Competence of the Tricuspid Valve in Pulmonary Heart Disease (Cor Pulmonale)

By Warren T. Sherman, M.D., M. Irené Ferrer, M.D., and Réjane M. Harvey, M.D.

The alleged occurrence of tricuspid regurgitation in association with, and as a physiologic consequence of, right ventricular failure in cases of cor pulmonale has been reported during the past decade. Emphasis has been laid upon hepatic pulsations, distended or pulsating neck veins, and a systolic murmur of appropriate timing and placement as evidence of this phenomenon. It has been suggested that the appearance of these findings should alert the clinician to the presence of this lesion. It has also been reported that in patients with cardiac failure a progressive change in the form of the right atrial pulse suggesting tricuspid incompetence occurred at a right atrial mean pressure of approximately 5 mm. Hg.

Several methods have been employed to detect regurgitant flow across the atrioventricular valves. Most depend upon changes in the contour of the atrial pressure curve, or upon the demonstration of regurgitation by indicator technics or visualization of reflux of radiopaque material. Impairment of the X descent on the right atrial pressure curve has long been utilized as an index of tricuspid regurgitation. The absence of the X descent is presumably produced by the transmission of regurgitant blood flow during isovolumic contraction of the ventricle when closure of the AV valve is incomplete. This change in contour of the atrial pressure pulse as a reflection of valvular regurgitation has not been questioned, although attempts to quantitate the degree of regurgitant flow from the pressure pulse have been queried. It is the purpose of this investigation to explore the incidence of tricuspid regurgitation as judged by the right atrial pressure pulse in a group of patients with right ventricular disease in normal sinus rhythm.

Materials and Methods

The right atrial pressure curves of all patients with cor pulmonale studied by cardiac catheterization in this laboratory since 1943 were examined. All patients met the criteria of pulmonary heart disease (cor pulmonale) as outlined by the New York Heart Association. The range of pulmonary diagnoses included chronic bronchitis, bronchiolitis and emphysema, sarcoidosis and other granulomatous lesions of unknown etiology, multiple pulmonary thromboemboli, fibrosarcoma with diffuse interstitial fibrosis, and pulmonary tuberculosis with extensive fibrosis. The presence of clinical right ventricular failure at the time of cardiac catheterization was determined from case summaries.

Patients were studied under a variety of conditions during the course of other investigations. Pressure pulses were recorded either from Hamilton manometers or in the great majority of instances from Statham gage-pressure transducers with a photo-oscillographic method of recording. The reference level for the pressure determinations was 5 cm. below the angle of Louis.

The right atrial pressure curves of the 134 catheterizations performed on 98 patients were examined by two or more members of this laboratory. Seventy right atrial tracings (70 separate studies in 55 patients) were judged to be of sufficient amplitude and quality to be meaningfully
interpreted. All curves were accompanied by a simultaneously recorded electrocardiogram. A right atrial pressure curve was considered to show normal tricuspid function when a fall in pressure (X descent), below the level prior to atrial systole, occurred with the onset of ventricular ejection. Any degree of impairment of the X descent to a level not definitely below that prior to atrial systole was considered as evidence of tricuspid regurgitation.

Various hemodynamic and clinical data were obtained for each of the 70 right atrial pressure curves studied. The right atrial and pulmonary artery mean pressures were measured by the planimetric method. Right ventricular end-diastolic and pulmonary artery mean pressures were those recorded closest in time to the right atrial pressure tracing and in most instances all three determinations were measured during withdrawal of the cardiac catheter from the pulmonary artery into the atrium. In no instance were the right ventricular or pulmonary artery pressure values included in the data if determined under different physiologic conditions from those during the recording of the atrial curves.

The cardiac output for each study was measured in almost all cases by the direct Fick prin-

Figure 1
Pressure curves from a patient with cor pulmonale whose right atrial mean pressure was 9 mm. Hg. The P-R interval measured 0.24 second. The onset of ventricular systole is indicated on the atrial curve at which time there is a rise in atrial pressure.

ciple, although in some instances flow was obtained by dye-dilution technics with T-1824 or by a radiocardiographic method with radio-iodinated human serum albumin. This measurement was not included unless determined under the same physiologic state as were the right atrial pressure curves.

Results

Of the 70 right atrial pressure curves selected for analysis, only one showed a pattern of tricuspid regurgitation as evidenced by a rise in pressure coincident with the onset of right ventricular ejection (fig. 1). This exception was accompanied by a prolonged P-R interval (0.24 second). The remaining 69 pressure tracings were interpreted as showing normal tricuspid function, with a definite fall in pressure to a level below that prior to atrial systole.

The distribution of 68 right atrial mean pressures of the 70 right atrial curves included for analysis appears in figure 2. The mean pressures could not be determined for two pressure curves as calibration of the manometers was no longer available. The right atrial mean pressures encountered ranged from −4 to 18 mm. Hg; 24 per cent exceeded 5 mm. Hg, the upper limit of normal accepted by this laboratory. The patient who demonstrated tricuspid regurgitation had a right atrial mean pressure of 9 mm. Hg.

The distribution of pulmonary artery mean

Figure 2
Distribution of right atrial mean pressures in 68 studies. Of these 24 per cent exceeded 5 mm. Hg. The patient with tricuspid regurgitation had a right atrial mean pressure of 9 mm. Hg., as indicated by the black area.

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 Similarly represented in figure 4. This value was recorded in 55 studies and ranged from -1 to 22 mm. Hg. In 38 per cent of the cases it exceeded 5 mm. Hg. The patient who demonstrated tricuspid regurgitation had a right ventricular end-diastolic pressure of 11 mm. Hg.

Figure 5 illustrates the wide range of blood flow encountered. Thirty-one per cent of the 59 determinations were above 3.5 L./min./M.² BSA and 22 per cent fell below 2.7 L./min./M.² BSA. Differently stated, in 78 per cent of the cases the cardiac output was at normal or higher than normal levels. The patient with tricuspid regurgitation had a cardiac output of 3.1 L./min./M.² BSA.

An estimation of the presence of right ventricular failure at the time of cardiac catheterization was made in 67 of the 70 studies. Forty-six per cent of the cases were clinically in right ventricular failure, 42 per cent had been in clinical failure some time prior to study, the remaining 12 per cent never having manifest evidence of peripheral venous congestion. The case with tricuspid regurgitation was in clinical failure at the time of cardiac catheterization.

The right atrial pressure curves of two patients with severe right ventricular failure are reproduced in figures 6 and 7. In neither is there evidence of tricuspid regurgitation, despite the high levels of the mean pressure and the large pulse pressure during atrial
systole. This change in the character of the atrial pressure pulse is attributed to altered pressure-volume relationships in a congested and hence poorly distensible system.4

Discussion

As is apparent from the results the incidence of tricuspid regurgitation in patients with cor pulmonale is extremely low. There was but one right atrial pressure curve that demonstrated a regurgitant pressure wave despite a wide range of right atrial mean, right ventricular end-diastolic, and pulmonary artery mean pressures, and despite the presence of clinical congestion at the time of study in 46 per cent of the cases.

The one patient who showed tricuspid regurgitation had a prolonged P-R interval (0.24 second), an unusual finding in cor pulmonale alone,2 hence implying a separate defect which existed by chance in this one subject. In view of recent knowledge of the hemodynamic effects of misplaced atrial systole,16 namely, the appearance of AV valvular regurgitation after prolongation of the atrial systole-ventricular systole interval in dogs, it is possible that in this patient the delay between atrial and ventricular systole was responsible for the loss of complete closure of the tricuspid valve and the resultant pattern of tricuspid regurgitation.

The commonly cited causes of tricuspid regurgitation include distortion of the valve leaflets, shortening of the chordae tendineae and disease of the papillary muscles, dilatation of the valve annulus, and absent or misplaced atrial systole. In the group of patients with cor pulmonale included in this report, there were several individuals who, subsequent to these studies, showed striking radiologic evidence of rapid reduction in the size of the right atrium and ventricle, a reduction most certainly attributable to reversal of chamber dilatation concomitant with therapy. One might have expected to see an occasional patient with a dilated annulus and consequent tricuspid incompetence, but evidence for this was absent.

The levels of pressures in the right atrium, right ventricle, and pulmonary artery were similar to those encountered by other investigators in reports on tricuspid insufficiency and incompetence.1,3,17 Muller and Shillingford1 included three patients with pulmonary heart disease among a group of 21 patients who were purported to have tricuspid incompetence. Thirteen had rheumatic heart disease and all but four were in atrial fibrillation. It is not clear from their data, however, if the patients with cor pulmonale had sinus mechanism or atrial fibrillation. Similarly, of
the 60 cases of tricuspid insufficiency reported by Sepulveda and Lukas from a group of patients with rheumatic heart disease, 96.7 per cent had atrial fibrillation.

The high incidence of the pressure pattern of tricuspid regurgitation as a hemodynamic consequence of atrial fibrillation has been described by Ferrer, Harvey, and others. More recently Daley and Skinner, using experimental technics in dogs, demonstrated mitral regurgitation as a consequence of atrial fibrillation. The pressure pattern is presumed to be due to the absence of atrial systole with consequent impaired closure of the AV valves immediately prior to ventricular contraction. Under these circumstances one need not invoke dilatation of the annulus as a cause of valvular incompetence nor infer that disease of the valve leaflets or their anchoring mechanisms had produced tricuspid insufficiency.

Although other technics may eventually reveal the presence of tricuspid regurgitation in the absence of impairment of the X descent, such evidence is lacking at the present time. Therefore, in view of the rare occurrence of tricuspid regurgitation in patients with cor pulmonale and sinus rhythm, with or without peripheral venous congestion, it is suggested that the clinical signs, often attributed to this lesion, might better be ascribed to a sudden reversal of flow from atrium to veins, which is consequent to the large pressure variations originating in a congested right atrium.

**Summary and Conclusions**

Seventy right atrial pressure curves obtained by cardiac catheterization of patients with cor pulmonale were reviewed. Only one right atrial tracing showed a regurgitant pressure wave, despite a wide distribution of right atrial mean, right ventricular end-diastolic, and pulmonary artery mean pressures, systemic blood flow, and despite the presence of peripheral venous congestion at the time of study in 46 per cent of the cases. This tricuspid regurgitation was probably due to the hemodynamic consequence of a prolonged P-R interval, an unusual finding in cor pulmonale. Hence it can be concluded that uncomplicated cor pulmonale rarely produced tricuspid regurgitation, as judged by the right atrial pressure curve.

The clinical signs often attributed to tricuspid regurgitation when noted in the presence of right ventricular failure are most probably explained by the sudden reversal of flow from atrium to veins, which is consequent to the large pressure variations originating in a congested right atrium.

**References**

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The Spirit of Science

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