Noninvasive Assessment of Left Ventricular Performance in Patients with Chronic Obstructive Pulmonary Disease*

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In patients with chronic obstructive pulmonary disease (COPD), the clinical differentiation between dyspnea due to left ventricular dysfunction and that due to pulmonary events is difficult. Invasive techniques have been the only reliable diagnostic approach. To assess the potential value of noninvasive techniques in this context, 27 patients with COPD and with clinically suspected left ventricular dysfunction were studied by echocardiography, radionuclide angiography, and right cardiac catheterization. In 20 (74 percent), adequate echocardiograms were obtained. Of these 20 patients, 17 had normal pulmonary arterial wedge pressures at rest and during submaximal handgrip exercise. Sixteen of these 17 had normal left ventricular performance by all three echocardiographic criteria used; in one patient, two criteria were not interpretable, but the third was normal. Results of radionuclide studies were normal in 15 patients, borderline in one, and not measurable in one. Of the three patients with abnormal wedge pressures, at least one echocardiographic criterion was abnormal in all. Radionuclide data were abnormal in two and not measurable in one. We conclude that left ventricular dysfunction is infrequently present in patients with COPD in whom such dysfunction is clinically suspected, that the two noninvasive techniques described here can be applied successfully to a high percentage of patients with COPD, and that the agreement among echocardiographic, radionuclide, and wedge pressure data is excellent.

In the patient with chronic obstructive pulmonary disease (COPD), definition of the cause for an exaggeration in dyspnea can be difficult. Distinction between progression of pulmonary disease and the onset of cardiac dysfunction usually cannot be made on clinical grounds. Rapaport has emphasized the difficulty of this differential diagnosis, and Unger and associates have demonstrated that the usual clinical findings of left ventricular failure (such as orthopnea, paroxysmal nocturnal dyspnea, gallops, rales, edema, and cardiomegaly) may be unreliable in this group of patients, because these findings occur in patients with pulmonary disease alone without evidence of left ventricular dysfunction. Furthermore, the empiric use of digitalis can pose some hazard in patients with COPD, both directly, because of the hypoxemia and acid-base disturbances that are frequently present, and also indirectly, because such treatment may distract attention from some remediable decrement in pulmonary performance.

Left ventricular catheterization and cineventriculographic studies may provide a precise answer to the question of whether a particular patient has an element of left ventricular dysfunction contributing to his dyspnea; however, these procedures are not without risk and are difficult to justify in either clinical or investigative contexts. The advent of balloon-tipped flow-directed catheters has greatly facilitated the acquisition of right cardiac and pulmonary arterial pressures, and in most circumstances the pulmonary arterial wedge pressure accurately reflects the left ventricular end-diastolic pressure. Right cardiac catheterization at bedside has been extremely useful for identifying dyspneic patients who have left ventricular failure in addition to pul-

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monary disease. Recently, echocardiography and radionuclide angiography have been established as valuable noninvasive techniques for evaluating left ventricular performance in patients without coexistent pulmonary disease; however, these have been applied rarely to patients with COPD, in whom technical problems might be anticipated to interfere with both performance and analysis. Therefore, we designed a study in patients with COPD who had clinically suspected left ventricular failure, in order to determine the following: (1) whether echocardiographic techniques and radionuclide techniques for ejection fraction could be successfully applied; (2) if so, the frequency with which abnormalities of left ventricular performance were detected by these techniques; and (3) the relationship between echocardiographic and radionuclide data and the behavior of the directly measured pulmonary capillary ("wedge") pressure.

METHODS

Twenty-seven patients with COPD were referred to us for evaluation because of clinically suspected left ventricular dysfunction. All patients were dyspneic on walking one block or less and had evidence of COPD based on clinical evaluation, chest x-ray films, and tests of pulmonary function.

Twenty (74 percent) of these patients had technically acceptable echocardiograms, were studied further, and form the basis of this report. Fifteen were men, and five were women, with an age range of 49 to 83 years (mean, 62 years). The study was described in detail to each patient, and informed consent was obtained. The ten patients receiving digoxin had it discontinued at least one week prior to right cardiac catheterization.

Studies of Pulmonary Function

The best of three maneuvers for forced vital capacity (FVC) was used to determine the one-second forced expiratory volume (FEV1) and the mean forced expiratory flow during the middle half of the FVC (formerly maximal midexpiratory flow). The pulmonary volumes were expressed as a percentage of the predicted normal values. The functional residual capacity and airway resistance (Raw) were determined in the body plethysmograph using the method of Dubois et al.

Echocardiographic Studies

Just prior to right cardiac catheterization, echocardiograms were recorded with an ultrasonic scope (Picker) using a 1.6-MHz transducer focused at 10 cm and a subxiphoid approach. The output signal was recorded on an oscillograph (Honeywell Visicorder model 1850), and each patient had an electrocardiogram and an external carotid arterial pulse tracing recorded simultaneously with the echocardiogram. Left ventricular dimensions were measured between the endocardial surfaces of the posterior wall and the left side of the septum (Fig 1A). The end-diastolic dimension (EDD) was measured at a point coincident with the peak of the R wave of the simultaneously recorded ECG. The end-systolic dimension (ESD) was measured as the smallest distance separating the left ventricular endocardial surfaces. The

![Figure 1](image-url)
ejection time (ET) was determined from a simultaneously recorded carotid arterial pulse tracing. Using these measures, ejection-phase indices of left ventricular performance were calculated as previously described. Briefly, the mean normalized rate of shortening of the left ventricular dimension was calculated as EDD - ESD/EDD × ET, and the ejection fraction was calculated using the formula, EDD³ - ESD³/EDD³ × 100 percent.

**Right Cardiac Catheterization**

Right cardiac catheterization was accomplished with a No. 7 balloon-tipped, flow-directed thermomilab catheter according to the method of Swan et al.6 Pressure transducers were either of two commercially available models (Statham P23db or P37a), and the pressures were recorded on a strip-chart recorder (Electronics for Medicine IR-4). Zero reference for the measurements of pressure was at the midthoracic line. The position of the catheter was verified by examining the phasic contour of the pressure pulse in the wedge position. Pulmonary arterial and pulmonary arterial wedge pressures were electronically analyzed for the mean, and all pressures were averaged over at least three respiratory cycles. Cardiac outputs by the technique of thermodilution were determined using a computer (Xerox Sigma III).

After recording all resting data in the basal state, isometric handgrip exercise was performed for one minute at 50 percent of maximal voluntary contraction using a calibrated dynamometer. This amount of exercise led to a mean increase in systolic blood pressure of 24 ± 16 mm Hg and a mean increase in heart rate of 10 ± 10 beats per minute. The pattern of breathing of the patients was carefully observed, with special care to avoid Valsalva's maneuver. Pulmonary arterial wedge pressure was recorded continuously throughout the period of exercise.

**Radionuclide Ejection Fraction**

Subsequently, all patients had a radionuclide ejection fraction calculated during the first pass of a 15-microcurie bolus of radioactive technetium-labeled albumin injected through a peripheral vein. Precordial activity was recorded during the first circulation through the heart with a gamma scintillation camera (Searle Pho/Gamma HP) equipped with a converging low-energy collimator in a 30° right anterior oblique position and was stored in "real time" on magnetic tape (Searle Data Storage/Accessory). To define the left ventricular counts precisely, the raw data were displayed using a digital computer (Nuclear Data Med II, Palatine, IL). The image of the bolus could be followed through the heart, and the left ventricular cavity could be clearly identified. The region of interest was then assigned with the computer light pen, with special care to precisely fit the left ventricular silhouette and exclude the aortic root. A time-activity curve of the left ventricle was generated, and the ejection fraction was derived from the early monoexponential portion of the downward slope when the period of mixing of radioactive tracer with the left ventricular blood pool was most complete.

**RESULTS**

**Normal Pulmonary Arterial Wedge Pressure**

Seventeen of the patients had a normal pulmonary

<table>
<thead>
<tr>
<th>Pulmonary Arterial Pressure (Mean), mm Hg</th>
<th>Cardiac Index, L/min/m²</th>
<th>Heart Rate, beats per minute</th>
<th>Blood Pressure, mm Hg</th>
<th>Pulmonary Arterial Wedge Pressure, mm Hg</th>
<th>Ejection Fraction, percent</th>
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<tbody>
<tr>
<td>Case</td>
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<td>&gt;250</td>
<td>&gt;120</td>
<td>&gt;120</td>
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<td>280</td>
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<tr>
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<td>101</td>
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<tr>
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<td>183</td>
<td>122</td>
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<td>16</td>
<td>29/17 (22)</td>
<td>4.2</td>
<td>99</td>
<td>92</td>
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<td>46/23 (28)</td>
<td>3.0</td>
<td>144</td>
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<td>108</td>
</tr>
</tbody>
</table>

*EDD, End-diastolic dimension by echocardiogram; PSM, paradoxical septal motion; PVR, pulmonary vascular resistance; TR, tricuspid regurgitation; and Vd, mean normalized rate of shortening of left ventricular dimension per second (by echocardiogram).*
arterial wedge pressure at rest and during exercise (Table 1). Four of these 17 had mean pulmonary arterial pressures above 22 mm Hg, and six had a cardiac index of 2.8 L/min/sq m or below. The resting heart rate tended to be increased but did not correlate with other hemodynamic abnormalities.

Echocardiographic studies disclosed that all 17 patients had normal end-diastolic dimensions, and 16 had a normal rate of left ventricular shortening and ejection fraction as measured by echocardiographic techniques. In one patient the rate of left ventricular shortening and the ejection fraction could not be determined by echocardiographic studies because paradoxical septal motion was present. Thus, in no patient with a normal pulmonary arterial wedge pressure in whom echo measurements could be made were there echocardiographic abnormalities. The radionuclide ejection fraction was within normal limits in 16 patients and could not be measured in one, due to tricuspid regurgitation.

**Abnormal Pulmonary Arterial Wedge Pressure**

Three patients had an abnormal pulmonary arterial wedge pressure; two patients (cases 18 and 20) had such abnormal values both at rest and during exercise, and one patient (case 19) had an abnormal value during exercise only. All three of these patients had a mean pulmonary arterial pressure above 22 mm Hg. All had cardiac indices above 2.8 L/min/sq m.

By echocardiographic studies the end-diastolic dimension and rate of left ventricular shortening were abnormal in two of these three patients. These two variables could not be measured in one patient because of paradoxical septal motion, and in one patient the ejection fraction was borderline; however, in this last patient the end-diastolic dimension and rate of left ventricular shortening were both abnormal. Thus, in no patient with an abnormal pulmonary arterial wedge pressure were all three echocardiographic indices normal. The radionuclide ejection fraction was abnormally low in two of these three patients and could not be measured in the third because of tricuspid regurgitation.

**Comparison of the Three Techniques**

Of these 20 patients with COPD who were suspected of having left ventricular dysfunction, only three (15 percent) had left ventricular abnormalities detected by the three techniques employed. Agreement among the three techniques, in terms of separation into normal and abnormal, was of high degree.

Of the 17 patients with normal pulmonary arterial wedge pressures, none was abnormal by echocardiographic criteria or by the radionuclide technique. Only one patient had a borderline radionuclide ejection fraction, but his echocardiographic data were normal.

Similar agreement was observed among the three patients with an abnormal pulmonary arterial wedge pressure. Echocardiographic data and radionuclide data on ejection fraction were abnormal in each instance in which they could be measured, except one patient (case 20) who had a normal end-diastolic dimension but an abnormal rate of left ventricular shortening and an abnormal ejection fraction.

**Tests of Pulmonary Function**

All 20 patients had results of tests of pulmonary function that were compatible with COPD, as illustrated in Figure 2.12 There was no correlation between any measurement of pulmonary function and abnormalities of left ventricular function, as reflected by echocardiographic data, radionuclide ejection fraction, or pulmonary arterial wedge pressure; for example, two of the three patients with

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**Figure 2.** Data on pulmonary function in 20 patients with COPD.12 RV/TLC, Ratio of residual volume to total lung capacity; MMEF\textsubscript{25-75}, maximal midexpiratory flow rate (or FEF25-75%); \textit{Po}₂, arterial oxygen pressure; and \textit{PCO}₂, arterial carbon dioxide tension. Solid bars indicate normal limits, and dotted lines indicate mean values.
Table 2—Clinical Data

<table>
<thead>
<tr>
<th>Abnormality</th>
<th>Total*</th>
<th>Normal**</th>
<th>Abnormal†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angina pectoris</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Orthopnea</td>
<td>14</td>
<td>12</td>
<td>2</td>
</tr>
<tr>
<td>Paroxysmal nocturnal dyspnea</td>
<td>12</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>Edema</td>
<td>6</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Gallop rhythm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Third heart sound</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Fourth heart sound</td>
<td>10</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>Increased cardiothoracic ratio</td>
<td>4</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Prominent left ventricle‡</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Previous therapy with digitalis</td>
<td>10</td>
<td>8</td>
<td>2</td>
</tr>
</tbody>
</table>

*Total number of patients with indicated abnormality of 20 patients studied.
**Number of patients with indicated abnormality but normal left ventricular function.
†Number of patients with indicated abnormality but abnormal left ventricular function.
‡Greater than 1.2 on standard chest x-ray film.
§Prominent left ventricular configuration in hyperinflated chest on standard chest x-ray film.

increased pulmonary arterial wedge pressure (cases 18 and 20) had values for Raw that were less than the group's mean of 3.7 cm H₂O/L/sec, while the third patient (case 19) had a markedly elevated Raw of 8.4 cm H₂O/L/sec.

Clinical Data

Clinical criteria did not serve to separate the patients with left ventricular dysfunction from those without (Table 2). A "history" of apparent left ventricular failure proved unreliable. Ten of the patients had been placed on therapy with digitalis previously because of a "compatible history." Eight proved to have normal left ventricular function, and two had abnormal function. No patient had a known history of prior myocardial infarction.

The lack of differential diagnostic value also applied to symptoms, such as orthopnea or angina; signs, such as peripheral edema; physical findings, such as third and fourth heart sounds; and laboratory findings, such as electrocardiographic abnormalities or assessment of cardiac size by chest x-ray films.

Discussion

The first question we posed in this investigation was whether the echocardiogram and the radionuclide angiogram could be successfully applied to assessment of left ventricular performance in patients with COPD. The utility of both of these non-invasive techniques has been well established in the evaluation of left ventricular function in patients without COPD. The application of these techniques to patients with COPD poses special problems because the left ventricle is displaced postero-medially away from the chest wall by increased pulmonary volume and enlargement of the right cardiac chambers. Using a left sternal approach and a standard transducer, we were unable to record echoes from the septal and posterior wall endocardial surfaces in most patients with COPD; however, the 1.6-MHz echocardiographic transducer can penetrate tissues much more effectively than transducers with higher frequency, and the subxiphoid approach allows better access to the cardiac chambers by avoiding air interfaces in the lung. With these modifications, satisfactory echocardiographic data were obtained in 75 percent (20) of the original 27 patients with COPD. The data of Chang and Feigenbaum indicated that these modifications do not alter the validity of the measurements. In 23 patients, these investigators found a high degree of correlation between the end-diastolic dimension measured by the left sternal border and subxiphoid approaches. Furthermore, in our study the same structural landmarks observed with the method using the left sternal border are seen when using the subxiphoid approach. This suggests that the heart is placed more medially in these patients with COPD and that the measured chord may actually be very close to the true minor chord. If the measured chord were not the true minor chord, it should have been longer, and most of our patients had end-diastolic dimensions in the normal range. Thus, we believe that the subxiphoid approach is acceptable for obtaining the minor dimension of the left ventricle in patients with COPD. There was no obvious difference (age, chest x-ray film, or extent of pulmonary functional abnormality) between the seven patients in whom satisfactory tracings could not be obtained and the other 20 patients.

The validity of echocardiographically determined ejection fractions or rates of left ventricular shortening as measures of overall left ventricular performance are dependent on the assumption that the left ventricle is contracting symmetrically. Thus, regional abnormalities of wall motion, such as paradoxical septal motion, invalidate the technique. Also the single-beam echocardiographic technique utilized in this study does not visualize all areas of the left ventricle. Thus, undetected regional abnormalities of wall motion could exist and be responsible for depressed overall left ventricular function, de-
spite a normal ejection fraction or rate of left ventricular shortening; however, in none of the 17 patients with a normal pulmonary arterial wedge pressure was an abnormality present in measured ejection fraction or rate of left ventricular shortening. The radionuclide technique for ejection fraction depends neither on the shape of the heart nor on the uniformity of left ventricular contraction. Rather, that technique measures the relative difference in counts between the end of diastole and the end of systole during a single pass of radionuclide through the left ventricle. Therefore, accurate measurements depend on proper positioning of the gamma camera over the left ventricle and on the careful elimination of background and aortic outflow activity. Since the heart is displaced in many patients with COPD, assessing the cardiac position before injection of the bolus and displaying the raw data on a digital computer provide an accurate way of identifying the left ventricular cavity. We encountered no difficulty in applying this technique to these patients with COPD; however, the radionuclide technique for ejection fraction is not reliable in the presence of tricuspid regurgitation, since the passage of a single bolus of the radionuclide through the heart will not occur, as was the case of two of our patients.

Thus, our data and experience indicate that these two noninvasive techniques can be successfully applied to a high percentage of patients with COPD. Furthermore, the techniques complement each other, in that factors which invalidate one do not alter data obtained by the other. The echocardiogram provides quantitative anatomic details of the minor axis of the left ventricle, and the radionuclide angiogram assesses relative stroke output independent of the geometry of the chambers.

The second question we posed was the frequency of abnormalities of left ventricular performance in patients with COPD who are clinically suspected of having left ventricular failure contributing to their dyspnea. Our data indicate that this suspicion was unfounded in 85 percent (17) of these 20 patients. This is a point of more than academic interest. Since therapy with digitalis is of no proven value in patients with right ventricular dysfunction, its application to the patient with COPD for the treatment of nonexistent left ventricular failure and normal sinus rhythm needlessly exposes the patient to the risks of digitalis toxicity. Such risks are especially high if hypoxemia is present. Another hazard exists in this situation; namely, that attribution of the patients' symptoms to left ventricular failure may obscure recognition of and therapy for the true cause of their symptoms, (ie, their pulmonary disease).

Thus, there appear to be sound reasons for defining whether or not left ventricular failure is the etiologic basis of progression of dyspnea in the patient with COPD.

The final question of interest to us was the relationship between the three methods employed to assess left ventricular performance. Clearly, each has its limitations and imperfections; for example, all of the measurements reported relate to the pumping function of the left ventricle and do not provide direct information regarding the compliance of the left ventricle. Furthermore, while the pulmonary arterial wedge pressure is widely used and accepted as an indicator of left ventricular filling pressure, there are instances in which these pressures are not in close agreement. With regard specifically to patients with pulmonary disease, it is evident that all hemodynamic measurements of pressure in the thorax are influenced by swings in intrathoracic pressure, which, in turn, are related to the mechanical behavior of the lungs and chest wall.\textsuperscript{16-22} Despite such complexities, which remain to be fully defined, the pulmonary arterial wedge pressure in patients with COPD appears to be an accurate measurement of left atrial and left ventricular end-diastolic pressure. We have found and reviewed 63 reported instances in which pulmonary arterial wedge pressure, left atrial pressure, and left ventricular end-diastolic pressure were measured simultaneously in patients with COPD. In 88 percent (55) of these 63 instances, the difference between these pressures was less than 5 mm Hg.\textsuperscript{19,23-27} Beyond this specific point concerning the accuracy of measurements of pressure is the broader one regarding what technique is the most sensitive indicator of left ventricular dysfunction and the even more difficult question as to a definition of left ventricular "failure." Resolution of such questions is beyond the scope of this discussion; however, it is an unwarranted oversimplification to place too great a reliance on measurements of pressure alone. Probably, measurement of ejection-phase indices of left ventricular performance are more reliable than any single measurement of pressure. Therefore, the echocardiographic and radionuclide methods are not only safe and noninvasive but may be even more definitive methods of assessing left ventricular dysfunction than is measurement of pulmonary arterial wedge pressure; however, it is likely that the onset of dyspnea due to left ventricular dysfunction appears when the pulmonary arterial wedge pressure becomes elevated.

Despite these semantic and physiologic questions, our data have nevertheless shown that there is close agreement among the three methods. When the pul-

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monary arterial wedge pressure was normal at rest and during exercise, echocardiographic data and radionuclide ejection fraction were normal; when pulmonary arterial wedge pressure was elevated, these same data were abnormal with one exception. Our experience indicates that these noninvasive techniques should allow proper identification of the cause of progressive dyspnea in a given patient with COPD and allow application of appropriate therapy.

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