Doppler-Detected Paradoxus of Mitral and Tricuspid Valve Flows in Chronic Lung Disease

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An echocardiographic Doppler study in a patient with pulsus paradoxus of respiratory origin demonstrated a large inspiratory increase of tricuspid flow velocity and a corresponding decrease of mitral flow velocity. This “flow paradoxus” is therefore not specific for cardiac tamponade, and provides evidence that decreased left ventricular filling is an important mechanism of pulsus paradoxus observed in severe chronic lung disease. (J Am Coll Cardiol 1986;8:706–9)

Although most physicians recognize that cardiac tamponade is in large measure a clinical diagnosis, a number of M-mode and, especially, two-dimensional echocardiographic methods to determine the presence and assess the hemodynamic severity of tamponade have been proposed (1–3). Nonetheless, the diagnosis of cardiac tamponade after cardiac surgery remains a difficult problem (4). Recently, it was demonstrated that during cardiac tamponade there is an inspiratory increase in velocity through the tricuspid and pulmonary valves and a decrease in velocity through the mitral and aortic valves measured by Doppler echocardiography, which is termed, somewhat inappropriately, “flow velocity paradoxus” (5,6). However, the specificity and clinical value of this sign have not been determined. Accordingly, we describe a patient with a postoperative pericardial effusion and severe lung disease who demonstrated flow paradoxus that was respiratory, not pericardial, in origin.

Case Report

A 66 year old Caucasian man who has severe chronic lung disease required prednisone for control of symptoms. He underwent a four vessel coronary artery bypass graft operation on March 11, 1985. Two weeks later, he complained of severe dyspnea and orthopnea, but had no chest pain. The blood pressure was 150/70 mm Hg with 35 mm Hg of pulsus paradoxus. The pulse rate was 110 beats/min, the temperature 102°F and the respiratory rate 28/min. Sternocleidomastoid retractions obscured the jugular venous pulse, but the carotid pulsations were brisk. The patient's breath sounds were diminished, particularly in the left lung base. Expiration was prolonged and diffuse wheezes were heard. The apical cardiac impulse was not displaced and the heart sounds were normal. A fourth heart sound, but no pericardial friction rub, was present. The liver was not enlarged; there was no edema. The electrocardiogram confirmed sinus tachycardia and showed low voltage and poor R wave progression across the precordial leads. A lingular infiltrate was noted on the chest radiogram. Arterial blood gas analysis while the patient was receiving 3 liters of oxygen revealed pH 7.44, partial pressure of oxygen 76 torr and partial pressure of carbon dioxide 34 torr.

Doppler echocardiographic and cardiac catheterization findings. Real-time echocardiography demonstrated a moderate-sized anterior pericardial effusion. There was no right ventricular diastolic collapse or right atrial compression. During inspiration, the right ventricular dimension increased from 1.4 to 3.1 cm, the ventricular septum bowed to the left and the left ventricular dimension decreased from 5.9 to 4.7 cm. Also during inspiration, mitral flow velocity (Fig. 1A) dropped from 95 to 55 cm/s (a decrease of 42%) and tricuspid flow velocity (Fig. 2A) increased from 55 to 90 cm/s (an increase of 64%).

Right heart catheterization was performed to exclude cardiac tamponade. The right atrial pressure was 6 mm Hg; the waveform showed a normal y descent. The right ventricular pressure was 28/7 mm Hg, the pulmonary artery pressure 25/15 mm Hg and the pulmonary artery wedge pressure 9 mm Hg. The arteriovenous oxygen difference was 4.8 vol% and a thermodilution cardiac output was 7.0 liters/min. After vigorous treatment with prednisone and
intravenous theophylline, symptoms decreased and pulsus paradoxus fell to 15 mm Hg. Another Doppler echocardiographic study (Fig. 1B and 2B) showed a reduction in respiratory variation of both mitral (from 85 to 60 cm/s) and tricuspid (from 60 to 80 cm/s) flow velocities, but no change in the pericardial effusion.

**Comments**

**Respiratory variations in transvalvular flow and velocity.** This is the first report of which we are aware of transvalvular flow (velocity) alteration associated with pulsus paradoxus of respiratory origin. The clinical importance of this case is that the presence of a pericardial effusion and a paradoxical pulse made the clinically crucial distinction between exacerbation of chronic lung disease and perioperative tamponade difficult. Although respiratory variation in flow velocity across the atrioventricular and semilunar valves may occur in pulsus paradoxus because of cardiac tamponade, our case is the first to demonstrate that the same respiratory variation in flow velocity also occurs in pulsus paradoxus of respiratory origin. The changes we observed are consistent with the findings of Pandian et al. (6). Working with a canine model of cardiac tamponade, they found a 17 ± 2% inspiratory increase in tricuspid flow velocity during the control period, and a 117 ± 19% increase during cardiac tamponade. The inspiratory decrease in mitral flow velocity was 10 ± 2% during the control period, and 42 ± 3% during tamponade.

Whereas Doppler-determined flow velocity correlates well with volume flow, estimates of volume flow require measurement of the cross-sectional area of the flow channel (7). Although we did not measure annular diameter or flow area, our findings and those of others (8) that left ventricular dimension decreases during inspiration suggest that trans-mitral volume flow was reduced and that this reduction might be underestimated by velocity tracings alone. Similarly, both the velocity across the tricuspid valve and the dimension of the right ventricle increased during inspiration, indicating augmented flow.

**Mechanisms of pulsus paradoxus.** A paradoxical pulse may result from a combination of several mechanisms. Posterior ventricular septal bulging and competition between the two ventricles for a reduced intrapericardial space are thought to be the primary mechanisms causing pulsus paradoxus in cardiac tamponade (9). Impedance to left ventricular ejection (10) and impairment of left ventricular filling resulting from a variety of mechanisms play lesser roles (11,12).

The genesis of pulsus paradoxus in obstructive lung disease is more controversial. Shabetai et al. (13) stated that pulse pressure and left ventricular stroke volume do not decrease abnormally in patients with obstructive lung disease who have pulsus paradoxus or in animals with experi-

![Figure 1. Mitral flow velocity before (PRE) (A) and after (POST) (B) treatment of severe chronic lung disease. Note the prominent A wave on the velocity tracing during inspiration (Insp.). See text for details.](image-url)
Figure 2. Tricuspid flow velocity before (A) and after (B) treatment of severe chronic lung disease. Note that early (E wave) filling remains dominant throughout the respiratory cycle. See text for details. Abbreviations as in Figure 1.

imental tracheal obstruction. Studies of the effect of intrathoracic pressure of left ventricular performance using Valsalva and Mueller maneuvers have suggested that negative intrathoracic pressure increases left ventricular transmural pressure, resulting in a reduced ejection fraction (14). However, the paradoxical pulse in patients with severe asthma may not be explicable merely by the direct transmission of intrapleural pressure changes. Rather, it also results from the interplay of enhanced venous return to the right heart chambers and impedance to right ventricular ejection during inspiration, leading to reduced left heart filling and an exaggerated inspiratory decrease in arterial pressure.

Settle et al. (15) obtained echocardiograms in nine patients with chronic obstructive lung disease who had pulsus paradoxus. M-mode echocardiograms obtained with the patient in the subxiphoid position showed the average dimension of the right ventricle during inspiration to be $2.96 \pm 0.38$ cm and during expiration to be $1.4 \pm 0.2$ cm. The average dimension of the left ventricle was $3.7 \pm 0.63$ and $4.8 \pm 0.61$ cm during inspiration and expiration, respectively. The mean dimensional difference between inspiration and expiration was $1.56$ and $1.1$ cm for the right and the left ventricle, respectively. This contrasted with a failure to demonstrate abnormal inspiratory or expiratory ventricular dimension changes in a group of normal volunteers. The findings of Settle et al. (15) are similar to the differences we observed in dimension during respiration of $1.7$ and $1.2$ cm for the right and the left ventricle, respectively. In a small subset of their patients whose left ventricular ejection time was measured, calculated left ventricular stroke volume diminished with inspiration. Alteration of left ventricular shape by inspiration reduces left ventricular compliance (16), explaining the shift we observed to a late diastolic filling pattern (A wave dominant) during inspiration. This mechanism may explain in part the limitation of mitral inflow during inspiration.

Although previous studies have speculated on the mechanism of pulsus paradoxus in chronic lung disease, respiratory alteration in stroke volume and ventricular filling have been implied from respiratory variation in ventricular dimensions (17), aortic and pulmonary artery pressures (12) or left and right ventricular ejection times (12,17), but were not directly measured.

Conclusions. In this case of chronic lung disease, decreased filling of the left ventricle rather than transmission of declining intrapleural pressure was responsible, at least
in part, for pulsus paradoxus. Thus, transvalvular flow velocity paradoxus is nonspecific for cardiac tamponade, but is observed in pulsus paradoxus of chronic lung disease.

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References


