Noninvasive Assessment of Cardiovascular Performance in Interstitial and Chronic Obstructive Lung Disease

The natural history and pathogenesis of cardiovascular performance in patients with chronic interstitial and obstructive lung disease have been difficult to unravel. The absence of appropriate noninvasive quantitative techniques for assessing right and left ventricular structure and function has been a limiting factor. Until recently, quantitative information regarding right ventricular and pulmonary vascular hemodynamics required right heart catheterization. During the past eight years, however, a number of noninvasive radiographic techniques have been developed to assess the cardiovascular status of patients with lung disease, particularly chronic obstructive pulmonary disease (COPD).

It has been established that measuring the widest diameters of the right and left descending pulmonary arteries on plain chest films of COPD patients reveals the presence and relative severity of pulmonary arterial hypertension. Unfortunately, this technique is not generally applicable in patients with diffuse interstitial lung disease and suspected pulmonary artery hypertension due to frequent obscuring of the descending pulmonary arteries on the chest radiograph.

However, radionuclide angiography, another recently developed noninvasive technique, offers considerable promise in assessing cardiovascular function in patients with both obstructive and interstitial lung disease. In this issue (see page 301) Baughman and colleagues describe the application of radionuclide angiography to assess right and left heart performance at rest and exercise in patients with sarcoidosis. Since 1978, this modality has been applied widely in patients with lung disease, particularly COPD, to evaluate right and left ventricular ejection fraction, a measure of systolic pump performance of the heart. Berger et al developed a first-pass technique to assess right ventricular ejection fraction (RVEF) at rest in patients with COPD, and demonstrated that occult abnormalities in right ventricular performance could be detected with this method. Subsequently, Mathay et al utilized this technique to demonstrate an abnormal RVEF response to submaximal upright bicycle exercise in 23 of 30 patients with COPD. Although pulmonary artery catheterization was not performed in this study, it was concluded that...
an abnormal increase in pulmonary artery pressures and pulmonary vascular resistance likely accounted for the failure of RVEF to increase normally with exercise (≥5 percent).4 Mahler and coworkers5 repeated these exercise studies in COPD patients, utilizing both first pass angiocardiography and right heart catheterization. Their study revealed an inordinate exercise-related rise in both mean pulmonary artery pressure (PAP) and pulmonary vascular resistance index (PVRI). Since neither left ventricular dysfunction nor arterial oxygen desaturation occurred in their patients, Mahler et al6 concluded that the increase in right ventricular afterload (ie, ↑ PAP and ↑ PVRI) and the attendant failure of right ventricular ejection fraction to increase with exercise was due to an overall reduction of the pulmonary vascular bed.

Applying alternate radionuclide techniques, gated first pass and equilibrium ventriculography, Baughman et al describe an abnormal response to symptom-limited maximal supine bicycle exercise in 14 patients with sarcoidosis. RVEF failed to increase with exercise in 12 patients; in only two patients, both with only a mild restrictive ventilatory defect, did RVEF increase normally (≥5 percent). There are two plausible explanations for this abnormal exercise response: 1) myocardial involvement with sarcoidosis, and 2) an exercise-induced increase in right ventricular afterload due to sarcoid lung disease. Myocardial injury is unlikely since no cardiac abnormality, such as arrhythmia or overt left or right ventricular failure, was clinically evident. Moreover, left ventricular function was normal in all but two patients. Right ventricular involvement is extremely unlikely in sarcoidosis without concomitant left ventricular disease. Although the authors did not measure central circulatory pressures, it is likely that the right ventricular performance abnormalities noted were due to an increase in right ventricular afterload, namely: an increase in pulmonary artery pressures and pulmonary vascular resistance. In turn, there are at least two explanations for this increase in right ventricular afterload, both dependent upon diffuse pulmonary involvement with sarcoidosis. The first, hypoxic pulmonary vasoconstriction, is possible since arterial oxygen tension (Pao2) dropped with exercise in nine of ten patients studied. However, only three of these ten had an exercise Pao2 level less than 60 mm Hg. The second explanation, obliteration of the pulmonary capillary bed by abnormal parenchymal tissue, is more plausible. The authors show a significant correlation between the decrease in total lung capacity, diffusing capacity and the exercise right ventricular ejection fraction. In a more invasive study of interstitial lung diseases including sarcoidosis, Enson et al8 found no relation between the level of pulmonary vascular resistance and alveolar oxygen tension; they concluded that vasoconstriction does not contribute significantly to the observed increase in resistance to pulmonary blood flow. Instead, they found that with vital capacities above 50 percent of predicted, chronic inflammatory changes in the alveolar septa and interstitium cause the increase in pulmonary vascular resistance; at lower levels of vital capacity, fibrosis dominates the picture. Thus, Enson and colleagues9 postulate that compression and obliteration of the vascular bed is the primary reason for the increase in right ventricular afterload.

Brent et al10 described preliminary data which suggest that in COPD, resting right ventricular ejection fraction correlates well with mean pulmonary artery pressure. If similar data obtained at rest and/or exercise in interstitial lung diseases such as sarcoidosis show a good correlation between radionuclide angiocardiography and mean pulmonary artery pressures or pulmonary vascular resistance, radionuclide angiocardiography may prove useful clinically in serially assessing the cardiovascular status of these patients.

Richard A. Matthay, M.D., F.C.C.P.; and Jacob Loke, M.D., New Haven

Pulmonary Section, Department of Internal Medicine, Yale University School of Medicine.

REFERENCES


