EDITORIALS

The Chest Roentgenogram
Its Role in Evaluating Cardiomegaly and Chronic Obstructive Pulmonary Disease

The usefulness and limitations of the chest x-ray film in the evaluation of both cardiac size and chronic obstructive pulmonary disease are often poorly understood. The problem is compounded if cardiomegaly is to be evaluated in the presence of chronic obstructive pulmonary disease because of its well-known effects on the configuration of the heart. An additional limiting factor is the perceptual and cognitive difficulties in the interpretation of chest roentgenograms.1-2

Numerous radiologic signs are reported to be useful in the evaluation of chronic obstructive pulmonary disease, including (1) increased generalized or localized radiolucency, (2) widened intercostal spaces, (3) flattened diaphragm and increased anteroposterior diameter, (4) wide costophrenic angles, (5) enlargement of the pulmonary conus and hilar pulmonary arteries, and (6) increased clarity and decreased diameter of pulmonary vessels of the second and third order. From recent studies, it is apparent that the only reliable sign of chronic obstructive pulmonary disease is flattening of the domes of the diaphragm.3-4 The degree of diaphragmatic flattening correlates well with the measured radiographic total lung capacity. The radiographic total lung capacity, in turn, shows excellent agreement with the values for total lung capacity obtained by helium dilution and bodyplethysmographic studies.5 In essence, therefore, the radiologic assessment of chronic obstructive pulmonary disease is limited to estimation of the total lung capacity, which will be increased in patients with moderate to advanced emphysema; however, the vast majority of patients with chronic obstructive pulmonary disease, including those who have symptoms of chronic bronchitis, will have normal chest x-ray films.4

The most commonly used radiologic sign of cardiomegaly is an increased transverse diameter of the heart, as seen on the frontal chest film. It was suggested as early as 1919 that the transverse diameter of the heart should be compared with the total transverse diameter of the thorax; and, thus, the concept of the cardiothoracic ratio was introduced.6 The assumption is that a cardiothoracic ratio greater than 0.5 indicates cardiomegaly, primarily left ventricular enlargement. In recent studies, the cardiothoracic ratio was correlated with the angiographic left ventricular end-diastolic volume. It was found that in moderate enlargement of the left ventricular end-diastolic volume, the cardiothoracic volume may be less than 0.5. Only in patients with marked left ventricular enlargement (left ventricular end-diastolic volume more than 125 ml/sq m) was the cardiothoracic ratio greater than 0.5.7

The evaluation of cardiomegaly is even more difficult in patients with moderate to severe emphysema, because the diaphragm is in a lower position and the heart, which is suspended in the thorax, will be “stretched,” decreasing the apparent cardiothoracic ratio. In this issue of Chest (see page 712), Murphy et al report that patients with advanced chronic obstructive pulmonary disease and autopsy-proven cardiomegaly may have a cardiothoracic ratio below 0.5. Computerized feature extraction is a potentially useful approach in improving the assessment of cardiac size on the chest x-ray film of patients with chronic obstructive pulmonary disease. By this method the cardiac size can be corrected for the level of the diaphragm.

The chest x-ray film in patients without chronic obstructive pulmonary disease is a good indicator of pulmonary hemodynamic events and provides a reasonable estimate of pulmonary venous and arterial pressures. Unfortunately, in patients with chronic obstructive pulmonary disease, the pulmonary vascular distribution may be significantly altered. This makes roentgenographic evaluation difficult.

It is apparent that the radiologic method does not detect early and potentially reversible chronic obstructive pulmonary disease.4 On the other hand, the method permits recognition of moderate to ad-
Tricuspid Stenosis Secondary to Pericardial Disease

Most medical students, from their earliest exposure to heart disease, are taught that tricuspid stenosis, in the absence of rheumatic mitral or aortic disease, does not occur as an isolated entity. The known exceptions are malignant carcinoid, intracardiac tumors, endocarditis, and external cardiac compression.

In this issue of Chest (see pages 770 and 772), there are two reported cases of tricuspid stenosis caused by disease of the pericardium, and in both cases the pericardial disease is well documented. In one case the pericardial disease was believed to be due to rheumatoid etiology, and the other case was thought to be viral infection. Both cases have reports from either autopsy or surgical pathologic specimens documenting the disease. Both cases demonstrated a clear-cut limitation in the tricuspid valvular orifice of 2 sq cm and 1.4 sq cm, respectively. The one patient who survived has done well after surgical intervention. There have been other reports in the literature of chronic pericardial disease involving the atrioventricular groove, in which constriction takes place. To this date, there have been only five previously reported cases of tricuspid disease secondary to this phenomenon, and the cases reported in this issue of Chest are the sixth and the seventh such cases reported.

Tricuspid stenosis occurs in 5 to 10 percent of patients with rheumatic disease of the mitral valve. There has only been one case in the literature that may represent tricuspid stenosis occurring without mitral stenosis in a patient who was thought to have a rheumatic etiology, although this patient later developed mitral stenosis. One could question whether adequate proof exists that tricuspid stenosis preceded the mitral stenosis. The occurrence of cases of rheumatic tricuspid disease has a predominant female-to-male ratio; however, the two cases reported in this issue occurred in men.

As our ability to detect occult valvular disease increases with medical advances, we may find that there are many other causes of tricuspid valvar disease that were heretofore not well recognized. It would appear that more and more atypical constrictive pericardial processes are beginning to emerge as more and more median sternotomies are being performed. It has been interesting to watch the course of development of this operation where, initially, much of the pericardium was left open, and the anterior mediastinal space was not packed with tissue, and how the heart, especially the right side of the heart, at times would become entrapped. Now, more and more measures are being employed to see if entrapment can be prevented. The use of mediastinal radiation for Hodgkin’s disease and other malignant diseases may increase the amount of constricting pericardial disease. One will have to be on guard for the development of pseudotricuspid stenosis as a result of these kinds of therapy.

On physical examination, the hallmark of tricuspid stenosis, besides the diastolic murmur, is an elevated venous pressure with an increased “a” wave and a delayed “y” descent noted on a tracing of the jugular pulse. With the advent of other noninvasive techniques, such as echocardiography, we may begin to recognize more definitive patterns of tricuspid valvular motion. At present, it is very difficult to distinguish echocardiographically a pattern of tricuspid valvular motion that is specific for tricuspid stenosis. With the advent of echocar-