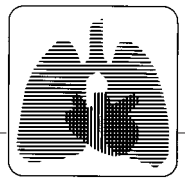

reviews

The Acutely Decompensated Right Ventricle*

Pathways for Diagnosis and Management

Gregory Piazza, MD; and Samuel Z. Goldhaber, MD, FCCP

Decompensated right ventricular (RV) failure is becoming increasingly common as the prevalence of predisposing conditions grows. Advances in diagnosis and management have granted insights into the following pathophysiologic mechanisms of RV dysfunction: impaired RV contractility, RV pressure overload, and RV volume overload. Emerging imaging modalities, such as cardiac MRI, and new therapeutic agents, such as pulmonary selective vasodilators, have expanded our options for evaluation and management, respectively. An improved understanding of pathophysiology and technologic progress provides us with new pathways in the diagnosis and hemodynamic support of these often critically ill patients. ***(CHEST 2005; 128:1836–1852)***

Key words: cor pulmonale; diagnosis; management; pathophysiology; prognosis; right ventricular dysfunction; right ventricular failure

Abbreviations: BNP = brain-type natriuretic peptide; LV = left ventricle, ventricular; PE = pulmonary embolism; RV = right ventricle, ventricular

Learning Objectives: 1. Understand the pathophysiological mechanisms of right ventricular dysfunction. 2. Focus upon learning key patient signs and symptoms from conditions associated with right ventricular failure, specifically, impaired right ventricular contractility, right ventricular pressure overload, and right ventricular volume overload. 3. Identify six diagnostic modalities used to determine the best treatment options for patients with right ventricular failure. 4. Identify and understand appropriate and evolving teatments for right ventricular failure, including pharmaceutical, mechanical, and surgical interventions.

As the prevalence of predisposing conditions, such as left ventricular (LV) dysfunction, pulmonary embolism (PE), pulmonary hypertension, and adult congenital heart disease, grows, the number of patients presenting with decompensated right ventricular (RV) failure is increasing. Decompensated RV failure refers to the clinical syndrome of RV dysfunction, which may be suggested by a variety of diagnostic modalities. RV failure may occur in the acute and acute-on-chronic settings and may result from cardiac or pulmonary disease. The specific case of RV failure from pulmonary or pulmonary vascular origins is referred to as *cor pulmonale* and may develop acutely, as in PE, or complicate an exacerbation of chronic respiratory disease, as with COPD. Cardiac biomarkers, such as troponins and brain-type natriuretic peptides (BNPs), and an increasing experience with cardiac MRI help to elucidate the pathophysiology of RV failure. The use of inotropic agents and pulmonary-selective vasodilators, along with novel surgical techniques, have expanded our therapeutic options. In this review, we provide a scheme for the evaluation and management of patients with acute and acute-on-chronic RV failure.

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The authors have indicated to the ACCP that no significant relationships exist with any company/organization whose products or services may be discussed in this article submission.

Manuscript received May 11, 2004; revision accepted February 15, 2005.

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Pathophysiology

Pathophysiologic changes in the acutely decompensated RV vary according to the underlying cause. Often, patients experience RV failure secondary to a combination of decreased RV contractility, RV pressure overload, and RV volume overload.

In conditions that depress RV function, such as ischemia and infarction, the ventricle is unable to handle even "normal" loading conditions. RV ischemia leads to chamber dilation and an impaired diastolic function with a concomitant rise in RV end-diastolic pressure. An elevation in RV diastolic pressure causes a shift of the interventricular septum toward an already underfilled LV. RV dilation in the setting of limited pericardial compliance leads to increased intrapericardial pressures and an additional constraint on both the RV and LV filling.[7] These changes in RV mechanics lead to depressed right-sided output, decreased LV preload, and, subsequently, a reduced overall cardiac output.[8]

In contrast to the muscular LV, the thin-walled RV is poorly suited to compensate for acute increases in the afterload, such as in PE (Fig 2).[9] In PE, the extent of pulmonary arterial obstruction appears to be a crucial factor in predicting the degree of RV dysfunction.[10] The sudden increase in the RV afterload increases wall tension and leads to chamber dilatation and impaired diastolic and systolic function.[11] The interventricular septum paradoxically shifts toward the LV and leads to impaired filling of this chamber under the constraint of a noncompliant pericardium.[12] Acute tricuspid regurgitation resulting from RV dilatation and systolic dysfunction also leads to a diminished right-sided cardiac output and a reduction in the LV preload. Increases in RV wall tension along with decreases in systemic cardiac output and perfusion pressures may alter the equilibrium between the myocardial oxygen supply and demand, leading to ischemia and possibly infarction. In ARDS, circulating vasoconstrictors, increased sympathetic tone, and microvascular obstruction increase the RV afterload.[13,14]

In addition to the pressure effects, conditions including adult congenital heart disease and acquired valvular heart disease may place substantial volume loads on the RV. Compared with pressure overload, RV volume overload is more detrimental and more profoundly affects the LV systolic function. One study[15] evaluated 10 patients with severe tricuspid regurgitation and 10 patients with severe pulmonary arterial hypertension, respectively, as models of isolated RV volume overload and RV pressure overload. In the RV pressure overload

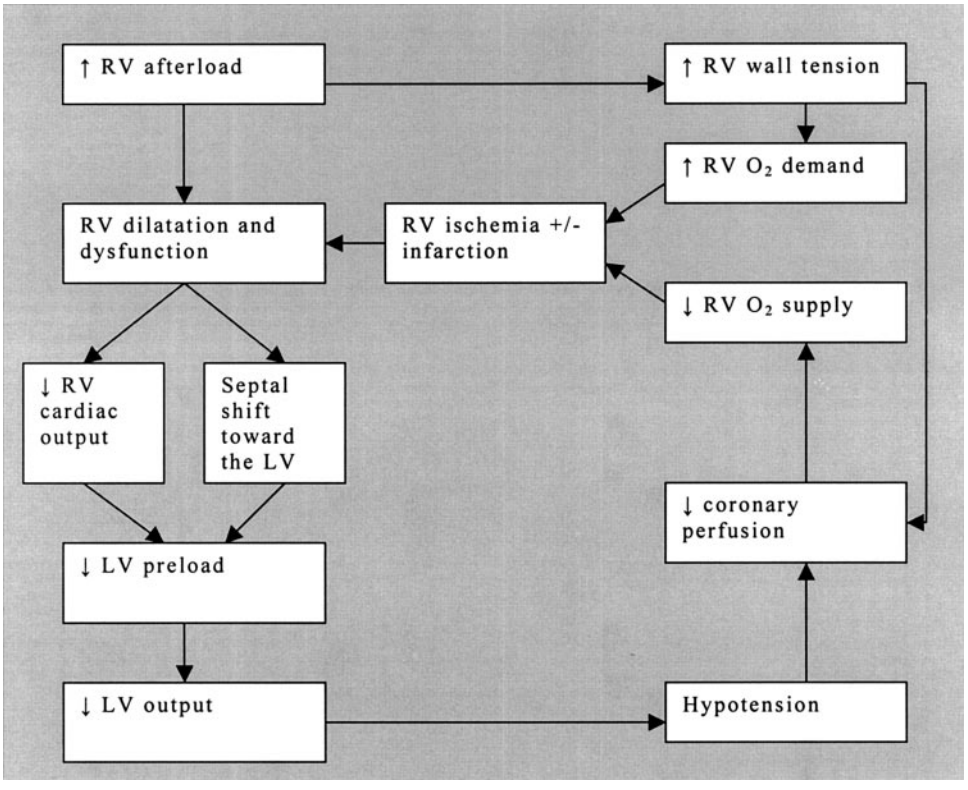

Figure 2. Pathophysiologic changes seen in RV failure resulting from increased afterload. Adapted with permission from Lualdi and Goldhaber.[10]

group, echocardiography revealed significant underfilling of the LV with relatively preserved LV ejection fraction. In the RV volume overload group, the LV exhibited similar end-diastolic volumes, compared with healthy control subjects, but marked depression in the LV ejection fraction. Echocardiography additionally revealed that in the RV pressure overload, the interventricular septum shifted toward the left at the end of systole and augmented systolic shortening of the septal-to-free wall dimension. In RV volume overload states, the leftward shift of the septum was seen at end-diastole with paradoxical movement away from the LV-free wall during systole, resulting in a depression of systolic shortening of the septal-to-free wall dimension. Based on these findings, the interventricular septum appears to play differing roles in purely RV pressure overload compared with RV volume overload.

Clinical Presentation

The symptoms of RV failure are nonspecific and often vary according to the precipitating condition (Table 1). Patients with conditions leading to increased RV afterload may report dyspnea, lightheadedness, and syncope.[10] In both RV infarction and PE, chest discomfort may be an important presenting symptom. Patients with acute-on-chronic RV failure may report right-upper-quadrant discomfort from hepatic congestion, as well as lower-extremity swelling. Of note, many of the acute symptoms and signs of RV compromise may be observed in chronic RV failure and vice versa.

On physical examination, the signs of RV failure may include systemic hypotension, tachycardia, tachypnea, cyanosis, elevated jugular venous pressure, a parasternal heave, an RV third-heart sound, and tricuspid regurgitation (Table 2).[10,16] In addition, signs of elevated pulmonary arterial pressures may be elicited, including an accentuated sound of pulmonic valve closure. Hepatic enlargement and lower-extremity edema are observed in acute-on-chronic RV failure. Other findings may also be present, such as coexisting LV failure or valvular lesions.

Table 1—*Symptoms of RV Failure*

Acute
Dyspnea
Lightheadedness
Syncope
Chest discomfort
Chronic
Acute symptoms
Right-upper-quadrant abdominal pain
Lower-extremity swelling

Table 2—*Signs of RV Failure*

Acute
Hypotension
Tachycardia
Tachypnea
Cyanosis
Elevated jugular venous pressure
Parasternal heave
RV third-heart sound
Tricuspid regurgitation
Accentuated sound of pulmonic valve closure
Chronic
Acute signs
Hepatic enlargement and ascites
Lower-extremity edema

The symptoms and signs of acute RV failure may be superimposed on those of chronic cor pulmonale in patients with exacerbations of chronic pulmonary disease, such as COPD. The findings of RV failure in such a setting may present both a diagnostic and a therapeutic challenge to the clinician, because the presentation of a patient may resemble decompensated congestive heart failure or an exacerbation of chronic lung disease, and management of these two disorders differs considerably. In addition, the treatment of the underlying pulmonary disorder, such as with mechanical ventilation, may additionally exacerbate the RV failure.

Diagnostic Testing

The current diagnostic options include ECG, chest radiography, cardiac biomarkers, echocardiography, cardiac MRI, and right-heart catheterization.

ECG

Depending on the cause of RV failure, patients may have ECG evidence of right-heart dysfunction with or without evidence of RV infarction (Table 3).

Table 3—*ECG Changes in RV Failure*

Sinus tachycardia
T-wave inversion in leads III and aVF or in leads V1-V4
Incomplete or complete right-bundle-branch block
QRS axis $> 90°$ or indeterminate axis
Concurrent deep S wave in lead I, Q-wave and T-wave inversion in lead III
Qr pattern in lead V1
S waves in lead I and aVL > 1.5 mm
Q waves in leads III and aVF but not in lead II
Transition zone shift to V5
Low limb lead voltage
Atrial fibrillation

A Qr pattern has been described[17] in patients with PE and RV dysfunction. ST-segment elevations and loss of the R-wave in V1 and right-sided leads, V3R and V4R, suggest RV infarction (Fig 3).[7,18] Sinus tachycardia and atrial fibrillation may also be appreciated.

Chest Radiography

The RV is poorly visualized on a conventional chest radiograph because of its anatomic location. However, changes in the position of the heart, cardiac silhouette, and adjacent structures may suggest RV dilatation (Table 4).[19]

Table 4—*Radiographic Findings Associated With RV Failure*

Dilatation of the proximal pulmonary arteries with abrupt tapering of the distal branches
Filling of the retrosternal space secondary to RV enlargement
Inferior vena cava and azygous vein dilatation
Pleural or pericardial effusions
Increased curvature of the right-heart border secondary to right atrial dilatation seen on anterior-posterior or posterior-anterior view

Cardiac Biomarkers

Cardiac biomarkers, such as cardiac troponin and BNP, were initially developed for the evaluation of patients with myocardial ischemia and congestive heart failure, respectively. Whereas cardiac troponins have well-established diagnostic and prognostic roles in the evaluation of suspected ischemic heart disease, BNP is useful in the evaluation of acute dyspnea, as well as in risk assessment for mortality and cardiovascular events.[20,21] The elevated levels of serum cardiac troponin, BNP, and probrain-type natriuretic peptide have also been documented in RV dysfunction, particularly in the setting of PE.[22–31] In other conditions leading to pulmonary hypertension, the increasing levels of BNP also correlate with the degree of RV dysfunction and adverse outcomes.[32–34] In RV failure, cardiac troponins are suspected to be elevated secondary to RV ischemia or microinfarction resulting from an increased wall tension, metabolic demand, and reduced coronary perfusion with or without atherosclerosis (Fig 4).[22,35,36] The myocardium synthesizes and secretes BNP as a result of increased RV shear stress.[22,37]

Cardiac troponins and BNP have been extensively studied in the evaluation of patients with RV dysfunction secondary to acute PE (Table 5). Both cardiac troponins and BNP accurately identify low-risk PE patients with negative predictive values for in-hospital death ranging from 97 to 100%. However, cardiac troponins and BNP have demonstrated

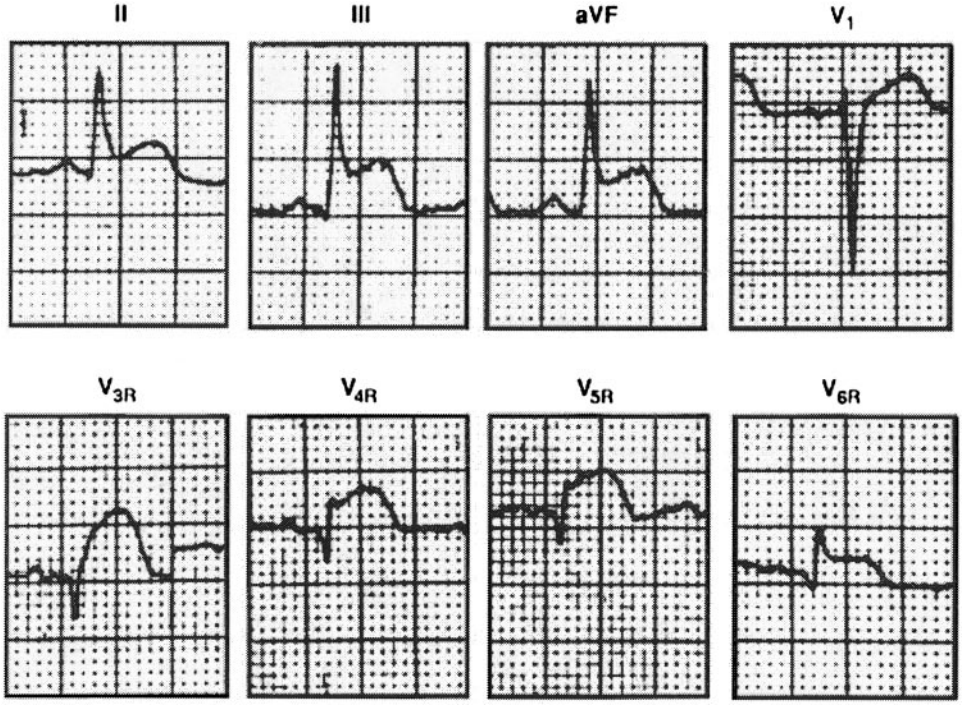

FIGURE 3. ECG findings of RV infarction complicating an inferior myocardial infarction. Loss of the R wave along with ST-segment elevation in V1 and right-sided leads suggest RV infarction. Reprinted with permission of Kinch and Ryan.[18]

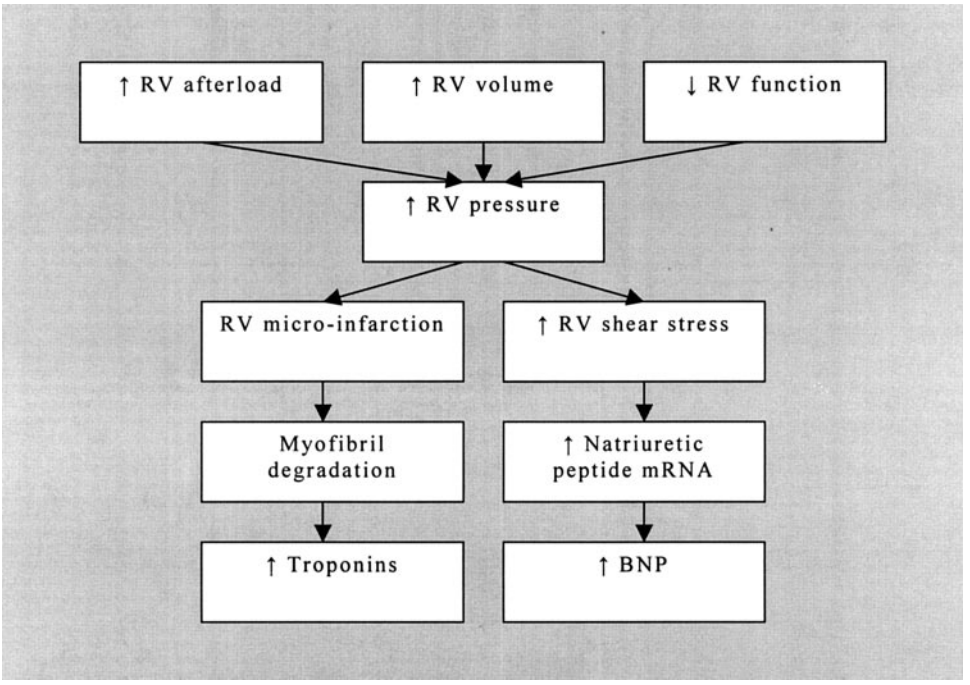

FIGURE 4. Proposed mechanism of cardiac biomarker elevation in RV failure. Adapted with permission from Kucher and Goldhaber.[22]

low positive predictive values, a wide range of sensitivities, and low specificity for adverse events, including in-hospital deaths in patients with acute PE and RV failure. Although the negative predictive values for an adverse prognosis are consistently high in the literature, several studies[29,30] have demonstrated relatively wide confidence intervals, with a range from 80 to 100%. In general, the cutoff values for troponins in PE prognostication are identical to those for the diagnosis of myocardial ischemia. The cutoff values for BNP in PE prognostication are usually lower than those used for congestive heart failure. In a patient with acute PE, elevated cardiac biomarkers may suggest the presence of RV failure. A confirmation with transthoracic echocardiography may help identify this high-risk population.[22]

The BNP levels are elevated in other conditions of RV volume and pressure overload.[32–34] In a study[32] of 18 patients with RV volume overload because of atrial septal defect and 26 patients with RV pressure

Table 5—*Accuracy of Cardiac Biomarkers for the Prediction of In-Hospital Death in Acute PE

Study	Patients, No.	Biomarker	Assay	Cutoff Level	Positive Test Result	Sens	Spec	NPV	PPV
Konstantinides et al[23]	106	cTnI	Centaur (Bayer‡)	0.07 ng/mL	41	86	62	98	14
Konstantinides et al[23]	106	cTnT	Elecsys (Roche§)	0.04 ng/mL	37	71	66	97	12
Giannitsis et al[24]	56	cTnT	TropT (Roche)	0.10 ng/mL	32	88	78	97	44
Janata et al[25]	106	cTnT	Elecsys (Roche)	0.09 ng/mL	11	80	92	99	34
Pruszczyk et al[27]	64	cTnT	Elecsys (Roche)	0.01 ng/mL	50	100	57	100	25
ten Wolde et al[31]	110	BNP	Shionoria (CIS Bio‖)	21.7 pmol/L	33	86	71	99	17
Kucher et al[30]	73	Pro-BNP	Elecsys (Roche)	500 pg/mL	58	95	57	100	12
Kucher et al[29]	73	BNP	Triage (Biosite¶)	50 pg/mL	58	95	60	100	12
Pruszczyk et al[26]	79	Pro-BNP	Elecsys (Roche)	153–334 pg/mL†	66	100	33	100	23

*Values are given as %, unless otherwise indicated. Sens = sensitivity; Spec = specificity; NPV = negative predictive value; PPV = positive predictive value; cTnI = cardiac troponin I; cTnT = cardiac troponin T. Adapted with permission from Kucher and Goldhaber.[22]

†Age- and gender-adjusted cutoff levels according to manufacturer.

‡Leverkusen, Germany.

§Nutley, NJ.

‖Bagnols Sur Ceze, France.

¶San Diego, CA.

overload secondary to pulmonary arterial hypertension or thromboembolic pulmonary hypertension, the BNP levels were elevated in both patient groups, although to a greater extent under the pressure overload conditions. The increasing BNP levels were positively correlated with mean pulmonary artery pressure, pulmonary vascular resistance, mean right atrial pressure, RV end-diastolic pressure, and RV mass, while inversely correlated with cardiac output and RV ejection fraction.[32] In 60 patients with pulmonary arterial hypertension, elevated baseline and posttreatment BNP were independent prognostic markers of mortality.[33]

Echocardiography

Echocardiography provides a rapid and effective means for diagnosing RV failure as well as several associated conditions, including pulmonary hypertension, valvular disease, adult congenital heart lesions, left-sided cardiomyopathies, and pericardial disease. Although transthoracic echocardiography provides direct visualization of the RV, technical and anatomic limitations may reduce its sensitivity and reproducibility, especially among the critically ill, mechanically ventilated patient population. In such patients, transesophageal echocardiography may provide a better assessment of RV structure and function.

The echocardiographic findings include tricuspid regurgitation, pulmonary artery systolic hypertension as estimated by the modified Bernoulli equation ($p = 4V^2$, where P is the peak pressure gradient between the right atrium and the RV, and V is the peak velocity of the tricuspid regurgitant jet), RV dilatation and hypokinesis, change from the typical crescent-shape RV chamber morphology to a more concentric one, right atrial enlargement, and paradoxical septal motion (Table 6).[13,38] Pulmonary artery pressure may be approximated by adding an estimate of the right atrial pressure to the calculated peak pressure gradient. The right atrial pressure may be estimated by examination of the jugular veins, direct catheter measurement of the central venous pressure, or echocardiographic evaluation of inferior vena cava size, degree of inferior vena cava collapse with inspiration, ratio of systolic to diastolic hepatic vein velocity, and magnitude of retrograde hepatic vein atrial velocity.[39] Because of the different accuracies of each of these techniques, the estimated right atrial pressure is a significant source of variability in the calculation of systolic pulmonary artery pressure. RV dilatation may be defined as a ratio of RV end-diastolic area to LV end-diastolic area of > 0.6.[40] RV dilatation is severe when the ratio is > 1.0.[40] In RV failure secondary to acute PE, echocardiography may reveal a McConnell sign, which is a distinct regional pattern of dysfunction with diffuse hypokinesis of the RV-free wall sparing the apex.[41] Mild increases in RV wall thickness may be evident.[13] Impaired LV diastolic function may be suggested by alterations in the Doppler mitral flow such that the A wave exceeds the E wave.[13] Pulmonary artery dilatation and a lack of the normal inspiratory collapse of the inferior vena cava may also be noted.[10] Pericardial effusions may be observed in patients with RV failure.[42,43]

Table 6—*Echocardiographic Findings Associated With RV Failure*

RV dilatation and hypokinesis
RV hypertrophy
Change to a more concentric RV morphology
Paradoxical septal motion
Impaired LV diastolic function
Right atrial enlargement
Tricuspid regurgitation
Pulmonary artery hypertension as estimated by the modified Bernoulli equation
Pulmonary artery dilatation
Lack of inspiratory collapse of the inferior vena cava
Pericardial effusions

Cardiac MRI

A cardiac MRI provides a direct evaluation of RV size, mass, morphology, and function (Fig 5).[19] A cardiac MRI has not been specifically validated in the evaluation of patients with acute RV failure but holds particular promise as a technique that may be more reproducible than transthoracic echocardiography. Cardiac MRI findings in RV failure include RV dilatation, tricuspid regurgitation, hypertrophy, interventricular septal flattening or paradoxical motion, and change in chamber morphology from a normal crescent shape to a more concentric form.[19] A right-sided myocardial disease, such as arrhythmogenic RV dysplasia, is often best evaluated by cardiac MRI.[19]

A cardiac MRI is superb for evaluating congenital heart disease. In adults with RV failure, a cardiac MRI identifies uncorrected congenital heart disease and defines the complex anatomy encountered in corrected lesions. Cardiac MRI is also useful in imaging pericardial disease and mediastinal pathology.[19] Gadolinium-enhanced cardiac MRI can detect ischemic myocardium, and delayed contrast-enhanced images help differentiate ischemia from infarction.[44] Severely ill patients with decompensated RV failure should be stabilized before considering imaging with a cardiac MRI.

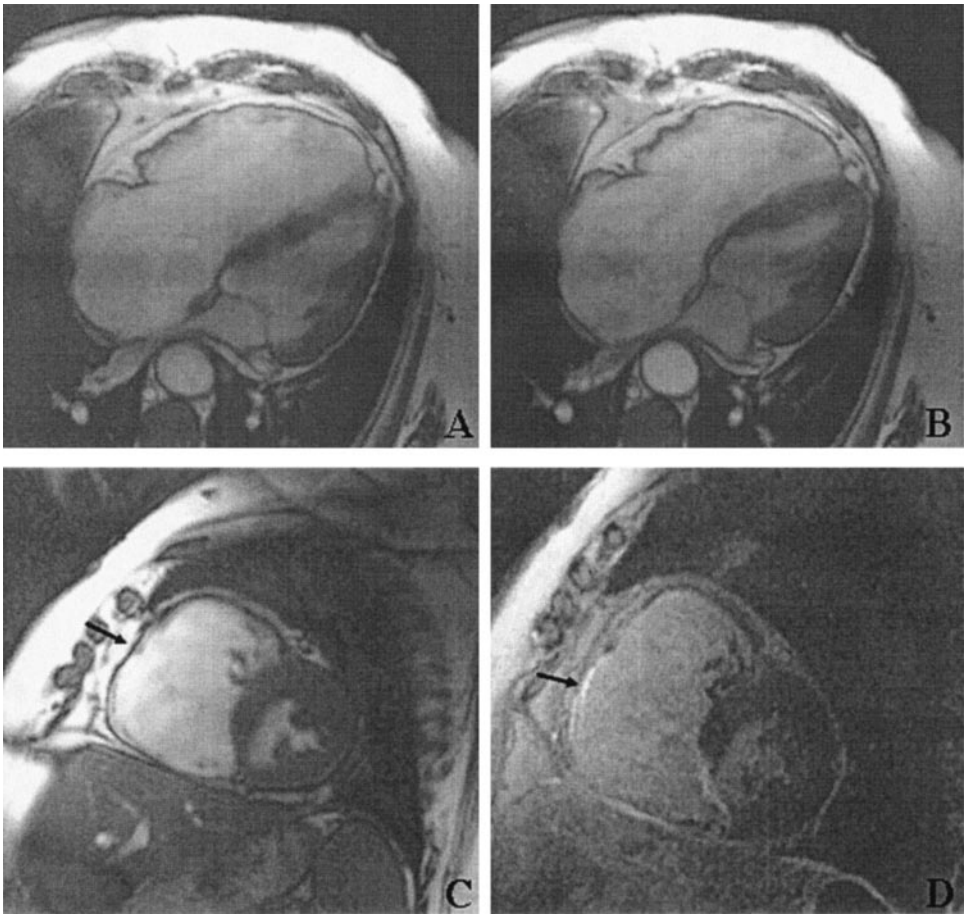

FIGURE 5. Cardiac MRI evaluation of a patient with RV infarction. RV dilatation and hypokinesis are observed with minimal change in chamber size from diastole (*top left*, *A*) to systole (*top right*, *B*). Marked RV dilatation is appreciated (arrow; *bottom left*, *C*). On the delayed contrast-enhanced imaging, increased signal intensity along the RV wall indicates areas of infarction (arrow; *bottom right*, *D*). Images courtesy of Dr. Raymond Kwong.

Right-Heart Catheterization

Although invasive, right-heart catheterization may be the diagnostic study of choice in patients with suspected RV failure when noninvasive imaging studies, such as echocardiography, are technically limited or if continuous minute-to-minute hemodynamic monitoring is required. In RV infarction, impaired diastolic function results in elevated RV diastolic pressures and a steep pressure-volume curve.[7] Pericardial constraint, in addition to limiting the compliance of both the RV and LV, eventually leads to an equalization of diastolic pressures and a rapid rise and early tapering of the RV diastolic pressure waveform classically described as a "dip-and-plateau" tracing.[7] The RV systolic waveform is depressed, and RV pulse pressure is narrow.[7,45] The RV pressure tracing is broad because of a delayed relaxation and bifid secondary to septal contraction (Fig 6).[18] The right atrial pressure is elevated, and its waveform may exhibit enhanced contractility as evidenced by a rapid upstroke, increased peak A wave amplitude, sharp x descent, and a blunted y descent.[7] Right atrial pressure may exceed pulmonary capillary wedge pressure.[18] If the right atrium is also ischemic, a depressed A wave and x descent may be seen.[7]

Tricuspid regurgitation may result from either RV dilatation or an elevation in the RV systolic pressure because of an increased RV afterload. In tricuspid regurgitation, the right atrial pressure tracing reveals a large systolic wave that comes before and may fuse with the normally observed venous filling wave.[46] As the tricuspid regurgitation worsens, the right atrial waveform more closely resembles the RV waveform (Fig 7).[46]

In conditions of increased RV afterload, RV systolic and diastolic pressures are often increased. When right-sided cardiac output is preserved, a widened RV pulse pressure may be seen in pulmonic

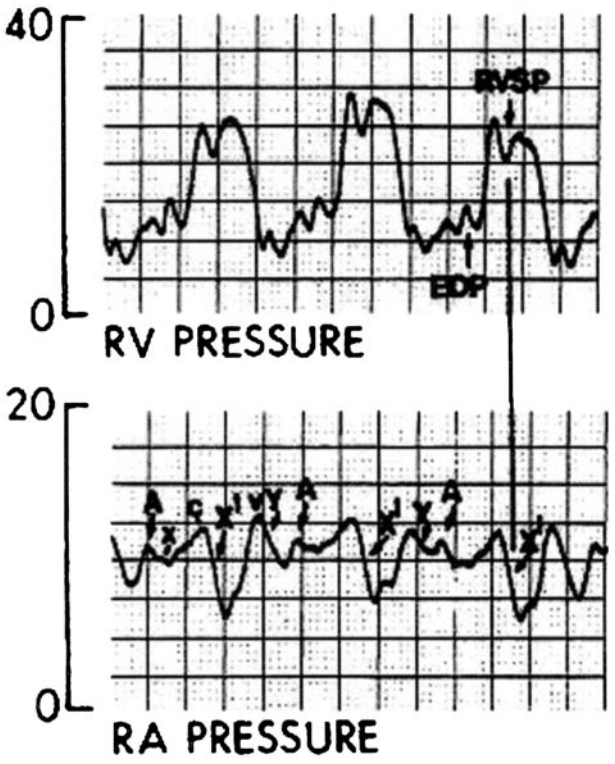

FIGURE 6. Right atrial and RV pressure tracings from a patient with RV infarction. The right atrial pressure tracing reveals a depressed A wave, prominent x descent, and a blunted y descent. The RSVP is depressed, and the waveform is bifid. Delayed relaxation and an elevated EDP are also characteristic of the RV pressure tracing. RVSP = RV systolic pressure; EDP = end-diastolic pressure. Reprinted with permission of Goldstein.[7]

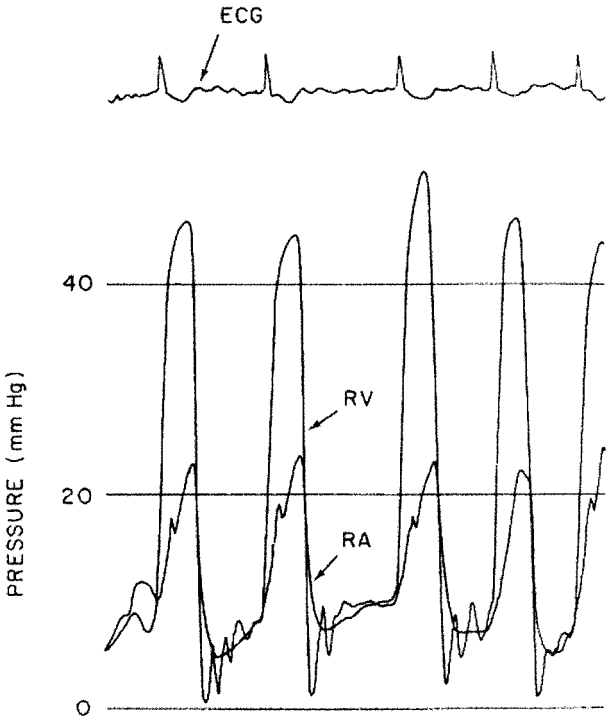

FIGURE 7. Right atrial and RV tracings from a patient with severe tricuspid regurgitation secondary to rheumatic heart disease. The right atrial waveform closely resembles the RV waveform in severe tricuspid regurgitation. Reprinted with permission of Grossman.[46]

stenosis and pulmonary arterial hypertension, because RV systolic pressure increases out of proportion to the diastolic pressure. Hemodynamic measurements may also demonstrate elevated mean pulmonary artery pressures (> 20 mm Hg). However, pulmonary artery pressures may be normal or paradoxically low in severe RV failure because of decreased right-sided cardiac output. Occasionally, the pressure tracing may "dampen" in the proximal pulmonary artery without balloon inflation mimicking the transition from pulmonary artery to pulmonary capillary wedge. This suggests a large proximal PE.[47] An elevated pulmonary capillary wedge pressure may be observed if left-sided dysfunction is contributing to RV failure.

As a result of ventricular interdependence and pericardial constraint, the hemodynamics of RV failure because of increased RV afterload may cause an equalization of diastolic pressures.[45] With RV failure, a rapid y descent, steep A wave, and precipitous x descent give the right atrial tracing a "W" configuration (Fig 8).[45] The RV tracing will often reveal a steep "dip" followed by a rapid rise in RV diastolic pressure, indicating decreased compliance.

A Pathway for Diagnosis of RV Failure

An integrated approach to the acutely decompensated RV requires assessment of the underlying cause (Fig 9). After the initial history and physical, laboratory studies, including serum cardiac biomarkers, should be obtained along with an ECG and chest radiograph. Cardiac biomarkers will aid in the diagnosis of RV failure, as well as suggest conditions that might have been unsuspected, such as myocardial infarction, PE, and congestive heart failure. After these initial studies, a transthoracic echocardiogram can confirm the diagnosis of RV failure. Frequently, the echocardiogram will help triage patients into the following four groups: LV failure, RV failure with elevated pulmonary artery pressures, RV failure without elevated pulmonary artery pressures, and pericardial disease. The finding of LV failure should prompt an additional evaluation for acute myocardial infarction, left-sided cardiomyopathy, and valvular heart disease. The patients with RV failure and elevated pulmonary artery pressures should undergo workup for causes of pulmonary hypertension. RV failure without pulmonary hypertension suggests intrinsic RV disease, such as RV infarction, right-sided valvular heart disease, and right-sided cardiomyopathy. Finally, pericardial dis-

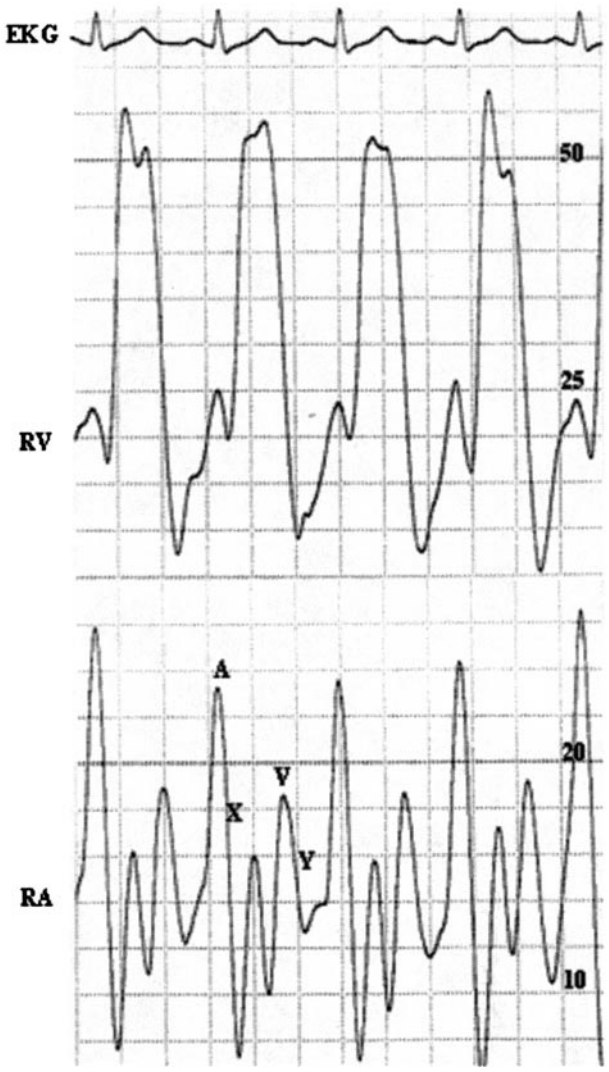

FIGURE 8. RA and RV pressure tracings from a patient with acute PE. Tracings courtesy of Dr. Nils Kucher.

ease may require additional evaluation with cardiac MRI and right-heart catheterization.

PROGNOSIS

RV failure is often a marker of the severity of the underlying disease process and a poor prognostic sign. In patients with heart failure, RV function is an important predictor of exercise tolerance and survival.[48,49] Cardiogenic shock because of RV failure is associated with a high mortality rate similar to LV shock.[8] The underlying disorder and its degree of reversibility also influence the prognosis among patients with RV failure.

MANAGEMENT

Definitive therapy for the acutely decompensated RV requires primary treatment of the underlying condition in addition to hemodynamic support. The RV is very resilient and can recover substantial function if the underlying condition is successfully addressed. Examples include percutaneous coronary intervention for RV infarction and thrombolysis or open surgical embolectomy for massive PE.[50] Even hemodynamically stable patients with acute PE and evidence of RV failure appear to derive a benefit from thrombolysis with a more rapid improvement in pulmonary artery pressures and RV function, as well as reductions in recurrent events and the need for escalation of therapy.[51–53] However, thrombolysis in hemodynamically stable patients with acute PE and RV failure remains controversial, because studies[54] have not shown a survival benefit.

In patients with chronic thromboembolic pulmonary hypertension and RV failure, thromboendarterectomy may be considered.[55] Patients with severe tricuspid regurgitation and RV dysfunction may be considered for tricuspid surgery, although the outcomes may be poorer after the onset of RV failure.[56]

Hemodynamic management of patients with RV failure has remained controversial, especially with regard to the use of volume loading, pressors, and inotropes. The current therapeutic options include oxygen, IV fluids for volume expansion, pressors, inotropes, pulmonary vasodilators, mechanical assist devices, and surgery.

The impact of such supportive measures on long-term outcomes of patients with acutely decompensated RV failure has not been clearly established. However, the ability to treat the underlying cause is an important factor in prognosis.

IV Fluids

Although patients with RV failure are often preload dependent, volume loading has the potential to overdistend the ventricles and cause increased wall tension, decreased contractility, increased ventricular interdependence, impaired LV filling, and reduced systemic cardiac output.[10,57] The utility of volume loading appears to depend on various factors, including the baseline cardiovascular function of the patient, degree of RV afterload, and volume status (Fig 10).[58] An initial trial of the volume may be appropriate for patients with decompensated RV failure, provided there is no evidence of pulmonary edema or increased right-sided preload conditions.[7,10] If signs of RV volume overload, including a central venous pressure of $>$ 12 to 15 mm Hg, are noted, the initiation of pressors and inotropes without additional volume administration may be prudent. Pulmonary artery catheterization may be helpful in determining the ideal volume loading conditions.[59]

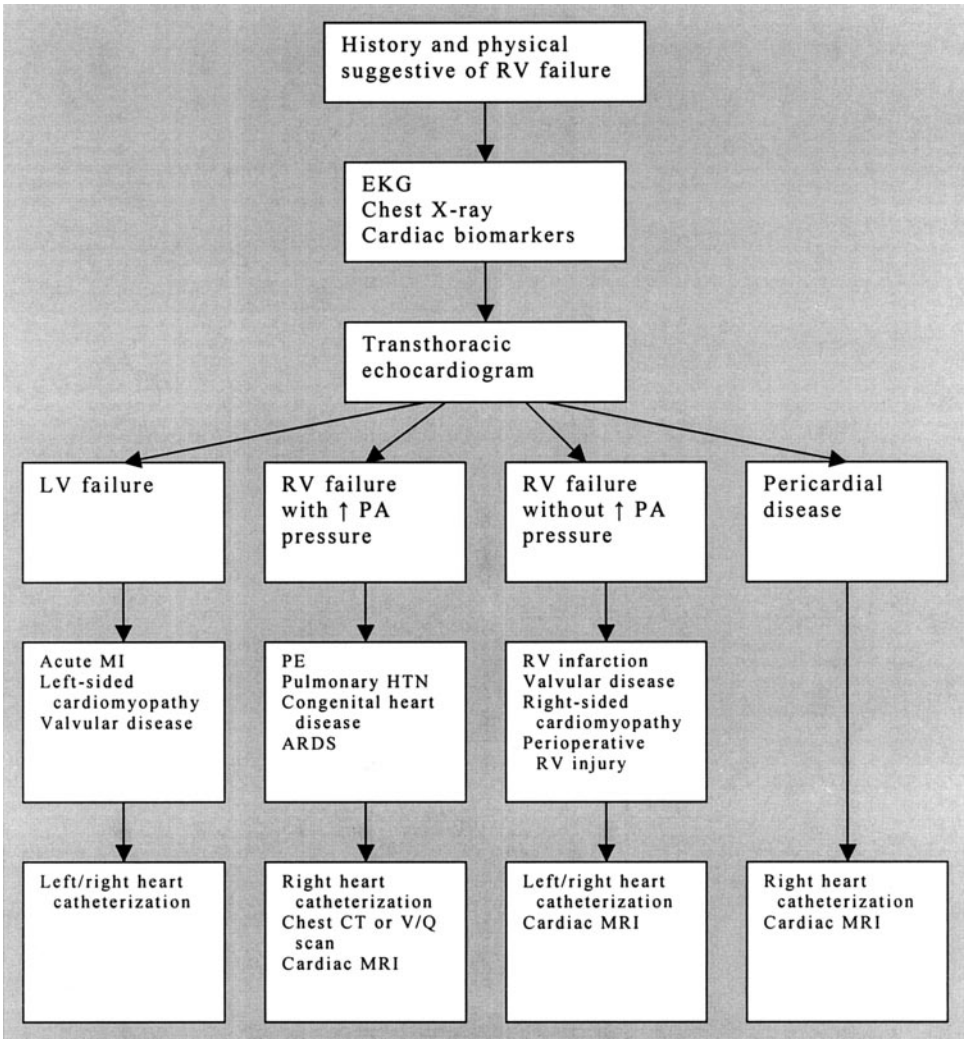

FIGURE 9. Evaluation of RV failure. PA = pulmonary arterial; MI = myocardial infarction; HTN = hypertension; V/Q = ventilation-perfusion.

Pressors and Inotropes

While awaiting primary therapy directed at the cause of RV failure to take effect, supportive use of pressors and inotropes is often necessary. The ideal agent should enhance RV function through positive inotropic effects and improve perfusion through peripheral vasoconstriction without increasing pulmonary vascular resistance (Fig 11). Norepinephrine, epinephrine, and high-dose dopamine have demonstrated favorable hemodynamic effects in the setting of acute PE and circulatory failure.[10,57] Vasopressors are also started initially to compensate for systemic hypotension that may ensue because of the vasodilatory effects of inotropes, which are frequently utilized. Vasopressin, a potent vasopressor with some positive inotropic effects, has been used in low doses to treat milrinone-induced hypotension without detriment to cardiac output or pulmonary artery pressures.[60]

In RV failure, inotropes enhance biventricular function and, thus, cardiac output. However, inotropes also have potent vasodilatory effects, which may improve RV afterload but also worsen or precipitate systemic hypotension.[7,10,59] Hemodynamic support with inotropes, as well as pressors, may be complicated by proarrhythmic effects. When used successfully in conjunction with vasopressors, the inotropes retain their beneficial effects on cardiac output without causing hypotension and diminished systemic and coronary perfusion. The commonly used inotropes include dobutamine and milrinone. Isoproterenol, a nonselective β-agonist with positive inotropy and chronotropy, has been favored in post-cardiac transplant patients with acute RV failure because of its pulmonary vasodilatory effects.[5] A new class of inotropes, the calcium sensitizers, may play an important role in the support of patients with RV failure.[61] Currently under investigation for the treat-

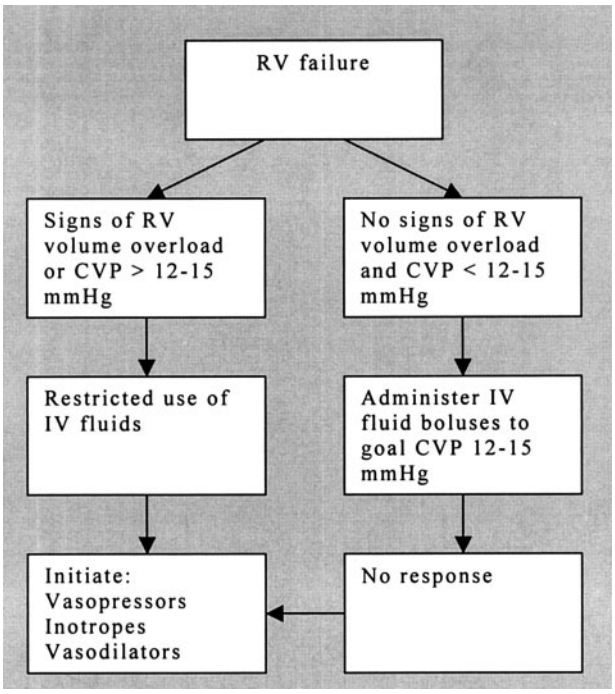

FIGURE 10. Volume administration in RV failure. CVP = central venous pressure.

ment of LV failure, the calcium sensitizer levosimendan appears to increase myocardial contractility without detriment to diastolic function and without an increase in myocardial oxygen demand.[62–64] Although it has not been studied in isolated RV failure, levosimendan appears to reduce pulmonary vascular resistance while also improving RV contractility among patients with chronic LV failure.[63,65]

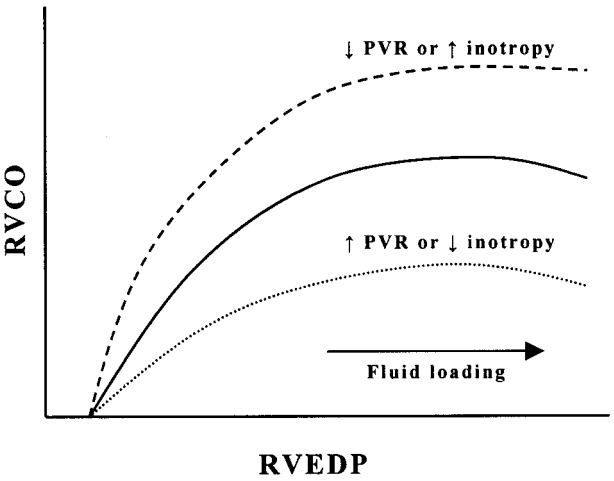

FIGURE 11. The effect of pulmonary vascular resistance and inotropy on the relationship of RVCO to RVEDP. PVR = pulmonary vascular resistance; RVCO = RV cardiac output; RVEDP = RV end-diastolic pressure.

Hemodynamic support of the patient with decompensated RV failure often requires combinations of vasopressors and inotropes (Fig 12). The normotensive patient with evidence of decreased cardiac output should be initiated on inotropic therapy with vasopressors added if a hypotensive response develops. The hypotensive patient with decreased cardiac output should receive vasopressors first and then inotropes if cardiac output remains low. Vasopressin may avoid exacerbation of tachycardia. If cardiac output remains low despite vasopressors and inotropes, a pulmonary vasodilator trial with nitric oxide may be beneficial when pulmonary hypertension is present.

Vasodilators

The goal of vasodilator use in RV failure is to improve right-sided cardiac output by reducing afterload. With many conventional vasodilators, such as nitroglycerin, nitroprusside, and hydralazine, the reductions in pulmonary vascular resistance and RV afterload come at the expense of decreased systemic vascular resistance, hypotension, and, possibly, worsening ischemia.[57] Systemic vasodilators may also decrease RV preload, thereby reducing right-sided cardiac output.[8]

Pulmonary vasodilators, such as prostacyclin (epoprostenol and prostaglandin I_2), may be useful in RV failure. However, these agents may worsen ventilation-perfusion mismatch or increase pulmonary capillary wedge pressure in patients with concurrent LV dysfunction. These agents should not be administered in patients with severe LV dysfunction.

Prostaglandin E_1 has been shown to reduce pulmonary vascular resistance and increase the cardiac index in patients with RV failure and pulmonary hypertension after a mitral valve replacement.[66] Prostacyclin is a very effective pulmonary vasodilator used in pulmonary hypertension patients with severe RV failure.[14] Both prostaglandin E_1 and prostacyclin may cause systemic hypotension.[5]

Inhaled nitric oxide may be useful in managing RV failure. Administered through an endotracheal tube or noninvasive positive pressure mask, nitric oxide decreases pulmonary vascular resistance without reducing systemic pressures.[14] In addition, nitric oxide appears to improve the ventilation-perfusion mismatch by increasing perfusion only to areas that are well-ventilated.[14] Nitric oxide may also enhance the efficacy of inotropic therapy by reducing the afterload and allowing for greater right-sided cardiac output.[14] The effect of nitric oxide on RV function has been evaluated in patients with ARDS; it reduced pulmonary vascular resistance and increased

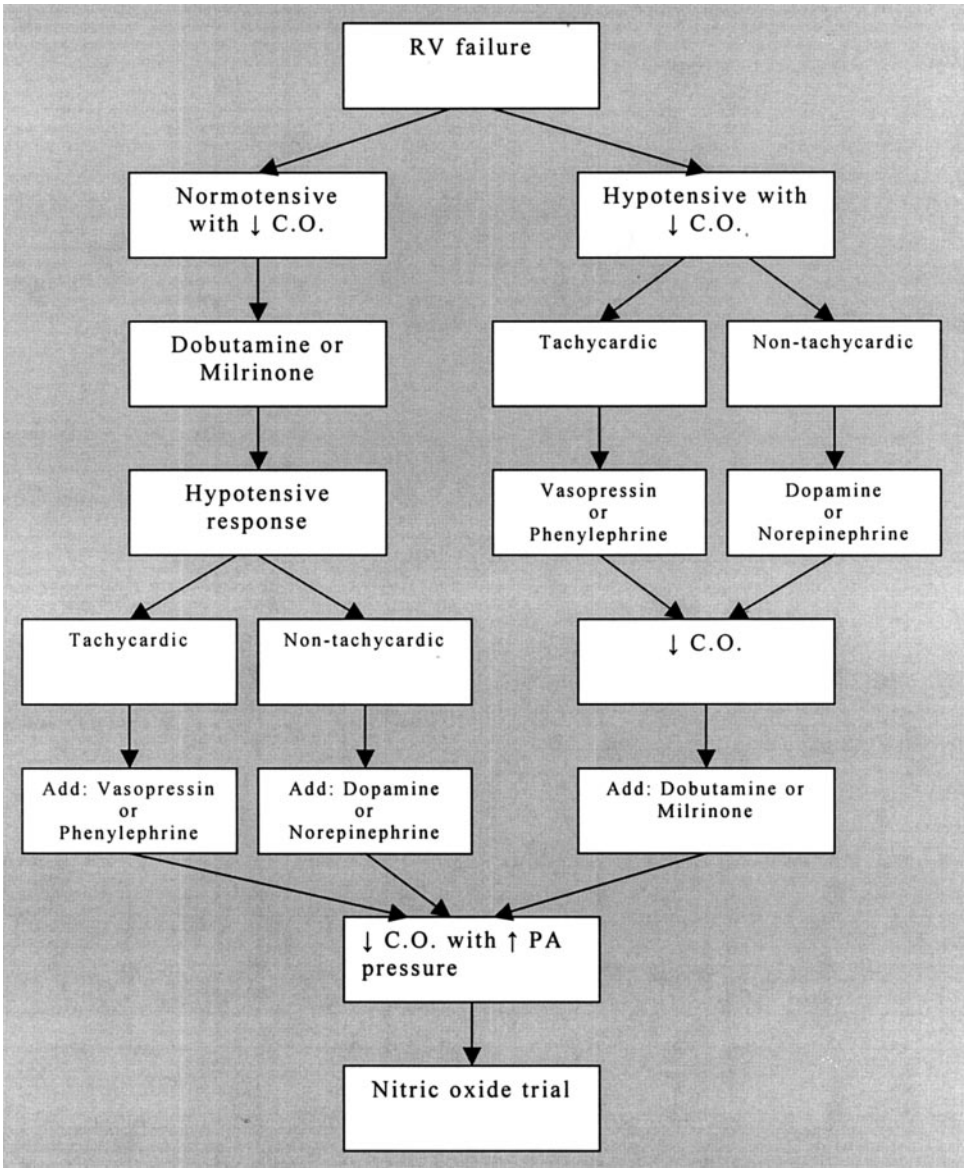

FIGURE 12. Hemodynamic support in RV failure. C.O. = cardiac output; PA = pulmonary arterial.

RV ejection fraction.[67] A subsequent study[68] evaluated inhaled nitric oxide in critically ill patients with pulmonary hypertension and echocardiographically diagnosed acute RV failure. The etiologies of RV failure included ARDS, pulmonary hypertension, COPD, PE, and obstructive sleep apnea.[68] In responders, inhaled nitric oxide significantly reduced the pulmonary artery pressures and pulmonary vascular resistance and consequently increased cardiac output, stroke volume, and mixed venous oxygen saturation.[68]

Newer vasodilators include cyclic guanosine monophosphate-specific phosphodiesterase inhibitors, such as sildenafil, and endothelin antagonists, such as bosentan.[69–71] In pulmonary hypertension patients, bosentan has been shown to improve RV systolic function and LV early diastolic filling, decrease RV dilation, and increase LV size.[72] Patients receiving bosentan require periodic monitoring for hepatotoxicity.

Mechanical Support

Patients with RV failure may require mechanical support to maintain coronary artery perfusion, as well as systemic BP. Intraaortic balloon pumps have been used in RV failure to augment right coronary artery perfusion, reduce ischemia, and allow for the

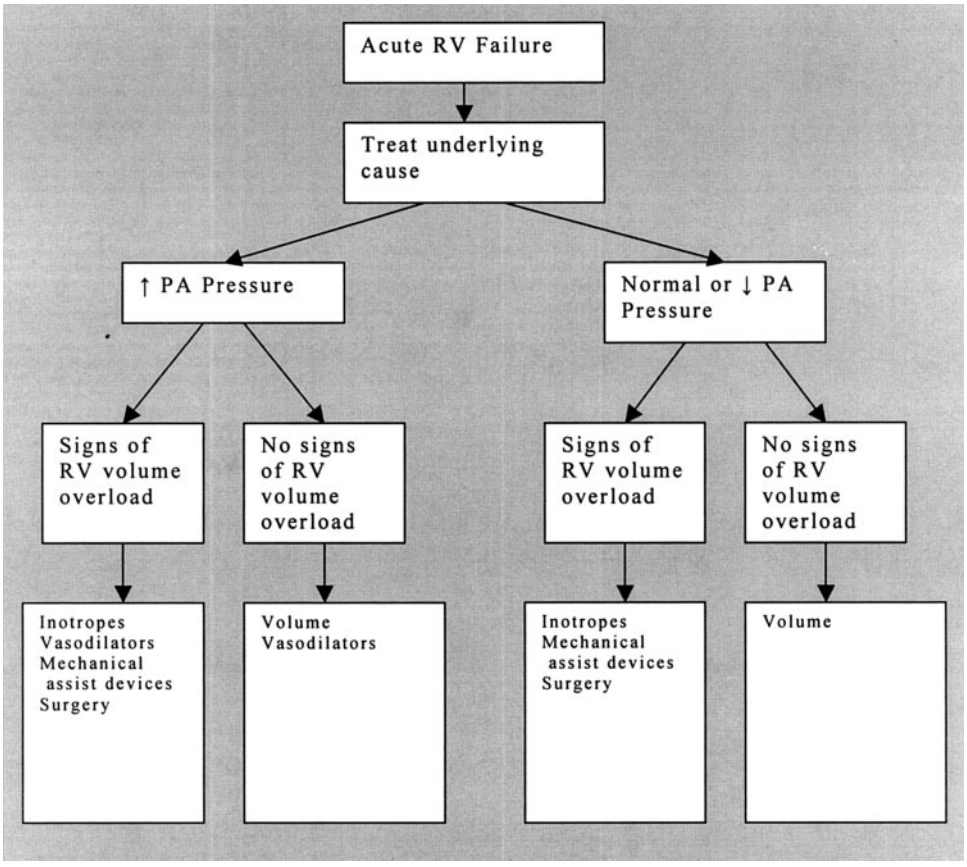

FIGURE 13. Overall management of decompensated RV failure. See Figure 12 for other abbreviations not used in the text. Adapted with permission from Hines.[77]

weaning of vasopressors that may have adverse effects on pulmonary vascular resistance.[14] RV assist devices may improve hemodynamics and act as bridges to cardiac transplantation in patients with RV failure secondary to disease intrinsic to the ventricle.[7]

The mechanical ventilatory support for patients with acute RV failure should aim to improve oxygenation and ventilation without worsening RV impedance, venous return, or diastolic function. Transpulmonary pressures should be limited in order to avoid increases in pulmonary vascular resistance.[6] A low respiratory rate should be used to limit gas trapping, which may increase pulmonary vascular resistance and elevate pleural and pericardial pressures leading to impaired diastolic filling.[6] Lower positive end-expiratory pressure settings may also limit the effect of mechanical ventilation on pulmonary vascular resistance.[6]

Surgical Interventions

The surgical strategies to manage RV failure remain limited. Current options include atrial septostomy, total RV exclusion procedures, and ultimately, cardiac transplantation.

Atrial septostomy has been used in severe pulmonary hypertension with concomitant RV failure. The creation of a shunt at the atrial level allows for right-sided decompression, a reduction in RV end-diastolic pressure, decreased wall tension, and improved contractility.[14] Although the right-to-left shunt leads to oxygen desaturation, an increased left-sided filling augments cardiac output and appears to improve oxygen delivery.[14] Atrial septostomy is generally considered when all of the other interventions have failed.[7]

A total RV exclusion procedure has been developed to treat end-stage isolated RV failure in the setting of right-sided volume overload.[73,74] The procedure involves resection of the entire RV-free wall along the atrioventricular groove, closure of the tricuspid valve, coronary sinus diversion to the left atrium, and creation of a cavopulmonary connection.[73] This procedure has been evaluated in patients with arrhythmogenic RV dysplasia and Ebstein anomaly.[73,74] A similar approach has been evaluated in postcoronary artery bypass graft patients with refractory RV dysfunction secondary to infarction.[75]

Cardiac transplantation may be considered in patients with RV failure, although they are often unsuitable can-

didates. Severe RV failure itself is a risk factor for unsuccessful bridging to transplantation.[76] RV failure secondary to recurrent PE causing chronic thromboembolic pulmonary hypertension may be treated with surgical pulmonary thromboendarterectomy.[55]

A Pathway for Management of RV Failure

The management of the acutely decompensated RV requires treatment of the underlying causes and hemodynamic support based on pathophysiology (Fig 13). Patients with elevated pulmonary arterial pressures and RV volume overload should receive inotropes, vasodilators, mechanical assist devices, and, possibly, surgery if supportive care is unsuccessful. Patients with pulmonary hypertension and no signs of RV volume overload may be treated with volume administration followed by pulmonary vasodilators. Patients with RV volume overload and normal pulmonary artery pressures should receive inotropic support followed by mechanical assist devices or surgery if all else fails. Patients with neither pulmonary hypertension nor RV volume overload may initially be managed with volume administration.

Conclusions

RV failure has a high mortality rate and has been increasing in frequency. We now have an enhanced understanding of the pathophysiology of RV failure, improved approaches to diagnosis and risk stratification, and extensive therapeutic options. The treatment depends on the assessment of RV function under varying pressure and volume-loading conditions. An approach that integrates pathophysiology and combines therapy for underlying causes of RV failure with supportive measures is essential.

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The Acutely Decompensated Right Ventricle* : Pathways for Diagnosis and Management

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Chest 2005;128; 1836-1852
DOI 10.1378/chest.128.3.1836