nosocomial infection. With this in mind, transbronchial biopsy may be used when the diagnosis of ARDS is in question and the patient may have a potentially treatable condition presenting with diffuse pulmonary densities and hypoxemia, but not necessarily for identifying pulmonary fibrosis.

If the diagnosis of fibroproliferation is entertained clinically or confirmed histologically, can the process be halted or reversed? At present, there is no recognized treatment of fibroproliferation in patients with late ARDS. However, our group and others have reported a favorable response to a sustained course of glucocorticosteroids administered as rescue treatment in patients receiving prolonged mechanical ventilation who fail to improve their lung function. Several large reports have consistently shown that inability to improve gas exchange by day 7 of mechanical ventilation in patients with ARDS carries a poor prognosis, with a mortality rate as high as 97%. Our patients had no improvement in gas exchange and lung mechanics for an average of 15 ± 7 days of mechanical ventilation. Rapid improvement in lung injury score was seen in patients without end-stage fibrosis (by open-lung biopsy) or liver failure. The three patients failing to improve lung injury score died, while mortality was only 14% in the 22 responders, indicating that reversal of fibroproliferation improves outcome. Surveillance cultures for early recognition and treatment of nosocomial pneumonia was an integral part of our protocol and may have contributed to improved outcome. Efforts are in progress to create a multicenter (blinded and randomized) study to clarify the role of antiinflammatory-fibrotic treatment of fibroproliferation in patients with late ARDS. Unfortunately, industry enthusiasm for supporting trials investigating glucocorticosteroids rapidly vanished once these drugs lost their profitability. It is my hope that adequate support can soon be found to investigate the only treatment option shown so far to reverse the leading contributor to morbidity and mortality in patients surviving the early phase of ARDS.

G. Umberto Meduri, MD, FCCP
Memphis, Tennessee

REFERENCES
2 Meduri GU, Headley S, Kohler G, et al. Persistent elevation of inflammatory cytokines predicts a poor outcome in ARDS: plasma IL-1β and IL-6 levels are consistent and efficient predictors of outcome over time. Chest (in press)
5 Meduri GU. Late adult respiratory distress syndrome. New Horizons 1993; 1:563-77

Does Cardiac Dysfunction Cause Pulmonary Restriction?

The association between cardiac dysfunction and abnormal pulmonary function has been known for years. This association has been shown in comparative clinical studies as well as physiologic studies of the relationships between the heart and lungs. However, translation of these observations to clinical practice has lacked quantitative scaling. Although we know that patients with cardiac dysfunction may have pulmonary dysfunction as a result, it has been difficult to say, in an individual patient, that the degree of pulmonary dysfunction observed is, or is not, consistent with the known degree of cardiovascular dysfunction.

In recent years, pulmonary function testing has become increasingly standardized and reliable. Standards have been published for equipment performance, testing techniques, methods for calculating results, maneuver quality control, predicted values, and interpretation of data. Using the best available techniques, it is possible to obtain acceptable and reproducible pulmonary function data in most subjects.

Techniques for noninvasive assessment of cardiac function have also improved in recent years. Cardiac ultrasonography is the most commonly used imaging test for the assessment of cardiac function. With good technique and equipment, reliable results can be obtained from most patients.

The Cardiovascular Health Study (CHS) was developed to determine the clinical correlates of cardiovascular morbidity and mortality in the elderly. A valuable by-product of the study was the opportunity to establish standards of normal cardiac and pulmonary function in the elderly. The CHS is
unique by virtue of the number of participants (5,201) and the quality of both pulmonary function testing and echocardiographic testing. In addition to establishing the range of normal function, it provides the opportunity to compare the results of testing and relate them to disease states.

The study by Enright and colleagues in this issue (see page 28) provides quantitative information, which pulmonary function interpreters have long needed. The question is, to what degree does cardiac dysfunction affect pulmonary function? The answer is, in the case of mild cardiac dysfunction, not very much. That by itself is helpful information.

The authors studied a cohort of elderly (over age 64) subjects who were somewhat healthier than average for that population. They excluded subjects who were current smokers or had smoked more than 20 pack-years. They also eliminated subjects with asthma, chronic bronchitis, and emphysema. The measurements were made at least 3 months after cardiac surgery or myocardial infarction.

In this healthier than average cohort, the authors found slightly decreased values of FEV₁ and FVC in subjects with coronary heart disease, hypertension, or congestive heart failure (greatest for the latter). The effects appear to be additive when more than one condition is present.

If these results are used for interpretation of pulmonary function tests, the degree of reduction in FEV₁ or FVC, compared with a previous baseline study or to a predicted value, may be interpreted in light of these findings. These effects are in the range of the magnitude of day-to-day variation in the same variables. Only a small degree of restriction may be attributed to these conditions. Whether more severe degrees of cardiac dysfunction would cause further reduction in the same variables is not proven by this study, but it is likely. It is unlikely that other studies of this magnitude will be performed in patients with more severe cardiovascular dysfunction. Therefore, future studies of this same cohort may provide those important further observations. Other efforts to ex-

pand the scale of the current observation may be helpful as well.

Other contributing factors to consider in patients with a restrictive pattern in pulmonary function tests may include a pulmonary parenchymal disorder, pleural effusion, chest wall deformity, muscle weakness, obesity, or poor effort. The current study helps give quantitation to what was previously a qualitative interpretation. In light of the data presented, physicians can better determine the importance of different factors contributing to pulmonary function abnormalities.

Paul D. Scanlon, MD Rochester, Minnesota

Division of Pulmonary and Critical Care Medicine, Mayo Clinic and Mayo Foundation, Rochester

REFERENCES