Han et al, lactates were checked within the appropriate time frame in approximately what proportion of the time on the wards?
 - a. 33%
 - b. 50%
 - c. 75%
 - d. 90%
4. Based on the BICAR-ICU study, sodium bicarbonate therapy in ICU patients with severe metabolic acidosis:
 - a. increased mortality compared to controls.
 - b. decreased mortality compared to controls.
 - c. increased mortality in patients with an Acute Kidney Injury Network (AKIN) score of 2-3.
 - d. decreased mortality in patients with an AKIN score of 2-3.

CME/CE OBJECTIVES

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

- identify relevant topics in the practice of critical care medicine;
- utilize recommendations from current clinical guidelines; and
- manage common critically ill patient and ICU administration scenarios.

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