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Vol. 25, Issue 9, December 2017
AHCMedia.com

Critical Care [ALERT]

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SPECIAL FEATURE

Right Heart Failure in the ICU

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Dr. Shah reports he is on the speakers bureau of Gilead Sciences.

Right ventricular (RV) failure continues to be a formidable clinical challenge in critical care medicine. Although no consensus definition of RV failure exists, it is defined commonly as the clinical result of RV dysfunction with onset of hypotension or end organ damage.[1] Clinically, RV failure is characterized by reduced cardiac output (cardiac index < 2.5 L/min/m^2) and elevated right-sided filling pressures (right atrial pressure > 8 mmHg).[2] Regardless of the etiology, pulmonary hypertension and RV failure carries a poor prognosis. Prompt identification and treatment is warranted.[1,3-6] Unfortunately, there is considerable lack of evidence in the management of RV failure in the medical ICU. Most recommendations are based on expert opinion or low-quality evidence.[1,7]

PATHOPHYSIOLOGY AND ETIOLOGY OF RV FAILURE

Normal pulmonary circulation is a low-resistance, low-pressure, high-compliance, high-flow system. Generally, RV afterload is low, and the normal thin-walled RV generates significantly lower pressures compared to the left ventricle (LV). The most common causes of RV failure include RV ischemia (e.g., RV infarction) or a sudden increase in RV afterload (e.g., pulmonary embolism, worsening of pulmonary arterial hypertension). If RV afterload increases gradually, the RV can undergo remodeling and adapt, but it adapts poorly to sudden increases in afterload.[8,9] A normal RV is unable to acutely increase mean pulmonary pressures > 40 mmHg, whereas a chronically remodeled RV can mount systematic level pressures.[8,9]

When decreased contractility causes RV failure, as in the case of RV infarction, afterload generally is low, and cardiac output is dependent on preload. However, in many other etiologies of RV failure, there is an acute increase in RV afterload (e.g., acute respiratory distress syndrome, pulmonary embolism, pulmonary arterial hypertension). Timing of RV failure depends on the severity of the increase in afterload and pre-event RV function. For example, patients with severe pulmonary arterial hypertension might exhibit preexisting

Financial Disclosure: *Critical Care Alert*'s Physician Editor Betty Tran, MD, MSc, Nurse Planner Jane Guttendorf, DNP, RN, CRNP, ACNP-BC, CCRN, Peer Reviewer William Thompson, MD, Executive Editor Leslie Coplin, Editor Jonathan Springston, and AHC Media Editorial Group Manager Terrey L. Hatcher report no financial relationships relevant to this field of study.

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Critical Care Alert, (ISSN 1067-9502) is published monthly by AHC Media, a Relias Learning company, 111 Corning Road, Suite 250, Cary, NC 27518

Periodicals Postage Paid at Atlanta, GA, and at additional mailing offices.

GST Registration Number: R128870672.
POSTMASTER: Send address changes to *AHC Media, LLC, P.O. Box 74008694, Chicago, IL 60674-8694*

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Editor's Note

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RV dysfunction and carry little reserve to compensate for acute increases in preload or afterload. Also, the RV and LV are interdependent through the presence of the interventricular septum and pericardium. Normally, LV systolic function helps RV ejection because of this interdependence.[10,11] However, in right heart failure, increased RV pressures and pericardial pressures lead to septal wall displacement toward the LV, causing underfilling and LV diastolic dysfunction.[10-12] In this case, pulmonary capillary wedge pressure (PCWP) becomes unreliable as an estimate of LV preload. Transmural pressures (PCWP — right atrial pressure) should be used instead.[12] Thus, a dilated and overloaded RV can lead to decreased LV filling and decreased cardiac output. Normal right coronary blood flow is dependent on the pressure gradient between the aorta and RV, and occurs during both systole and diastole because of normal low RV pressures. However, in RV failure, increases in RV pressure and oxygen demand from increased contractility may cause RV ischemia, which further worsens RV function.[13]

Mechanical ventilation can contribute to RV failure by preload reduction due to increased intrathoracic pressures and decreased venous return. It can also increase afterload as a result of alveolar over distention due to high positive end-expiratory pressure (PEEP) and/or high tidal volumes.[14] Higher tidal volumes, plateau pressures, and driving pressures also can increase cytokine release, leading to endothelial injury and dysfunction in the pulmonary circulation.[14]

Similarly, cytokine release in sepsis can induce pulmonary vascular endothelial dysfunction and cause an increase in pulmonary vascular resistance (PVR).[15] Hypoxemia, hypercapnia, and acidosis lead to pulmonary vasoconstriction.[16] In certain situations, such as during cardiac surgery, cardiopulmonary bypass can induce pulmonary vascular dysfunction and an increase in PVR in addition to lung injury.[17]

DIAGNOSIS OF RV FAILURE

A high index of suspicion is needed for the diagnosis of RV failure. A history of the underlying cause might be a first clue to diagnosis. Transthoracic echocardiogram (TTE) is a reasonable screening tool to assess severity of right heart failure as well as to rule out left heart failure and valvular heart disease as the cause of pulmonary hypertension. TTE should be used first, prior to more invasive measures like pulmonary artery catheters (PAC).

Although PAC is the gold standard for diagnosing pulmonary hypertension, routine use of a PAC is not recommended for most ICU patients.[18] Recommendations against the use of PACs have been more clear in acute lung injury and cardiogenic shock secondary to left heart failure, but these studies excluded patients with group 1 pulmonary hypertension.[18]

In the latter subset of patients with RV failure, PACs can be used to establish a diagnosis or guide management in select patients in whom TTE is not sufficient for diagnosis or management. In postoperative cardiac surgery patients, it is reasonable to use PACs for postoperative management. However, in these cases, PACs should be removed as soon as their utility is over. Various serum markers of organ dysfunction, like creatinine, liver enzymes, and lactic acid, can be used to assess severity of RV failure and response to therapy.

N-terminal pro-brain natriuretic peptide (NT-proBNP) and BNP are important markers. Elevated levels predict increased mortality.[19-21] For all patients with unexplained RV failure, pulmonary

embolism should be suspected. Pulmonary embolism is a common cause of RV failure, even in patients who present with preexisting RV dysfunction. CT angiogram should be considered for acute pulmonary embolism, while a ventilation-perfusion scan should be considered if chronic thromboembolism is suspected. A right-sided ECG and troponin levels should be obtained if RV infarction is suspected.

MANAGEMENT OF RV FAILURE

Management of RV failure includes treating the underlying disease, optimizing volume status, providing hemodynamic support, and avoiding further injury to the RV or an increase in afterload. Management of these patients should be handled at an expert pulmonary hypertension center and considered in a stepwise manner.[2]

Step 1: Treatment of triggering factors and supportive measures. In the current era, RV failure is an uncommon first presentation of pulmonary arterial hypertension (PAH) because of improvement in diagnosis and treatment of this disease. However, from time to time, these cases occur and must be identified and treated promptly. Most of these cases feature a triggering factor that leads to acute decompensation of the patient's pulmonary hypertension.[21] Forty-one percent of patients in a French series experienced a triggering factor, most commonly infection, anemia, arrhythmia, pulmonary embolism, volume overload, or myocardial infarction.[21] These factors should be identified and treated promptly.

Infection needs special mention since documented infection was the strongest predictor of death in that study. In addition to usual sources of infection, patients in right heart failure demonstrate increased bacterial translocation from the bowel due to gut edema from elevated central venous pressures, leading to loss of intestinal barrier function.[2] PAH patients do not tolerate atrial arrhythmias well because of loss of atrial kick.

A goal should be to restore sinus rhythm with either electric or chemical cardioversion. Rate-controlling medications like calcium channel blockers and beta-blockers should be avoided as they are poorly tolerated in these patients. Anemia is common, mainly because of gastrointestinal (GI) bleeding. To optimize the oxygen-carrying capacity of blood, optimal hemoglobin levels should be maintained. Again, this subpopulation has not been included in most studies; hence, maintaining a hemoglobin level > 8 g/dL is reasonable. Endotracheal intubation and mechanical ventilation should be avoided if at all possible. If absolutely necessary, sedation for anesthesia should be considered carefully as the drop in systemic vascular resistance (SVR) is poorly tolerated in RV failure patients. Because of its minimal cardiovascular effects, etomidate is the induction agent of choice.[22] Another option is to use awake fiber optic intubation with local anesthesia. Pretreatment with a vasopressor medication should be considered, too. Post-intubation PEEP, plateau pressures, driving pressures, hypercapnia, hypoxia, and acidosis should be minimized as much as possible.[14]

Step 2: Volume optimization. Most patients exhibit volume overload and need prompt diuresis and/or hemofiltration. However, a minority demonstrate hypovolemia and might need a small amount of fluid. Most routinely used measures of volume status in the ICU are not studied in this population. More than one test should be used prior to fluid administration. Generally, fluid administration should be in small amounts (e.g., 250 cc normal saline or lactated Ringer's).

Step 3: Reduce RV afterload. In patients with PAH with RV failure, inhaled or IV prostanoids are the drugs of choice. IV epoprostenol has been shown to offer a mortality benefit in these patients.[23] However, these drugs cause a drop in SVR in addition to decreasing PVR. These patients may need administration of vasopressor medications to maintain adequate SVR. For the same reasons, these medications must be started at a lower dose and increased gradually. Oral medications (e.g., PDE-5 inhibitors, endothelin receptor antagonists) generally are not useful in the setting of right heart failure, but can be used as an adjunct to prostanoids.

Step 4: Optimize cardiac output. Inotropes can be used to augment RV contractility. PDE-3 inhibitors (e.g., milrinone) and dobutamine are used commonly. As dobutamine causes more tachyarrhythmias, PDE-3 inhibitors are preferred. Additionally, PDE-3 inhibitors also can induce pulmonary vasodilation and help with afterload reduction.[24] Systemic hypotension is a common limitation for using these medications. Concomitant vasopressors often are needed.

Step 5: Optimize perfusion pressure. With the use of pulmonary vasodilators like IV prostanoids and inotropes, systemic hypotension is common; hence, vasopressors are needed commonly. Triggering factors like sepsis and GI bleed also cause a decrease in SVR and may require vasopressor support. Based on current evidence and experience, low-dose vasopressin appears to be beneficial, but higher doses may be harmful.[25,26] Vasopressin at 0.01-0.04 U/min can be used to increase SVR. High-dose dopamine should be avoided, as dopamine causes more arrhythmias as compared to norepinephrine and an increased rate of

death in a subgroup of patients with left-sided cardiogenic shock.[27] Lower-dose dopamine might help with inotropic and chronotropic support, but data to support this are weak. Norepinephrine should be used as the vasopressor of choice. Data to support its use in PAH are weak but are extrapolated from studies in circulatory and cardiogenic shock.[27] Epinephrine can be used as the next vasopressor.

Step 6: Advanced therapies. Atrial septostomy can be considered after stabilization of the patient. This helps decrease RV filling pressures and subsequently helps the failing RV by decreasing preload.[28] Since this procedure causes a right to left shunt, it is only appropriate for patients with a room air SpO_2 > 80%. Lung or heart-lung transplantation remains an important and last treatment option for patients with progressive RV failure despite the above measures. For patients who are transplant candidates, venous arterial extracorporeal membrane oxygenation is used often as a bridge to transplant.[29]

SUMMARY

Right heart failure in the ICU remains an important challenge. Prompt identification and treatment of the underlying disease is warranted. Additionally, most patients with RV failure need volume optimization and inotropic and vasopressor support, too. In patients with PAH, a stepwise approach to treatment of RV failure should be adopted with special effort to identify and treat triggering factors. ■

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ABSTRACT & COMMENTARY

Deflating Recruitment Maneuvers

By Richard Kallet, MS, RRT, FCCM

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Mr. Kallet reports he is a major stockholder in the Asthma & Allergy Prevention Company, and receives grant/research support from Nihon Kohden.

SYNOPSIS: Compared to the ARDSNet lower positive end-expiratory pressure (PEEP)/low tidal volume strategy, use of an "open lung ventilation" strategy consisting of aggressive alveolar recruitment maneuvers and higher PEEP is associated with higher mortality in patients with moderate to severe acute respiratory distress syndrome.

SOURCE: Cavalcanti AB, et al. Effect of lung recruitment and titrated positive end-expiratory pressure (PEEP) vs low PEEP on mortality in patients with acute respiratory distress syndrome: A randomized clinical trial. *JAMA* 2017;318:1335-1345.

The authors of this multinational, prospective, randomized, controlled trial enrolled more than 1,000 subjects with moderate to severe acute respiratory distress syndrome (ARDS) within 72 hours of onset. These patients received either 1) an open lung ventilation (OLV) strategy in which a neuromuscular blockade was administered, followed by a lung recruitment maneuver (RM), with incremental positive end-expiratory pressure (PEEP) levels titrated to the best respiratory-system static compliance, or 2) a conventional low-PEEP lung-protective ventilation (LPV) strategy. Patients had to be hemodynamically stable and without evidence of barotrauma. At baseline, there was no difference between treatment arms in terms of demographics, illness severity, comorbidities, pulmonary mechanics, gas exchange, or in achieved LPV goals. Sixty-two percent of subjects demonstrated a pulmonary source of ARDS and 67% exhibited septic shock. Of those randomized to OLV, 96% received an initial RM, and 78% received an RM following the PEEP decrement trial. Approximately 63% of subjects required no further RMs vs. 9% who required three or more maneuvers during the trial. On the first two study days, mean PEEP levels were approximately 4 cm H_2O higher in the OLV vs. low-PEEP groups (16.2 vs. 12.0 and 14.2 vs. 10.5 cm H_2O, respectively). Mean plateau and driving pressures were significantly higher in the OLV group, yet both variables were well within accepted LPV boundaries for each group. Mortality in the OLV group was significantly higher at both day 28 and at six months compared to the control low-PEEP group (55.3 vs. 49.3% and 65.3 vs. 59.9%, respectively). The OLV group experienced slightly fewer ventilator-free days than the conventional low-PEEP group; however, both intensive care and hospital lengths of stay were not different.

■ COMMENTARY

ARDS presenting with severe hypoxemia refractory to high PEEP levels often reflects the contribution of enormous compressive forces emanating from reduced chest wall compliance. Over several decades, mounting evidence suggests that inspiratory pressures between 40-60 cm H_2O are necessary to fully recruit dorsal-caudal regions and reverse intractable hypoxemia. While the mechanical foundation for a recruitment maneuver is sound, its application in ARDS has remained uncertain given both the heterogeneous nature of lung injury and the inability to ascertain the amount of potentially recruitable lung vs. consolidated lung. Moreover, the degree to which atelectrauma contributes to lung injury and mortality risk in ARDS has (up to this point) remained unanswered.

Clearly, the Cavalcanti et al study indicates that an open lung ventilation strategy with recruitment maneuvers and higher PEEP should not be used routinely in the management of ARDS. Nonetheless, more in-depth analysis of the study results is needed to determine how OLV should be used going forward. At this juncture, it would be unwise to reject the use of RM in ARDS categorically. First, 63% of subjects received only two RMs, the duration of which was a total of eight minutes at plateau pressures of 40-60 cm H_2O at a safe driving pressure. Given what is known about ventilator-induced lung injury, it's implausible that such brief exposure could affect mortality that profoundly.

What is plausible is that the 46 patients who received three or more RMs may have been harmed. Subjects requiring repeated RMs may have been less recruitable and more susceptible to aggravating a proinflammatory state in a study sample characterized by pulmonary ARDS and septic shock. This would include regional lung overdistension, the possibility of bacterial translocation, and repeated gastrointestinal ischemia/reperfusion injury.[1,2] The higher mean PEEP levels in the OLV group would not explain the increased mortality, as it was only 3-4 cm H_2O above the conventional low-PEEP group. In contrast, dif-

ferences in mean PEEP in three previous major LPV studies (ALVEOLI, EXPRESS, LOVS) was 6-8 cm H_2O. A similar relationship existed for higher mean plateau and driving pressures in those studies compared to the ART study.

What can we reasonably conclude at this juncture? The OLV strategy is not necessary for managing the vast majority of ARDS cases, and atelectrauma is unlikely to be a mortality driver when LPV incorporates reasonable levels of PEEP early in the acute phase (e.g., ~12-16 cm H_2O). However, there exists a small subset of ARDS where these levels of PEEP and other ancillary therapies are insufficient. These situations are likely restricted to cases of severe lung injury complicated by markedly reduced chest wall compliance that necessitate the OLV strategy as a temporizing measure to stabilize oxygenation and reduce long-term exposure to hyperoxia. Unfortunately, given that these cases represent such a small minority of ARDS, we will never see an adequately powered study to provide conclusive evidence in a timely manner. ■

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ABSTRACT & COMMENTARY

Oncologists and Intensivists Perceive Prognosis Differently for Critically Ill Patients with Cancer

By Elaine Chen, MD

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

SYNOPSIS: When considering prognostication or limitation of care in critically ill patients with different cancers, oncologists may focus on cancer characteristics, whereas intensivists may focus on multiple organ failure.

SOURCE: Nassar AP Jr, et al. Oncologists' and intensivists' attitudes toward the care of critically ill patients with cancer. *J Intensive Care Med* 2017 Jan 1:885066617716105. doi: 10.1177/0885066617716105. [Epub ahead of print].

Approximately 20% of all ICU admissions present with cancer, and ICU mortality for patients with cancer is approximately 30%. Intensivists might be overly pessimistic about their patients with cancer, and oncologists might be overly optimistic about their patients' survival. Metastatic cancer often is associated with refusal of ICU admission in some countries. The aim of this study was to identify whether oncologists and intensivists would make different decisions in clinical scenarios for two critically ill patients with cancer.

The authors created a survey that included demographic information and two hypothetical patient vignettes, which the authors administered to oncologists and intensivists at two academic cancer centers in Brazil. The case vignettes were identical, other than the type of cancer: metastatic pancreatic or triple-positive metastatic breast cancer. The patient developed septic shock after two days of inpatient therapy for community acquired pneumonia. Participants were asked about their decision-making at this juncture and again three days later when the patient was deteriorating despite mechanical ventilation, vasopressors, and renal replacement therapy.

At the first juncture, options included no ICU admission, ICU admission with discussion of eventual limitation of life support, and ICU admission without discussion of eventual limitation of life support. At the second juncture, options included comfort measures with withdrawal of life support, withholding (which investigators defined as maintenance of life support without additional measures), or full code. The authors hypothesized that oncologists would focus on cancer status and intensivists would focus on organ dysfunction when making decisions, and that oncologists would manage the patients differently, whereas intensivists would favor limitation of care for both patients.

Of 113 physicians invited, 106 completed the survey. Sixty were intensivists, and 46 were

oncologists. More oncologists had attended end-of-life or palliative care courses, but more intensivists had participated in end-of-life or palliative care educational activities within the past year.

For the pancreatic cancer patient, more intensivists (75%) than oncologists (52%) opted for admission to the ICU with discussion of eventual limitation of life support ($P = 0.02$). Most of the oncologists who chose otherwise opted for no ICU admission. At three days, most respondents opted for withdrawal of life support (76%), with no difference between oncologists and intensivists.

For the breast cancer patient, no participant selected no ICU admission, and most opted for ICU admission with discussion of eventual limitation of life support, with more intensivists (78%) than oncologists (59%) selecting this option ($P = 0.055$). At three days, more intensivists (54%) than oncologists (21%) favored withdrawal of life support ($P < 0.001$).

For both patient types, those who had attended palliative care education at any time opted for withdrawal more frequently than those who had not (90% vs. 53% for the pancreatic patient, $P = 0.018$; 46% vs. 12% for the breast cancer patient, $P = 0.007$). More oncologists favored full code for the breast cancer patient compared to the pancreatic cancer patient (27% vs. 0%, $P < 0.001$), whereas there were no differences among the intensivists (13% vs. 3%; $P = 0.094$).

These results suggest that oncologists and intensivists use different clinical information in their decision-making processes. The authors hypothesized that oncologists may pay more attention to cancer survival rates, and intensivists pay more attention to organ dysfunction survival rates. In the pancreatic cancer patient, this led to the same conclusion of a dismal prognosis, but different conclusions in the breast cancer patient. While this simple study does not further evaluate decision-making processes, it does point out potential cognitive biases and areas of conflict between oncologists and intensivists. The study authors also noted that receiving updates on palliative care and end-of-life issues is associated with a higher tendency to withdraw life support.

■ COMMENTARY

This study provides important perspective to help intensivists be mindful in their conversations with referring colleagues. An intensivist's perspective is appropriately narrowly focused on the patient's current critical status. However, referring colleagues may have a long-term outpatient relationship with strong emotional connections and the perspective of the patient's prior excellent functional status.[1] Personally speaking, as a pulmonologist, my outlook for my chronic stable outpatients who are admitted to the ICU often is more positive than it would be for similar patients I first meet when they are critically ill.

Conflicts are common in critically ill patients requiring life-sustaining therapy. Up to 50% of these conflicts arise among hospital staff,[2] and up to 20% of conflicts involve ICU staff and consultants.[3] These conflicts may arise between oncologists and intensivists regarding the aggressiveness of management of critically ill patients with cancer. Conflict may not necessarily lead to negative outcomes, but can present an opportunity for constructive conversation.

This study notched an exceptionally high response rate of 94%. The survey was short and simple (four questions). The simplicity of the survey likely contributed to the high response rate but limited the extent of potential analysis. Other limitations include the fact that survey responses may not actually reflect clinical practice, and that only cancer centers in Brazil were included. Do these results generalize to community hospitals without cancer centers elsewhere in the world? Are there practice differences in Brazil compared to the United States? For example, refusing admission to the ICU from an acute care unit is a rare practice in the United States, regardless of presenting illness. Many oncologists in this study recommended deferring ICU admission for the pancreatic cancer patient, while intensivists recommended admission; this result warrants further investigation.

As cancer treatment improves, worldwide ICU use by cancer patients has increased, and outcomes have improved.[4] Given the increasing use of ICUs by cancer patients, intensivists and oncologists should respectfully remember that perspectives are different and communicate regularly about their perspectives in hopes of reducing conflict. ■

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CME/CE QUESTIONS

1. **What is the recommended first screening tool to diagnose right ventricular (RV) failure in an ICU patient?**
   a. Pulmonary artery catheterization
   b. Transthoracic echocardiogram
   c. Brain natriuretic peptide level
   d. CT pulmonary angiogram
   e. Transesophageal echocardiogram

2. **In a patient with a history of pulmonary arterial hypertension (group 1) presenting with RV failure in the ICU, what is the first step in management?**
   a. Identify and treat triggering factors, supportive measures, and volume optimization.
   b. Start IV prostaglandins.
   c. Start milrinone.
   d. Start norepinephrine.
   e. Refer patient for lung transplantation.

3. **Which of the following statements is *false* regarding the Cavalcanti et al study?**
   a. The majority of enrolled subjects had an extrapulmonary source of acute respiratory distress syndrome (ARDS).
   b. The majority of enrolled subjects had a pulmonary source of ARDS.
   c. The majority of enrolled subjects had septic shock as a co-diagnosis.
   d. The majority of subjects enrolled into the experimental arm received an initial recruitment maneuver.

4. **In the report by Nassar et al, which of the following is *true* regarding oncologists' and intensivists' decision-making?**
   a. Regardless of specialty, those who had participated in end-of-life or palliative training were less likely to recommend no ICU admission for patients with metastatic cancer.
   b. Regardless of specialty, those who had participated in end-of-life or palliative training were less likely to recommend withdrawal of life support after three days of life-sustaining therapy.
   c. Oncologists apply similar decision-making processes to patients with metastatic pancreatic cancer and metastatic breast cancer when considering admission to an ICU.
   d. In a patient with breast cancer and multi-organ failure despite three days of life-sustaining therapy, both oncologists and intensivists favor withdrawal of life support at similar rates.
   e. In a patient with pancreatic cancer and multi-organ failure despite three days of life sustaining therapy, both oncologists and intensivists favor withdrawal of life support at similar rates.

5. **In the report by Nassar et al, which of the following was found regarding decision-making for patients with metastatic cancer?**
   a. Oncologists more often favor full code status for patients with breast cancer than pancreatic cancer.
   b. Intensivists more often favor full code status for patients with breast cancer than pancreatic cancer.
   c. Intensivists are more likely to recommend no ICU admission for patients with metastatic pancreatic cancer than patients with breast cancer.
   d. Intensivists are more likely to recommend no ICU admission for patients with metastatic pancreatic cancer than are oncologists.
   e. The cancer characteristics of a patient with metastatic cancer is likely to equally affect decision-making in oncologists and intensivists.

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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Critical Care [ALERT]

Vol. 25, Issue 9, December 2017
Special Supplement
AHCMedia.com

Authoritative, evidence-based summaries for the critical care clinician

Figure 1. Treatment Algorithm for Right Ventricular Failure in the ICU

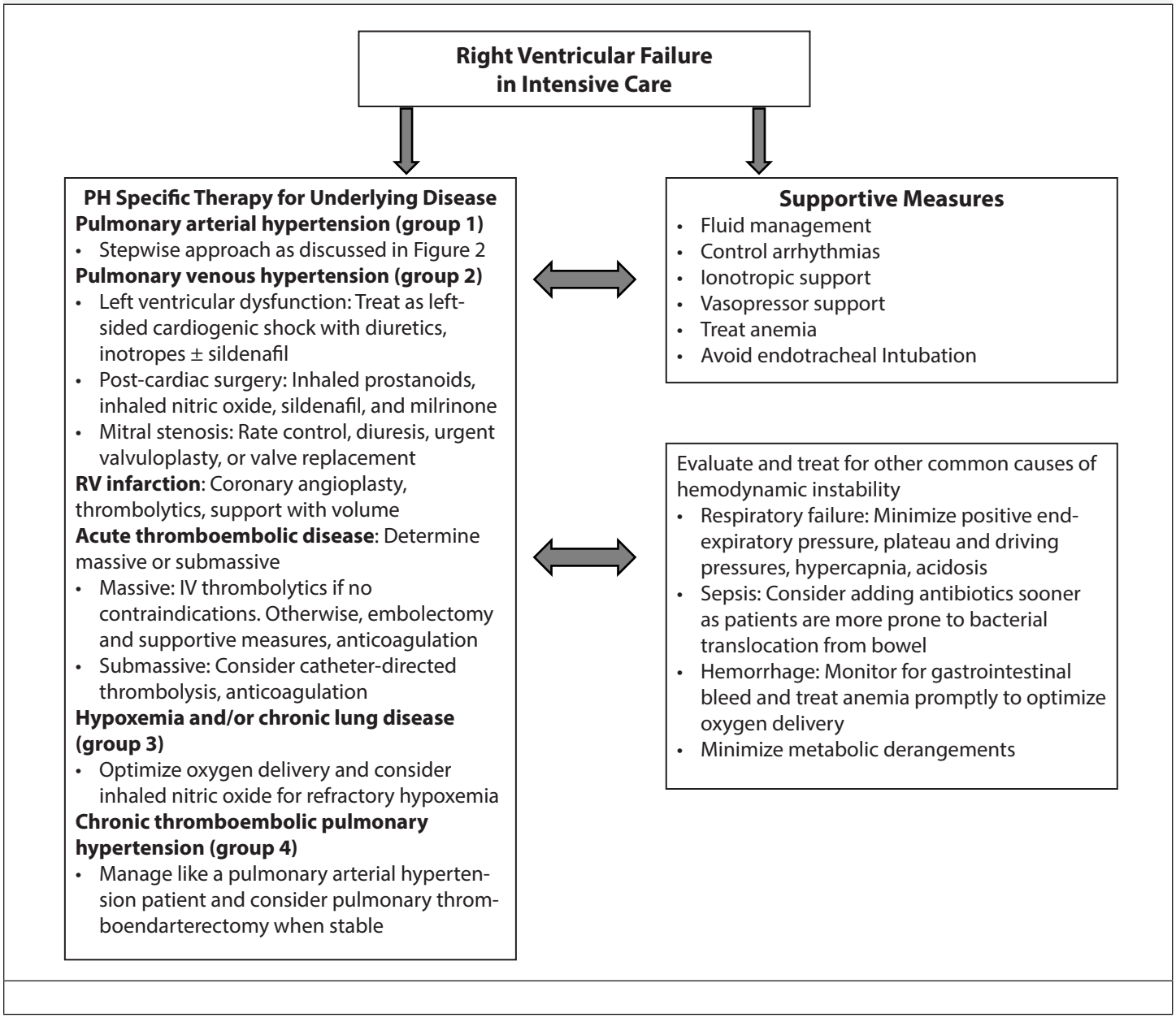

Figure 2. Stepwise Approach to Right Ventricular Failure in Pulmonary Arterial Hypertension Patients

Step 1 and 2 are needed in every patient. Escalation to the next step is recommended if not responding adequately.

Step 1: Treatment of triggering factors and supportive measures
- Treat infections, anemia, arrhythmias
- Rule out pulmonary embolism, myocardial infarction
- Correct hypoxemia with oxygen supplementation, avoid intubation
- Contact PH referral center

Step 2: Volume optimization
- Diuresis and/or hemofiltration for volume overload
- IV fluids for suspected hypovolemia (confirmed by objective tests)

Step 3: Reduce RV afterload
- IV prostaglandins are treatment of choice
- Inhaled prostanoids and nitric oxide can be considered, especially if severe hypoxemia is present from pneumonia/acute lung injury
- Oral therapy is only adjunctive to above

Step 4: Optimize cardiac output
- Dobutamine or PDE-3 inhibitors to goal central venous oxygen saturation ($ScVO_2$) > 70%, mixed venous oxygen saturation (SVO_2) > 65 %, or cardiac index > 2.0 L/min/m^2

Step 5: Optimize perfusion pressure
- Vasopressors like norepinephrine, vasopressin, and epinephrine

Step 6: Advanced therapies
- Consider lung transplantation, atrial septostomy, and VA ECMO as bridge to transplant