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SPECIAL FEATURE

Mechanical Circulatory Support for Cardiogenic Shock

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

Cardiogenic shock (CS) occurs because of reduced cardiac output, which can lead to severe end-organ hypoperfusion. Primarily, CS is a clinical diagnosis with hypotension, defined as a systolic blood pressure (SBP) level < 90 mmHg for a prolonged period (> 30 minutes) or requiring vasopressors to maintain SBP > 90 mmHg, pulmonary congestion (i.e., elevation in left ventricular end-diastolic pressure [LVEDP]), and clinical evidence of hypoperfusion (e.g., altered mental status, oliguria, cold and hypoperfused skin, and/or elevation in serum lactate levels).[1,2] The most common cause of CS is acute myocardial infarction (AMI) or acute coronary syndrome. Nonischemic causes include myocarditis, valvular heart disease, acute decompensated heart failure, and recalcitrant arrhythmias. CS occurs in up to 5-15% of patients with AMI and carries a mortality between 40% and 50%.[1,2] Standard treatments for CS include fluid resuscitation, vasopressors, and inotropes, along with early reperfusion tactics. Refractory shock may require initiation of mechanical circulatory support (MCS). What other options exist for temporary percutaneous mechanical support for CS associated with AMI?

INTRA-AORTIC BALLOON PUMP

The oldest and still most frequently used mechanical assist device is the intra-aortic balloon pump (IABP). It consists of a 30-40 mL balloon mounted on a 7.5 Fr or 8 Fr catheter, which is placed percutaneously via an 8 Fr or 8.5 Fr sheath into the femoral artery and advanced so that the tip of the balloon catheter lies in the descending thoracic aorta, distal to the left subclavian artery. The balloon inflation is timed from the ECG or arterial pressure waveform to occur during diastole in which displacement of blood, both antegrade and retrograde, augments coronary artery filling and propels blood to the periphery. The balloon deflates at the start of systole. This results in a decrease in afterload for the next cardiac cycle. Additional hemodynamic benefits of the IABP include an increase in diastolic blood pressure, an increase in mean arterial pressure, a decrease in myocardial oxygen consumption, and improvement in cardiac

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output of approximately 0.5-1 L/minute.[3,4] IABP use is contraindicated in patients with aortic insufficiency and severe peripheral vascular disease.

Careful attention is made to ensure correct placement so the balloon does not migrate proximally and impede blood supply to the left subclavian artery or distally and impede flow to renal arteries or mesenteric vessels. Patient mobility is limited. Potential complications include vascular insufficiency to the distal limb, thromboembolism, thrombocytopenia, and bleeding.

The IABP is available in almost all cardiac catheterization labs. It can be deployed quickly and with relative ease, thus making it popular for initial support in acute CS or as an adjunct either immediately before or after percutaneous coronary revascularization. Widespread use of IABP was based largely on U.S. guidelines that supported IABP use as a Class IB recommendation and European guidelines that supported IABP use as a Class IC recommendation in patients with CS.[5,6]

More recently, the authors of a large, multicenter, randomized, controlled trial (RCT) of IABP vs. medical therapy in CS due to myocardial infarction (IABP-SHOCK II) enrolled almost 600 patients. All patients were expected to receive standard medical therapy and early percutaneous coronary revascularization. There was no significant difference in the primary outcome of 30-day mortality between groups (39.7% in IABP group vs. 41.3% in control group; $P = 0.69$). There were no significant differences in bleeding, stroke, peripheral ischemic complications, or sepsis.[7] Additionally, long-term outcomes were evaluated at six and 12 months and demonstrated no significant differences in 12-month mortality (52% in IABP group vs. 51% in control group; $P = 0.91$).[8]

The authors of a meta-analysis of IABP use in AMI included 12 RCTs (2,123 patients) and 15 observational studies (15,530 patients) published between 1981 and 2013. IABP was not found to improve mortality among patients with AMI in the RCTs. There was significant heterogeneity among the observational studies, but no clear improvement in mortality using of IABP.[9] Current guidelines recommend using IABP only for mechanical complications of AMI.

PERCUTANEOUS VENTRICULAR ASSIST DEVICES

Considering the limitations of support and the lack of mortality benefit with IABP, other means of MCS have gained popularity. Use of the percutaneous ventricular assist devices (pVADs) has been expanding. There are two primary FDA-approved pVADs in use for short-term MCS in CS: the TandemHeart and the Impella.

The TandemHeart is an extracorporeal, continuous flow, centrifugal pump. It requires two cannulas, one placed via the femoral vein (21 Fr) passed up to the right atrium, then across into the left atrium (LA) via a trans-septal puncture. This draws blood from the LA, reducing forward flow to the left ventricle and resulting in an unloading of the left ventricle. The blood circulates through the pump and returns via a femoral arterial cannula (15 Fr to 19 Fr) advanced to the level of the iliac arteries (provides LA to aorta bypass). Depending on the size of the arterial cannula, flows of 3.5-5 L/minute can be achieved. Hemodynamic effects are reduced LV preload and wall stress, lower myocardial oxygen demand, and some increase in afterload from the retrograde return of blood via the femoral vessels.[1,3] To prevent thrombus formation, the pump requires anticoagulation, provided as a continuous infusion of heparinized saline into the lower chamber of the pump. Complications include vascular compromise and distal limb ischemia. The trans-septal catheter placement is technically difficult, generally requiring a skilled interventional cardiologist using either fluoroscopic guidance or transesophageal echocardiography to guide catheter placement. Caution must be taken not to dislodge the trans-septal catheter from the LA, which could result in significant hypoxemia from a large volume right-to-left shunt.[3] Careful assessment of right ventricular (RV) function is necessary to ensure adequate LA filling. RV assist device (RVAD) support may be required if RV function is poor. The patient must be supine and mobility is limited by the femoral cannulations.

The Impella is another pVAD that provides direct unloading of the LV (LV to ascending aorta bypass). The Impella is a nonpulsatile axial flow pump placed intracorporeally within the LV. Usually, access is obtained via the femoral artery. The catheter is threaded

through the descending aorta, ascending aorta, and across the aortic valve, seating the pump within the LV. Blood is drawn from the left ventricle and returned to the ascending aorta. There are three different size devices: 12 Fr (Impella 2.5), 14 Fr (Impella CP), and 21 Fr (Impella 5.0). These permit flows of 2.5, 3.0-4.0, and 5.0 L/minute, respectively. The Impella 5.0 is placed via a cutdown; the others are placed percutaneously. The Impella 2.5 provides a better augment to cardiac output than the IABP, but not as much as the TandemHeart, whereas the Impella 5.0 and CP provide flow support more comparable to that of the TandemHeart. Complications relate to the transfemoral access, limb ischemia, and bleeding, as well as hemolysis from the pump. Systemic anticoagulation is required. Native RV function must be maintained to adequately fill the LA, or RVAD support may be required.[1,3]

The authors of a systematic review and meta-analysis of four RCTs concerning pVADs in CS evaluated TandemHeart and Impella implants compared to control, which was the IABP. A total of 148 patients were represented in the four trials (77 in the MCS groups, 71 in the control groups). There was no difference in 30-day mortality between MCS and IABP. MCS increased mean arterial pressure more than IABP ($P = 0.02$) and lowered lactate levels ($P = 0.02$). There were no differences in leg ischemia between groups, but the incidence of bleeding was significantly higher in the MCS group ($P < 0.001$). The meta-analysis authors concluded the results did not support the unselected use of MCS in patients with CS complicating AMI.[10]

Investigators from the University of Virginia reported a retrospective review of 55 patients treated with the TandemHeart for CS to evaluate predictors of survival. Hemodynamic parameters, including better cardiac index and lower pulmonary capillary wedge pressure, improved significantly after the TandemHeart implant. Indication for implant influenced survival. In patients bridged to LVAD or surgery, survival was 51%. In patients supported with the aim of recovery of function, survival was 23.8% ($P = 0.04$). Only younger age predicted survival to discharge ($P = 0.004$).[11]

The Impella has been studied more widely. In a single-center study of 112 consecutive patients treated with Impella for CS associated with AMI, mortality at six months remained high at 60.7%.[12] The Detroit Cardiogenic Shock Initiative reported outcomes after the first eight months of enrolling patients in an early MCS initiative using Impella for patients with CS after AMI following early reperfusion therapy. Forty-one patients were enrolled. Survival to explant was 85% for the cohort, a significant improvement over historical control of 51% ($P < 0.001$), and survival to discharge was 76%.[13] A large, multicenter registry that included more than 15,000 patients supported with Impella for CS associated with AMI demonstrated survival to explant of 51%. Survival rates were higher if MCS was initiated as first-line treatment (59%) rather than as salvage therapy (52%; $P < 0.001$). Likewise, survival rates improved in centers with larger implant volumes.[14] A study comparing outcomes of Impella support in AMI complicated by CS matched one group of patients to subjects from the IABP-SHOCK II trial. The primary endpoint was 30-day mortality. Patients were matched for age, sex, mechanical ventilation, ejection fraction, lactate, and prior CPR. The authors matched 237 patients treated with Impella to 237 control patients from the IABP-SHOCK II trial. There were no significant differences in 30-day all-cause mortality (48.5% vs. 46.45%, respectively; $P = 0.64$). Bleeding ($P < 0.01$) and peripheral vascular complications ($P = 0.01$) were significantly higher in the Impella group.[15]

VENOARTERIAL EXTRACORPOREAL MEMBRANE OXYGENATION

Venoarterial extracorporeal membrane oxygenation (VA-ECMO) is the MCS of choice for patients with biventricular failure or CS with concomitant hypoxemic respiratory failure. For cardiac support, peripheral VA-ECMO can be instituted easily at the bedside with percutaneous femoral arterial and venous cannulation. The cannulae are connected to an extracorporeal pump (nonpulsatile centrifugal pump), membrane oxygenator, and heat exchanger. Systemic anticoagulation is required. Venous cannulas range in size from 17-21 Fr and arterial cannulas from 15-19 Fr. Depending on cannula size and type of pump, flows of 4-7 L/minute can be achieved.[1,3] Typically, a perfusionist or ECMO specialist is present to maintain the machine.

One disadvantage of peripheral VA-ECMO is that it does not unload the LV. Peripheral VA-ECMO increases afterload and may cause LV distention and pulmonary edema. This can be mitigated by placement of a vent to drain the LV. With percutaneous cannulation, drainage can be achieved by concomitant placement of an Impella device to decompress the LV or a TandemHeart catheter placed trans-septally to drain the LA. An alternative technique is placement of a transapical vent via a left minithoracotomy approach, which is then Y'd into the venous drainage cannula.[16] Other complications include vascular injury, limb compromise, bleeding, and stroke. Placement of a smaller distal perfusion cannula to maintain blood flow to the distal limb can help minimize the risk of limb ischemia.

In a single-center review of 76 consecutive VA-ECMO patients supported for post-MI CS (51%) or other causes (49%), overall 90-day mortality was 49%. Forty-six percent of patients died on ECMO, 37% were weaned, 13% bridged to heart transplant, and 4% bridged to

LVAD.[17] In a study comparing outcomes after use of Impella (48 patients) and VA-ECMO (46 patients) for CS, there was no significant difference in ICU survival (65% for ECMO and 63% for Impella) or in long-term survival (four years). Even adjusting for disease severity using the Survival after VA-ECMO (SAVE) score, there was no difference in survival between groups.[18]

In another single-center retrospective analysis of 88 patients treated with PCI after AMI and admitted to the ICU with CS, researchers evaluated early MCS (within 72 hours). Impella was placed in 19 patients, and 23 patients had ECMO. The ECMO group was sicker at time of initiation of MCS (higher lactate levels, higher inotrope and vasopressor support). ECMO was identified as the technique of choice in profound CS, and Impella was appropriate for less profound shock. There was no significant difference in six-month survival: 52% in the ECMO group and 58% in the Impella group.[19]

Traditionally, patients with biventricular failure have been treated with VA-ECMO as the choice of mechanical support. However, case reports using biventricular Impella devices support both the RV and LV, so-called Bi-Pella support. The Impella RP device is used for RV support. One of the other standard Impella devices (2.5, CP, or 5.0) is used for LV support.[20] The Impella RP is placed via the femoral vein and advanced to the inferior vena cava into the right atrium, across the tricuspid valve into the right ventricle, and across the pulmonic valve into the pulmonary artery. The inlet to the pump is in the inferior vena cava. Blood is delivered to the tip of the catheter near the pulmonary artery.

TIMING

Initiating mechanical support in a timely fashion is important, although there are few discrete triggers. Recognition of CS, initiation of early reperfusion with PCI, and early initiation of MCS is important. While IABP is readily available, there is no demonstrated mortality benefit in CS due to AMI. Placement of an IABP at the time of PCI and prior to transfer to a tertiary facility may be appropriate. Other devices may be more complicated to insert but offer improved hemodynamic benefits. Despite improved hemodynamics seen with TandemHeart and VA-ECMO, there are no data to suggest mortality benefit with either device. Respiratory failure and hypoxemia are indications for VA-ECMO rather than other MCS options. Patients in cardiac arrest would be candidates for ECMO as opposed to other MCS options. Those with profound biventricular failure should be treated with VA-ECMO. Most often, in acute CS associated with MI, the patient is in extremis and the device is placed with an unknown trajectory. While hopeful that reperfusion of the culprit lesion, unloading the ventricle, and time will allow myocardial recovery, this is not always realized. Often, patients treated with urgent MCS in CS are on a "bridge to decision" pathway. Support is provided until clinicians can assess neurologic status, reassess hemodynamics, and obtain formal echocardiography to ascertain etiology of shock. Patients may bridge to recovery and device explant, bridge to a more formal long-term ventricular assist device, bridge to heart transplant, or (in some cases) transfer to hospice care. At each step, careful communication with the patient and family about expectations of care, outcomes, complications, and goals should be transparent.

SUMMARY

Several MCS devices are available for use in acute CS complicating AMI. There are little primary data to support one device over another. Evaluation of patient size, access for cannula placement, hemodynamics, and symptoms are important considerations. Close monitoring for complications, particularly limb ischemia, vascular injury, bleeding, and hemolysis, is necessary. Early intervention is warranted. Mortality in CS remains high, but aggressive and timely intervention may help mitigate this. ■

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ABSTRACT & COMMENTARY

Late Awakening Among Cardiac Arrest Survivors Predicts Worse Short- and Long-Term Outcomes

By Vibhu Sharma, MD, MS

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Dr. Sharma discloses no financial relationships relevant to this field of study.

SYNOPSIS: In this retrospective single-center study, late awakening after cardiac arrest was associated with a higher rate of unfavorable outcomes immediately after awakening and at three months.

SOURCE: Rey A, et al. Late awakening in survivors of postanoxic coma: Early neurophysiologic predictors and association with ICU and long-term neurologic recovery. *Crit Care Med* 2019;47:85-92.

Rey et al conducted a retrospective study of consecutive post-cardiac arrest (CA) comatose adult patients. All were admitted to the ICU between 2009 and 2016 and underwent standard post-arrest care, including targeted temperature management (TTM) for 24 hours using surface cooling to 33° C (until December 2014) or 36° C (after December 2014). All patients were sedated for the first 24 hours with either a midazolam drip (first choice) or propofol; a fentanyl drip was administered for analgesia. Opiates and sedatives were discontinued after the initial period of TTM. All patients were mechanically ventilated; mean arterial pressure (MAP) was maintained at > 70 mmHg using vasopressors, if necessary. It is unclear what proportion of these patients experienced CA in the community and how many episodes occurred in the hospital.

Certified neurologists performed a daily standardized exam to assess neurologic recovery, including Glasgow Coma Scale motor response (GCS-M) to pain and brainstem reflexes (pupillary reaction to light, corneal reflexes, and oculocephalic reflexes). The absence of pupillary and corneal reflexes or myoclonus within 72 hours were considered poor prognostic markers. A standard 10-20 lead montage video-electroencephalography (EEG) was performed on day 1 (during TTM and sedation) and then on day 2 after cessation of TTM, off sedation. EEG patterns in response to painful stimuli also were assessed by certified neurophysiologists on days 1 and 2. Absent normothermic EEG and absent bilateral somatosensory evoked potentials (SSEPs) were considered poor prognostic signs. Irreversible cerebral damage within 72 hours was defined as occurring if at least two of the following signs were observed: absent brainstem reflexes, myoclonus, nonreactive EEG, or absent SSEP bilaterally. Serum neuron-specific enolase (NSE) levels were drawn on days 1 and 2 after cardiac arrest. NSE levels > 33 ng/mL at any time within seven days of CA predict persistent coma

with a high degree of specificity.[1] Overall, 228 of 402 patients in the cohort awoke; the rest never regained consciousness and died. One hundred and fifty of the 228 who awoke did so early. Late awakening occurred in 78 of the 228 who awoke, with a median time to awakening of five days (range, five to 23 days). Late awakening was associated with more absent motor responses to painful stimuli and absent brainstem reflexes by day 3 after CA and discontinuous EEG on days 1 and 2 after CA.

Regarding prediction of late awakening, the highest odds were associated with findings of day 2 discontinuous EEG (odds ratio [OR], 3.459), followed by day 3 absence of brainstem reflexes (OR, 3.352), day 3 absent motor response (OR, 3.35), and the use of midazolam alone as the sedating agent during TTM (OR, 1.71). Time to return of spontaneous circulation after CA and day 1 Sequential Organ Failure Assessment score also were significantly associated with late awakening but the association was less robust. TTM strategy (33°C vs. 36°C) did not influence outcomes with respect to awakening.

Despite early unfavorable neurophysiologic signs, 73% of patients with late awakening eventually exhibited good neurologic recovery defined as Cerebral Performance Category (CPC) 1 and 2 outcomes ("Normal life" and "Disabled but independent," respectively). Nevertheless, in this cohort, late awakening was associated with a higher likelihood of CPC categories 3-5 ("Conscious but dependent on others," "Coma or vegetative state," and "Brain death," respectively).

■ COMMENTARY

The results of this study do not apply to patients with catastrophic neurologic injury. Predicting who might awaken after a cardiac arrest can be frustrating because most post-CA patients look "the same." The most important concern for families in this setting may be when and if their loved one will awaken. The relevant finding of interest from the perspective of clinicians and families in this study is that prognostication remains a moving target.

For example, when queried early in admission during a family meeting, it may be reasonable to offer prognostication based on a 48-hour timeline from the point sedation is discontinued. Based on this single-center study, the longer it takes to awaken, the more likely it is that delirium will occur and the longer the patient will be in the ICU. Those awakening late are more likely to be severely disabled or die within three months. When and how much neurologic recovery will occur cannot be predicted with certainty, but early awakening (responding to pain and following commands within 48 hours of sedation discontinuation) makes meaningful recovery more likely.

The single most important recommendation for clinicians is to avoid the use of midazolam for sedation in the CA patient. Clinical findings within 72 hours of CA that convey a poor outcome or nonsurvivability include absent pupillary and corneal reflexes, myoclonus, nonreactive EEG background, and absent bilateral SSEP. In scenarios with disparate neurophysiologic findings, NSE levels may complement prognostication, with higher levels predicting a worse outcome. Prediction of outcomes early after CA remains a daunting task. Caution is warranted in prognostication despite early adverse results on clinical and neurophysiological testing. Sedation using midazolam ought to be avoided in the immediate post-CA phase of ICU care and is the single most modifiable factor influencing time to awakening. ■

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ABSTRACT & COMMENTARY

Rethinking the Prophylactic Use of Proton Pump Inhibitors in the ICU

By Betty Tran, MD, MSc, Editor

SYNOPSIS: The authors of this multicenter, blinded, randomized trial found that among critically ill adults at risk for gastrointestinal (GI) bleeding, fewer patients in the pantoprazole group exhibited clinically important GI bleeding compared to placebo, although mortality at 90 days was similar in both groups.

SOURCE: Krag M, et al. Pantoprazole in patients at risk for gastrointestinal bleeding in the ICU. *N Engl J Med* 2018;379:2199-2208.

Prophylactic use of proton pump inhibitors (PPIs) in critically ill patients at risk for gastrointestinal bleeding (GIB), such as those receiving mechanical ventilation, is standard protocol in the ICU. In fact, it is a component of the daily ICU checklist as an important reminder to ensure compliance. However, data supporting their use are limited in terms of quality, and there are concerns surrounding higher risks of pneumonia and *Clostridium*

difficile that may limit any benefits.[1,2] The Stress Ulcer Prophylaxis in the Intensive Care Unit (SUP-ICU) trial was conducted in 33 ICUs in Denmark, Finland, the Netherlands, Norway, Switzerland, and the United Kingdom between 2016 and 2017. Investigators screened critically ill adults (age > 18 years) who were admitted to the ICU for an acute condition with at least one factor for GIB (shock; use of anticoagulants, nonsteroidal anti-inflammatory drugs, acetylsalicylic acid, and IV thrombolysis; acute renal replacement therapy; mechanical ventilation > 24 hours; chronic liver disease; and coagulopathy).

Ultimately, 3,298 patients were enrolled in the trial and were randomized either to IV pantoprazole 40 mg daily until ICU discharge or death (maximum 90 days) or placebo. If a patient was readmitted to the ICU within 90 days, the original assigned regimen was resumed. The primary outcome was death by 90 days after randomization.

Secondary outcomes included clinically important GIB, new infection with either pneumonia or *C. difficile*, percentage of days alive without life support, serious adverse reactions (e.g., anaphylaxis, pancytopenia, acute hepatic failure), and a composite outcome of clinically important ICU events (GIB, pneumonia, *C. difficile* infection, or myocardial ischemia).

In addition to analyzing the per-protocol population, investigators assessed the primary outcome in predefined subgroups, including presence of liver disease, history of or ongoing coagulopathy, medical vs. surgical ICU, presence of shock, use of mechanical ventilation, and a Simplified Acute Physiology Score (SAPS) II above 53 at baseline (a score of 53 was chosen as predictive of a 50% mortality rate).

At 90 days, similar mortality rates were seen in both the pantoprazole and placebo groups (31.1% vs. 30.4%, respectively; relative risk [RR], 1.02; 95% confidence interval [CI], 0.91-1.13; $P = 0.76$). There was no heterogeneity in the effect of pantoprazole on 90-day mortality in any predefined subgroup except for the group of patients with SAPS II scores > 53, where the mortality appeared higher in the pantoprazole group (RR, 1.13; 95% CI, 0.99-1.30; $P = 0.05$).

In terms of secondary outcomes, the rate of clinically important ICU events was similar in both groups: 21.9% in the PPI group vs. 22.5% in the placebo group (RR, 0.96; 95% CI, 0.83-1.11). More patients in the placebo group demonstrated clinically important GIB compared to the PPI group (4.2% vs. 2.5%, respectively; RR, 0.58; 95% CI, 0.40-0.86). Rates of one or more new infections with pneumonia or *C. difficile*, serious adverse reactions, and days alive without life support were similar between the two groups. The authors did not provide any *P* values for the secondary outcomes, as no adjustments were made for multiple comparisons.

■ COMMENTARY

Although SUP-ICU was a large, multicenter, randomized, placebo-controlled, blinded trial, its results are unlikely to change current practice at this time. The trial did not reveal a significant difference in 90-day mortality between patients at risk for GIB who received a PPI vs. those who did not.

First, the trial was powered to detect an absolute between-group difference of 5%, which may be a large margin to detect. Second, there are no data regarding whether patients were receiving enteral nutrition at baseline, which can modify the effect of PPI prophylaxis on the development of stress ulcer-related GIB as well as risk of pneumonia.[3] Third, the finding of higher 90-day mortality among patients receiving a PPI with a SAPS II score > 53 is hypothesis-generating at best. The finding was borderline significant, and the trial was not powered to address the primary outcome in this subgroup. Finally, although it is difficult to interpret the secondary outcomes in this trial given no adjustment was made for multiple comparisons, the investigators did report an increase in the number of patients with clinically important GIB in the placebo group compared to those receiving a PPI without a difference in the rates of infection.

The composite secondary outcome that combined these measures (referred to as "clinically important events in the ICU") was similar in both groups, but inferences are difficult to draw from this finding as this composite endpoint combines outcomes that are affected by PPI prophylaxis in opposite directions (e.g., PPI prophylaxis theoretically increases the risk of infection but reduces the risk of GIB).

Currently, the standard of care in the ICU is stress ulcer prophylaxis in patients at risk for GIB. Considering that there was an observed higher rate of GIB in patients receiving only placebo without clear evidence of increased risk of infection or mortality in patients receiving PPI prophylaxis in this trial, this standard practice will continue until further evidence emerges to dictate otherwise. ■

REFERENCES

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3. Huang HB, et al. Stress ulcer prophylaxis in intensive care unit patients receiving enteral nutrition: A systematic review and meta-analysis. *Crit Care* 2018;22:20.

CME/CE INSTRUCTIONS

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CME/CE QUESTIONS

1. **Which of these devices requires an atrial trans-septal puncture for catheter placement for left ventricle assist?**
   a. Intra-aortic balloon pump
   b. Impella 2.5
   c. TandemHeart
   d. Venoarterial extracorporeal membrane oxygenation (ECMO)

2. **The best choice for mechanical circulatory support device in cardiogenic shock associated with biventricular failure and hypoxemia is:**
   a. intra-aortic balloon pump.
   b. Impella 2.5.
   c. Bi-Pella (Impella RP plus standard Impella).
   d. venoarterial extracorporeal membrane oxygenation (VA-ECMO).

3. **Which of these devices directly decompresses the left ventricle?**
   a. Intra-aortic balloon pump
   b. Impella CP
   c. Impella RP
   d. TandemHeart

4. **Which of the following findings is associated most strongly with likelihood of late awakening after cardiac arrest?**
   a. Day 2 discontinuous electroencephalography
   b. Day 3 absence of brainstem reflexes
   c. Day 3 absent motor response
   d. Use of midazolam alone as the sedating agent during targeted temperature management

5. **In the trial by Krag et al, compared to placebo, ICU patients assigned to pantoprazole:**
   a. demonstrated lower 90-day mortality.
   b. demonstrated a lower rate of clinically important gastrointestinal bleeding.
   c. demonstrated a higher rate of pneumonia.
   d. demonstrated a higher rate of *Clostridium difficile* infection.

6. **In the trial by Krag et al, which subgroup of patients showed a higher risk of 90-day mortality associated with pantoprazole?**
   a. Those who scored > 53 on the Simplified Acute Physiology Score II scale
   b. Those with mechanical ventilation at randomization
   c. Those in shock at randomization
   d. Those who presented with a history of liver disease

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.