Reversed Pulsus Paradoxus in Right Ventricular Failure

Maan Malahfji, M.D.[a]; *Salman Arain,* M.D.[b]

[a]HOUSTON METHODIST HOSPITAL, HOUSTON, TEXAS[b]; MCGOVERN MEDICAL SCHOOL, UNIVERSITY OF TEXAS HEALTH SCIENCE CENTER AT HOUSTON, HOUSTON, TEXAS

ABSTRACT: Reversed pulsus paradoxus was first described in 1973 as a rise in peak systolic pressure on inspiration in patients with idiopathic hypertrophic subaortic stenosis or isorhythmic ventricular rhythm and in patients with left ventricular systolic dysfunction on positive pressure ventilation. Positive pressure ventilation, for example, may impel blood from the pulmonary capillaries and venules into the left atrium. This may increase left ventricular preload and accelerate ventricular emptying, which in turn may cause the systolic arterial pressure to rise during inspiration. We observed this phenomenon in a patient with a large pericardial effusion, right ventricular failure, and pulmonary arterial hypertension, and we noted the lack of echocardiographic features of tamponade in the presence of right ventricular hypertrophy and pulmonary hypertension. This case report discusses the subsequent occurrence of acute congestive heart failure after pericardiocentesis.

CASE REPORT

A 41-year-old African American female with a medical history of systemic sclerosis complicated by severe pulmonary arterial hypertension and interstitial lung disease was admitted with worsening dyspnea and increasing oxygen requirement. Her physical exam demonstrated an elevated jugular venous pulsation, fine crackles in the lung bases, a right ventricular (RV) heave, and a loud S2 sound. She had a heart rate of 110 beats per minute, a respiratory rate of 36 breaths per minute, blood pressure (BP) of 118/65 mm Hg, and 93% oxygen saturation on high flow nasal cannula. A transthoracic echocardiogram revealed a circumferential pericardial effusion with a maximum diameter of 2.8 cm. There was severe right atrial and RV dilatation with reduced RV systolic function and flattening of the interventricular septum. There was no evidence of RV diastolic collapse but there was severe RV hypertrophy. Transthoracic echocardiography demonstrated reversal of normal respiratory variation when mitral inflow was assessed by pulsed wave Doppler (Figure 1). Right- and left-heart catheterization showed a reversed pulsus paradoxus pattern (Figure 2, respirometry was not available). The left ventricular (LV) end-diastolic pressure was lower

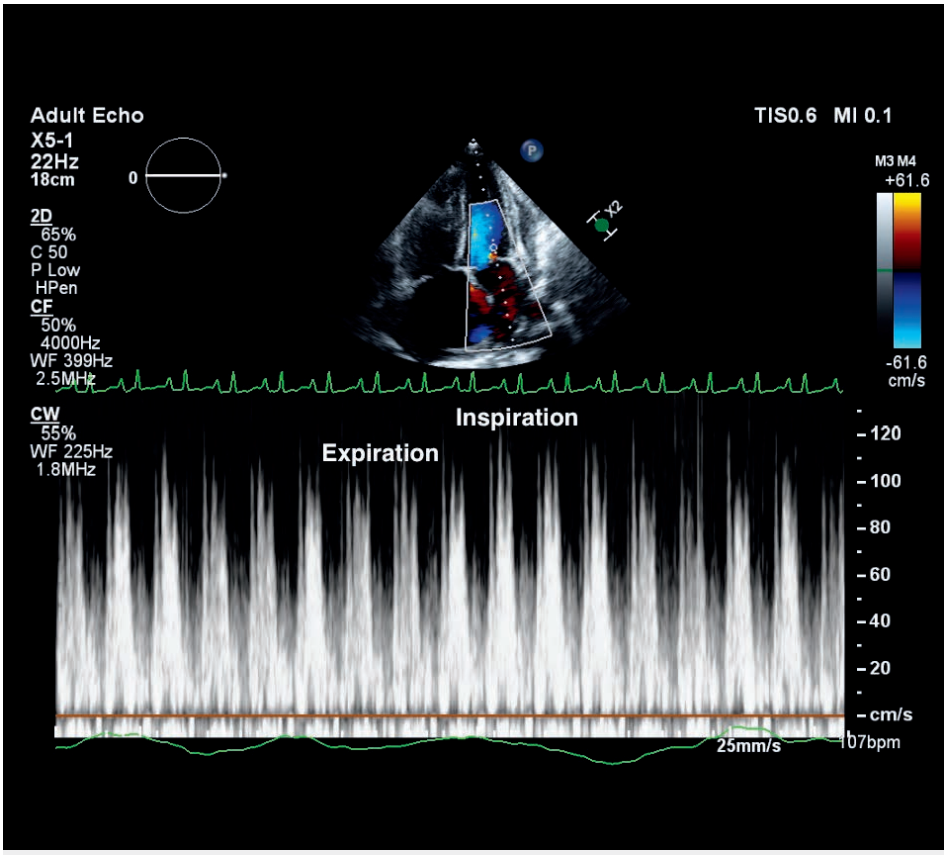

Figure 1.
Pulse wave Doppler recording of the mitral valve inflow showing reversal of the expected respiratory variation prior to pericardiocentesis.

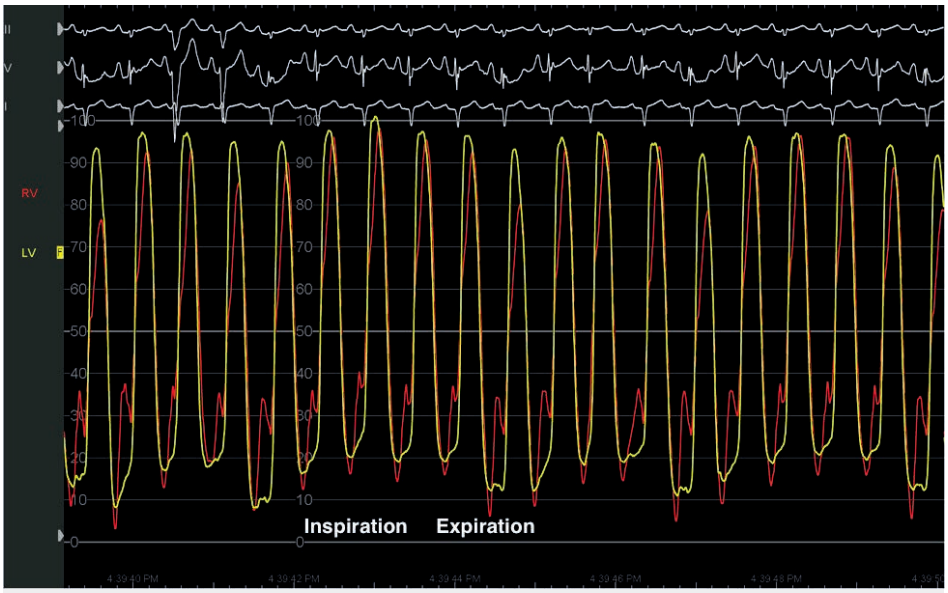

Figure 2.
Cardiac catheterization shows a decrease in left ventricular systolic and diastolic pressures during expiration.

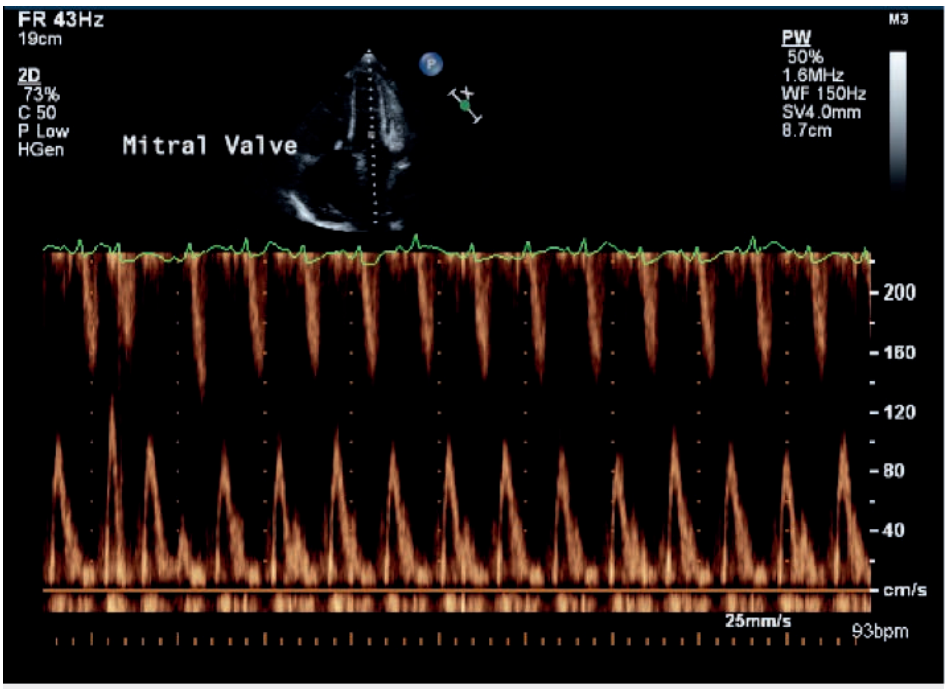

Figure 3.
Pulse wave Doppler recording of tricuspid valve inflow showing normal respiratory variation 1 week after pericardiocentesis. I: inspiration, E: expiration

than the RV end-diastolic pressure, and this relationship was exaggerated during expiration. Pericardiocentesis was performed under echocardiographic and fluoroscopic guidance, and 625 cc of serosanguinous fluid was removed. Cytologic analysis of the pericardial fluid revealed no malignant cells, and cultures were negative. Tricuspid and mitral inflow patterns by pulse wave Doppler are shown in figures 3 and 4.

The patient's condition worsened during the following 24 hours with progressive hypoxemia and hypotension (systemic BP 82/40 mm Hg). A follow-up catheterization showed elevation of pulmonary capillary wedge pressure (PCWP) to 36 mm Hg. She was placed on noninvasive positive pressure ventilation and intravenous diuretics. Five days later, PCWP decreased to 14 mm Hg and the patient returned to her clinical baseline.

DISCUSSION

Reversed pulsus paradoxus is a phenomenon first described by Massumi et al. in patients with hypertrophic obstructive cardiomyopathy or isorhythmic ventricular rhythm and in patients with LV systolic dysfunction who received positive pressure ventilation.[1] We report similar observations in a patient with pulmonary arterial hypertension, severe RV failure, and a large pericardial effusion.

Pulsus paradoxus describes an exaggeration of the normal inspiratory fall in systolic blood pressure. Exaggerated ventricular interdependence develops in the presence of a significant pericardial effusion, and the inspiratory increase in venous return to the right heart shifts the interventricular septum to the left, thereby decreasing LV filling and output.

Different mechanisms occur in reversed pulsus paradoxus, depending on the etiology. In patients with hypertrophic

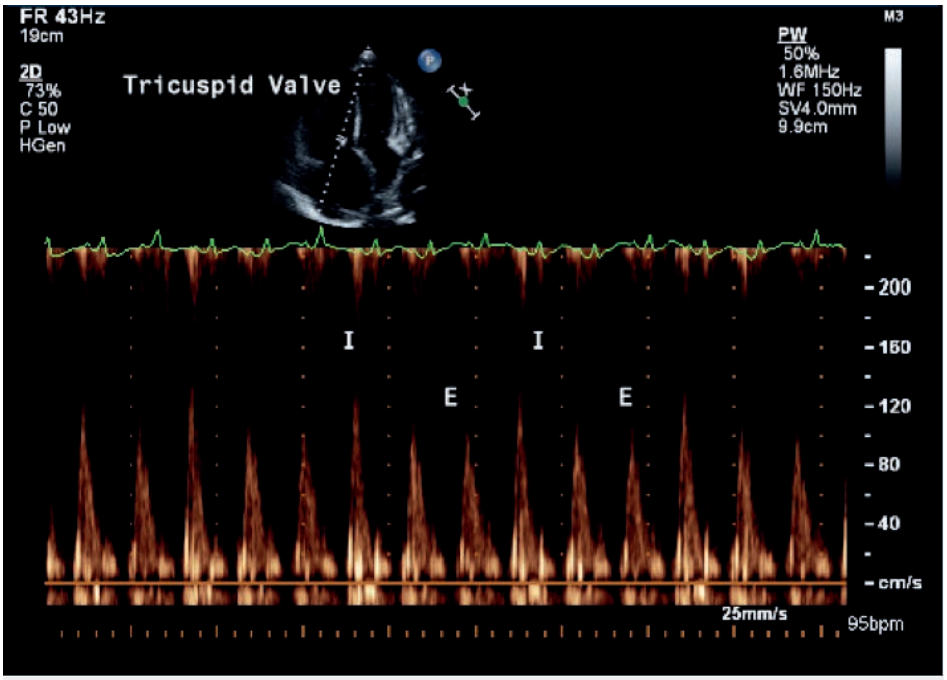

Figure 4.
Pulse wave Doppler recording of mitral valve inflow showing minimal respiratory variation 1 week after pericardiocentesis.

subaortic stenosis, now termed hypertrophic cardiomyopathy, there is expiratory exaggeration of LV outflow obstruction that causes decreased blood pressure with expiration. With inspiration, the increased venous return increases filling in the left side of the heart and lessens outflow obstruction, causing a relative increase in blood pressure. In our patient, we hypothesize that RV hypertrophy offers an explanation to our finding. The noncompliant RV wall is not compressed by the relatively modest elevation in pericardial pressure seen in early diastole with active LV relaxation. Therefore, echocardiographic signs of tamponade might be absent. Right ventricular hypertrophy and noncompliance may exaggerate the effects of respiration of LV filling and output. In the setting of tachypnea and possibly intrinsic positive end-expiratory pressure, LV filling may improve during inspiration while being impeded during expiration.

The occurrence of ventricular dysfunction and cardiogenic pulmonary edema following pericardiocentesis is of great clinical significance, but the pathogenesis is unclear. First described by Vandyke et al,[2] increasing ventricular preload after pericardiocentesis initially improves contractility due to the Frank-Starling mechanism. However, persistent increases in preload may lead to elevated ventricular wall stress and reduced contractility, in turn causing decreased stroke volume and cardiac output. Ischemic and sympathetic overdrive theories have also been suggested.[3] With a reported incidence of 4.8% to 11% and high mortality rates,[4,5] pericardial decompression syndrome warrants further investigation. There are no guideline recommendations that specify the upper limits on the quantity or rapidity of fluid removal during pericardiocentesis. Additional studies to identify optimal treatment strategies, specifically in patients with pulmonary hypertension, are warranted.

Conflict of Interest Disclosure:
The authors have completed and submitted the *Methodist DeBakey Cardiovascular Journal* Conflict of Interest Statement and none were reported.

Keywords:
reversed pulsus paradoxus, pulmonary hypertension, pericardial decompression

REFERENCES

1. Massumi RA, Mason DT, Vera Z, Zelis R, Otero J, Amsterdam EA. Reversed pulsus paradoxus. N Engl J Med. 1973 Dec 13;289(24):1272-5.

2. Vandyke WH Jr, Cure J, Chakko CS, Gheorghiade M. Pulmonary edema after pericardiocentesis for cardiac tamponade. N Engl J Med. 1983 Sep 8;309(10):595-6.

3. Ligero C, Leta R, Bayes-Genis A. Transient biventricular dysfunction following pericardiocentesis. Eur J Hear Fail. 2006 Jan;8(1):102-4.

4. Sabzi F, Faraji R. Predictors of post pericardiotomy low cardiac output syndrome in patients with pericardial effusion. 2015;7(1):18-23.

5. Pradhan R, Okabe T, Yoshida K, Angouras DC, DeCaro MV, Marhefka GD. Patient characteristics and predictors of mortality associated with pericardial decompression syndrome: a comprehensive analysis of published cases. Eur Hear J Acute Cardiovasc Care. 2015 Apr;4(2):113-20.