Left Ventricular Dysfunction in Deteriorating Patients With Chronic Obstructive Pulmonary Disease*

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Objective: To evaluate the effectiveness of simple clinical variables and radionuclide ventriculogram in separating those patients with isolated chronic obstructive pulmonary disease (COPD) from those with COPD and coexisting left ventricular dysfunction (LVD).

Design: Retrospective record review of 77 patients with increasing dyspnea, defined as recent deterioration in exercise tolerance, new use of corticosteroids, or recent hospital admission for COPD; referred to the outpatient Pulmonary Rehabilitation Program at the Cincinnati Veterans Affairs Medical Center from July 1987 to October 1992.

Setting: Outpatient medical clinic.

Patients: Veterans who were referred to the Pulmonary Rehabilitation Program.

Measurements: History and physical findings, pulmonary function tests, arterial blood gases, distance achieved in a 12-min walk, dyspnea score, electrocardiogram, chest radiograph, and radionuclide multigated ventriculography.

Results: Twenty-five of 77 patients evaluated in the Pulmonary Rehabilitation Program for increasing dyspnea were functionally more limited (12-min walk 10.4 vs 13.9 laps; MRC score 2.68 vs 2.06; *p<0.05) and had left ventricular dysfunction (LVD) (left ventricular ejection fraction <40%) associated with wall motion abnormalities on radionuclide ventriculogram. Careful standard clinical evaluation did not separate those patients with COPD from those with both COPD and LVD.

Conclusions: LVD was found in 32% of patients with COPD presenting with symptomatic deterioration. Since the therapeutic approach to these two disorders differs, the identification of patients with LVD is important. Prospective studies are needed to identify the most cost-effective approach to this problem of coexisting disease and to evaluate the benefit from therapy.

(Chest 1995; 107:162-68)

COPD=chronic obstructive pulmonary disease; Deo=single breath diffusing capacity; FEVi=forced expiratory volume in 1 s; FVC=forced vital capacity; LVD=left ventricular dysfunction; LVEF=left ventricular ejection fraction; MRC=modified British Medical Research Council dyspnea scale; MUGA=multigated radionuclide ventriculography; NL LV=the group of patients with normal left ventricular function and COPD; PA=posteroanterior; PFT=pulmonary function tests; PND=paroxysmal nocturnal dyspnea; VAMC=Veterans Affairs Medical Center

Key words: dyspnea; heart failure, congestive; lung diseases, obstructive; pulmonary emphysema; pulmonary heart disease; ventricular function, left

Most patients who develop left ventricular failure do so as a consequence of systolic left ventricular dysfunction (LVD) related to hypertension and/or coronary atherosclerosis, conditions that share risk factors with chronic obstructive pulmonary disease (COPD), particularly tobacco use and male gender.1 Epidemiologic studies confirm the relatively frequent coexistence of COPD and coronary artery disease.2 A physician’s ability to discriminate whether worsening dyspnea is the result of deterioration of pulmonary disease or the onset of decompenedated LVD is critically important as it will greatly influence therapeutic decisions.

Traditionally, texts3,4 and review articles5,6 suggest that dyspnea due to COPD can be readily distinguished from dyspnea due to left ventricular failure by clinical, radiographic, and spirometric abnormalities. However, COPD may obscure clinical signs of coexisting LVD. Both disorders produce similar symptoms: paroxysmal nocturnal dyspnea (PND), orthopnea, and cough. Radiographic and physical findings of pulmonary congestion and cardiomegaly may be obscured by the large barrel chest and hyperinflated lungs of the patient with emphysema. Evidence of airway obstruction and a bronchodilator response on pulmonary function tests is found not only in COPD but also in acute congestive heart failure.7-9

We designed this study to determine whether a standardized, routine outpatient evaluation, including history, clinical examination, exercise tolerance, chest radiographs, ECGs, and pulmonary function tests (PFT) was adequate to identify patients who...
were referred for increased dyspnea to a pulmonary rehabilitation clinic and in whom COPD and left ventricular dysfunction coexisted. The results indicate that close to one third of patients with COPD who presented with increasing dyspnea have left ventricular dysfunction, and that routine clinical evaluation fails to discriminate between patients with COPD alone and those who also have LVD.

**Methods**

**Patient Selection**

Patients were selected retrospectively from all of those with COPD evaluated in the Pulmonary Rehabilitation Program between July 1987 and October 1992. We defined COPD by American Thoracic Society (ATS) criteria. Patients referred to the clinic undergo a standardized evaluation consisting of a medical history, physical examination, subject assessment of dyspnea (British Medical Research Council [MRC] Dyspnea score) and exercise tolerance (estimate of distance patient can walk on a flat surface at their own pace without stopping), objective measurement of exercise tolerance (distance walked in 12 min, including pulse oximetry), resting arterial blood gas, posteroanterior (PA) and lateral chest roentgenogram, PFT, and 12-lead ECG. An multigated (equilibrium gated) radionuclide ventriculogram (MUGA) is obtained for patients with a deterioration of functional capacity defined as a complaint of increased dyspnea on exertion, recurrent hospitalization for COPD, or new initiation of corticosteroid therapy for COPD. From a total of 172 patients with COPD referred to the rehabilitation program, 92 experienced deterioration in function during this interval, defined as reduced reported exercise tolerance, new use of corticosteroids, or recent hospitalization, and they were evaluated with MUGA. We excluded 13 patients with overt cardiac disease who met at least one of the following criteria: positive exercise test for ischemia by ECG, intercurrent ischemic events (intercurrent myocardial infarction or unstable angina), or concurrent coronary revascularization; and two patients for the absence of data essential to this study (chest radiograph, PFTs, enrollment form). Based on the left ventricular ejection fraction (LVEF), we divided the patients into two groups based on criteria from large clinical trials; those with LVEF >40% (normal left ventricular function and COPD [NL LV]) and those with LVEF <40% (LVD).

**History and Physical Examination**

History and physical examination were performed by one of us (M.L.R.) prior to the radionuclide scan. A printed history form focused on respiratory, cardiac, and gastrointestinal review of systems was completed on each patient evaluated in the pulmonary rehabilitation program. All patients were asked about the presence of chest pain, paroxysmal nocturnal dyspnea, orthopnea, wheezing, cough and sputum production, chest infections in the past, childhood illness, use of corticosteroids, hemoptysis, and characteristics of their dyspnea (what, when, how much, how fast). Medication profiles were reviewed. Cardiac risk factors, hypertension (systolic blood pressure greater than 145 mm Hg or a diastolic blood pressure greater than 89 mm Hg on two consecutive readings or use of antihypertensive medication), diabetes (use of oral hypoglycemic or insulin), and positive family history of cardiovascular disease were also assessed. The presence of edema, neck veins, and hepatojugular reflex, site of maximal impulse of the heart on the chest wall, third or fourth heart sounds, lung sounds, including rales, rhonchi, and wheezing were recorded in specified lines of the printed history and physical form.

**Pulmonary Function Testing**

Complete PFT was performed using the PFT system (Collins DS/360 Pulmonary Function Testing System). Lung volumes were assessed using the helium dilution technique, and diffusing capacity (Dco) using the single-breath technique with carbon monoxide inhalation. Standard predictive equations were used. Arterial blood gas analysis was performed (on the ABL2, Radiometer, Copenhagen). We quantified exercise tolerance as the number of laps patients walked in 12 min around the perimeter of the gymnasium at the Veterans Affairs Medical Center (VAMC), Cincinnati. During that walk, we defined arterial oxygen desaturation (Nellcor pulse oximeter) as a sustained fall in oxygen saturation greater than 5% and below 85%.

**Chest Radiograph Review**

A PA and lateral radiograph from each patient was matched as closely as possible (±1 month) with the MUGA. One of us (A.S.W.) reviewed each radiograph for signs of heart failure and COPD blinded for all other data. Radiographic criteria for heart failure and COPD were those from standard texts.\[10,11\]

**Electrocardiograms**

One of us (A.S.B.) reviewed ECGs blinded to all other data. All ECGs selected for review had been acquired within 3 months of the isotope ventriculogram. The diagnosis of chamber enlargement (left atrium and ventricle; right atrium and ventricle), ischemia, infarction, and intraventricular conduction abnormalities met standard criteria outlined in a recent ECG text.\[12\]

**Isotope Ventriculography**

The Nuclear Medicine Department performed isotope ventriculogram using in evo labeling of red blood cells with technetium 99. The LVEF was calculated from the left anterior oblique projection of the equilibrium multigated acquisition. Patients with LVEF less than 0.40 were considered to have LVD; those with LVEF greater than 0.40 were considered to have normal left ventricular function. Right ventricular ejection fraction (RVEF) was calculated using the gated first pass technique and wall motion analyzed from three projections (left anterior oblique, PA, lateral) using the standard 15-segment map. The authors used the official clinical interpretation and substituted a value of 55% if RVEF was simply reported as “normal.”

**Data Analysis**

We analyzed all data using statistical software for personal computers. (TRUE EPISTAT Version 4.0, EPISTAT Σ Services). Our null hypothesis was that clinical testing was incapable of differentiating between patients with COPD alone and those with coexisting LVD. Group variables are presented as the mean ± standard error of the mean. We used a two-tailed t test to evaluate differences between the group with normal left ventricular function and the group with reduced left ventricular function and linear regression to evaluate relationships between two variables and multivariate regression to evaluate relationships among variables. A p value of <0.05 was considered significant; p values are provided for all relevant data.

**Results**

**Clinical Variables**

Patients with LVD compared with those with normal left ventricular function were similar in age (66.0 ± 1.2 vs 65.4 ± 0.86 years; p>0.10) (Table 1). All patients were men and had significant smoking his-
Table 1—Risk Factors and Clinical Findings in NL LV and LVD*  

<table>
<thead>
<tr>
<th>Age, yr</th>
<th>NL LV</th>
<th>LVD</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>52</td>
<td>25</td>
<td>0.640</td>
</tr>
<tr>
<td>Cardiovascular risk factor, % positive</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>9.7</td>
<td>16.0</td>
<td>0.421</td>
</tr>
<tr>
<td>Family history</td>
<td>41.7</td>
<td>31.6</td>
<td>0.473</td>
</tr>
<tr>
<td>Hypertension</td>
<td>41.7</td>
<td>36</td>
<td>0.777</td>
</tr>
<tr>
<td>Symptoms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distance, m</td>
<td>0.52 ±0.09</td>
<td>0.21 ±0.04</td>
<td>0.006†</td>
</tr>
<tr>
<td>MRC</td>
<td>2.06 ±0.13</td>
<td>2.68 ±0.17</td>
<td>0.008†</td>
</tr>
<tr>
<td>PND, %</td>
<td>55.0</td>
<td>70.8</td>
<td>0.17</td>
</tr>
<tr>
<td>Orthopnea, %</td>
<td>37.0</td>
<td>50.0</td>
<td>0.226</td>
</tr>
<tr>
<td>Chest pain, %</td>
<td>62.5</td>
<td>54.2</td>
<td>0.596</td>
</tr>
</tbody>
</table>

| Signs, % positive |        |       |         |
| Edema           | 32.6   | 18.2  | 0.193   |
| S3              | 4.5    | 0     |         |
| Wheeze          | 65.2   | 62.8  | 0.756   |
| Rales           | 17.4   | 23.8  | 0.823   |
| 12-min walk     | 13.9±0.8 | 10.4±0.8 | 0.010† |

*Signs, symptoms, cardiac risk factors, and physical findings in those with COPD and NL LV and in those with COPD and reduced LVD. †p≤0.05.

There were no statistically significant differences in the incidence of risk factors for coronary artery disease or heart failure (hypertension, positive family history of coronary artery disease, or diabetes mellitus), nor were the medication profiles different between the two groups, including the use of diuretics, nitrates, angiotensin-converting enzyme inhibitors, and calcium channel blockers. None of the patients were receiving β-blockers.

Patients with LVD reported greater functional limitation than did those with normal left ventricular function (Fig 1). Self-reported exercise tolerance was lower in those with LVD compared with those with normal left ventricular function (0.21 ±0.04 vs 0.52 ±0.09 miles, p=0.003) and functional limitation assessed by the modified MRC Dyspnea Scale was more severe in LVD (2.68 ±0.17 vs 2.06 ±0.12; p=0.008). Objectively, results of their 12-min walk (10.4 ±0.8 laps in LVD vs 13.9 ±0.8 laps NL LV; p=0.010) confirmed the greater functional limitation in patients with LVD but the two groups substantially overlapped. No other symptom or physical finding separated the groups (Table 1). Multiple linear regression analysis showed that FEV₁ (expressed as percent predicted) correlated with number of laps walked during the 12-min walk in the group with normal left ventricular function (p=0.0233) but not in those with LVD (p=0.295) whereas PaO₂, Dco, LVEF, and heart size did not correlate with exercise tolerance in either group.

We also analyzed a subset of 40 patients; 20 with normal left ventricular function and 20 with LVD. Patients in this subset were all patients with LVD and data sets that included laps in the 12-min walk; the 20 subjects with LVD were then matched by percent predicted FEV₁ to 20 subjects with normal left ventricular function. In this subset analysis, those with LVD had a reduced exercise tolerance compared with those with normal left ventricular function (10.30 ± 0.9 vs 14.6 ±1.1 laps; p=0.037). Exercise tolerance correlated positively with percent predicted FEV₁ (LVD, slope=−0.0056, r=0.1975, in-

Table 2—Pulmonary Function Tests (Mean±SE)*  

<table>
<thead>
<tr>
<th></th>
<th>NL LV</th>
<th>LVD</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PFTs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FVC, L</td>
<td>2.51±0.11</td>
<td>2.35±0.11</td>
<td>0.236</td>
</tr>
<tr>
<td>FEV₁, L</td>
<td>1.15±0.08</td>
<td>1.01±0.08</td>
<td>0.180</td>
</tr>
<tr>
<td>FEV₁/FVC, %</td>
<td>45.2±1.52</td>
<td>42.8±2.2</td>
<td>0.327</td>
</tr>
<tr>
<td>TLC, L</td>
<td>6.79±0.17</td>
<td>6.87±0.28</td>
<td>0.808</td>
</tr>
<tr>
<td>MVV, L/min</td>
<td>46.9±2.9</td>
<td>43.2±3.4</td>
<td>0.437</td>
</tr>
<tr>
<td>DCO, % pred</td>
<td>58.3±4.1</td>
<td>49.4±5.0</td>
<td>0.214</td>
</tr>
<tr>
<td>Arterial blood gas</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.42±.005</td>
<td>7.43±.005</td>
<td>0.695</td>
</tr>
<tr>
<td>PaO₂</td>
<td>60.3±1.45</td>
<td>65.8±2.1</td>
<td>0.079</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>39.9±1.11</td>
<td>40.9±1.43</td>
<td>0.6156</td>
</tr>
</tbody>
</table>

*Pulmonary function tests: spirometry, lung volumes, Dco, MVV, and arterial blood gases in COPD alone (NL LV) and in COPD with LVD. TLC=total lung capacity; PaO₂=partial pressure of oxygen in arterial blood; PaCO₂=partial pressure of carbon dioxide in arterial blood.

Figure 1. Exercise tolerance in patients with COPD with LVD (stipped bar) compared with those patients with NLLV (solid bar).
Table 3—Cardiac Evaluation*

<table>
<thead>
<tr>
<th></th>
<th>NL LV</th>
<th>LVD</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MUGA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEF, %</td>
<td>56 ± 1.1</td>
<td>31 ± 1.2</td>
<td>&lt;0.000001</td>
</tr>
<tr>
<td>RVEF, %</td>
<td>54 ± 0.9</td>
<td>45 ± 2.2</td>
<td>0.002</td>
</tr>
<tr>
<td>LV WMA, %</td>
<td>41</td>
<td>92</td>
<td>&lt;0.000001</td>
</tr>
<tr>
<td>Chest radiograph</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lung fields, % present</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Effusions</td>
<td>1.9</td>
<td>4.2</td>
<td>0.621</td>
</tr>
<tr>
<td>Hilar full</td>
<td>21</td>
<td>25</td>
<td>0.760</td>
</tr>
<tr>
<td>Kerley B</td>
<td>7.7</td>
<td>8.3</td>
<td>0.907</td>
</tr>
<tr>
<td>COPD</td>
<td>71</td>
<td>76</td>
<td>0.405</td>
</tr>
<tr>
<td>Heart</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>heart size, cm</td>
<td>13.5 ± 0.29</td>
<td>14.2 ± 0.41</td>
<td>0.160</td>
</tr>
<tr>
<td>CT ratio, %</td>
<td>41 ± 1.0</td>
<td>44 ± 1.1</td>
<td>0.161</td>
</tr>
</tbody>
</table>

*Cardiac and radiographic evaluation in the two groups: patients with COPD alone (NL LV) and patients with LVD. LV WMA=left ventricular wall motion abnormalities; CT ratio=cardiothoracic ratio.

intercept=10.71; vs NL LV; slope=0.2419, r=0.660, intercept=6.00) only in the subset with normal left ventricular function. There was a significant difference in the slopes of the lines (p=0.0094). The disparity in exercise tolerance was greatest in those whose pulmonary function was best.

Pulmonary Function

The two groups did not differ in the severity of airway obstruction, total lung capacity, DEO, or maximal voluntary ventilation assessed by PFT (Table 2). Differences in arterial oxygen tension were small (LVD: 65.8 ± 2.1; NL LV: 69.3 ± 1.5 mm Hg; p=0.078). The number of patients in each group that desaturated with exercise did not differ (p=0.41).

Nuclear, Radiographic, and ECG Analysis

By design, the patients with LVD had reduced LVEF compared with NL LV (31 ± 1.3% vs 56 ± 8.0%; p<0.000001) (Table 3). Right ventricular ejection fraction was also reduced in LVD (45.5 ± 2.2% vs 54.1 ± 6.6%; p=0.002). Wall motion abnormalities were present in 92% of patients with LVD compared with 42% of those with NL LV (p<0.000001, Fig 2).

No radiographic features alone or in combination discriminated LVD from NL LV. The average size of the heart silhouette on PA chest radiograph (Fig 3) in the patients with LVD (14.2 ± 4.1 cm) was similar to that in patients with normal left ventricular function (13.5 ± 2.1 cm; p=0.15). Only 29% of patients with LVD had cardiomegaly defined as a silhouette greater than 15.5 cm, compared with 15.4% of patients with NL LV. This difference was not statistically significant. The cardiothoracic ratio was greater than 50% in 16% of LVD (range: 51% to 53%) and 4% of those with normal left ventricular function (range: 53% to 57%) but on average was within normal limits in both groups (LVD: 43.8 ± 1.1% vs NL LV 41.2 ± 7.7%; p=0.161). Neither cardiac size nor cardiothoracic ratio was larger in the subgroup with biventricular dysfunction (RVEF and LVEF less than 40%). Chest findings, pleural effusions, Kerley's B lines, hilar infiltrates and enlargement, and parenchymal infiltrates did not differentiate LVD, and those with normal left ventricular function and linear regression failed to disclose a relationship between either heart size or cardiothoracic ratio and left ventricular ejection fraction. COPD was deemed present radiographically if two thirds of the follow-

Figure 2. Radionuclide scanning showed reduction in the RVEF in patients with COPD with LVD (stippled bar) compared with those with NL LV (solid bar) and an increase in WMA.

Figure 3. Relationship between LVEF and heart size on chest radiograph in patients with COPD. Plus signs=LVD; squares=NL LV.

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ing criteria were met—an obtuse angle of the dia-
phragm, an anterior clear space measuring greater
than 2 cm, or flattening of the diaphragm on the
lateral chest film. COPD was radiographically evi-
dent in 71% of patients with LVD compared with
76% of patients with normal left ventricular function
(p=0.4952).

Sixty-five patients in the two groups had ECGs
available for analysis. Tachycardia (rate >90/min)
was the most frequent abnormality occurring in
47.7% of all patients; its incidence was similar in both
groups. All but two patients had sinus rhythm.
Twelve of 65 (18.5%) had evidence of prior infarction
(22% LVD vs 16% NL LV) of which five were ante-
terior (3 NL, LV, 2 LVD). Overall the incidence of
myocardial infarction did not differ between the
groups. ECG evidence of atrial and ventricular
enlargement occurred equally in the two groups.

**Discussion**

In the outpatient pulmonary rehabilitation pro-
gram at the Cincinnati VAMC, 25 of 77 patients with
COPD referred for deteriorating functional status
had left ventricular dysfunction (LVEF <40%) and
92% of these had wall motion abnormalities. Patients
with COPD and LVD reported more severe dyspnea
and were both statistically and clinically more lim-
ited in their activities. An MRC score of 2.0 signifies
that the subject is unable to keep up with peers
and/or stops on level ground at his or her own pace,
while a score of 3.0 signals dyspnea limiting walking
to 100 yards (92 m) or less. However, physical find-
ings and symptoms alone or in combination did not
differentiate them from those with COPD and nor-
mal left ventricular function, nor did chest radio-
graph, ECG, and PFTs.

These data do not support statements made in
several texts that suggest that the cause of dyspnea
can be easily separated into cardiac and pulmonary
causes by clinical criteria.3,4 Left ventricular dys-
function can be suspected in individuals who have
disproportionate dyspnea and unexpected exercise
limitation for their degree of airway obstruction.
Others have shown that the relationship of FEV1 to
exercise capacity is variable. In our study, only
objective measurement of cardiac function identified
patients with cardiac dysfunction. Our patients were
a selected population: male smokers with COPD and
recent functional deterioration and thus the true
prevalence of cardiac dysfunction in COPD requires
prospective study.

All patients studied had a recent reduction in ex-
ercise capacity and were referred to the pulmonary
rehabilitation clinic for management of their COPD;
the diagnosis of LVD was not suspected by their re-
ferring physician. The conventional teaching is that
progression of symptoms in COPD is insidious while
symptoms in CHF are more abrupt.3,4 Cardiopul-
monary symptoms and signs were found in equal
proportions in both groups. Paroxysmal nocturnal
dyspnea, orthopnea, and chest discomfort are de-
scribed in both COPD and in symptomatic LVD.
Although some note that chest pain is uncommon in
COPD,13 more than half of our patients complained
of some type of chest discomfort. The chest pain was
frequently described as a chest tightness, nearly
always sequela of breathlessness. The overlap in
physical findings is also not surprising. Both cor pul-
monale with right ventricular failure and decomp-
ensated left heart failure may cause edema and an
S3 gallop, rales, and wheezing.

All our patients had chronic airway obstruction by
study design, but PFT patterns failed to separate the
group of patients with COPD and LVD from those
with COPD and normal left ventricular function,
despite differences in degree of dyspnea. Airway
obstruction with airtrapping and a reduced DCO
were usual in both groups; we did not observe the
restricted volumes and obstructive spirometry de-
scribed in acute heart failure.7

Routine PFTs do not evaluate aspects of cardio-
respiratory function that best correlate with impor-
tant mechanisms of “cardiac” dyspnea. The reduced
exercise capacity of patients with LVD is multifac-
torial; left ventricular failure limits cardiac output
during exercise, inducing a metabolic acidosis.14
Respiratory muscle weakness and hypoxia combined
with altered airway reactivity during exercise may
result in a ventilatory limitation.8,15 Abnormal skel-
etal muscle metabolism,15 abnormal behavior of
pulmonary and peripheral arterial circulations dur-
ing exercise,16 and respiratory muscle dysfunction,15,17
are now considered important factors in restricting
exercise tolerance in patients with symptomatic
LVD. As yet, no simple technique is known to be
useful in detecting serial changes in respiratory
muscle function. Our patients do not meet standard
definitions for decompensated LVD; and we do not
know what extent LVD played in their symptoms. All
patients in our study were outpatients with relatively
stable conditions and their MUGA and PFT evalua-
tions were performed at rest. The clinical differences
between the two groups might have been accentu-
ated had they been in the midst of an acute exacer-
bation of dyspnea or had invasive exercise testing
been performed.

Radiographic findings classically associated with
cardiovascular disease were rare in the group with
LVD. Experimental studies show that an acute
increase of 35% in extravascular lung water is
required to demonstrate radiographic evidence of
lung congestion.18 In chronic severe LVD, the chest
radiograph is a poor predictor of pulmonary capillary wedge pressure. Oth-
eras have described that radiographic signs of pulmonary edema are uncom-
mon in patients with COPD presenting to an emergency department for evaluation of dyspnea. Chronic in-
creases in lung water might have been insufficient to detect on the radiograph yet influenced lung me-
chanics and the perception of dyspnea. Because pa-
patients were seen in the outpatient clinic, chest films
were not necessarily contemporaneous with the ra-
dionuclide evaluation and parenchymal findings
therefore may have been less apparent. However, no
significant clinical event separated the two studies.
Neither cardiac size (Fig 3), usually a feature of
chronic LVD, nor cardiothoracic ratio was helpful in
identifying those with significant LVD.
We believe that reduced cardiac function in our
patients with COPD was most likely the result of
primary cardiovascular diseases and not an effect of
chronic pulmonary disease. The high incidence of
regional wall motion abnormalities noted in the
group with LVD is a diagnostic hallmark of coronary
artery disease. The lower average RVEF in those
with COPD and LVD is more likely linked to LVD
than pulmonary vascular disease. RVEF and LVEF
correlated well whereas PFT, arterial blood gases,
Deo, and exercise-induced oxygen desaturation, fac-
tors influencing the development of cor pulmonale,
were similar in both groups. Impaired right ventric-
ular function may limit exercise tolerance in LVD.

Unsuspected left ventricular disease in patients
with COPD has been reported previously. Steele et
al, reported that one third of out patients and one
half of inpatients with COPD had LVEF<55 per-
cent. Like our population, their patients were male
veterans who smoked and thus may have had an in-
creased prevalence of cardiac disease compared with
the general population. Their selection criteria dif-
fered from ours and their series did not evaluate the
utility of standard clinical evaluation. Unsuspected
abnormalities in coronary perfusion, identified using
thallium scanning, have been associated with pro-
longed mechanical ventilation. Earlier epidemi-
ologic studies of the natural history of COPD done
prior to technology of radionuclide scanning or ech-
ocardiography identified coronary artery disease
clinically as a significant cause of death. The study of
Burrows and Earle from 1969 found 5% and 10%
of patients died of acute myocardial infarction or
sudden death, respectively, while 34% of patients
died of cardiorespiratory failure with no obvious precipitating factor. The Veterans Administration
Cooperative Study, after excluding patients with
overt cardiac disease, found that 30% of patients with
an FEV₁ greater than 1.49 complained of dispropor-
tionate dyspnea (unable to walk more than 100 yards
(92 m) on the flat without resting, or breathless on
talking or undressing, or housebound). We specu-
late that some of those patients with disproportionate dyspnea and those with no obvious precipitating
cause for their cardiorespiratory death may have had
unsuspected LVD.

CONCLUSIONS

Our findings indicated that LVD is common in a
subset of referral patients with COPD whose func-
tional capacity deteriorates, and that the LVD con-
tributes separately to exercise intolerance. Only ob-
jective measures of ventricular function identified
individuals with both COPD and LVD. This was an
observational, retrospective analysis on a male refer-
ral population. Our conclusions apply only to this
group of patients in whom the prevalence of LVD
would be expected to be higher. The prevalence of
this problem in an unselected population, the most
cost-effective approach to the problem of coexisting
disease, and the relative benefits of therapy in this
population will need to be evaluated in a prospective
fashion. We speculate that these patients may be
helped substantially by vasodilators, especially an-
giotensin-converting enzyme inhibitors, whose estab-
lished benefits include improved quality of life,
reduced number of hospitalizations, and reduced mortal-
ity. In this population, therapy of LVD might reduce airway obstruction and improve symp-
toms. In contrast, with the exception of oxygen ther-
apy, no drug intervention prolongs life in COPD.
Furthermore, overtreatment with inappropriate and
potentially dangerous agents (sympathomimetics and
corticosteroids) may be avoided in patients whose
primary problem is decompensated LVD.

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