

The Clinical Physiologist

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Cardiac Tamponade in Severe Pulmonary Hypertension

A Therapeutic Challenge Revisited

Jacob R. Adams, Adriano R. Tonelli, Haala K. Rokadia, and Abhijit Duggal

Respiratory Institute, Cleveland Clinic, Cleveland, Ohio

In Brief

A 62-year-old man with a recent diagnosis of pulmonary hypertension presented with a new large pericardial effusion. Forty eight hours after admission, the patient developed intermittent episodes of hypotension, worsening organ perfusion, increased edema, and decreased urinary output. His blood pressure and organ perfusion improved after an intervention that was based on an understanding of the

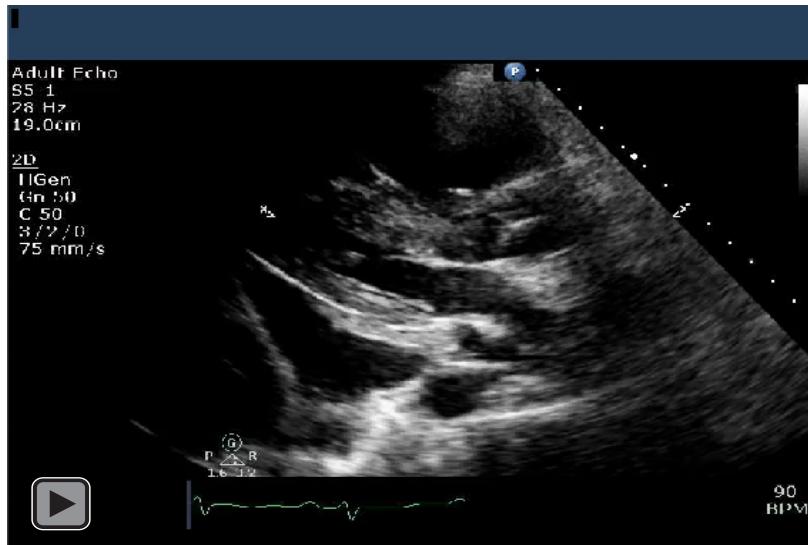
physiological effects of pericardial effusion in the setting of pulmonary hypertension and right ventricular failure.

interstitial lung disease (probable idiopathic pulmonary fibrosis), and a transthoracic echocardiogram had shown severe right ventricular dysfunction with an estimated right ventricular systolic pressure of 93 mm Hg. A left and right heart catheterization was scheduled, but the patient did not keep the appointment.

At the time of admission, the blood pressure was 104/74 mm Hg, the heart rate was 92 beats per minute, the respiratory rate was 24 breaths per

The Clinical Challenge

A 62-year-old man presented to the hospital with worsening bilateral leg swelling and dyspnea. Six months before presentation, he was diagnosed with



Video 1. Parasternal long-axis view reveals a large pericardial effusion with a small left ventricle and diastolic compression of the left atrium. The right ventricle is severely dilated. BPM = beats per minute.

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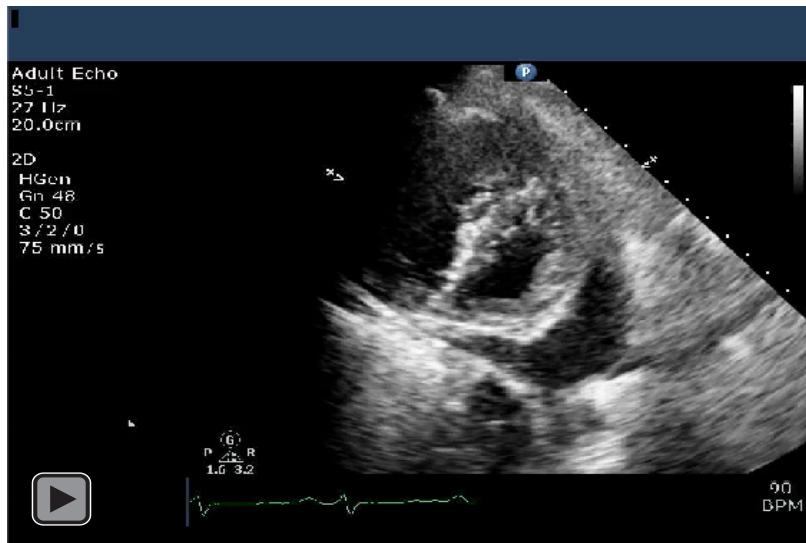
Correspondence and requests for reprints should be addressed to Jacob Adams, D.O., Respiratory Institute, Cleveland Clinic, Cleveland, OH 94965. E-mail: jake@skipatrick.com

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Video 2. Parasternal short-axis view showing a large pericardial effusion, systolic and diastolic flattening of the interventricular septum, and severely enlarged right ventricle. BPM = beats per minute.

minute, and the arterial oxygen saturation was 87% while the patient breathed ambient air. Jugular venous distension was present to the angle of the jaw. Cardiac examination revealed a 3/6 holosystolic murmur heard best at the left midsternal border. The lungs were clear on auscultation bilaterally. There was pitting edema of both lower extremities to the midthighs and peripheral cyanosis.

A transthoracic echocardiogram revealed an ejection fraction of 58%,

severe right atrial and ventricular dilation, and an estimated right ventricular systolic pressure of 95 mm Hg. Right ventricular systolic function was severely decreased without evidence of hypertrophy or diastolic dysfunction. Right atrial pressure was estimated to be 15 mm Hg. A large pericardial effusion measuring 2.8 cm was identified (Figure 2). There was systolic and diastolic flattening of the interventricular septum (Videos 1 and 2) and diastolic collapse of the left atrium (Figure 2,

Video 1). Right atrial and right ventricular diastolic collapse was not present. The inferior vena cava measured 2.3 cm, and there was no respiratory variation.

As shown in Table 1, right and left heart catheterization demonstrated severe pulmonary hypertension, a markedly elevated transpulmonary gradient, increased pulmonary vascular resistance, and a very low cardiac output. "Equalization" of the mean right atrial pressure, right ventricular end-diastolic pressure, pulmonary artery wedge pressure, and left ventricular end-diastolic pressure was not present. An arterial catheter showed a maximal inspiratory decline in peak systolic pressure of 8 mm Hg.

The patient subsequently developed worsening hypotension and signs of inadequate organ perfusion. He was intubated and mechanically ventilated, and dobutamine and norepinephrine were infused to increase cardiac output and blood pressure. Inhaled epoprostenol was started because of worsening hypoxemia.

Questions

1. Does this patient have cardiac tamponade, despite the absence of typical echocardiographic and hemodynamic findings?

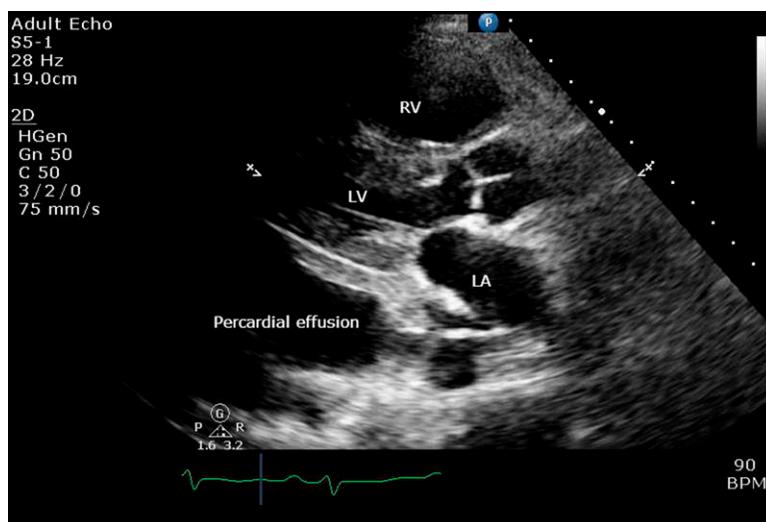


Figure 1. Parasternal long-axis view of the left atrium, left ventricle, and right ventricular outflow tract during ventricular systole. A large pericardial effusion is seen. BPM = beats per minute; LA = left atrium; LV = left ventricle; RV = right ventricle.

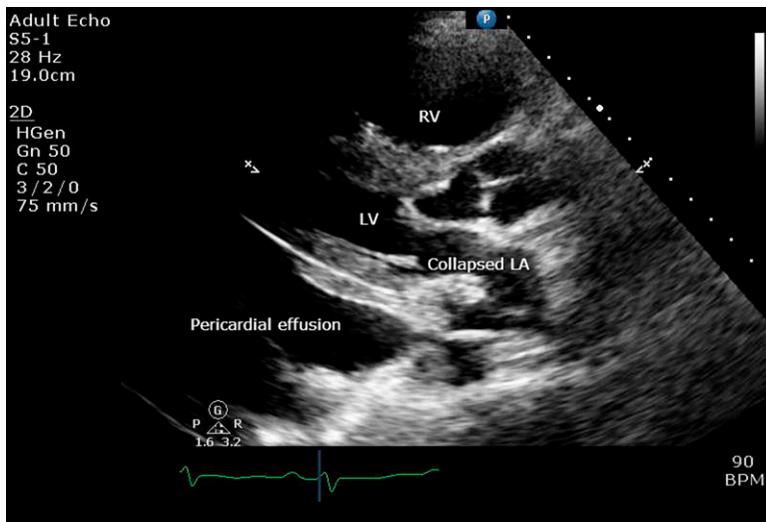


Figure 2. Parasternal long-axis view during ventricular diastole. Note the compression of the left atrium by the pericardial fluid. BPM = beats per minute; LA = left atrium; LV = left ventricle; RV = right ventricle.

2. What are the possible hemodynamic effects of pericardial drainage in patients with pre-existing right ventricular failure?

Clinical Reasoning

In clinical practice, cardiac tamponade is not an “all-or-none” phenomenon, but rather a continuum of hemodynamic impairment. Diagnosis depends on an overall assessment of clinical and echocardiographic findings, hemodynamic measurements, and other corresponding patient-level variables. As illustrated by this case, the recognition of

cardiac tamponade in the presence of severe pulmonary hypertension and right ventricular failure can be even more challenging, because the “classic” findings are often not observed. Our patient clearly had hemodynamic compromise from a large pericardial effusion, but did not exhibit the common features of tamponade. This can be explained by the pre-existing, markedly elevated right-sided pressures, which prevented pulsus paradoxus, right atrial and ventricular diastolic collapse, and equalization of diastolic pressures.

Drainage of a large pericardial effusion in patients with pulmonary

hypertension has been accompanied by catastrophic, sudden hemodynamic collapse. The underlying pathophysiology is not clearly understood, but it has been postulated that the presence of pericardial fluid limits right ventricular distension in response to pressure and volume overload. When the pericardial fluid is removed, rapid enlargement of the right ventricle causes: (1) reduced right ventricular systolic function due to muscle fiber distension; and (2) compression of the left ventricle, which leads to impaired diastolic filling and left ventricular outflow track obstruction. This complex interaction produces a sudden drop in cardiac output, which may reduce coronary blood flow during diastole and cause right ventricular ischemia, which further compromises cardiac output.

The Clinical Solution

Due to the posterior location of the pericardial effusion, our patient was taken to the operating room, where 1 liter of pericardial fluid was slowly removed. Dobutamine and norepinephrine were titrated off over the next 48 hours. The patient required an additional 2 weeks in the hospital, and was discharged home in stable condition.

The Science behind the Solution

Pathophysiology of Pericardial Tamponade

The hallmark of cardiac tamponade is elevation and equalization of diastolic pressures in all cardiac chambers. The pericardial space normally contains approximately 20–50 ml of fluid. In addition, the pericardium has both elastic and nonelastic properties allowing for compliance and stiffness, respectively. In the presence of a pericardial effusion, the pressure surrounding the heart uniformly increases with accumulation of fluid. The pericardial sac may stretch to accommodate the increased fluid until its elastic reserve is reached. This has two important effects. First, the pressure *within* the atria and ventricles (intracavitory pressure) rises, and,

Table 1. Heart catheterization

Hemodynamic Profile	Measurement
Systemic BP, mm Hg	102/71
HR, bpm	90
Aortic pressure, mm Hg	96/75
LVEDP, mm Hg	17
Right atrial pressure, mean, mm Hg	24
Right ventricular pressure, mm Hg	100/28
Pulmonary artery pressure, mm Hg	98/50
Pulmonary artery pressure, mean, mm Hg	64
Pulmonary capillary wedge pressure, mm Hg	14
Fick cardiac output, L/min	2.90
Fick cardiac index, L/min/m ²	1.60
Systemic vascular resistance, dyne s/cm ⁵	1,852
Pulmonary vascular resistance, dyne s/cm ⁵	1,488
Prepericardial drainage BP (mean), mm Hg	107/74 (85)
Postpericardial drainage BP (mean), mm Hg	138/79 (98)

Definition of abbreviations: BP = blood pressure; bpm = beats per minute; HR = heart rate; LVEDP = left ventricular end-diastolic pressure.

Table 2. Cardiac chamber collapse in tamponade

Chamber of Heart	Cardiac Cycle	Characteristics
RA collapse*	End diastole and early systole	More specific if right atrial collapse lasts longer than 30% of cardiac cycle
RV collapse*	Early diastole	Less sensitive, but more specific for cardiac tamponade
LA collapse	Early diastole	Seen in approximately 25% of cardiac tamponade and is highly specific
LV collapse	Early diastole	Usually only under specific conditions (i.e., regional tamponade or pulmonary hypertension)

Definition of abbreviations: LA = left atrial; LV = left ventricular; RA = right atrial; RV = right ventricular.

*Most characteristic of cardiac tamponade, however nonspecific.

second, the pressure gradient *between* the inside and the outside of the heart chambers (transmural pressure) falls. The collapse of the cardiac chambers during this fall in transmural pressure can be visualized by echocardiogram (Table 2). Because blood flow into the right atrium is proportional to the difference between systemic venous and right atrial pressure, the increase in intracavitory pressure reduces venous return to the heart. Simultaneously, the drop in transmural pressure decreases ventricular volume and compliance, which impairs filling during diastole. The decreased venous return and reduced diastolic filling cause a drop in right and left ventricular preload and stroke volume. If pericardial

pressure continues to rise, cardiac output, and eventually blood pressure, will fall.

Pulsus Paradoxus

Pulsus paradoxus is defined as a decrease in systolic blood pressure by more than 10 mm Hg during spontaneous inspiration. This is an important and characteristic finding in patients with pericardial tamponade. It reflects an exaggerated response to the normal ventilation-induced changes in intrathoracic (pleural) pressure, and can be explained by the concept of “ventricular interdependence.”

The right and left ventricles are mechanically coupled, because they

share a common septum, and the sum of right and left ventricular volume is limited by the size of the pericardial sac. This means that an increase or decrease in the diastolic volume of one ventricle must have the opposite effect on the volume of the other. Spontaneous inspiration reduces pleural and intracavitory right atrial pressure, which increases venous return and right ventricular volume. This decreases left ventricular volume and increases left ventricular diastolic pressure, which normally causes a small drop in diastolic filling, preload, and stroke volume.

Pericardial tamponade causes a marked reduction in diastolic volume, which places the ventricles on the steep portion of the ventricular function (Starling) curve. Therefore, even the relatively small changes in ventricular volume produced by inspiratory changes in pleural pressure cause a marked change in left ventricular stroke volume and blood pressure.

The Diagnosis of Pericardial Tamponade

Diagnosis is usually based on characteristic clinical, echocardiographic, and hemodynamic findings that reflect the underlying pathophysiology. Because the right ventricle is normally thinner and more compliant than the left ventricle and has much lower systolic and diastolic

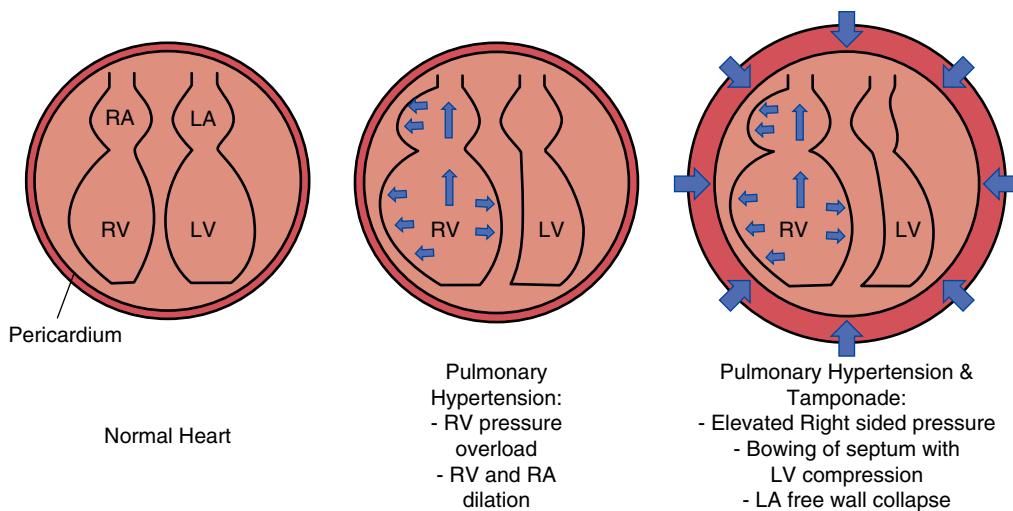


Figure 3. Schematic illustration of the effects of right ventricular dilation and a large pericardial effusion on left ventricular size and function. Arrows indicate the direction of force. LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle.

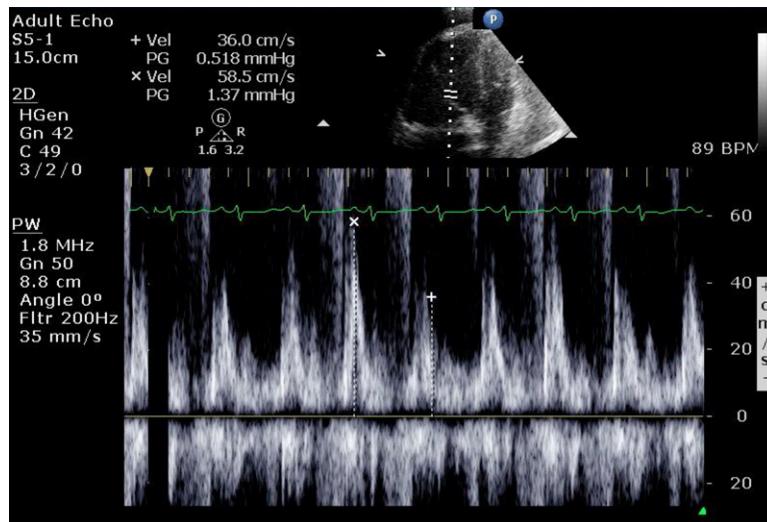


Figure 4. There is markedly exaggerated respiratory variation in transvalvular tricuspid inflow as assessed by pulsed wave Doppler. BPM = beats per minute; PW = pulsed wave.

pressures, echocardiography typically shows a large pericardial effusion that compresses the right ventricular free wall during diastole, when intracavitory and transmural pressure reach their lowest levels. If fluid is distributed evenly throughout the pericardial space, its effect on intracavitory pressure is fairly uniform, and right heart catheterization shows “equalization” of the mean right atrial pressure, right ventricular end-diastolic pressure, and mean pulmonary artery wedge pressure (which approximates mean left atrial pressure). By convention, “equalization” is said to be present when

these pressures differ by no more than 5 mm Hg.

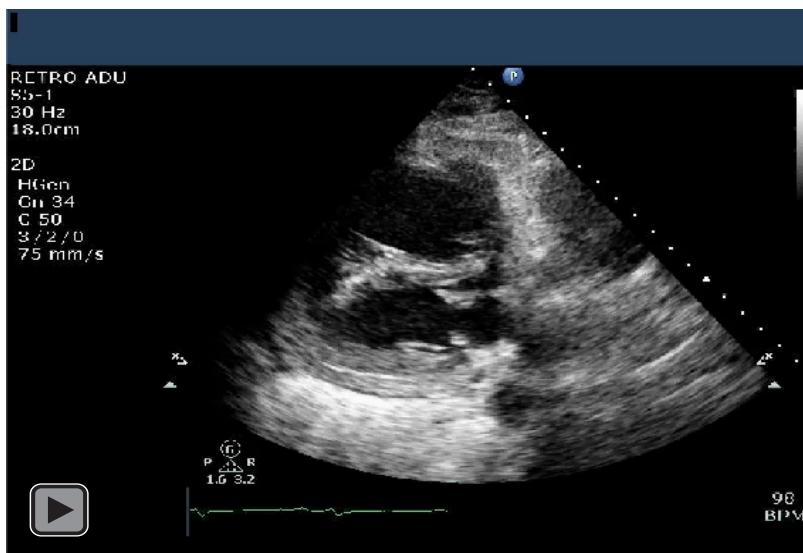
Pericardial Tamponade in Patients with Right Ventricular Failure

The presence of pre-existing right ventricular failure may significantly alter these typical diagnostic findings. Abnormally high intracavitory pressures make the right atrium and right ventricle much more resistant to collapse. Because of ventricular interdependence, right ventricular dilation causes a disproportionate decrease in the size of the left ventricle, which worsens

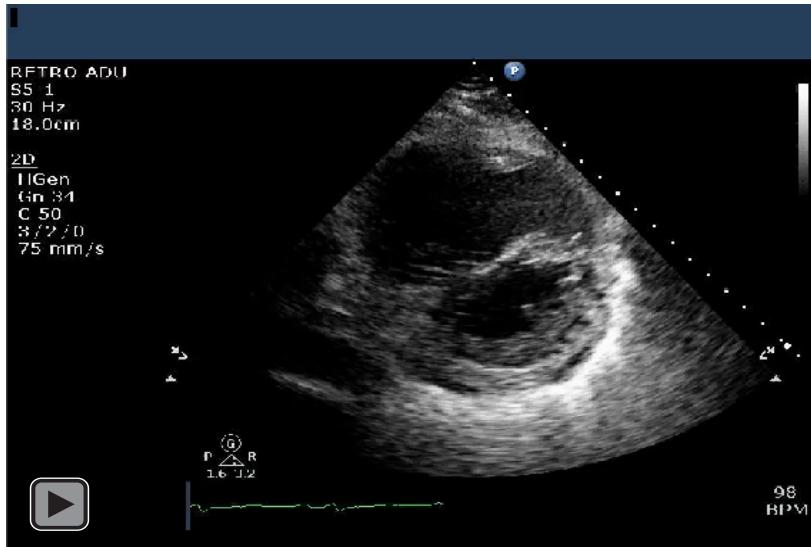
diastolic filling and stroke volume (Figure 3). Pre-existing right ventricular pressure overload may prevent equalization of pressures by causing mean right atrial and right ventricular end-diastolic pressure to significantly exceed mean left atrial pressure. In such cases, exaggerated respiratory variation in tricuspid transvalvular Doppler flow velocity can suggest the presence of cardiac tamponade (Figure 4). Finally, pulsus paradoxus is often absent in this subset of patients, because elevated right ventricular pressure and volume prevent the inspiratory drop in pleural pressure from further increasing right ventricular preload and stroke volume.

Although pericardial drainage is required to reverse the life-threatening effects of pericardial tamponade, it has been accompanied by further hemodynamic deterioration and death in patients with pre-existing right heart failure. It is believed that rapid fluid removal allows the already dilated right ventricle to further expand, thereby acutely decreasing left ventricular diastolic filling and cardiac output. Echocardiographic findings before (Videos 1 and 2) and after (Videos 3 and 4) pericardial drainage are shown.

We have demonstrated that great care must be taken when patients with pulmonary hypertension and right ventricular failure develop a large pericardial effusion.



Video 3. Parasternal long-axis view shows minimal pericardial effusion after drainage. The left atrium is no longer inverted. BPM = beats per minute.



Video 4. Parasternal short-axis view shows minimal pericardial effusion after drainage. Systolic and diastolic flattening of the interventricular septum persist. BPM = beats per minute.

Understanding the underlying pathophysiology is paramount to the

success of managing such a complicated patient. ■

Author disclosures are available with the text of this article at www.atsjournals.org.

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