CLINICAL SIGNIFICANCE OF PULMONARY FUNCTION TESTS

Use of Pulmonary Function Tests in the Management of Sarcoidosis*

Richard H. Winterbauer, M.D., F.C.C.P., and John F. Hutchinson, M.D.

The literature on pulmonary function testing in sarcoidosis is critically reviewed. The results indicate that pulmonary function tests are not a reliable means for detecting the presence of parenchymal sarcoidosis, nor do they provide an accurate estimate of the extent of parenchymal disease. There are at present no known pulmonary function criteria that allow the clinician to predict the natural cause of pulmonary sarcoidosis or the response to therapy. The major value of pulmonary function testing is to assess changes in the disease course of the individual patient through sequential measurements. Currently there is no conclusive evidence that measurements of arterial blood gas tensions or pulmonary compliance add significantly to the sensitivity and specificity of the vital capacity and diffusing capacity in the management of sarcoidosis.

Early studies of sarcoidosis emphasized radiologic evidence of pulmonary involvement, and subsequent pathologic studies have confirmed the universal involvement of lung parenchyma even in asymptomatic patients with normal lung parenchyma on x-ray film. In 1940, Bruce and Wassen demonstrated a reduction in vital capacity and total lung capacity in the first pulmonary function studies in sarcoidosis. Subsequent studies have shown that a reduced diffusing capacity, decreased pulmonary compliance, abnormal gas exchange, and abnormal airway function are other physiologic abnormalities associated with pulmonary sarcoidosis.

Current knowledge has thus established that a number of easily measured parameters of pulmonary physiology may be disrupted with the development of pulmonary sarcoidosis. It seems intrinsically logical that measurement of pulmonary function should provide useful information to the clinician in the recognition or treatment or both of sarcoidosis. To be helpful in clinical decision making, measurements of pulmonary function should provide a reliable basis for detection of disease, an assessment of the extent of disease, prediction of the natural history of the disease, measurement of changes in the disease resulting from either natural course or therapy, or all of these. Through a review of the literature, we examined the role of pulmonary function testing with each of these criteria. Recommendation for the use of pulmonary function tests in the management of sarcoidosis were then developed.

DETECTION OF PULMONARY SARCOIDOSIS

We must recognize from the outset that sarcoidosis does not lend itself to ready study of pathophysiologic correlations. The many easily accessible extrathoracic biopsy sites and the benign natural history of this disease combine to minimize the amount of biopsy and necropsy lung tissue available for clinical study. Although we emphasize studies with morphometric grading of lung tissue when available, in most studies roentgenographic estimates of the degree of pulmonary involvement provide the basis of comparison with pulmonary function testing. In any patient with clinical features and extrathoracic biopsy results typical of the disease, a diffuse abnormality of lung parenchyma on chest x-ray film would be accepted by most physicians as evidence of pulmonary sarcoidosis. However, lung biopsies in patients with stage 1 disease have clearly established that parenchymal involvement is universal. In our discussion, we will presume that all patients with sarcoidosis have parenchymal involvement whether they have stage 1, 2, or 3 roentgenographic changes.

Tables 1 and 2 show the frequency of pulmonary function abnormalities in patients with and without abnormal lung parenchyma on x-ray film. These investigators presented their data in a manner that
allowed us to calculate the frequency of abnormal tests. The data in Table 2 on patients with abnormal chest x-ray films comes from the laboratories quoted in Table 1. Individual pulmonary function tests are obviously quite inefficient in detecting sarcoidosis when the chest x-ray film is normal. Only 21 percent of such patients have a reduction in vital capacity and 30 percent a reduction in diffusing capacity (Table 2). The sensitivity of measurements of lung compliance may be greater than the 20 percent noted in Table 1, however. Marshall and Karlish found an abnormal compliance in only one of 44 patients, while the three other studies listed in Table 1 have a combined 45 percent incidence of abnormal compliance.\(^5,18,24,27\) Further investigation is needed to resolve this discrepancy. When vital capacity, DLCO, and compliance are taken collectively, a more sensitive index of the presence of sarcoidosis results. Only four of 18 stage 1 patients described by Sharma et al\(^28\) were normal on all three tests. Once parenchymal sarcoidosis is roentgenographically visible, the frequency of abnormal pulmonary function tests increases significantly. Table 2 shows that nearly two thirds of patients with roentgenographically demonstrable sarcoidosis will have a reduction in either vital capacity, DLCO, or static lung compliance. Approximately one-half of such patients have abnormal gas exchange at rest and 59 percent a PaO\(_2\) of less than 85 mm Hg during exercise. Huang et al\(^9\) found only five patients with a combination of normal vital capacity, normal DLCO, and normal arterial oxygen tension at rest and exercise among 62 sarcoid patients with abnormal lung parenchyma on chest x-ray film. However, Table 2 also shows that any single test may be normal in at least one-third of patients with abnormal x-ray films, again indicating poor sensitivity for detection of disease.

**Correlation With Extent of Parenchymal Disease**

In the past 13 years, four studies have attempted to correlate lung pathology with pulmonary physiologic measurements. Young and colleagues\(^4\) in 1967 graded granuloma density and alveolar septal thickness on open lung biopsies from 22 patients. The correlations between lung histology and specific pulmonary function tests are listed in Table 3. Measurements of vital capacity, carbon monoxide diffusing capacity, arterial blood gas at rest and exercise, and

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**Table 1—Sensitivity of Individual Pulmonary Function Tests in Detecting Pulmonary Sarcoidosis in Patients With Normal Lung Fields on Chest Roentgenogram**

<table>
<thead>
<tr>
<th>Author, Reference</th>
<th>No. Patients</th>
<th>Vital Capacity, &lt;85%</th>
<th>DLCO, &lt;80%</th>
<th>PaO(_2) at Rest, &lt;85 mm Hg</th>
<th>PaO(_2) Exercise, &lt;85 mm Hg</th>
<th>Abnormal Lung Compliance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young et al(^4)</td>
<td>4</td>
<td>4</td>
<td>0</td>
<td>3</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Holmgren, Svanborg(^\text{a}1)</td>
<td>11</td>
<td>2</td>
<td>8</td>
<td>4</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Huang et al(^9)</td>
<td>19</td>
<td>8</td>
<td>6</td>
<td>8</td>
<td>6</td>
<td>NM*</td>
</tr>
<tr>
<td>Sharma, Colp, Williams(^c)</td>
<td>18</td>
<td>7</td>
<td>13</td>
<td>2</td>
<td>NM</td>
<td>5</td>
</tr>
<tr>
<td>Richert, Klocke(^\text{a}1)</td>
<td>26</td>
<td>3</td>
<td>NM</td>
<td>NM</td>
<td>NM</td>
<td>NM*</td>
</tr>
<tr>
<td>Marshall, Karlish(^77)</td>
<td>44</td>
<td>2</td>
<td>2</td>
<td>NM</td>
<td>NM</td>
<td>1</td>
</tr>
<tr>
<td>Total (% abnormal)</td>
<td>122</td>
<td>26/(21)</td>
<td>29/(30)</td>
<td>15/(29)</td>
<td>13/(38)</td>
<td>15/(20)</td>
</tr>
</tbody>
</table>

\(^*\)NM indicates not measured.

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**Table 2—Sensitivity of Individual Pulmonary Function Tests in Detecting Pulmonary Sarcoidosis in Patients With Abnormal Lung Fields on Chest Roentgenogram**

<table>
<thead>
<tr>
<th>Author, Reference</th>
<th>No. Patients</th>
<th>Vital Capacity, &lt;85%</th>
<th>DLCO, &lt;80%</th>
<th>PaO(_2) at Rest, &lt;85 mm Hg</th>
<th>PaO(_2) Exercise, &lt;85 mm Hg</th>
<th>Abnormal Lung Compliance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young et al(^4)</td>
<td>18</td>
<td>17</td>
<td>6</td>
<td>7</td>
<td>9</td>
<td>13</td>
</tr>
<tr>
<td>Svanborg(^\text{a}1)</td>
<td>26</td>
<td>12</td>
<td>21</td>
<td>16</td>
<td>16</td>
<td>17</td>
</tr>
<tr>
<td>Huang et al(^9)</td>
<td>62</td>
<td>40</td>
<td>43</td>
<td>29</td>
<td>38</td>
<td>NM*</td>
</tr>
<tr>
<td>Total (% abnormal)</td>
<td>106</td>
<td>69 (65)</td>
<td>70 (66)</td>
<td>52 (49)</td>
<td>63 (59)</td>
<td>30 (68)</td>
</tr>
</tbody>
</table>

\(^*\)NM indicates not measured.
dynamic lung compliance showed no statistically significant correlation with the two histologic variables. However, measurements of granuloma density showed a statistically significant correlation with increased tidal volume, increased minute ventilation, and increased anatomic dead space. The thickness of the alveolar septa correlated with increased respiratory rate, increased air velocity index, and abnormal single-breath nitrogen washout.

A subsequent study by Young and colleagues quantitatively graded the tissue obtained by percutaneous lung biopsy from 34 patients with sarcoidosis. Contradicting the earlier study, there was a significant correlation between the reduction in diffusing capacity, reduced PaO₂ with exercise, and degree of lung parenchymal changes (Table 3). There was a great deal of overlap in the range of pulmonary function tests in each pathologic group. The authors concluded that “in any individual patient one cannot predict the degree of lung parenchymal change based on the value of any given function study or roentgenographic change, nor can one predict functional studies from parenchymal changes.”

Open biopsy specimens from 47 patients with pulmonary sarcoidosis were analyzed by Carrington et al. Approximately 80 histologic features were graded for distribution and severity. The authors calculated a “mean interstitial cell index,” which was the mean of weighted numbers quantitating interstitial lymphocytes, interstitial plasma cells, and interstitial monocytes. The mean interstitial cell index exhibited a significant correlation with FVC, DLCO, DLss exercise, and D[A-a]O₂ with exercise. A marked degree of overlap was again obvious. For example, patients with a nearly normal interstitial lung index exhibited a range of from 10 to 30 ml/min/mm Hg in their exercise DLCO. These authors also concluded there were “no significant correlations between roentgenographic and histologic findings.”

The largest group studied consisted of 81 patients with open lung biopsies quantitatively graded for density of granuloma, degree of interstitial pneumonitis, presence of granulomatous angiitis, and severity of parenchymal fibrosis. Granulomatous and interstitial pneumonitis were found in all 19 patients with stage 1 roentgenographic findings, six of whom had normal pulmonary function tests (VC, DLCO, PaO₂ at rest, and PaO₂ after two minutes of exercise). Five patients with roentgenographic stage 2 or 3 disease had normal pulmonary function. Quantitative comparisons among pathologic changes and pulmonary function measurements were done with t tests comparing the mean values of each physiologic measurement for patients with mild, moderate, or extensive overall pathologic changes. The mean values for individual pulmonary function tests were normal in patients with mild degrees of overall lung pathology and became increasingly abnormal with more advanced disease. Each specific pulmonary function test readily differentiated mild overall lung pathology from moderate and severe changes. However, none of the tests could distinguish moderate from severe overall lung pathology. No correlation was found between granulomatous angiitis and pulmonary function tests. The authors concluded, “when stage 1 findings were combined with normal pulmonary function tests only minimal lesions were present. . . . Normal pulmonary function tests may not be sufficiently sensitive for demonstrating minimal pathologic changes in pulmonary sarcoidosis. Nonetheless, the presence of normal pulmonary function tests makes the findings of extensive lesions and diffuse fibrosis unlikely.”

Thus, four studies (each using a different morphometric grading technique) involving lung biopsies from a total of 184 patients have failed to identify a common physiologic parameter that accurately predicts the histologic severity of disease. The results suggest that an increased respiratory rate at rest may correlate as well with histologic change as more sophisticated measurements of pulmonary function (Table 3).

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Table 3—Correlation Between Lung Histology and Pulmonary Function Tests in Patients With Sarcoidosis

<table>
<thead>
<tr>
<th>Author, Reference</th>
<th>No. Patients</th>
<th>Biopsy Technique</th>
<th>Vital Capacity</th>
<th>DLco</th>
<th>PaO₂ Rest</th>
<th>PaO₂ Exercise</th>
<th>Static Lung Compliance</th>
<th>Respiration Frequency at Rest</th>
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<tbody>
<tr>
<td>Young et al⁴</td>
<td>22</td>
<td>Open</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td>Young et al⁴</td>
<td>34</td>
<td>Needle</td>
<td>–</td>
<td>+</td>
<td>–</td>
<td>+</td>
<td>NM</td>
<td>NM</td>
</tr>
<tr>
<td>Carrington et al⁷</td>
<td>47</td>
<td>Open</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>NM</td>
<td>+</td>
</tr>
<tr>
<td>Huang et al⁸</td>
<td>81</td>
<td>Open</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>NM</td>
<td>NM</td>
</tr>
</tbody>
</table>

*Minus indicates no significant correlation with histologic severity of disease; +, significant correlation with histologic severity of disease; NM, not measured.
CHANGES IN PULMONARY FUNCTION IN UNTREATED SARCIOIDOSIS

In 1958, Marshall and colleagues17 found a residual reduction in diffusing capacity in five of six patients with complete, spontaneous roentgenographic clearing of a parenchymal infiltrate. The authors concluded that "function is as impaired in patients whose lung fields have cleared as in those with diffuse reticulonodular shadows. This emphasizes that there is no close relationship between the radiographic appearances and pulmonary function in sarcoidosis."

A loose relationship between roentgenographic change and pulmonary function tests does exist, however. Table 4 compares sequential pulmonary function testing in patients with roentgenographic improvement (frequently total clearing) and patients with unchanged or worsening parenchymal involvement. Seventy percent of patients with roentgenographic improvement had a 10 percent or greater increase in vital capacity, while only 30 percent exhibited a significant increase in DLco. No patient had worsening of vital capacity or diffusion capacity in the face of roentgenographic improvement. In contrast, only two of 23 patients with persistent parenchymal disease on chest x-ray film had improvement in vital capacity, and none showed an improved DLco. Persistent parenchymal disease was associated with a falling vital capacity in 26 percent of patients and a reduction in DLco in 43 percent.

The measurement of oxygen saturation at rest and exercise did not provide a more sensitive index of disease activity than the vital capacity and DLco.32 There was no change in hemoglobin saturation at rest or exercise in the five patients with roentgenographic improvement reported by Emirgil et al.32 Also, none of his 11 patients with unchanged or worsening chest roentgenograms had significant hemoglobin desaturation at rest, and only two exhibited desaturation with exercise.32

CORRELATION WITH NATURAL HISTORY OF DISEASE

Pulmonary function tests must also be examined for their ability to predict the disease course. Sarcoidosis is well known for its benign natural history, with most patients showing stability or spontaneous improvement with time, while a minority exhibit progressive lung disease.29-33 Can pulmonary physiologic testing define a quantitative or qualitative pattern that would aid in separating progressive from nonprogressive sarcoidosis?

Colp36 separated 78 untreated sarcoidosis patients into those above and those below 65 percent of predicted normal on initial measurements of vital capacity and diffusion. Sixty-seven patients had a vital capacity initially greater than 65 percent predicted. During a mean two-year, eight-month follow-up, 70 percent of this group showed no change in vital capacity, 4 percent improved, and 26 percent worsened. The 11 patients with a vital capacity less than 65 percent predicted were similar, as 81 percent were unchanged, 19 percent improved, and none worsened. The diffusing capacity showed a greater tendency to spontaneous change. In the 57 patients who had an initial diffusing capacity greater than 65 predicted, the DLco improved in 16 percent, worsened in 42 percent, and remained unchanged in 42 percent. The 20 patients with a diffusing capacity less than 65 percent of predicted normal again were similar, as 20 percent improved, 55 percent were unchanged, and 25 percent showed further reduction. Thus, both the patients above and patients

Table 4—A Comparison of Sequential Pulmonary Function Testing and Roentgenographic Changes in Untreated Stage 2 or 3 Sarcoidosis

<table>
<thead>
<tr>
<th>Author, Reference</th>
<th>No. Patients</th>
<th>Vital Capacity</th>
<th>DLeo</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Increase</td>
<td>No Change</td>
<td>Decrease</td>
</tr>
<tr>
<td>Sharma et al17</td>
<td>5</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Emirgil et al18</td>
<td>5</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Total (% abnormal)</td>
<td>10</td>
<td>7 (70)</td>
<td>3 (30)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author, Reference</th>
<th>No. Patients</th>
<th>Vital Capacity</th>
<th>DLeo</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Increase</td>
<td>No Change</td>
<td>Decrease</td>
</tr>
<tr>
<td>Sharma et al17</td>
<td>12</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Emirgil et al18</td>
<td>11</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>Total (% abnormal)</td>
<td>23</td>
<td>2 (9)</td>
<td>15 (65)</td>
</tr>
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below 85 percent of predicted normal on vital capacity and DLCO showed similar patterns of spontaneous variations in disease, and this arbitrary standard did not serve to separate distinct clinical patterns of disease. Additional studies with emphasis on patients with greater degrees of impairment will be necessary to clarify the issues.

Changes in Pulmonary Function Coincident With Treatment

Controversy remains over the role of corticosteroids in the treatment of pulmonary sarcoidosis. Our discussion will not address this issue, and, indeed, the reader should be careful not to misinterpret this section of our review. We devoted attention to the role of pulmonary function tests in monitoring corticosteroid therapy. The pertinent issue here is the best means of measuring a change with physiologic tests, not whether the frequency or magnitude of change is different in the treated and untreated patient groups. Consequently, the studies reviewed are those that do show a change in pulmonary function with corticosteroid therapy. A significant body of literature that fails to show improved pulmonary function with steroids has been excluded.

The results of individual pulmonary function tests from studies showing improvement with corticosteroid therapy are listed in Table 5. Details of therapy, such as dose and duration, have been purposely deleted as not pertinent to this discussion. Sixty percent of 291 patients with parenchymal sarcoidosis had a 10 percent or greater increase in vital capacity coincident with therapy. The DLCO improved in 49 percent of patients, and there was a 19 percent and 27 percent frequency of improvement in the arterial oxygen levels at rest and exercise, respectively. Sequential measures of compliance have been obtained in only 19 patients, with nine (47 percent) showing improvement. If we are to accept the premise that improvement in pulmonary function tests truly reflects improvement of parenchymal disease, the vital capacity appeared to be the most sensitive index of response, with DLCO and compliance slightly less responsive, while arterial oxygen studies were least sensitive. Table 6 lists the change in pulmonary function tests in 26 patients with roentgenographic improvement in parenchymal disease (not necessarily complete clearing). Two thirds of such patients had an improved vital capacity and one half increased their DLCO. The vital capacity and DLCO rarely decreased in the face of roentgenographic improvement. Arterial oxygen studies were unchanged or worse in more than 90 percent of patients and correlated poorly with roentgenographic improvement. A single study suggested that measurement of static compliance raveled the vital capacity in its correlation with the x-ray film, but observations are available for only six patients. Despite an unchanged or progressively abnormal chest x-ray film, pulmonary function test results frequently improved during steroid therapy. Forty-eight percent of such patients increased their vital capacity, 41 percent increased the DLco, and 26 percent and 30 percent of patients, respectively, increased their rest and exercise oxygen levels (Table 6).

Summary and Recommendations

This synopsis summarizes information that we believe is pertinent to the rational use of pulmonary

<table>
<thead>
<tr>
<th>Table 5—Improvement in Pulmonary Function Tests Coincident With Corticosteroid Therapy*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Author, Reference</strong></td>
</tr>
<tr>
<td>Colp et al**</td>
</tr>
<tr>
<td>Johns et al**</td>
</tr>
<tr>
<td>Emirgil et al**</td>
</tr>
<tr>
<td>Sharma et al**</td>
</tr>
<tr>
<td>Stone and Schwartz**</td>
</tr>
<tr>
<td>Boushy et al**</td>
</tr>
<tr>
<td>Smellie et al**</td>
</tr>
<tr>
<td><strong>Total (%)</strong></td>
</tr>
</tbody>
</table>

*NM indicates not measured.
function tests in the management of sarcoidosis.

1. Eighty percent of patients without roentgenographic evidence of parenchymal sarcoidosis will have a normal vital capacity, and 70 percent will have a normal DLco.

2. Thirty-five percent of patients with roentgenographic evidence of parenchymal sarcoidosis will have a normal capacity, and 34 percent a normal DLco. Most patients with a reduction in vital capacity will also have a reduced DLco.

3. Pulmonary function tests have a poor correlation with histologic severity of disease but, in general, tend to sort the extremes in separating mild from severe illness.

4. There is no known difference in the natural history of disease between stage I sarcoidosis with normal lung function and stage I sarcoidosis with abnormal lung function.

5. There are no known pulmonary function criteria that allow the clinician to predict the natural course of parenchymal sarcoidosis or response to therapy.

6. The vital capacity and DLco share a common direction of change on sequential testing in two-thirds of patients with parenchymal sarcoidosis. The remaining one-third of patients show a change in only one of the two measured functions. The DLco and vital capacity will change in opposite directions in less than 5 percent of patients.

7. Sixty-seven percent of patients with roentgenographic improvement in parenchymal sarcoidosis (either spontaneously or with corticosteroids) have a coincident increase in vital capacity, and 45 percent will increase their DLco. Less than 5 percent of patients with roentgenographic improvement will exhibit a coincident reduction in either vital capacity or DLco.

8. Virtually all untreated patients with roentgenographically stable or worsening sarcoidosis will show no change or a decline in the vital capacity and DLco. In contrast, 48 percent of patients with roentgenographic stability during corticosteroid therapy will increase their vital capacity, and 41 percent demonstrate an increase in DLco.

9. At present, there is no conclusive evidence that measurements of arterial blood gas tensions or pulmonary compliance add significantly to the sensitivity and specificity of the vital capacity and DLco in detecting the presence of parenchymal sarcoidosis, assessing the extent of disease, predicting the natural history of the disease, or measuring changes in the disease course.

The fragmentary state of current knowledge does not provide an adequate basis for formulation of precise, all-encompassing rules about the use of pulmonary function tests in the management of sarcoi-
dosis. The authors' experience and bias must play a large role in formulating recommendations. The following observations from our personal experience were thought to be important in formulating guidelines for the use of pulmonary function testing.

1. Clinicians often overemphasize the value of pulmonary function testing in the management of sarcoidosis.
2. Pulmonary function measurements by themselves can rarely, if ever, be immediately translated into clinical decisions.
3. Pulmonary function data should be correlated with roentgenographic and symptomatic information to best facilitate clinical decision making.
4. The greatest clinical value of pulmonary function tests is to assess changes in the disease course through sequential measurements, comparing an individual with himself through time.
5. Patient comfort and cost are important considerations in formulating a pattern of pulmonary function use.

With considerable temerity, we offer the following schema for the clinical use of pulmonary function tests in sarcoidosis (Fig 1).

Our recommendations use simultaneous measurements of vital capacity and DLco. Clinicians frequently order a profile of pulmonary function tests that include some combination of lung volumes, diffusion, and gas exchange. The interpretation of multiple tests may be difficult, however, in estimating improvement in the individual patient. For example, Emirgil et al measured vital capacity, exercise DLco, and hemoglobin saturation at rest and exercise in 22 patients with parenchymal sarcoidosis given corticosteroids. Reducing their tabular data to a simple increased, unchanged, or decreased in the measured function exemplifies the dilemma. Only four patients showed similar results in all four measurements (two improved in all categories, and two were unchanged). Fourteen patients showed similar changes in vital capacity and DLco (ten increased, two unchanged, and two decreased). In four patients, the vital capacity increased or decreased with an unchanged DLco, three patients had changes in the DLco with a stable vital capacity, and significant changes in opposite directions were seen in the vital capacity and DLco of one patient.

The interpretation of disparate results remains an enigma. The temptation is to believe that a patient with improvement in multiple parameters has had a more favorable change in his disease than a patient with improvement in a single test. When the vital capacity and DLco are disparate in direction and magnitude of change, there is currently no basis from which to select which test is more accurate in reflecting disease course. Although it is inherently more comforting to the clinician to have two corroborative laboratory results than one, we harbor the suspicion that future studies may show the vital capacity alone to be equal to the combination of vital

![Figure 1. Suggested use of pulmonary function tests in sarcoidosis.](http://journal.publications.chestnet.org/pdfsaccess.ashx?url=/data/journals/chest/21174/)

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capacity and DLCO as an aid in the management of sarcoidosis. Pulmonary physiologists may pale at this thought, but the suspicion remains viable in the face of present knowledge.

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