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Acute Right Ventricular Dysfunction
Real-Time Management With Echocardiography

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VIDEO

In critically ill patients, the right ventricle is susceptible to dysfunction due to increased afterload, decreased contractility, or alterations in preload. With the increased use of point-of-care ultrasonography and a decline in the use of pulmonary artery catheters, echocardiography can be the ideal tool for evaluation and to guide hemodynamic and respiratory therapy. We review the epidemiology of right ventricular failure in critically ill patients; echocardiographic parameters for evaluating the right ventricle; and the impact of mechanical ventilation, fluid therapy, and vasoactive infusions on the right ventricle. Finally, we summarize the principles of management in the context of right ventricular dysfunction and provide recommendations for echocardiography-guided management. CHEST 2015; 147(3):835-846

ABBREVIATIONS: LV = left ventricular; LVAD = left ventricular assist device; PEEP = positive end-expiratory pressure; PVR = pulmonary vascular resistance; RAP = right atrial pressure; RV = right ventricular; RVFAC = right ventricular fractional area change; S′ = peak systolic velocity; TAPSE = tricuspid annular plane systolic excursion; TEE = transesophageal echocardiography; TTE = transthoracic echocardiography

In critically ill patients with circulatory shock, the role of the left ventricle has long been appreciated. The right ventricle, in contrast, is considered "forgotten" perhaps because it is thinner walled, more difficult to image, and coupled indirectly to the systemic circulation. The ascendance of intensivist-conducted echocardiography has forced us to revise this view.[1] New evidence shows acute right ventricular (RV) dysfunction to be common, readily detected with simple bedside imaging, amenable to basic ICU interventions, yet often lethal. We review the epidemiology of RV dysfunction; the crucial role both transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) play to reveal shock due to acute RV dysfunction; and how ventilator changes, fluid therapy, and vasoactive drug infusions can be titrated based on real-time imaging. Although some patients with acute RV dysfunction have preexisting pulmonary hypertension, we restrict this review to patients without preexisting disease.

Knowledge of RV physiology is essential to the intensivist because several supportive therapies, including mechanical ventilation and fluid management, interact with RV dysfunction, potentially exacerbating shock. We briefly review the epidemiology of acute RV dysfunction and echocardiographic

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parameters for judging RV function and conclude with principles and recommendations for using echocardiography to guide management. Strong evidence for the use of this echocardiographic-based approach is currently lacking; thus, the majority of our recommendations are based on experience, physiologic reasoning, and logical extension of the existing literature.

Epidemiology

Acute RV dysfunction causes and exacerbates many common critical illnesses (Table 1). The right side of the heart, comprising the right atrium and right ventricle, accepts the entire cardiac output and pumps it through the pulmonary circulation. Because the pulmonary vasculature is characterized by low resistance and high compliance, pulmonary artery pressures are quite low, typically 25/10 mm Hg. Moreover, the right ventricle operates below its unstressed volume (ie, increasing its volume does not raise its pressure) so that atrial pressure does not reflect preload.[2] The thin-walled right ventricle is much more sensitive to increases in afterload than the left ventricle, making it vulnerable to systolic failure in disease states that raise RV afterload, a condition known as acute cor pulmonale.

ARDS is one of the most common conditions to challenge the right ventricle. The incidence of acute RV failure is around 25% with lung protective ventilation, a value corroborated by several studies, although it can be much higher depending on the severity of lung injury and the chosen ventilator strategy.[3-6] Acute RV dysfunction (defined as a dilated right ventricle with septal dyskinesia) was found in 22% of 226 patients studied by TEE within the first 3 days after diagnosis of moderate to severe ARDS,[7] with acute RV dysfunction an independent risk factor for 28-day mortality. Another survey revealed similar findings, with 22.5% of 204 patients with ARDS who were ventilated showing acute RV dysfunction.[8]

In unselected patients with pulmonary embolism, echocardiographic features of RV strain or dysfunction are present in between 29% and 56%.[9,10] The presence of RV dysfunction imparts a sixfold increase in the risk of in-hospital mortality. Even among patients with pulmonary embolism who are hemodynamically stable at presentation, the presence of acute RV dysfunction correlates with an increased risk of developing shock and dying in the hospital.[11]

Acute RV dysfunction is the hallmark of RV infarction, complicating roughly one-third of cases of ST-elevation infarction of the left ventricular (LV) inferior wall. In patients with inferior myocardial infarction, RV infarction increases the risk of complications and death. Distinguishing RV infarction from other causes of acute RV dysfunction can be challenging.

The pathophysiology of postcardiotomy RV failure is related to ischemia and myocardial depression during cardiopulmonary bypass and aortic cross-clamping, alteration of RV size and shape due to LV unloading after left ventricular assist device insertion (LVAD),[12] and donor heart ischemia and preexisting pulmonary vascular disease in heart transplant recipients.[13] On occasion, allograft rejection or mechanical obstruction at the pulmonary artery anastomosis in heart transplants, preexisting pulmonary hypertension, coronary embolism or graft occlusion, arrhythmias, pulmonary hypertension related to protamine or acute lung injury, postoperative pulmonary embolism, or sepsis could also contribute to postcardiac surgery RV dysfunction. Acute RV dysfunction after cardiothoracic surgery is a major cause of morbidity and mortality in specific patient populations. Although the overall incidence of RV failure after cardiotomy is 0.1%, it occurs in 2% to 3% of patients after heart transplantation and up to 30% of patients after LVAD insertion. Significantly, acute RV dysfunction after cardiac surgery is associated with mortality rates as high as 80%.[14] Excellent reviews on the topic have been published.[15,16]

Patients undergoing lung resection are at postoperative risk for increased pulmonary vascular resistance (PVR) due to loss of pulmonary tissue. When combined with the effects of atelectasis, hypoxia, and hypercarbia, acute RV dysfunction may follow.[17] Preexisting pulmonary vascular disease or RV dysfunction and the extent of pulmonary parenchymal resection predict the severity of postoperative RV dysfunction in these patients.

Diagnosis of Acute RV Dysfunction

Clinical clues to acute RV dysfunction include elevated right-sided filling pressures, a right-sided third heart sound, tricuspid regurgitation, a pulsatile liver, and peripheral edema. Systemic hypotension, alterations in liver function tests,[18] creatinine level, urine output, neurologic function, mixed and central venous saturation, and lactate and natriuretic peptide levels provide insight into the degree of venous congestion and tissue hypoperfusion and are clues about the clinical impact of RV dysfunction. ECG signs of RV dysfunction are varied and inconsistent.[19] Especially in light of the fading relevance of the pulmonary artery catheter,[20] diagnosis typically relies on echocardiography. Intensivist-performed point-of-care echocardiography has become particularly

TABLE 1] Causes of Acute RV Dysfunction

Causes
Acute pressure overload
Increased pulmonary venous pressure
LV dysfunction
Mitral regurgitation or stenosis
Pulmonary veno-occlusive disease
Increased pulmonary artery pressure
Worsening of preexisting pulmonary hypertension
Pulmonary vasoconstriction due to hypoxia or hypercarbia
Mechanical ventilation (related to elevated PEEP and plateau airway pressure)
ALI and ARDS (including postsurgical ALI due to post-CPB or lung transplantation)
Extensive lung resection
Pulmonary embolism (thromboembolic, air, cement, tumor, or amniotic fluid)
Acute chest syndrome in sickle cell disease
Congenital cardiac disease with pulmonary outflow obstruction
Drugs (eg, protamine)
RV outflow tract obstruction
Congenital cardiac disease
Postcardiac surgery
Acute decrease in contractile function
RV ischemia or infarction
Relative RV ischemia (inadequate coronary perfusion related to oxygen consumption in overload conditions)
LV dysfunction (through ventricular interdependence)
Cardiomyopathy (eg, septic)
Myocarditis (viral, bacterial)
Post-CPB
LVAD-related change in RV geometry
Chest trauma with cardiac contusion
Arrhythmias
Acute volume overload
Valvular disease
Severe tricuspid regurgitation
Severe pulmonary regurgitation
Hypervolemia (in susceptible patients)
Intracardiac shunts
Acute decrease in diastolic filling
Cardiac tamponade
Constrictive pericarditis
Mechanical ventilation

ALI = acute lung injury; CPB = cardiopulmonary bypass; LV = left ventricular; LVAD = left ventricular assist device; PEEP = positive end-expiratory pressure; RV = right ventricular.

important for detecting acute RV dysfunction and both guiding specific therapies and monitoring the impact of supportive care, such as fluid therapy and mechanical ventilation.

Echocardiographic Views for RV Assessment

RV size and function can be evaluated through both TEE and TTE. Examples of commonly acquired TEE and TTE views to evaluate RV function are shown in Videos 1 to 9. Visualization of the right ventricle can be challenging on TTE because of its complex three-dimensional geometry and sonographic interference from the lungs. Although TTE provides adequate imaging in 80% to 90% of critically ill patients,[21] particularly in reference to the identification of RV failure in patients with ARDS, TEE provides adequate image quality more frequently than does TEE (87% vs 61%).[8] Moreover, in the setting of postcardiac surgery acute RV dysfunction, incisions, drains, dressings, and pericardial air make finding adequate TTE imaging windows difficult, resulting in inadequate imaging of the right ventricle in 37% of cases.[21] More recently, a miniaturized TEE probe for continuous hemodynamic monitoring (hemodynamic TEE, or hTEE) has been used for guiding management in patients who are ventilated,[22] monitoring RV recovery in sepsis, and monitoring patients on venoarterial extracorporeal life support.[23] The probe is designed to be left in place for 72 h and provides acceptable image quality in the majority of patients. This probe can provide two-dimensional and color Doppler echocardiographic evaluation but no spectral Doppler echocardiographic measurements.

An accurate assessment of RV structure and function requires integration of multiple echocardiographic views because of its crescentic shape.[24] The reader is referred to comprehensive reviews of transesophageal examination designed for perioperative hemodynamic assessment[25,26] and a review comparing common transthoracic and transesophageal views.[27] The midesophageal four-chamber, midesophageal RV inflow-outflow, and transgastric short-axis views on TEE and the parasternal long- and short-axis, apical four-chamber, and subcostal four-chamber views on TTE are commonly used to evaluate the right ventricle.

Echocardiographic Parameters for RV Assessment

Common echocardiographic parameters used to detect RV dysfunction are described in this section and are listed in Table 2.

RV:LV Area Ratio

An acute elevation in RV afterload is manifested with RV dilatation (Videos 10, 11). Normal values for RV dimensional quantification on two-dimensional echocardiography have not been established in patients receiving mechanical ventilation.[24] However, comparison with the LV size provides a quick estimate of RV size. The RV:LV area ratio is measured in end diastole by tracing the areas of the two chambers in the apical four-chamber view on TTE or the midesophageal four-chamber view on TEE[28] (Figs 1, 2, Videos 12, 13). A ratio of >0.6 suggests moderate RV dilation, whereas a ratio >1.0 indicates severe RV dilation. Because the measurement of RV size is greatly altered by small rotations of the imaging plane, care must be taken to avoid foreshortening of the RV chamber. An RV-focused view should be obtained with the transducer plane through the center of the LV cavity and with RV size maximized, a maneuver that can be more reliably performed with TEE. As a word of caution, RV linear dimensions correlate poorly with RV volume under conditions of increased preload and increased afterload.[29,30]

LV Eccentricity Index

Normally, the interventricular septum is curved toward the right ventricle. The eccentricity index, a ratio of two perpendicular diameters of the LV cavity in the short-axis view (the anteroposterior and septolateral dimensions), is a quantitative measure of septal bowing and increases with RV volume and pressure overload[31,32] (Fig 3). Normally, the LV eccentricity index is 1 both at end diastole and at end systole (Videos 4, 7). An index >1 signifies RV pressure or volume overload and denotes a distorted septum with a D-shaped left ventricle (Videos 14, 15). Aberrant ventricular conduction can make analysis of septal motion challenging.

RV Fractional Area Change

Right ventricular fractional area change (RVFAC) is the ratio of the change in RV area to the RV end-diastolic area (Fig 4). It is easier to measure; correlates well with the RV ejection fraction measured by MRI[33]; and is a predictor of adverse outcomes in patients after pulmonary embolism,[34] cardiac surgery,[14] and myocardial infarction.[35] The trabeculations and papillary muscles should be included in the RV cavity when tracing the endocardial border. Normal values for RVFAC are $\geq 32\%$, and RVFAC values $<17\%$ represent severely decreased RV systolic function.[24]

Tricuspid Annular Plane Systolic Excursion

In the apical four-chamber view, the right ventricle contracts largely along its length. M-mode imaging through the lateral tricuspid valve plane captures this longitudinal motion, a simple, reproducible measure with low interobserver variability (Fig 5, Videos 5, 8, 13). It has shown good correlation with two-dimensional RVFAC and radionuclide-derived RV ejection fraction.[36] Values of tricuspid annular plane systolic excursion (TAPSE) <16 mm indicate RV dysfunction and are associated with increased mortality in patients with chronic right-sided heart failure.[37,38] TAPSE values are reduced in freshly transplanted hearts and correlate with ischemic time of the transplanted organ.[39]

Peak Velocity of Systolic Excursion

Lateral tricuspid annular motion is also judged by its peak velocity using tissue Doppler imaging in the apical four-chamber view. The sample volume is placed at the tricuspid annulus or the middle of the basal segment of the RV free wall, and the peak systolic velocity (S′) is

TABLE 2] Echocardiographic Parameters of RV Function

Parameter	View		Abnormal Value
	TEE	TTE	
RV:LV area ratio	ME four chamber	Apical four chamber	>0.6
LV eccentricity index	TG midpapillary short axis	Parasternal midpapillary short axis	>1
RVFAC	ME four chamber	Apical four chamber	$<35\%$
TAPSE	Deep TG RV	Apical four chamber	<1.6 cm
Peak velocity of systolic excursion at the annulus	Deep TG RV	Apical four chamber	<10 cm/s
Pulmonary artery flow acceleration time	Ascending aortic short-axis	Parasternal RV outflow	<100 ms

ME = midesophageal; RVFAC = right ventricular fractional area change; TAPSE = tricuspid annular plane systolic excursion; TEE = transesophageal echocardiography; TG = transgastric; TTE = transthoracic echocardiography. See Table 1 legend for expansion of other abbreviations.

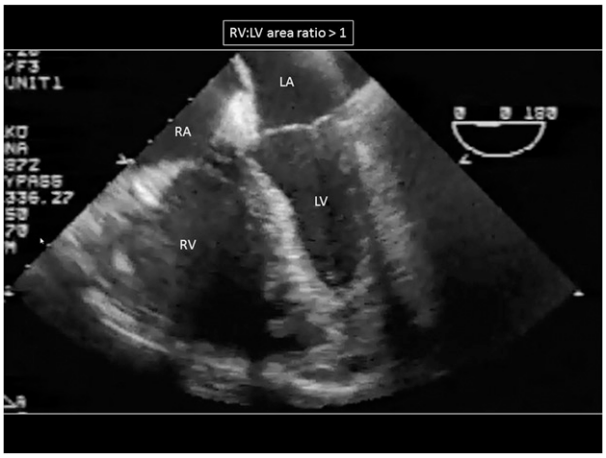

Figure 1 – *Dilated RV in midesophageal four-chamber view on transesophageal echocardiography at end diastole. LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle.*

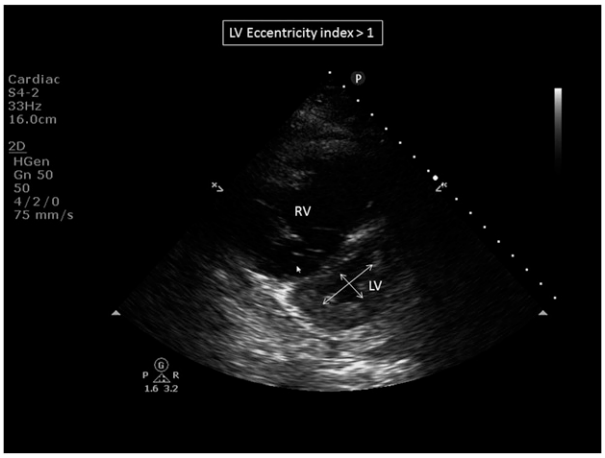

Figure 3 – *Parasternal short-axis view on transthoracic echocardiography showing the anteroposterior and septolateral dimensions of the LV with LV eccentricity index > 1 at end systole. See Figure 1 and 2 legends for expansion of abbreviations.*

determined (Fig 6). S′ is easy to measure and reproducible, with values < 10 cm/s indicating RV systolic dysfunction.[24] Interpreting both TAPSE and S′ to reflect overall RV systolic function assumes that the basal segment is representative of global function.

Systolic RV Pressure

When tricuspid regurgitation is present, the RV-right atrial pressure gradient can be estimated by measuring the peak regurgitant jet velocity using continuous-wave Doppler echocardiography and the modified Bernoulli equation[40] (Fig 7, Videos 16-19). Adding this pressure to the right atrial pressure (RAP) yields the peak systolic RV pressure, which unless pulmonic stenosis is present, equals the systolic pulmonary artery pressure. To measure regurgitant velocity accurately, the ultrasound beam should be parallel to the jet (within 15°). Doppler evaluation of tricuspid regurgitation is best achieved in the modified bicaval view and the deep transgastric view. Aligning the ultrasound beam can usually be more effectively done on TTE in the modified parasternal long-axis, parasternal RV inflow-outflow, apical four-chamber, and subcostal four-chamber views.

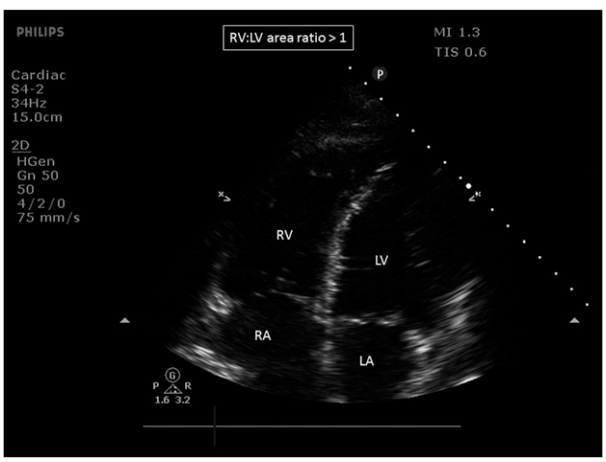

Figure 2 – *Dilated RV in apical four-chamber view on transthoracic echocardiography at end diastole showing a right ventricular area larger than the left ventricular area. 2D = two dimensional. See Figure 1 legend for expansion of other abbreviations.*

Right Atrial Pressure

RAP can be estimated by measuring the inferior vena cava size and respiratory collapse on the subcostal view[41] (Fig 8, Video 20). We caution that even direct, accurate measurement of RAP fails to predict fluid responsiveness[42] and should not be used for that purpose.

McConnell's Sign

Acute RV pressure overload often is accompanied by a specific pattern of abnormal regional wall motion with akinesia of the mid-free wall but normal motion at the apex[43] (Video 21). This pattern, called McConnell's sign, has been shown to have prognostic implications in patients with pulmonary embolism but is not specific for this diagnosis in the setting of acute RV dysfunction.[44]

Pulsed Doppler Echocardiographic Evaluation of Pulmonary Artery Flow

Respiratory variation in pulmonary artery flow and a biphasic flow pattern, reflecting obstruction to RV ejection, can be visualized in the midesophageal ascending aortic short-axis view on TEE and occasionally in the

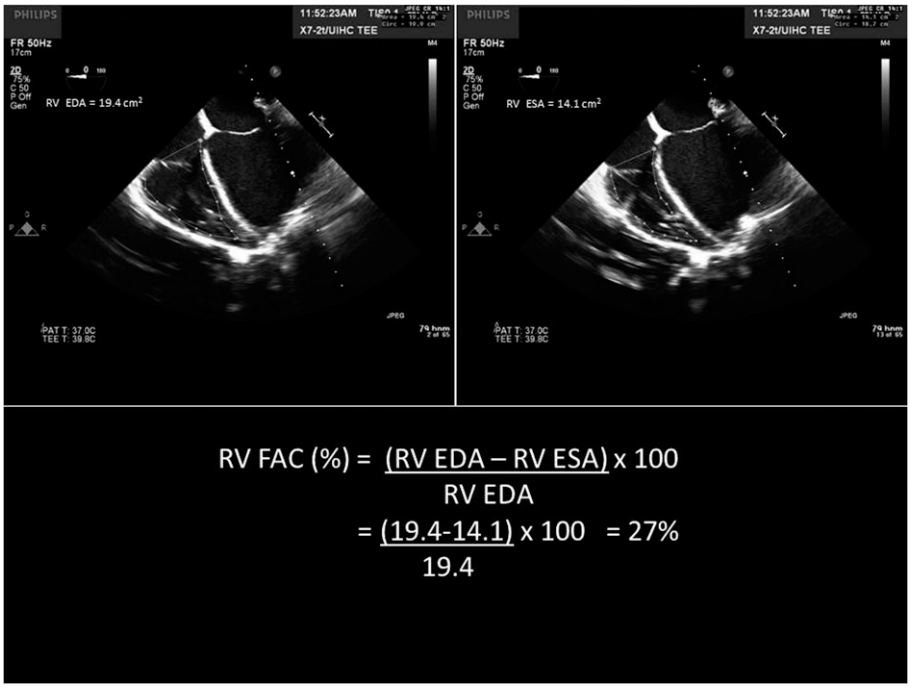

Figure 4 – *Decreased RV FAC on midesophageal four-chamber view on TEE. The calculated value of 27% is below the lower limit of normal (35%). FR = frame rate; RV EDA = right ventricular end-diastolic area, RV ESA = right ventricular end-systolic area; RV FAC = right ventricular fractional area change; TEE = transesophageal echocardiography; UIHC = University of Iowa Health Care. See Figure 2 legend for expansion of other abbreviation.*

parasternal RV outflow view on TTE using pulsed Doppler echocardiography[45] (Fig 9, Video 7). Shortening of the acceleration time (< 100 milliseconds) in pulmonary arterial flow suggests pulmonary hypertension[45] (Fig 10). Doppler echocardiographic evaluation of the pulmonary artery can also be used to estimate changes in stroke volume.

Clinical Utility of Real-Time Echocardiography of the Right Ventricle

The failing right ventricle is exquisitely sensitive to changes in loading conditions, such as those produced by the ventilator, fluid therapy, and vasoactive infusions. Even body positioning during ventilation can have meaningful effects, sometimes acting more powerfully than pharmacotherapy. Blind changes in ventilator settings and other therapies risk further crippling the circulation, suggesting that real-time echocardiographic guidance would lead to more rational therapy. Elevated RAP may reveal a patent foramen ovale, risking shunting of venous blood to the left side and exacerbating hypoxemia, and this can be detected echocardiographically. We next address

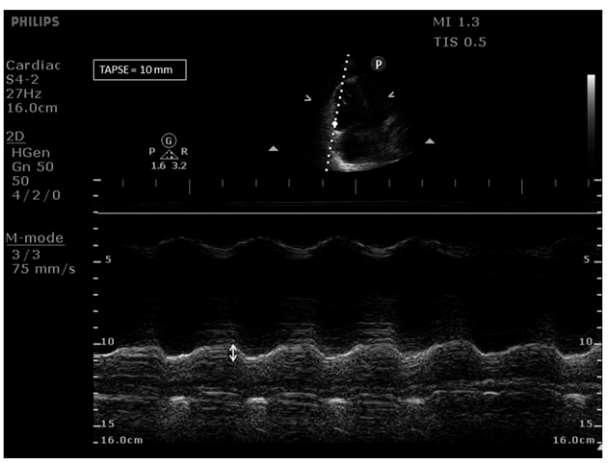

Figure 5 – *Decreased TAPSE on apical four-chamber view on transthoracic echocardiography. The measured value of 10 mm is well below normal (> 16 mm). TAPSE = tricuspid annular plane systolic excursion. See Figure 2 legend for expansion of other abbreviations.*

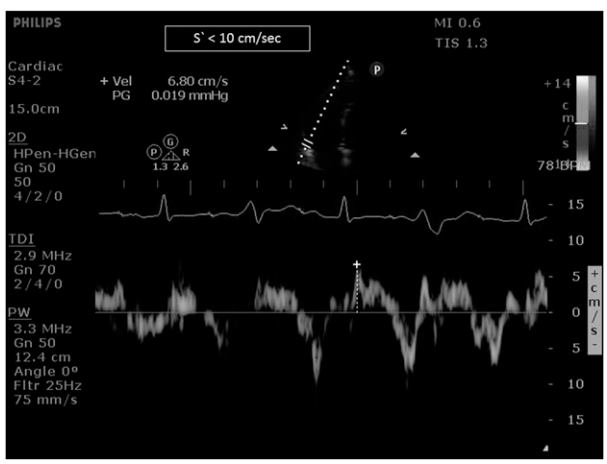

Figure 6 – *Decreased S′ on apical four-chamber view on transthoracic echocardiography. The measured value of 7 cm/s is below the cutoff for abnormal (10 cm/s). PG = pressure gradient; PW = pulse wave; S′ = peak systolic velocity; TDI = tissue Doppler imaging; Vel = velocity. See Figure 2 legend for expansion of other abbreviations.*

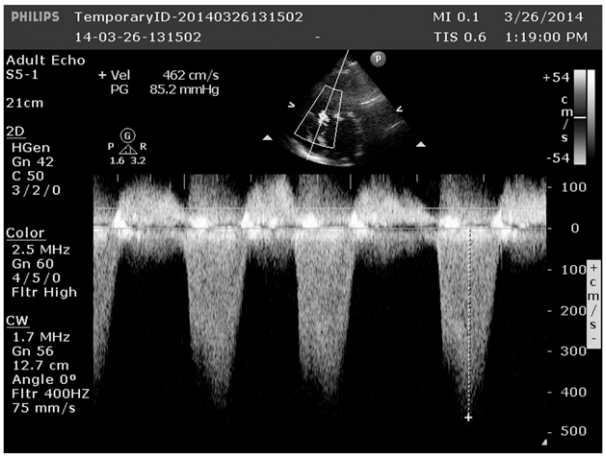

Figure 7 – *Calculation of peak gradient of tricuspid regurgitation on apical four-chamber view on transthoracic echocardiography for the estimation of systolic pulmonary artery pressure. The measured velocity of 462 cm/s corresponds to a pressure gradient of 85 mm Hg. CW = continuous wave. See Figure 2 and 6 legends for expansion of other abbreviations.*

the supportive care of acute RV dysfunction and how echocardiography can guide ventilation, fluid management, and vasoactive therapy. Specific therapy for the underlying cause of acute RV dysfunction, such as thrombolysis, coronary revascularization, management of LVAD pump speed (Videos 22, 23), or treatment of ARDS, is beyond the scope of this article. Furthermore, we do not discuss the use of mechanical therapies such as RV assist devices, extracorporeal membrane oxygenation, atrial septostomy, or intraaortic balloon counterpulsation, although these occasionally may play a role.[46]

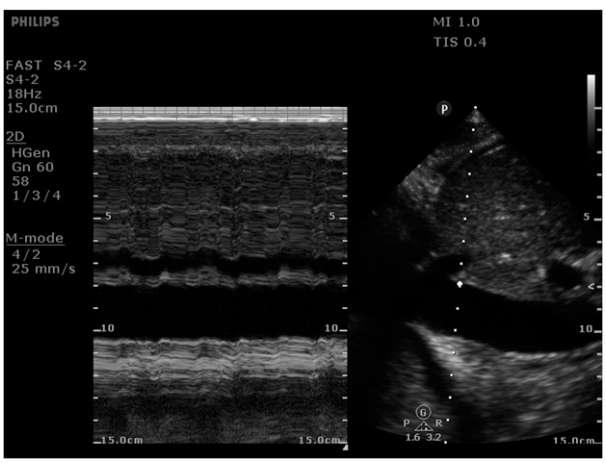

Figure 8 – *M-mode of the inferior vena cava (IVC) in subcostal view on transthoracic echocardiography, demonstrating a dilated IVC with minimal respiratory variation. The right side shows the 2D image where the cursor has been placed just below the entry of the hepatic vein into the IVC. The left side shows the M-mode image along this cursor. See Figure 2 legend for expansion of other abbreviation.*

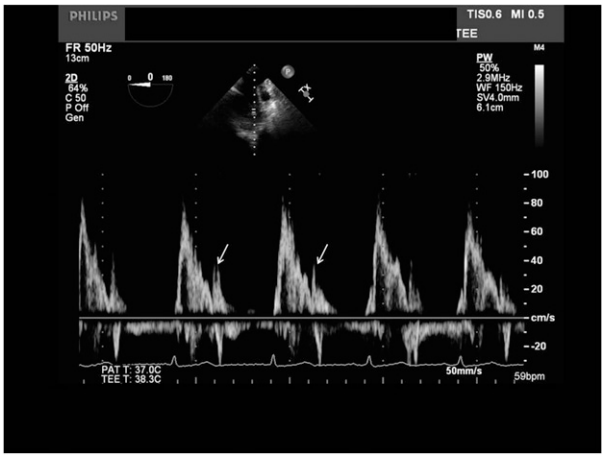

Figure 9 – *Biphasic pulmonary artery flow (arrows) on pulsed Doppler echocardiographic evaluation in the ascending aortic short-axis view on TEE. bpm = beats per min. See Figure 2 and 4 legends for expansion of other abbreviations.*

Mechanical Ventilation

In the patient who is passively ventilated, the dominant RV effects are to reduce preload (mediated through the rise in pleural pressure) and raise RV afterload. The impact on RV preload is proportional to the rise in juxtacardiac pressure, depending on tidal volume, positive end-expiratory pressure (PEEP), chest wall compliance, and lung recruitability. Tidal inflation and PEEP also affect PVR in a complex manner, depending on their magnitude and the degree of lung recruitment. At moderate levels, especially when PEEP opens alveoli, PVR falls.[47] At high levels, PEEP (or excessive tidal volume) compresses the pulmonary vascular bed, reducing blood flow.[48] The importance of this mechanism has been revealed through focused

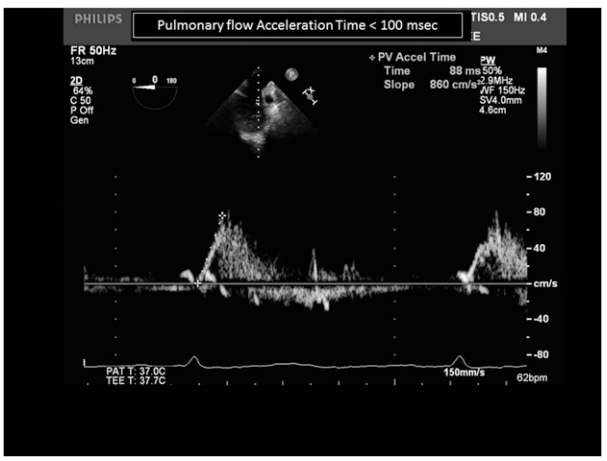

Figure 10 – *Pulmonary artery flow acceleration time < 100 ms on pulsed Doppler echocardiographic evaluation in the ascending aortic short-axis view on TEE. PV = pulmonary valve. See Figure 2, 4, and 9 legends for expansion of other abbreviations.*

echocardiographic study. When 21 patients with ARDS were studied at a PEEP of 5 cm H_2O and again after PEEP was raised until the plateau airway pressure was 30 mm Hg (resulting in a final PEEP that averaged 13 cm H_2O), cardiac output fell, which was associated with a rise in RV end-diastolic area and PVR.[49] In these patients, therefore, PEEP depressed cardiac output by raising RV afterload and impeding ejection, not by reducing preload. Similar findings are seen during tidal ventilation.[50] A plateau pressure of 27 mm Hg has been proposed as a cutoff for preventing RV dysfunction in patients who are ventilated.[51]

Ventilating patients with severe ARDS ($Pao_2/Fio_2 < 150$) in the prone position has been shown to reduce mortality,[52] but the mechanism of this effect is uncertain and may be based on higher Pao_2, enhanced lung protection,[53] or unloading of the right ventricle. A detailed hemodynamic assessment showed that prone positioning reduced PVR[54]; reduced RV:LV end-diastolic area ratio; reduced LV eccentricity index; and, in preload-responsive patients, raised cardiac output.[55] On the other hand, high-frequency ventilation impairs RV function[56] and may worsen survival.[57]

The principles of mechanical ventilation for patients with acute RV failure are as follows: (1) limit tidal volume and PEEP, (2) avoid hypercapnia, and (3) prevent hypoxic pulmonary vasoconstriction. These principles are potentially conflicting because limiting tidal volume may produce hypercapnia. Similarly, lower PEEP may leave alveoli collapsed, reducing their blood flow. Nevertheless, ventilator adjustment has the potential to significantly improve the circulation. For example, in patients with severe ARDS ($Pao_2/Fio_2 < 150$ but hemodynamically stable; not restricted to those with RV dysfunction), raising PEEP from 5 to 10 cm H_2O (at constant plateau airway pressure) depressed the cardiac index by 28%.[32] TEE showed that RV end-diastolic volume and the end-systolic eccentricity index both rose, corroborating the hypothesis that PEEP reduces perfusion by impeding RV systolic function rather than by limiting right-sided heart filling. This effect was more pronounced in those with the least recruitable lungs. A multifactorial analysis pointed to hypercapnic acidosis, rather than the PEEP level per se, as the basis for RV impairment. Others have also associated hypercapnia ($Paco_2 > 60$ mm Hg) with acute RV dysfunction.[8] Raising the respiratory rate may not help because hypercapnia tends to resist increases in respiratory rate and may produce dynamic hyperinflation, further impeding pulmonary perfusion.[32,58]

Echocardiography-Guided Management: The complexity of competing influences of PEEP, tidal volume, $Paco_2$, and intravascular volume creates a powerful argument for intensivists to master echocardiography, including advanced techniques, to identify and manage these patients optimally. Ventilator settings and prone positioning are likely to be most valuable in patients with ARDS, with less benefit seen in those with pulmonary embolism or postcardiac surgical shock. For patients with known acute RV dysfunction and for hemodynamically unstable patients with ARDS, we suggest that echocardiography be used to guide significant changes in mode, tidal volume, or PEEP. The eccentricity index or RV:LV end-diastolic area ratio could be measured before and after adjustments, using changes to guide further care. At centers that perform TEE frequently, monitoring the pulmonary artery flow waveform can be used to evaluate the impact of intrathoracic pressure on RV outflow.[45]

Fluid Management in Shock

When the circulation is impaired by acute RV dysfunction, increasing blood volume generally will not augment perfusion. Fluid administration is likely to worsen hypotension through decreased RV systolic performance, increased tricuspid annular diameter (worsening tricuspid regurgitation), and decreased LV filling through ventricular interdependence.[59,60] This is true even in acute RV infarction, where vasoactive drug infusion raises cardiac output but fluids do not.[61-63] Excessive volume loading and septal deviation can diminish the contribution of septal contraction to RV function.[64] In canine models of RV failure, volume administration results in RV dysfunction, whereas norepinephrine administration improves RV performance.[65,66] On the other hand, reducing intravascular volume (through diuresis or ultrafiltration) might improve circulatory function.[67] In a retrospective analysis of 70 normotensive patients with acute pulmonary embolism, diuresis improved creatinine level, oxygenation, and systolic BP compared with fluid expansion.[68] In volume-overloaded patients unable to tolerate diuretic-guided fluid loss, slower volume removal through continuous renal replacement therapy might be necessary.

Increasingly, intensivists use dynamic fluid responsiveness predictors to guide fluid therapy. These predictors, such as variations in pulse pressure, stroke volume, arterial flow velocity, and LV outflow tract velocity-time integral, are based on the preload-related effects of

ventilation (which dominate when RV function is normal). When beat-to-beat stroke volume also depends on changing RV afterload, as is typical in RV failure, these dynamic indices may falsely signal preload dependency.[69] In this situation, further fluid therapy will not boost perfusion and through ventricular interdependence, could produce an ever-worsening spiral of shock. The principles of fluid management are (1) detect hypovolemia (although this is unusual) but recognize that many dynamic predictors are invalid in acute RV dysfunction, (2) avoid excessive fluid therapy because this may compromise overall perfusion through ventricular interdependence, and (3) confirm objectively the impact of treatments that change intravascular volume.

Echocardiography-Guided Management: Before infusing fluid empirically in the patient with known or suspected acute RV dysfunction, echocardiography should be considered for measurement of baseline parameters (RV:LV area ratio, eccentricity index, magnitude of tricuspid regurgitation). It may be possible to predict the effect of fluid infusion by a passive leg-raising maneuver or of diuresis by reverse Trendelenburg positioning, with objective measurement of RV echocardiographic parameters and stroke volume, although this has not been tested in patients with acute RV dysfunction. Following therapies directed at intravascular volume, reimaging the heart could measure the effect of intervention.

Vasoactive Drug Therapy

RV systolic function can be enhanced with catecholamines (norepinephrine, epinephrine, dobutamine), phosphodiesterase 3 inhibitors (milrinone), and calcium sensitizers (levosimendan), whereas pulmonary vasodilators (inhaled nitric oxide, inhaled prostacyclin, inhaled milrinone) may reduce RV afterload. Many of these drugs have complex effects on RV contractility, LV systolic function (which may crucially sustain RV function in acute RV dysfunction), PVR, ventricular interdependence, and adequacy of coronary blood flow to the right ventricle. If these drugs cause systemic hypotension, coronary perfusion pressure may fall, threatening subendocardial ischemia.

Vasoactive drugs have differing effects on pulmonary vascular tone. For example, vasopressin has no constrictive properties, whereas norepinephrine does. In patients undergoing cardiopulmonary bypass, both drugs raise systemic vascular resistance, but only vasopressin decreases the ratio of PVR to systemic vascular resistance.[70] However, a norepinephrine-induced increase in PVR is likely to occur only at high doses.[71]

The inhaled pulmonary vasodilators prostacyclin and nitric oxide reduce PVR and mean pulmonary artery pressure in patients with acute lung injury while avoiding systemic hypotension.[72,73] These agents have nearly identical effects to reduce shunt, raise Pao_2, and lower PVR,[72-74] although these effects do not confer a survival benefit.[75,76] Nevertheless, withdrawal of inhaled nitric oxide has been associated with hemodynamic collapse due to acute RV loading.[77] As a newer agent, levosimendan has garnered significant research interest for managing ventricular failure of various etiologies,[78] although it is not yet available in the United States. Like dobutamine and milrinone, levosimendan can cause systemic hypotension, necessitating close hemodynamic monitoring, especially in patients with acute RV dysfunction. The principles of vasoactive drug use in acute RV dysfunction are as follows: (1) emphasize the use of inotropes; (2) avoid drugs that tend to provoke pulmonary vasoconstriction; (3) maintain systemic perfusion pressure, which is vitally important to RV oxygen supply; (4) titrate drugs to objective measures of the adequacy of perfusion, such as venous oximetry, stroke volume, LV outflow tract velocity-time integral, and cardiac index, rather than systemic BP; (5) measure the impact of drugs on ventricular interdependence through the RV:LV area ratio or eccentricity index; and (6) repeat imaging in concert with stepwise changes in therapy to reverse shock rapidly.

Echocardiography-Guided Management: Because vasoactive drugs have variable effects on systolic function, heart rate, venous capacitance, and both pulmonary and systemic arterial tone, the impact on the failing right ventricle can be complex. We advocate using these drugs guided by TAPSE, RV:LV area ratio, and eccentricity index. Any deterioration should prompt consideration of alternate drug strategies. Of note, dynamic RV outflow tract obstruction might be present in postcardiac surgery patients, necessitating a reduction in inotropic dose.[79]

Conclusions

Acute RV dysfunction is both common and potentially lethal in critically ill patients. Because echocardiography is the de facto standard for recognizing its presence and judging severity, intensive care physicians must master this tool and be capable of imaging the right ventricle. The intensivist is well positioned to use real-time

echocardiographic images, combined with knowledge of RV physiology, to guide management. Only in this way can the complex effects of mechanical ventilation, fluid therapy, vasoactive drug infusions, and other therapies be integrated to resuscitate the patient optimally.

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Additional information: The Videos can be found in the Multimedia section of the online article.

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