Echocardiographic Study of the Paradoxical Arterial Pulse in Chronic Obstructive Lung Disease

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SUMMARY In nine subjects with chronic obstructive pulmonary disease (COPD) and pulsus paradoxus, M-mode echocardiograms showed inspiratory augmentation of right ventricular dimensions and inspiratory decrease of left ventricular diastolic dimensions. In five subjects in whom the echocardiographic transducer was in the subxiphoid position, mean right ventricular dimensions increased during inspiration from 1.4 ± 0.20 to 2.96 ± 0.38 cm (p < 0.01). With inspiration, mean left ventricular diastolic dimensions decreased from 4.8 ± 0.61 to 3.7 ± 0.63 cm (p < 0.01) in these five subjects. Two-dimensional echocardiograms, performed in three subjects, confirmed inspiratory augmentation of right ventricular cross-sectional area. Similar changes were produced in two normal volunteers by artificial obstruction to breathing. Left ventricular ejection time measurements demonstrated an inspiratory decline in left ventricular stroke volume.

Inspiratory filling of the right ventricle is not hampered, but rather is exaggerated in patients with COPD and pulsus paradoxus, and left ventricular stroke volume is reduced during inspiration. Exaggerated variations in intrathoracic pressure alone did not explain pulsus paradoxus. Increased right ventricular filling and stroke volume during inspiration probably play a part.

Subjects

Nine subjects with severe chronic airway disease and a paradoxical pulse were studied by echocardiography. These patients included all patients with chronic obstructive pulmonary disease and a paradoxical pulse found at a pulmonary clinic, during a 3-year period. Patients were included if a satisfactory echocardiogram for measurement of ventricular dimensions could be recorded. The clinical diagnosis of chronic obstructive lung disease was based on evidence of arterial blood oxygen undersaturation, clinical evidence of hyperinflated lungs, and, in eight of the nine, confirmation by pulmonary function studies (table 1). Eight of nine patients had had episodes of right-sided cardiac failure, manifested by increased cervical venous pressure, pedal edema and fourth heart sounds audible along the left lower sternal edge or in the epigastrium.

We also studied two normal young men, ages 24 and 28 years. These volunteers' echocardiograms were studied before, during and after induced airway obstruction.

Methods

Measurements of forced expiratory flow were made with a Collins 9-liter spirometer in the standard manner. Lung volumes were calculated from rebreathing helium-containing gas mixtures with the Collins bag-in-box system.

The standard echocardiographic examination was performed with a commercially available ultrasonoscope (Smith Kline Instruments, Ekoline 20) interfaced to either a Honeywell 1856 visicorder strip-chart recorder or an Irex multichannel Continutrace 101 recorder. A 0.5-inch diameter, 2.25-MHz trans-
A transducer focused at 7.5–10 cm with a repetition rate of 1000/sec was used. The patients were examined in the supine or semi-erect position. The transducer was positioned at the fourth intercostal space adjacent to the left sternal border or in the subxiphoid area, and the standard sweeps from the aorta to the left ventricle were obtained. Starling et al.\textsuperscript{7} reported that right ventricular volume and ejection fraction could be accurately evaluated in patients with chronic obstructive airway disease by subxiphoid echocardiography. Systolic septal motion and left ventricular dimensions, as measured by the subxiphoid transducer, showed good correlation with those measurements obtained with the transducers in the standard left parasternal position.\textsuperscript{8}

An electrocardiographic lead was recorded during each study. Instantaneous respiratory wave forms were recorded simultaneously with a tungsten wire respirometer\textsuperscript{9} from patients studied with the Irex Continutrace recorder. In those studied with the aid of the Honeywell recorder, the phases of respiration were signaled by appropriate manipulation of the baseline of the electrocardiographic tracing. Measurements were made during both inspiration and expiration of right and left ventricular internal diastolic and systolic diameter, and interventricular septal motion was evaluated. Patients with chronic obstructive airway disease and pulsus paradoxus in whom these measurements could not be made were not included in this study.

The left ventricular internal diameter during diastole was measured as follows: With the patient in a supine position, measurements were made at the level of the chordae tendineae from the left side of the interventricular septum to the endocardial surface of the left ventricular posterior wall. Diastole was defined by the initial inscription of the electrocardiographic QRS complex, and systole was defined by the smallest vertical septal–posterior wall endocardial distance. Minimal inspiratory values and maximal expiratory values were averaged for three to five respiratory cycles.

Right ventricular measurements were made as follows: The ultrasound beam was directed along the course of the left ventricular endocardial surface, the right and left sides of the interventricular septum, the anterior and posterior mitral leaflets, and the endocardial and epicardial surfaces of the left ventricular posterior wall. The measurements were made from the right ventricular endocardial surface to the right ventricular side of the septum at end-diastole.\textsuperscript{10} The dimension measurements were reported only when structures defining the dimensions were recorded clearly. Maximal inspiratory and minimal expiratory values were averaged for three to five respiratory cycles.

The reliability of left and right ventricular dimension measurements in our echocardiographic laboratory was estimated from the coefficient of variation (CV) (standard deviation divided by mean). Intraobserver CV for left ventricular diastolic dimension was 4.3%, for left ventricular systolic dimension was 4.1%, and for right ventricular dimension was 11.3%. Interobserver CV for left ventricular diastolic dimension was 0.7%, for left ventricular systolic dimension was 2.9%, and for right ventricular dimension was 16.2%.

Cross-sectional echocardiograms were performed in three of the nine subjects was a commercially available mechanical sector scanner (Ekosector I, Smith-Kline Instruments), which uses a 2.25-MHz transducer swept through a 30° sector at 30 Hz. Adequate real-time two-dimensional visualization of the heart could be obtained only by using a subxiphoid window in two of the three subjects so studied. Images were recorded on videotape using a Sanyo VTC–7100 cassette recorder. Polaroid photographs were obtained from individual video tape frames at the same time in each cardiac cycle, as judged from the simultaneously recorded ECG.

### Table 1. Pulmonary Function Tests in Patients with Chronic Obstructive Airway Disease

<table>
<thead>
<tr>
<th>Pt</th>
<th>Inspiratory decline of systolic blood pressure (mm Hg)</th>
<th>TLC (% pred)</th>
<th>RV (ml BTPS)</th>
<th>RV (% pred)</th>
<th>RV TLC (%)</th>
<th>FEV-1 (ml BTPS)</th>
<th>FRC (ml)</th>
<th>FRCP (% pred)</th>
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<td>86</td>
<td>3298</td>
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<td>1400</td>
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<td>45</td>
<td>1300</td>
<td>1479</td>
<td>54</td>
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</table>

Abbreviations: TLC = total lung capacity; pred = predicted; RV = residual volume; BTPS = volume corrected to body temperature, and standard atmospheric pressure; FEV-1 = 1-second forced expiratory volume; FRC = functional residual capacity; FRCP = functional residual capacity, percent of predicted.
Left Ventricular Ejection Times

In four subjects, left ventricular ejection time (LVET) was measured by ear densitography.11-18 The densitograph11 is a photoelectric plethysmograph, attached to the pinna of the ear, that is sensitive to variations in the amount of blood in the tissues between a light source and a photoconductive cell. These variations produce proportional changes in light reaching the photocell; the consequent variation in the resistance of the photocell results in a pulse curve that correlates well with ejection times derived from carotid arterial pulse records.18, 18

In our laboratory, in five normal subjects, the correlation coefficient between the two methods of measuring LVET was 0.97 when the first derivative of the ear densitogram was used. LVET has been found to correlate well with changes in left ventricular stroke volume during atrial fibrillation,14 complete atrioventricular block,15 or during head-up tilting.18

In normal subjects, external obstruction to inspiration and to expiration was produced as follows: The subjects breathed through a double Douglas valve while the nose was completely obstructed with a flexible nosepiece. Varying degrees of inspiratory and expiratory obstruction were produced by adjusting C clamps placed around 2.5-cm diameter plastic tubing attached to the inspiratory and expiratory ports of the double Douglas valve. The degree of obstruction was estimated by measuring instantaneous pressure within the double Douglas valve by means of a Statham P23d gauge. These pressure variations, together with the echocardiogram and the densitogram, were recorded with the Irex Continutrace recorder. Inspiratory and/or expiratory obstruction was increased gradually as arterial blood pressure was measured by cuff manometry. Respiratory obstruction was increased until an inspiratory fall of systolic blood pressure of 10 mm Hg or more (pulsus paradoxus) was produced. Subjects then remained at a steady degree of airway obstruction for 5 minutes or more while echocardiograms and densitograms were recorded.

We tested the relationship between respiratory changes in LVET and in systolic blood pressure in normal subjects by means of the correlation coefficient $r_{xy}$. We also evaluated the relationship between respiratory changes in echocardiographic left and right ventricular dimensions and respiratory changes in systolic blood pressure by means of the correlation coefficient. Respiratory changes in LVET were evaluated by $t$ test.

Results

Arterial Blood Gases

Arterial blood PO$_2$ values were 28–64 mm Hg partial pressure (mean 44 mm Hg). The PCO$_2$ values were 26–60 mm Hg (mean 46 mm Hg). The arterial blood pH values were 7.32 –7.57 (mean 7.41). The degree of paradoxical pulse did not correlate with the level of PO$_2$ or PCO$_2$.

Ventilatory Studies

Pulmonary function studies were obtained in eight of the nine patients (table 1). All patients had a paradoxical arterial pulse at the time of this study. The residual lung volume was above 3200 ml except in patient 9, who had evidence of both restrictive and obstructive lung disease. The residual volume was increased in each, except in patient 9. The residual volume–total lung capacity ratio was 45–75% (normal 25–35%). The functional residual capacity (FRC) was 180% of predicted value or above, except in patient 9. The 1-second forced expiratory volume was 517–1400 ml (normal, minimum of 3 l).

Electrocardiograms

ECGs revealed evidence of right atrial enlargement in four of the nine patients.

Five of the nine patients had a rightward QRS axis exceeding 90° in the frontal plane, and seven of nine had an S wave in lead V$_6$ equal to or greater than the R wave in the same lead. Only patient 7 did not have any of these findings. Two patients had electrocardiographic evidence of right ventricular hyper trophy in lead V$_1$.

Echocardiographic Findings

Every patient demonstrated enlargement of the right ventricular internal dimension in inspiration (range 2–4 cm) (normal 1.9 ± 0.14 cm)$^9$ (fig. 1, table 2). In five patients studied with the transducer in the subxiphoid position, the average inspiratory dimensions of the right ventricle was 2.96 cm. The average expiratory diameter of the right ventricle was 1.4 cm. The mean difference between inspiration and expiration was 1.56 cm ($p < 0.01$). The left ventricular internal diastolic dimension in inspiration was 3.7 cm, and in expiration was 4.8 cm. The mean difference of 1.1 cm was statistically significant ($p < 0.01$). The systolic diameter in inspiration averaged 2.4 cm and in expiration 3.1 cm, with a difference in inspiration and expiration of 0.74 cm ($p < 0.02$) (table 2).

The two-dimensional echocardiograms obtained in three of the nine subjects with chronic obstructive airway disease and pulsum paradoxus showed inspiratory augmentation of the right ventricular cross-sectional area and inspiratory diminution of the left ventricular cross-sectional area, (figs. 2 and 3).

Artificially Induced Paradoxical Arterial Pulse

The control record with the normal volunteer breathing through the apparatus without any inspiratory or expiratory obstruction failed to demonstrate abnormal inspiratory or expiratory ventricular dimension changes by echocardiogram (fig. 4). In adults, we found no more than 2 mm inspiratory augmentation of right ventricular dimensions.$^5$ Feigenbaum found no respiratory change in left ventricular dimension exceeding 2–3 mm.$^7$ When 8 mm Hg of inspiratory and expiratory resistance was
measured by the mouthpiece, 14 mm Hg of paradoxical pulse was produced. With 20 mm Hg of inspiratory and 15 mm Hg expiratory resistance, 24 mm Hg of paradoxical arterial pulse was produced (fig. 5). The echocardiogram recorded during pulsus paradoxus thus revealed right and left ventricular dimensional changes similar to those reported in cardiac tamponade, and similar to those observed in our patients with chronic obstructive airway disease and a paradoxical arterial pulse. A paradoxical pulse could be produced when the inspiratory or expiratory pressure alone was −10 mm Hg or 10 mm Hg, respectively.
More important, in one subject the maximum paradoxical pulse did not appear immediately upon the institution of a given degree of expiratory obstruction, but only after 3–5 minutes. This observation suggests that increased variations in intrapleural pressure alone did not account for the increased variations in blood pressure.

In volunteer JH, the left ventricular diastolic respiratory dimension decreased 2–3 mm during inspiration in the control period. When there was pulsus paradoxus caused by airway obstruction, the left ventricular diastolic dimension decreased 4–8 mm. The relationship between inspiratory decrease in systolic blood pressure and the decline in left ventricular

**TABLE 2. Echocardiographic Measurements of Ventricular Dimensions in Nine Subjects with Chronic Obstructive Airway Disease**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Transducer position</th>
<th>RV₁ (cm)</th>
<th>RVₑ (cm)</th>
<th>LVD₁ (cm)</th>
<th>LVDₑ (cm)</th>
<th>LVS₁ (cm)</th>
<th>LVSE (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Subxi</td>
<td>2.2</td>
<td>1.5</td>
<td>2.4</td>
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<td>Subxi</td>
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<td>1.1</td>
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<td>2.1</td>
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<tr>
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<td>LSE</td>
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<tr>
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<td>—</td>
<td>3.7</td>
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<td>4.8</td>
<td>4.8</td>
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<tr>
<td>6</td>
<td>Subxi</td>
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<tr>
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<tr>
<td>8</td>
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<td>0.9</td>
<td>2.8</td>
<td>4.0</td>
<td>2.2</td>
<td>2.5</td>
</tr>
<tr>
<td>9</td>
<td>LSE</td>
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<td>1.9</td>
<td>3.0</td>
<td>3.6</td>
<td>2.0</td>
<td>2.0</td>
</tr>
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</table>

Average ± SD (subxiphoid): 2.96 ± 0.84 1.4 ± 0.44 3.7 ± 1.42 4.8 ± 1.38 2.4 ± 0.98 3.1 ± 1.22

Δ I - E ± SD (subxiphoid): 1.56 ± 0.36 1.1 ± 0.24 0.74 ± 0.19

*p < 0.01  p < 0.001  p < 0.02

Abbreviations: Subxi = subxiphoid transducer position; LSE = fourth left intercostal space transducer position; RV₁ = inspiratory right ventricular dimension; RVₑ = expiratory right ventricular dimension; LVD₁ = inspiratory left ventricular dimension; LVS₁ = inspiratory left ventricular systolic dimension; LVDₑ = expiratory left ventricular diastolic dimension; LVSE = expiratory left ventricular systolic dimension.

**Figure 2. Cross-sectional echocardiogram from same patient as in figure 1.** Views are from the subxiphoid window in expiration and inspiration during the same portion of cardiac cycle. Arrowheads indicate timing in cardiac cycle relative to previous R wave. The scanning plane is oriented along the short axis of the left ventricle (LV). The accompanying line drawings are for clarity. With expiration, the septum is slightly convex in an anterior direction. During inspiration the septum is seen to shift posteriorly and flatten. RV = right ventricle; IVS = interventricular septum.
Figure 3. Cross-sectional echocardiogram from another patient with chronic obstructive pulmonary disease and pulsus paradoxus. Arrowheads indicate timing in cardiac cycle relative to previous R wave. (A) Long-axis view of left ventricle (LV) in mid- to late diastole. Note increase in right ventricular dimension and flattening of interventricular septum (IVS) during inspiration. (B) Apical view of IVS in early systole. Note that IVS becomes concave toward the LV during inspiration. (C) Short-axis view of LV from same patient at end-diastole. During expiration, the IVS is concave toward the right ventricle (RV); during inspiration the septum is seen to flatten toward the LV. AML = anterior mitral leaflet; RA = right atrium; TV = tricuspid valve; LA = left atrium; MV = mitral valve.

Figure 4. Echocardiogram of normal control subject without obstruction to breathing. The inspiratory and expiratory changes in the double Douglas valve pressure are very small (for scale, see figure 5). Left ventricular diastolic dimension shows a minimal respiratory change (from 4.3 cm in expiration to 4.1 cm in inspiration). The right ventricular dimension increases from 1.4 cm in expiration to 1.6 cm in inspiration. Insp = inspiration; Exp = expiration;\( LV_i \) = left ventricular inspiratory diastolic dimension;\( LV_e \) = left ventricular expiratory end-diastolic dimension;\( RV_i \) = right ventricular inspiratory end-diastolic dimension;\( RV_e \) = right ventricular expiratory end-diastolic dimension.
diastolic dimension was significant \((r = 0.8546, p < 0.001)\) (fig. 6). Right ventricular dimensions increased 0–4.5 mm without airway obstruction and 5–8 mm with airway obstruction. The relationship between inspiratory fall in systolic blood pressure and inspiratory increase of right ventricular dimension was significant \((r = 0.503, p < 0.02)\). In HK, the left ventricular diastolic dimension decreased with inspiration by 2–3 mm during the control period, and by 3–11 mm when pulsus paradoxus was present during airway obstruction. The relationship between the inspiratory decrease of systolic blood pressure and the decline in left ventricular diastolic dimension was significant \((r = 0.6749, p < 0.01)\). The right ventricular dimensions increased by 3 mm or less during inspiration in the control period, and by as much as 8 mm during induced pulsus paradoxus. The relation between inspiratory right ventricular dimension increase and systolic blood pressure decline was significant \((r = 0.732, p < 0.001)\).

**LVET Variations from Ear Densitometer**

Patient EA was studied with a paradoxical pulse of 18–30 mm Hg and an LVET of 0.244 ± 0.002 (mean ± SEM) during expiration and 0.226 ± 0.003 during ins-

**Figure 6.** Scatter diagram showing correlation between inspiratory decline in systolic blood pressure and inspiratory decline in echocardiographic left ventricular dimension during artificial obstruction to breathing in a normal subject (JH). Insp OBS = inspiratory obstruction; Exp OBS = expiratory obstruction.
spiration. The difference was significant statistically ($p < 0.001$). Patient CM, studied while he had a paradoxical pulse of 10 mm Hg, had an LVET of 0.252 ± 0.002 second in expiration and 0.236 ± 0.002 second in inspiration. The difference was significant ($p < 0.001$). Control subject JH had an LVET of 0.290 in inspiration and 0.300 second in expiration when he had no airway obstruction. When the paradoxical pulse was 34 mm Hg, with mouthpiece pressure during obstruction varying from −10 mm Hg to 10 mm Hg, he had an LVET during inspiration of 0.255 second and 0.305 second in expiration. The relationship between the inspiratory decrease in systolic blood pressure and the inspiratory decrease in LVET was significant ($r = 0.8016, p < 0.001$). Subject HK showed a control LVET in inspiration of 0.295 second and LVET in expiration of 0.305 second. With a pulsus paradoxus of 22 mm Hg, his LVET during inspiration was 0.265 second and 0.305 second in expiration. The relationship between the inspiratory decrease in systolic blood pressure and the inspiratory decline in LVET was significant ($r = 0.8776, p < 0.001$).

### Discussion

Several investigators have studied the causes of the paradoxical arterial pulse in severe asthma and chronic airway disease.

The paradoxical pulse has been documented as a valuable indicator of the severity of the obstructive process in asthma. The appearance of paradoxical pulse in patients with chronic lung disease or asthma correlates best with a decrease in the 1-second forced expiratory volume. Reubuck and Pengelly observed a paradoxical pulse in normal subjects after they breathed for 3½–5 minutes through a resistance circuit. The paradoxical pulse was detected when the rise in functional residual capacity exceeded the resting level by 54–78%. When subjects voluntarily breathed at high lung volumes with no airway obstruction, no abnormality was detected in blood pressure. Thus, they postulated that the paradoxical pulse of severe asthma and chronic lung disease is produced by pulmonary overdistention and high intrapulmonary pressure, and that right heart filling was impeded. During the inspiratory phase, a sharp inspiratory drop in pulmonary venous pressure was believed to further hamper filling of the left heart.

Investigations of the mechanism of paradoxical pulse in airway obstruction have concentrated upon changes in left ventricular stroke volume and changes in intrathoracic pressure.

Ruskin et al. found an average of 25% inspiratory decrease in left ventricular stroke volume in eight patients with airway obstruction and paradoxical pulse. Shabetai et al. found that in anesthetized dogs, the paradoxical pulse associated with airway obstruction created by partially occluding an endotracheal tube was not accompanied by abnormal respiratory variations of left ventricular stroke volume. These investigators postulated that increased respiratory variations in intrathoracic pressure transmitted to the left ventricle and aorta were the chief cause of pulsus paradoxus in their experiments. Shabetai et al. also found that brachial artery blood velocity in a patient with chronic airway obstruction did not vary as much as arterial pressure. Lange and Tsagaris, on the other hand, using arterial pulse pressure as an index of left ventricular stroke volume in eight patients with a paradoxical pulse caused by airway obstruction, concluded that there was a brief inspiratory decrease in left ventricular filling caused by increased pulmonary vascular storage of blood during inspiration. Ruskin et al. observed in eight patients with airway obstruction and in normal patients no definite overshoot in left ventricular stroke volume after the inspiratory fall. Thus, they could not find a definite role of right ventricular stroke volume changes as a cause of the paradoxical pulse.

Our studies suggest that there is decreased inspiratory filling of the left ventricle in patients with pulsus paradoxus and chronic obstructive airway disease. Not only were echocardiographic left ventricular dimensions smaller in inspiration, but the significant decrease of inspiratory LVET was consistent with an inspiratory decrease of left ventricular stroke output.

Although an inspiratory increase in left ventricular impedance might alter LVET, this factor should have prolonged LVET rather than decreasing it, as in our study. Increased right ventricular dimensions during inspiration imply that inspiration did not decrease right ventricular filling.

In normal subjects with induced airway obstruction, increased respiratory variations in right and left ventricular diameters were always found when systolic blood pressure fell more than 10 mm Hg during inspiration. In these experiments also, the right ventricular dimensions increased during inspiration, while left ventricular dimensions fell. The progression of pulsus paradoxus and of inspiratory decline of LVET without further respiratory changes in ventricular dimensions suggested the operation of an additional mechanism when pulmonary functional residual capacity began to rise.

Inspiration increases right-heart filling, so what is the normal effect of inspiration upon echocardiographic ventricular dimensions? In adults we found no more than 2 mm of inspiratory augmentation of right ventricular dimension, and Feigenbaum found no respiratory change of left ventricular dimension greater than 2–3 mm.

Brenner and Waugh studied 30 normal subjects with echocardiography during phasic respiration, and found that the left ventricular end-diastolic dimension decreased by 0–13% with inspiration. The inspiratory decrease of left ventricular dimensions in our nine patients averaged 19% (range 8–36%). Only two fell within the normal range described by Brenner and Waugh.

In each of our nine patients with obstructive airway disease, the inspiratory effort apparently caused overdistention of the right ventricle. This overdistention, in
turn, displaced the interventricular septum posteriorly into the left ventricle, and might impair filling of the left ventricle.

This argument is further supported by our observations of inspiratory augmentation of right ventricular dimensions by two-dimension echocardiograms. This technique also demonstrated posterior motion of the interventricular septum with inspiration. Weiss and associates, in a two-dimensional echocardiographic study of nine normal men, reported leftward septal movement during right ventricular loading produced by the Mueller maneuver.29

Abnormal anterior systolic motion of the interventricular septum appeared during inspiration in one of our patients (fig. 1). This observation is consistent with right ventricular volume overload during inspiration.30, 31

We conclude that the paradoxical arterial pulse in obstructive lung disease is not caused by decreased filling of the right ventricle on inspiration, as initially postulated by Rebuck and Pengelly.2 Indeed there appears to be increased filling of the right ventricle during inspiration in patients with chronic obstructive lung disease and a paradoxical arterial pulse. The paradoxical pulse may result from an interplay of several mechanisms:

1. The increased respiratory variations of intrathoracic pressure are transmitted to the left ventricle and to the arterial vessels in the thoracic cavity, with a decrease in blood pressure during inspiration and an increase in blood pressure during expiration.

2. The increased filling of the right ventricle on inspiration tends to increase right ventricular stroke volume. This, in turn, may cause more than the usual increase of left ventricular filling in the subsequent expiration, increasing left ventricular stroke volume and systolic blood pressure in expiration. The increased inspiratory filling of the right ventricle is judged by comparison of right ventricular dimensions during inspiration and expiration in the same patient. Because there is no baseline observation during normal breathing, we cannot tell whether there is an absolute decrease in right ventricular filling during expiration, nor can we judge the degree of absolute as opposed to relative increase above that to be found during normal inspiration.

3. Increased filling of the right ventricle in inspiration causes posterior displacement of the interventricular septum (demonstrated by echocardiogram); this may limit inspiratory filling of the left ventricle by reducing its compliance,28 thus lowering left ventricular stroke volume and decreasing the arterial blood pressure during inspiration. Alternatively, abnormal systolic motion of the interventricular septum during inspiration may reduce left ventricular stroke volume without compliance changes.

Increased inspiratory capacity of pulmonary veins may also limit left ventricular filling as the functional residual capacity increases. Robotham et al. suggested another factor that may limit left ventricular output during inspiration in patients with obstructive airway disease.28 In a study of anesthetized dogs, they found that decreased left ventricular filling pressure during inspiration did not explain the inspiratory fall of left ventricular stroke volume. The increased negative intrathoracic pressure surrounding the left ventricle means that the left ventricle must pump against a higher peripheral mean pressure; thus, there is an inspiratory increase of left ventricular afterload that might be a major mechanism in the inspiratory decline of left ventricular stroke volume.

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