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Critical Care [ALERT]

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SPECIAL FEATURE

Treatment and Follow-Up of Acute Pulmonary Embolism: A Multidisciplinary Team Approach

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Venous thromboembolism (VTE) is common, affecting about 900,000 Americans and leading to 100,000 deaths per year.[1] Overall, about 50% of VTEs occur after or during hospitalization and/or surgery. Approximately 150,000 to 250,000 patients require hospitalization for pulmonary embolism (PE). After diagnosis of PE, further management is dictated by risk stratification. Different risk-stratification models are available for use that can include the Pulmonary Embolism Severity Index (PESI) score, the Simplified PESI (sPESI) score, biomarkers (e.g., troponins, brain natriuretic peptide [BNP]/Nt-proBNP), and findings on echocardiography (e.g., presence of right ventricular [RV] strain). Please refer to the August 2017 issue of *Critical Care Alert* for Samuel Nadler's excellent discussion for more details.[2] Management of PE is addressed after patient classification as high, intermediate, or low risk. This article will review management of pulmonary embolism based on risk stratification, with an emphasis on the role of a Pulmonary Embolism Response Team as well as appropriate follow-up.

PULMONARY EMBOLISM RESPONSE TEAM

Although anticoagulation remains the cornerstone of treatment, some patients may benefit from further treatment with catheter-directed thrombolysis/thrombectomy, systemic thrombolytics, or surgical embolectomy and may even warrant consideration for veno-arterial extracorporeal membrane oxygenation (VA-ECMO). Given the acuity of PE events, associated high mortality, and the need for urgent mobilization of resources, many healthcare systems have moved to a multidisciplinary team approach in the form of

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a Pulmonary Embolism Response Team (PERT).[3]

The structure of a PERT varies by institution, but generally it consists of a multidisciplinary team of specialists from pulmonary/critical care, interventional radiology, emergency medicine, cardiothoracic surgery, anesthesiology, cardiology, and hematology.[3] Usually there is one point person who gathers information on the patient, and the team convenes in real time over telephone or video conferencing. This expedites formulation of a consensus treatment plan and ensures immediate implementation.[3-5] Several studies have shown benefits with the PERT approach as compared to historical controls.[6-8] For example, a PERT approach is associated with expedited decision and treatment of intermediate- and high-risk PE and has been shown to improve mortality in high-risk PE patients.[6,7,9] Implementation of a PERT also has been associated with decreased length of intensive care unit (ICU) stay and hospitalization.[8]

ICU MANAGEMENT OF ACUTE PULMONARY EMBOLISM

All patients with PE should receive anticoagulation as first-line treatment unless there is an absolute contraindication. Whenever possible, low molecular weight heparin (LMWH) should be considered first as opposed to unfractionated heparin (UFH) for initial anticoagulation for PE. A Cochrane review of 23 studies comparing LMWH vs. UFH showed that LMWH as an initial therapy is safer and more effective than UFH. LMWH significantly reduced the incidence of thrombotic complications, occurrence of major hemorrhage, and overall mortality at follow-up.[10]

HIGH-RISK PE

Based on various risk stratification models, patients are classified further as high-, intermediate-, or low-risk PE. Low-risk PE can be managed safely as an outpatient or on a general medical floor with anticoagulation alone and does not need PERT consultation.[3] High-risk PE is defined as PE with cardiac arrest, signs of shock, and/or persistent hypotension. Shock is associated with significant early risk of death, up to 30% to 50%.[11] High-risk PE patients may not be stable enough to confirm the diagnosis with a computed tomography (CT) pulmonary angiogram, and the diagnosis often is made by the presence of RV dysfunction on echocardiogram in the setting of shock.

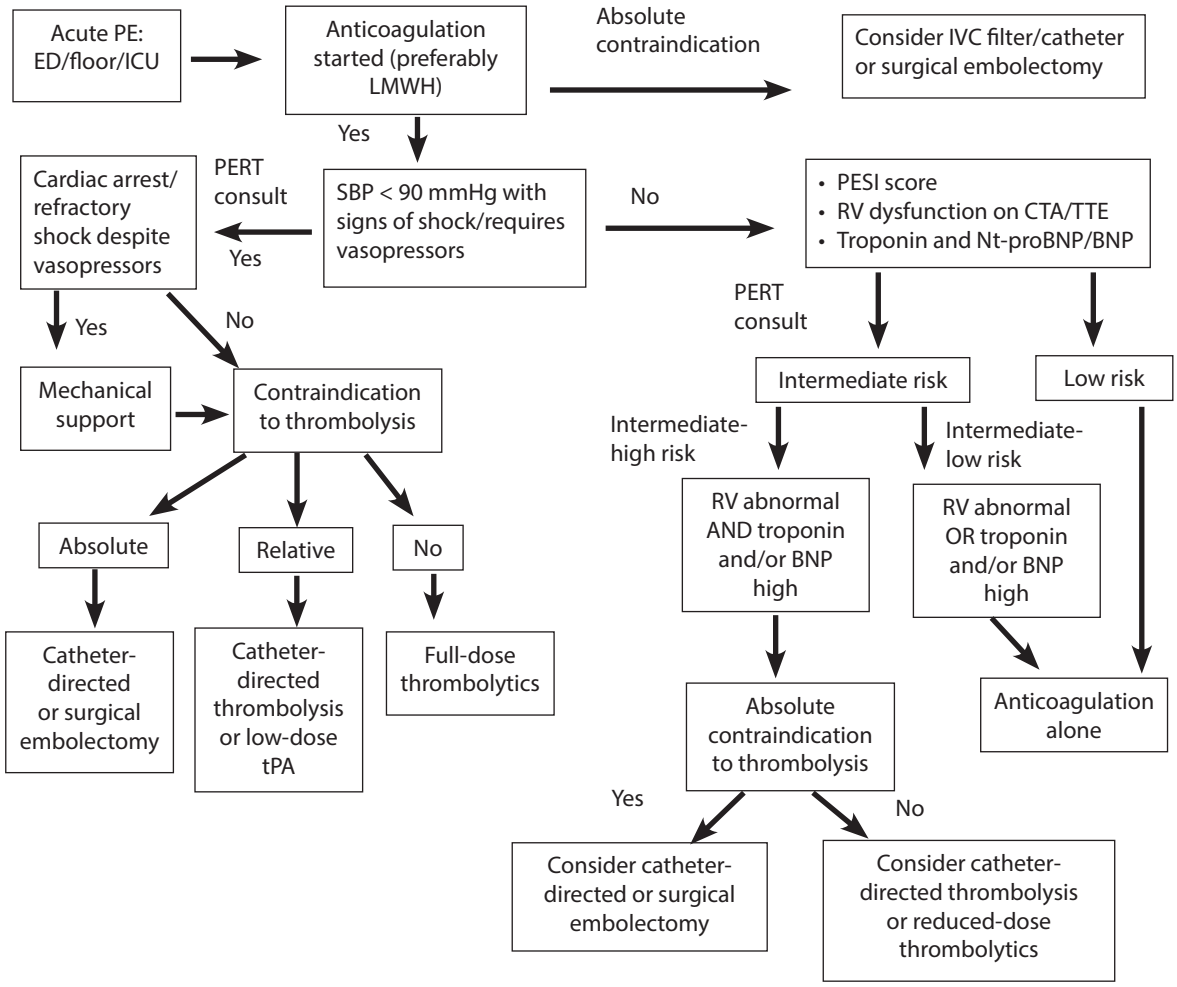
PERT consultation has been shown to expedite the diagnosis of high-risk PE and the implementation of immediate treatment (e.g., systemic thrombolytics).[7] It also can facilitate mechanical circulatory support in the setting of refractory shock/cardiac arrest. Patients with contraindications to thrombolytics should be considered for catheter-directed thrombectomy or surgical embolectomy.

INTERMEDIATE-RISK PE

Patients with intermediate-risk PE are hemodynamically stable with the presence of RV dysfunction and/or elevated biomarkers (i.e., troponin, BNP). These patients need immediate attention, since they are at risk of decompensation, especially within the first 24 hours of presentation. The presence of RV dysfunction, elevated troponin, and elevated BNP is associated with poor prognosis and increased mortality.[12-13] Although systemic thrombolytics are the cornerstone of management in high-risk PE, management of intermediate-risk PE varies according to further prognostication. Intermediate-risk PE is divided further into intermediate-high risk (RV dysfunction and elevated troponin and/or BNP) and intermediate-low risk (RV dysfunction or elevated troponin/BNP).

For intermediate-low risk patients, anticoagulation alone will suffice, and no adjunct treatment modality is needed. For intermediate high-risk patients, reduced-dose thrombolytics or catheter-directed thrombolytics should be considered. It should be noted that the use of both reduced-dose thrombolytics and catheter-directed thrombolytics in intermediate-high risk PE patients mainly expedites improvement in RV function, pulmonary hypertension, and quality of life, but these therapies have not demonstrated any mortality benefit.[14-16] In intermediate-high risk patients with a contraindication to thrombolysis, catheter-directed or surgical

Figure 1: Proposed Treatment Algorithm of Acute Pulmonary Embolism



PE: pulmonary embolism; ED: emergency department; ICU: intensive care unit; LMWH: low molecular weight heparin; IVC: inferior vena cava; PERT: Pulmonary Embolism Response Team; SBP: systolic blood pressure; PESI: Pulmonary Embolism Severity Index; RV: right ventricle; CTA: computed tomography angiogram; TTE: transthoracic echocardiogram; BNP: brain natriuretic peptide; tPA: tissue plasminogen activator

embolectomy can be considered. Note that further risk stratification of intermediate-risk patients is not perfect, and the patient's individual presentation should be considered to determine further management. (*See Figure 1.*) A PERT team provides multidisciplinary opinions in such cases and can help with timely decision-making.

FOLLOW-UP AFTER ACUTE PE

Intermediate- and high-risk PE patients are at increased risk of clinical worsening during the hospitalization and should be monitored closely for any signs of deterioration.[17] PERT should be reconsulted in case of deterioration, and further intervention should be planned as per risk stratification. One of the important components of PERT is assuring appropriate follow-up of PE patients after hospitalization. The INFORM study by Tapson et al showed that the majority of patients with persistent symptoms after PE did not undergo diagnostic testing or further imaging.[18]

The post-PE follow-up clinic visit should focus on assessing the patient for persistent/recurrent symptoms, the type/dosage/duration of anticoagulation, and medication compliance; evaluating for underlying thrombophilia; screening for cancer as age-appropriate; arranging for temporary inferior vena cava (IVC) filter removal; and diagnosing sequelae of PE (e.g., post-PE syndrome, chronic thromboembolic disease [CTED], and chronic thromboembolic pulmonary hypertension [CTEPH]).[3]

If a patient has persistent symptoms of dyspnea on exertion, fatigue, lightheadedness, or pedal edema after three months of anticoagulation, further workup with a ventilation perfusion (VQ) scan and

echocardiogram should be considered to evaluate for CTED/CTEPH. CTED/CTEPH are treatable with pulmonary thromboendarterectomy (PTE) surgery, with subsequent significant improvement in prognosis.[19] Patients with high suspicion or confirmed CTED/CTEPH should be referred to an expert CTEPH center for consideration for PTE and further management. ■

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ABSTRACT & COMMENTARY

An Assessment of Hospital-Acquired Infections in Critically Ill SARS-CoV-2-Infected Patients

By Vibhu Sharma, MD

Associate Professor of Medicine, University of Colorado, Denver

SYNOPSIS: This retrospective analysis of prospectively collected data showed that critically ill patients with SARS-CoV-2 infection are at increased risk for hospital-acquired infections.

SOURCE: Grasselli G, Scaravilli V, Mangioni D, et al. Hospital-acquired infections in critically ill patients with COVID-19. *Chest* 2021;160:454-465.

This report of a retrospective analysis of prospectively obtained data describes the epidemiology and clinical outcomes of hospital-acquired infections (HAIs) in critically ill patients diagnosed with SARS-CoV-2 infection between February 2020 and May 2020 in eight Italian

hospitals. Routine surveillance cultures were the norm in the institutions where this study was done; this included weekly tracheal aspirates, perineal and nasal swabs, and urine cultures, with additional cultures as clinically indicated. A designated infectious disease physician and intensive care physician adjudicated final diagnoses at each institution, with intensive care and infectious disease physicians at the sponsoring institution available for consultation in case there were disagreements or questions. Overall, 774 consecutive patients were included in the analysis, most of whom received mechanical ventilation (n = 689, 89%). The median age of enrollees was 62 years, most of whom were men (77%).

In total, 359 patients (46%) were diagnosed with HAIs. The probability of an HAI increased rapidly with duration of intensive care unit (ICU) stay. The first HAI was described a median of 12 days (interquartile range [IQR], 8-18 days) from hospital admission and eight days (IQR, 5-12 days) from ICU admission. Importantly, none of the 82 non-intubated patients admitted to intensive care developed an HAI. Overall mortality in the ICU was 30%. Patients with an HAI complicated by septic shock had a mortality rate of approximately 52%. Ventilator-associated pneumonia (VAP) was the most common, accounting for 50% of total HAIs; bloodstream infections (BSIs) (34%) and catheter-related infections (10%) accounted for most of the rest. The proportion of BSIs was higher than what was described in similar critically ill patients prior to the pandemic.

With respect to bacteriology of the HAIs, as expected, most VAPs were caused by gram-negative bacteria, followed by *Staphylococcus aureus*. Extended-spectrum beta-lactamase (ESBL)-producing and carbapenamase-producing organisms accounted for 19% and 42% of these gram-negative bacteria, respectively. Slightly more than half (55%) of *S. aureus* isolates were methicillin-resistant (MRSA). Overall, one-third of all HAIs were the result of multidrug-resistant organisms (MDRO). Invasive fungal infections were rare; invasive aspergillosis was identified in 2% of patients and an identical number were diagnosed with invasive candidal infection. Patients with SARS-CoV-2 infections were treated with steroids and other immunomodulators that may increase the risk of serious HAI; however, use of these drugs was not associated with an increased risk of HAI.

■ COMMENTARY

The major limitation of this study is that it reports data from a single province in Italy, limiting generalizability of the results. An additional limitation that also may limit generalizability to current practice is that the study reports the bacteriology of HAIs early in the pandemic, with a short duration of study (three months). Since the end of the study in May 2020, there has been a substantial change in the management of critically ill patients with SARS-CoV-2 infection, with a shift to early institution of noninvasive ventilation and a more delayed strategy with respect to invasive ventilation compared to prior. Furthermore, there also has been an increase in the use of immunomodulatory therapies. Both of these factors may contribute to a change in the distribution of HAIs now compared to early 2020. However, it was encouraging that this report did not find an increase in infection with immunomodulatory therapies.

[Since the end of the study in May 2020, there has been a substantial change in the management of critically ill patients with SARS-CoV-2 infection.]

Routine microbiologic surveillance with weekly cultures from multiple sites is not the standard of care in most critical care units in the United States, and it is plausible that this may have led to an overdiagnosis of HAIs. VAP was diagnosed using cutoffs for bacterial growth of 10^4 colony forming units (CFU)/mL for bronchoalveolar lavage (BAL) and 10^5 CFU/mL for endotracheal aspirate in addition to clinical criteria. The use of quantitative thresholds in the diagnosis of VAP is controversial and not recommended for diagnostic use in this setting.[1,2] Approximately 30% of isolated organisms were MDRO, which is an unusually high proportion, a finding that also may limit generalizability.

The reports of invasive aspergillosis and invasive candidiasis are intriguing. Invasive aspergillosis was diagnosed definitively if positive cultures were obtained from lung tissue or positive stains were found on microscopy of needle aspirates/biopsies of lung tissue. The diagnosis was probable if positive stains were obtained from BAL fluid or if the patient worsened clinically (e.g., pleuritic chest pain, hemoptysis, recrudescent fever despite adequate antibiotic therapy) with suggestive imaging findings. Globally, bronchoscopies were not performed routinely early in the pandemic, and in many medical centers still are not performed routinely. Fungal infections may have been underdiagnosed and/or undertreated in other institutions with lower use

of invasive diagnostic strategies. While the authors reported invasive aspergillosis in ~2% of all HAIs, other larger studies have reported higher numbers ranging from 5% to 15%.[3] The reason for this difference is unclear and may reflect variation in diagnostic criteria or intensity of diagnostic studies.

Invasive candidiasis was diagnosed if blood cultures were positive or if serum (1,3)-beta-D-glucan was positive with no other diagnosis to explain sepsis, both of which are reasonable diagnostic strategies. Only two patients were diagnosed with *Clostridium difficile* infection; this may relate to almost universal use of personal protective equipment.

In summary, this study is an informative epidemiologic report on the incidence of HAIs in critically ill patients with SARS-CoV-2 infection, keeping in mind the caveats described. ■

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ABSTRACT & COMMENTARY

Efficacy of Neuroinvasive Goal-Directed Therapy in Hypoxic Ischemic Brain Injury After Cardiac Arrest

By Vibhu Sharma, MD

Associate Professor of Medicine, University of Colorado, Denver

SYNOPSIS: This retrospective observational study found the suggestion of favorable neurological outcomes at six months among out-of-hospital cardiac arrest patients who received interventions to optimally manage intracranial pressure and brain tissue oxygenation compared to standard care.

SOURCE: Fergusson NA, Hoiland RL, Thiara S, et al. Goal-directed care using invasive neuromonitoring versus standard of care after cardiac arrest: A matched cohort study. *Crit Care Med* 2021;49:1333-1346.

The investigators in this study tested the hypothesis that invasive management of hypoxic ischemic brain injury (HIBI) after cardiac arrest would lead to improved neurologic outcomes. This was a retrospective, matched, observational cohort study performed at a single center, enrolling adult patients admitted to the intensive care unit (ICU) after a cardiac arrest lasting at least 10 minutes (median, 21 minutes). Most patients had a pulseless electrical activity (PEA) cardiac arrest (~80% in both groups), with a minority having shockable rhythms. Patients were enrolled within 72 hours of return of spontaneous circulation (ROSC) and had a post-resuscitation Glasgow Coma Scale (GCS) score of less than 8. More than 70% in each group were deeply comatose with a GCS of 1. None of the cardiac arrests were inpatients. The median age of patients was 40 years for the intervention group (n = 21) and 58 years for the standard of care group (n = 44).

All patients received targeted temperature management (TTM) consistent with institutional guidelines. The intervention, or goal-directed care (GDC) group, received invasive neuromonitoring; patients were enrolled in this group based on availability of a neurosurgeon to place intracranial devices. Specifically, an intracranial intraparenchymal pressure monitor was placed in the non-dominant frontal lobe to measure intracranial pressure (ICP) and brain tissue oxygenation ($PbtO_2$). In addition, a jugular venous bulb catheter was placed in the dominant internal jugular vein using ultrasound guidance with the tip guided to the mastoid bone. This catheter measured jugular venous oxygen saturation ($SjvO_2$).

Goal-directed therapy targeted $PbtO_2$ of > 20 mmHg and an ICP < 20 mmHg. Interventions aimed at returning $PbtO_2$ to normal included first ensuring ICP was less than 25 mmHg and maintaining mean

arterial pressure (MAP) to an acceptable level based on "attending physician discretion" using vasopressors, with norepinephrine being the first choice. Packed red blood cell transfusion was used to bring hemoglobin to 9 g/dL if the previously noted interventions did not optimize $PbtO_2$. Partial pressure of peripheral blood oxygen was maintained between 80 mmHg to 100 mmHg. Body temperature was maintained using guideline-directed targeted temperature management.

With respect to ICP management, a multi-tiered approach was used to maintain ICP < 20 mmHg. An ICP > 20 mmHg for > 10 minutes was actionable. Head of bed elevation to > 30 degrees, optimization of sedation, and maintenance of normocapnia and normonatremia were tier 1 interventions. If ICP remained above 20 mmHg for more than 10 minutes despite these interventions, tier 2 interventions were initiated and included osmotherapy (3 mL/kg to 5 mL/kg bolus of 3% saline and/or 0.25 g/kg to 0.5 g/kg of 20% mannitol) and neuromuscular blockade. Escalation to tier 3 therapies included barbiturate therapy and induction of moderate hypothermia.

The standard of care (SoC) cohort was managed with standard targeted temperature management: 36ºC for 24 hours followed by slow rewarming over 48 hours to 37.5ºC. Normoxemia and normocapnia were maintained. Surface devices (e.g., Arctic Sun) were used for active cooling.

The primary outcome was neurologic outcomes at six months measured using the Cerebral Performance Category (CPC) score; a score of 3-5 is defined as unfavorable and 1-2 as favorable. The CPC was assessed by trained investigators over the phone who called the patient or an authorized representative. Favorable neurologic outcomes were more frequent in the intervention cohort (43%) compared with the standard of care cohort (10%) ($P = 0.016$). This was driven mostly by the GDC group having a significantly higher MAP (~90 mmHg) and lower temperature over the course of their ICU stay.

■ COMMENTARY

This study describes an intriguing concept of neuroinvasive management in the care of patients with post-cardiac arrest HIBI. The major factor for allocation to the GDC group was the availability of an attending neurosurgeon. It is possible that the perception of more severe neurological injury may have made it more likely for a neurosurgeon to be called, leading to concern for selection bias. Patients could be enrolled up to 72 hours after ROSC. The authors did not delineate time to intervention with GDC.

It is plausible that early intervention is associated with better outcomes. Most patients in this study were brought to the emergency department with PEA arrest, which typically is associated with worse outcomes than shockable arrests.[1] Significantly more patients in the GDC group had unreactive pupils at enrollment (43% vs. 18%, $P = 0.045$). It is possible that more aggressive therapy in severe HIBI may improve prognosis. Biomarker measurement may help neuro-prognostication[2] further, and application of invasive therapies may be triaged to those with higher levels of brain biomarkers in the setting of HIBI.

The duration of cardiac arrest was short and, therefore, longer arrests may not benefit from aggressive therapy. Significantly more arrests in the SoC group were of cardiac etiology than in the GDC group (32% vs. 10%, respectively). All of the patients in this study had an out-of-hospital cardiac arrest, and the benefits (if any) of GDC cannot be assessed for in-hospital cardiac arrests.

All patients with HIBI ought to have aggressive interventions to maintain normothermia. In the absence of invasive monitoring of ICP, point-of-care ultrasound may be used by trained intensivists to assess optic nerve sheath diameter (ONSD), and transcranial Dopplers can assess middle cerebral artery diastolic flow velocities. Both have been shown to correlate well with invasively assessed intracranial pressure.[3,4]

In the event that a clear benefit for invasive GDC is demonstrated eventually, this intervention may remain aspirational for all but the largest quaternary care medical centers, given the scarcity of qualified neurosurgeons. Larger studies are needed to validate the benefit of early invasive GDC in patients with HIBI after cardiac arrest. ■

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CME/CE QUESTIONS

1. **What is the treatment of choice for high-risk pulmonary embolism in a patient with no contraindication for thrombolysis?**
   a. Anticoagulation alone
   b. Catheter-directed thrombolysis and anticoagulation
   c. Surgical embolectomy and anticoagulation
   d. Full-dose systemic thrombolytics and anticoagulation

2. **A Pulmonary Embolism Response Team (PERT) is potentially associated with lower mortality in which situation?**
   a. High-risk pulmonary embolism
   b. Intermediate-risk pulmonary embolism
   c. Low-risk pulmonary embolism
   d. Chronic thromboembolic pulmonary hypertension

3. **Which of the following describes the findings related to hospital-acquired infections (HAIs) in critically ill patients with SARS-CoV-2 infection as described by Grasselli et al?**
   a. Ventilator-associated pneumonia (VAP) was most common.
   b. Most VAPs were caused by *Staphylococcus aureus* infection.
   c. Immunomodulatory therapies were associated with an increased risk of HAIs.
   d. Bloodstream infections were less common compared to pre-COVID-19 pandemic rates.

4. **In the article by Fergusson et al, which one of the following accurately describes a goal of invasive management in the early invasive goal-directed care group?**
   a. Intracranial pressure (ICP) < 20 mmHg
   b. Brain tissue oxygenation ($PbtO_2$) < 20 mmHg
   c. Mean arterial pressure (MAP) > 90 mmHg
   d. Partial pressure of arterial oxygen (PaO_2) < 80 mmHg

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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.