Echocardiographic Observations of Paradoxic Pulse Without Pericardial Disease*

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Echocardiograms were obtained in 10 normal patients and in 11 patients with respiratory insufficiency due to chronic obstructive lung disease (8) and to thoracic poliomyelitis (3). Only the eight patients with obstructive lung disease had paradoxic pulse. No patient had pericardial disease. The left ventricular internal dimension, stroke volume, and the mitral valve E-F slope and D-E excursion were measured. The inspiratory to expiratory ratio of each measurement was significantly lower in patients with obstructive lung disease than in normal subjects.

The patients with thoracic poliomyelitis demonstrated almost no respiratory change in these measurements. The magnitude of the change in the measured factors probably relates to the degree of negativity of intrathoracic pressure during respiration. The inspiratory reduction of mitral valve motion and left ventricular internal dimension is not specific to tamponade but may be seen in patients who exhibit paradoxic pulse due to other conditions.

Paradoxic pulse is a valuable sign in the clinical recognition of cardiac tamponade. This sign may also be present in several respiratory conditions associated with an exaggerated inspiratory effort. To evaluate the mechanism responsible for paradoxic pulse in patients without pericardial disease, the current study was performed.

METHODS

Fifty patients with chronic respiratory insufficiency were screened during quiet respiration for the presence of an inspiratory fall of the systolic arterial pressure by at least 10 mm Hg. Eight patients met these criteria and underwent echocardiography. Four of these patients were also studied with a sector scanner. Patients were studied during quiet respiration in the supine position with the transducer in the third or fourth left intercostal space. Each of the eight patients had typical historic, physical, and radiologic findings of chronic obstructive pulmonary disease. In addition, pulmonary function tests revealed reduction of FEV1 and maximal mid-expiratory volumes and an increase in the ratio of residual volume to total lung capacity. In six of eight patients, a clinical diagnosis of cor pulmonale was made. The presence of paradoxic pulse during performance of the echocardiograms was evaluated by cuff manometry in each patient and confirmed by arterial pulse tracings in six patients and by direct arterial measurement in two patients. Femoral arterial pulse recordings were employed to avoid the distortion introduced into carotid pulse tracings during use of the cervical accessory muscles of respiration. Respiration was recorded in each patient. Three patients with respiratory insufficiency due to thoracic poliomyelitis were similarly studied. Ten patients without lung disease or paradoxic pulse served as controls. No patient had clinical or echocardiographic evidence of pericardial disease. Two of the eight patients with obstructive lung disease, both of whom had cor pulmonale, died one day and one week after echocardiographic study. Autopsy revealed right ventricular dilatation, emphysema, and no evidence of pericardial disease in either patient.

To record the left ventricular internal dimension, the mitral valve was first identified. The transducer was then angled towards the apex, and the left ventricular internal dimension was measured at the point at which the mitral valve echoes disappeared. Systolic left ventricular internal dimension (LVIDs) was measured at the point of closest approximation of the left ventricular walls. Diastolic left ventricular dimension (LVIDd) was measured at the peak of the QRS complex from a simultaneously recorded ECG. The stroke volume was calculated by subtracting the cubed LVIDs from the cubed LVIDd. Each variable was measured at peak inspiration and peak expiration, and the ratio between the inspiratory and expiratory measurements was calculated. For example, Figure 1 indicates the technique used for calculation of the ratio of inspiratory to expiratory diastolic left ventricular internal dimension. If the ratio equals 1.0, there is no respiratory variation of the factor being evaluated. If the ratio is greater than 1, inspiration causes an increase of the measurement; finally, a ratio less than 1.0 indicates an inspiratory reduction. Ratios were employed to permit comparison of patients with varying chamber size and degrees of valvular motion. Each reported value represents the average of measurements taken during peak inspiration and end expiration from each of five respiratory cycles. Pericardial effusion was searched for by the technique of Feigenbaum.1 Statistical analysis was performed using Student's t-test.

RESULTS

No patient in this study had echocardiographic evidence of pericardial effusion. Respiratory rate ranged from 10 to 20 per minute. Each patient with

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CHEST, 78: 3, SEPTEMBER, 1980
paradoxic pulse demonstrated a marked inspiratory decrease in left ventricular internal dimension and a reciprocal increase in right ventricular internal dimension. Right ventricular dilatation (right ventricular internal dimension greater than 2.4 cm) was present in six patients. During each inspiration, the interventricular septum displayed exaggerated posterior displacement (Fig 2). The reduction in LVIDd and LVIDs occurred with a reduction of the peripheral pulse. The ratio of LVIDd in expiration

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\frac{D_{d_{\text{ins}}}}{D_{d_{\text{exp}}}} = 0.74
\]

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\frac{SV_{\text{ins}}}{SV_{\text{exp}}} = 0.37 = 0.43
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**Figure 1.** Echocardiogram of patient with obstructive lung disease and paradoxic pulse; note absence of pericardial effusion and diminution of left ventricular internal dimension occurring simultaneously with decrease in external arterial blood pressure tracing. Measurement of left ventricular dimensions is demonstrated and resultant stroke volume is indicated. Dd indicates ventricular internal diastolic dimension; Ds, left ventricular internal systolic dimension; EXP, expiration; INS, inspiration; RV, right ventricle; and SV, stroke volume. Vertical intervals are 10 mm. Horizontal intervals are 0.5 second. Stroke volume is expressed in square centimeters.

**Figure 2.** Low speed recording of right and left ventricles. An indirect arterial pulse tracing is recorded simultaneously. Arrows indicate inspiration. Diminution of left ventricular internal dimension occurs during inspiration and is associated with a transient fall in blood pressure. Abbreviations similar to Figure 1.
to LVIDd in inspiration was 0.4 to 0.81 (mean 0.73) in patients with paradoxical pulse as opposed to 0.77 to 1.0 (mean 0.91) in control patients (Fig 3A). This is a significant difference with a $P < 0.001$. The ratio of inspiratory stroke volume to expiratory stroke volume (Fig 3B) was 0.21 to 0.84 (mean 0.56) in patients with paradoxical pulse and 0.72 to 1.0 in controls (mean 0.91), $(P < 0.001)$. These observations were confirmed in four patients by sector scanning.

To assess the possible correlation between changes in stroke volume and the magnitude of the paradoxical pulse, these factors were plotted against one another (Fig 4). In patients with paradoxical pulse, there was a correlation between its severity, and the degree of inspiratory reduction of stroke volume. Control patients had little or no respiratory alteration of stroke volume. Patients with pulmonary insufficiency due to thoracic poliomyelitis had no respiratory change in either systolic pressure or stroke volume. A ratio of inspiratory to expiratory stroke volume that was less than 0.75 generally correlated with the presence of paradoxical pulse. Seven of eight patients with paradoxical pulse had an expiratory to inspiratory stroke volume ratio that was less than 0.75. In only one of ten control patients was this ratio less than 0.75. Mitral valve E-F slope (Fig 3C) and mitral valve D-E excursion (Fig 3D and 5) decreased during inspiration. The ratio of mitral valve E-F slope in inspiration to the mitral E-F slope in expiration was 0.37 to 0.73 (mean 0.58) in patients with paradoxical pulse and 0.73 to 0.96 (mean 0.87) in control patients $(P < 0.001)$. Tricuspid valve recordings were made in six of eight patients with paradoxical pulse. A representative example is presented in Figure 6. The respiratory change in the tricuspid valve E-F slope was the inverse of that observed for the mitral valve (Fig 3E). This value increased markedly during inspiration in patients with paradoxical pulse. The three patients with respiratory failure due to thoracic poliomyelitis demonstrated right ventricular dilatation and absence of respiratory alteration in left ventricular internal dimension.

![Figure 3](image-url) Inspiratory to expiratory ratio of each factor is displayed for controls and patients. Mean and two standard deviations are displayed for each group. See text for discussion of results. LVIDd indicates diastolic left ventricular internal dimension; MV, mitral valve; pp, patients with paradoxical pulse; Stroke Vol, stroke volume; and TV, tricuspid valve.

![Figure 4](image-url) Inspiratory reduction of systolic blood pressure is plotted against the ratio of inspiratory to expiratory stroke volume in control patients with obstructive lung disease (old), and those with thoracic poliomyelitis (polio). Values below horizontal dashed line are within range of normal inspiratory reduction of systolic blood pressure. A ratio of inspiratory to expiratory stroke volume less than 0.75 (vertical dashed line) correlates with the presence of paradoxical pulse.

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DISCUSSION

Paradoxic pulse is defined as an inspiratory reduction of the systolic blood pressure by 10 mm Hg or more. Paradoxic pulse occurs in several pathologic conditions, including cardiac tamponade, constrictive pericarditis, pulmonary embolization, chronic obstructive lung disease, and other forms of airway obstruction. A variety of hemodynamic hypotheses have been advanced to explain the pathophysiology of paradoxic pulse, indicating the

![Figure 5](http://publications.chestnet.org/pdfaccess.ashx?url=data/journals/chest/21170/)

**Figure 5.** Mitral valve echocardiogram demonstrates inspiratory reduction of mitral valve D-E excursion associated with reduction of left ventricular internal dimension and blood pressure. MV EXC indicates DE excursion of mitral valve.

![Figure 6](http://publications.chestnet.org/pdfaccess.ashx?url=data/journals/chest/21170/)

**Figure 6.** Inspiratory increase of the tricuspid valve E-F slope is displayed. TV is tricuspid valve.
absence of a uniformly accepted explanation for this phenomenon. Expansion of the chest produces a transient negative intrathoracic pressure. This causes an increase in systemic venous return and right ventricular stroke volume. Dornhorst et al\(^8\) speculated that the normal, small reduction of systemic blood pressure during inspiration might be due to a temporal delay in transmission of the inspiratory augmentation in right-sided heart output through the pulmonary circulation to the left side of the heart. Depending on the respiratory rate, the drop in arterial pressure could be induced either with inspiration or expiration. However, Ruskin et al\(^8\) demonstrated that inspiration after a period of apnea produced an immediate fall in blood pressure and left ventricular stroke volume. Shabetai et al\(^8\) found that in experimental tamponade, the aortic pressure fell when systemic venous return was increased during apnea. This finding suggested that the inspiratory increase in right ventricular volume resulted in encroachment upon the left ventricle, producing a reduction of left ventricular stroke volume. Guntheroth et al\(^10\) subsequently demonstrated that in tamponade, the left and right ventricular stroke volumes were not wholly reciprocal and that their sum was not constant throughout the respiratory cycle. They concluded that "almost all the changes of pulsus paradoxus reflect the normal respiratory effects on the right ventricular stroke volume delayed by transit to the pulmonary bed and exaggerated by the small left ventricular stroke volume in a vasoconstricted state."\(^10\) While the direct effect of the respiratory reduction of intrathoracic pressure cannot be excluded as a contributing factor to the reduction of the arterial pressure, the studies quoted suggest that many other factors are involved in this relationship of respiration and blood pressure.

D'Cruz et al\(^11\) and Vignola et al\(^12\) reported that patients with pericardial effusion and tamponade demonstrate inspiratory reduction in left ventricular internal dimension and in mitral valve excursion and early diastolic velocity. Such changes were also reported in a patient with pulmonary embolism\(^6\) and in two patients with chronic obstructive lung disease.\(^6\) To assess the pertinence of the echocardiographic obstructions in the setting of cardiac tamponade to other conditions associated with paradoxical pulse, we examined patients with paradoxical pulse but no pericardial effusion.

Systemic venous return and transvalvular tricuspid flow increase and pulmonary venous return and transvalvular mitral flow decrease during inspiration. These changes occur in normal individuals, but become marked when there is an exaggerated fall of intrathoracic pressure as seen in patients with obstructive lung disease. There appears to be a spectrum in the magnitude of the cyclic respiratory changes in left ventricular stroke volume and systemic blood pressure. While not confirmed by actual measurements, it appears likely that these changes relate to the degree of negativity of intrathoracic pressure during respiration. At one end of the spectrum there are individuals with impairment of thoracic musculoskeletal function, such as patients with thoracic poliomyelitis. They are unable to develop normally negative intrathoracic pressure. At the other extreme are patients with respiratory distress due to chronic obstructive lung disease. These patients have greater than normal falls in intrathoracic pressure during inspiration and also have greater than normal falls in systolic arterial pressure and left ventricular stroke volume. Brenner and Waugh\(^18\) have shown that inspiration in normal individuals produces small, but significant reductions of left ventricular diastolic dimension and stroke volume. The mechanism responsible for the fall in pulmonary venous return may be different in patients with tamponade and those with lung disease. For example, Golinko et al\(^14\) demonstrated an inspiratory reduction in the pressure gradient between the pulmonary veins and the left atrium during experimental tamponade. This finding may not be applicable to patients without tamponade.

In the present study, reproducible echocardiographic abnormalities were observed in each patient with paradoxical pulse. Inspiratory reduction of systemic pressure was associated with simultaneous decreases in LVIDd, left ventricular stroke volume, mitral valve E-F slope, and mitral valve D-E excursions. Mitral valve E-F slope has been previously correlated with the rate of left ventricular filling\(^16\) and the D-E excursion with left ventricular stroke volume.\(^16\) The cyclic respiratory changes in left ventricular size, and the inspiratory decrease of flow across the mitral valve and stroke volume are the hemodynamic correlates of paradoxical pulse. The absence of pericardial effusion in these patients excludes cardiac tamponade. The measurement of systemic venous pressure will be helpful in the patient with paradoxical pulse in whom there is coexistent respiratory insufficiency and pericardial effusion. The central venous pressure should be increased with cardiac tamponade but may be normal with airway disease in the absence of right ventricular failure.

**Conclusion**

Echocardiograms in eight patients with paradoxical pulse, but without pericardial disease, demonstrated an inspiratory reduction of diastolic left ventricular
internal dimension, left ventricular stroke volume, mitral valve E-F slope, and mitral valve D-E excursion and an inspiratory increase of right ventricular internal dimension and the tricuspid valve E-F slope. These changes are similar to those previously reported in patients with cardiac tamponade. We therefore suggest that these echocardiographic observations are correlates of paradoxical pulse of any etiology. Cardiac tamponade remains a clinical diagnosis and should not be made solely on the basis of echocardiographic findings. Patients with respiratory distress and moderate pericardial effusion may present with echocardiographic observations which are indistinguishable from those previously reported as indicative of cardiac tamponade.

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