A Study of the Venous Pulse in Tricuspid Valve Disease

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Diagnosis of lesions of the tricuspid valve is often difficult. Mechanically, however, the action of this valve is capable of study through its effect upon the peripheral veins, somewhat as the disturbances of the aortic valve are reflected in the peripheral arteries. The conditions to be differentiated are "organic" versus "functional" regurgitation, and obstruction of the valve from rheumatic stenosis. Graphic records of the venous pulse with suitable reference tracings may make this possible.

The presence of an unusually large positive ventricular systolic pulsation in the internal jugular veins has been recognized for years as a sign of tricuspid regurgitation. White and Cooke have suggested that, when this type of pulsation has been present for months or years without significant congestive heart failure, tricuspid stenosis also may be present. The value of noting the degree of venous distention and the presence or absence of venous pulsation is generally appreciated, but it is rare that actual timing of the pulse waves by simultaneous auscultation of the heart is attempted. Such observations can be made without difficulty unless the heart rate is rapid.

The clinical and pathologic features of tricuspid stenosis and regurgitation have been adequately discussed. In all of the cases reported the factors leading to the correct diagnosis ante mortem consisted in a high index of suspicion and the recognition of the peripheral manifestations of this lesion. When other peripheral signs of tricuspid regurgitation are present, i.e., expansile pulse of the liver, ascites, venous engorgement, and edema; the manifestations in the jugular veins will always be present. It is for this reason that careful observations of the character of the pulsation in the jugular veins is one of the most important clues to abnormality of the tricuspid valve.

Description of the normal venous pulse and the technic of graphic recording can be found in the monographs of Wiggers and Groedel. There are three important points in observing or recording the venous pulse. First, the internal jugular, not the external or superficial, is the most suitable vein to use. Second, because this vein lies beneath the sternocleidomastoid muscle it is important that there be no tension in this muscle, for tension can completely obliterate or greatly alter the pulsations. It is best to have the subject's head facing forward or rotated slightly toward the side used for recording or observing. The head should never be rotated greatly as this will cause excessive tension in the neck muscles. Third, the upper half of the patient's body must be elevated to the point where the veins will be seen to empty and fill with the greatest amplitude. This position must be determined individually for each patient.

Venous pulsation can be distinguished from arterial by the general configuration of rounded undulating waves of the former, by the fact that arterial pulsation is easily palpable whereas venous pulsation is not, and, most important, by timing the waves with the heart sounds during auscultation.

The venous pulse in tricuspid regurgitation tends to lift the sternocleidomastoid muscle slowly during the latter part of ventricular diastole; this elevation becomes suddenly more marked during systole and is followed by an abrupt and pronounced collapse of the vein immediately after the second sound. This diastolic collapse of the vein is one of the most striking features of tricuspid regurgitation.

We are reporting 10 patients who presented this type of pulsation. In 6 of the patients tricuspid regurgitation was associated with acute or chronic right ventricular failure and dilatation of the tricuspid valve ring without evidence of organic valvular disease. Four pa-
Patients had rheumatic heart disease with multiple valvular involvement, and, in addition to the abnormal venous pulse, had other peripheral signs of tricuspid valve disease. A diastolic murmur at the lower end of the sternum could be heard or recorded in all patients of the latter group. A comparison of the graphic records of those patients in whom tricuspid regurgitation was associated with dilatation of the right ventricle without stenosis of the

**Fig. 1.—A** (top tracing). Phonocardiographic, venous pulse, and electrocardiographic tracings made during heart failure unassociated with tricuspid regurgitation. A.G indicates presystolic (auricular) gallop; point x indicates the normal systolic collapse.

**B** (middle tracing). Tracings made three and one half months following those shown in **A** after tricuspid regurgitation had developed. The venous pulse shows an absence of the normal systolic collapse which has been replaced by a positive systolic wave, TR, owing to tricuspid regurgitation. The diastolic collapse, point y, is now quite striking.

**C** (bottom tracing). Phonocardiogram and venous pulse tracing, made after the congestive failure had cleared, showing a normal systolic (x) and diastolic (y) collapse.
mm. per second with a Sanborn Tri-Beam Stethocardiette and the venous pulse was re-
proved to be of greater value than the former because it provides the most accurate refer-
ence tracing for the beginning and ending of mechanical systole as well as for other events
during the cardiac cycle.

Fig. 2.—Phonocardiogram, venous pulse record, and electrocardiogram. There is a low energy systolic murmur (SM). The venous pulse shows a plateau-like positive wave following the c wave and replacing the normal systolic collapse; this wave (TR) is due to tricuspid regurgitation and ends in an abrupt diastolic collapse (y) when the tricuspid valve opens.

Fig. 3.—A (top tracing). Phonocardiogram, arterial pulse record, and electrocardiogram. The phonocardiogram shows a low energy systolic murmur (SM) and a low-frequency diastolic murmur (DM) at the apex.

B (bottom tracing). Phonocardiogram, venous pulse record, and electrocardiogram. The phonocardiographic tracing made over the tricuspid area shows a systolic murmur (SM) and diastolic murmur (DM). The venous pulse has a positive systolic wave (TR) due to tricuspid regurgitation. The deep diastolic collapse (y) coincides with the diastolic murmur.

Recorded by means of a crystal microphone.22-24 An electrocardiogram and phonocardiogram were recorded as reference tracings; the latter
Figures 1 and 2 illustrate venous pulse tracing from two patients with right ventricular dilatation and variable tricuspid regurgitation without evidence of stenosis. In figure 1, A, the venous pulse shows no evidence of tricuspid regurgitation although the patient had congestive failure with pulmonary congestion, high venous pressure, and peripheral edema. In figure 1, B, made after the patient had a sudden weight gain of 25 pounds associated with a marked increase in edema and ascites but little increase in dyspnea, the venous pulse shows tricuspid regurgitation. Figure 1, C, made after partial compensation, shows the normal systolic collapse of the venous pulse indicating a return of competence of the tricuspid valve. Figure 2 is an illustration of the venous pulse showing tricuspid regurgitation associated with dilatation of the tricuspid valve ring in another patient.

Figures 3 and 4 are examples of the type of venous pulse tracings we have obtained from patients with rheumatic heart disease and multiple valvular lesions with evidence of tricuspid regurgitation. None of these patients presented acute or recent congestive failure. This type of venous pulse together with chronic enlargement of the liver was present for several months to over a year without marked dyspnea or orthopnea being manifest in these patients.

**Discussion**

The positive late systolic wave is not an exaggeration of any of the normal waves in the jugular pulse but replaces what would normally be a negative wave or collapse of the vein. It is due to the regurgitation of blood through an incompetent tricuspid valve. This transmission to the jugular vein prevents the normal late systolic emptying of the veins into the venous reservoir within the thorax and is responsible for the systolic distention of these veins. At the end of systole there may occur some decline in the positive wave of regurgitation. However, the deep diastolic collapse does not occur until the tricuspid valve opens. This collapse always coincides with the diastolic murmur associated with blood flowing through the valve when tricuspid stenosis is also present.

A comparison between the graphic tracings of the patients with tricuspid regurgitation
due to dilatation of the tricuspid ring and those of patients with organic disease of the tricuspid valve demonstrates certain differences. In the latter group the positive systolic wave begins at a definite interval after the c wave, the rise is slower than in the former group, and the peak tends to occur somewhat later. The diastolic collapse is slower; however, the onset and maximum collapse coincide with the diastolic murmur associated with ventricular filling. The factors responsible for the delay in the rise of the systolic regurgitant wave seem to consist of (1) incomplete filling of the right ventricle due to the valvular obstruction and (2) a similar impedance at the valve during regurgitation, associated with transmission of the wave against a higher atrial pressure. This is shown in figure 4. The second systole occurs relatively early and the delay in the systolic venous wave (1A) is evident. However, the abrupt diastolic collapse occurs with the opening of the tricuspid valve and the onset of the diastolic murmur. When diastole is short, in the presence of mitral or tricuspid valve stenosis, the atria do not have sufficient time to empty and, at the onset of systole, the ventricles are less well filled than under normal conditions. The result is a smaller regurgitant stroke volume against a higher atrial pressure.

In those patients in whom tricuspid regurgitation is due to dilatation of the valve ring there is no mechanical interference in ventricular filling; hence, although the venous and atrial pressures may be high, the systolic wave of regurgitation occurs earlier in systole. In our records from patients with tricuspid regurgitation due to severe right-sided heart failure, the early rise of the positive systolic wave in the venous pulse was a constant feature, although the heart rates were relatively rapid and comparable to the faster rates of those patients thought to have tricuspid stenosis.

Conclusions

1. The normal phlebogram from the internal jugular vein shows a negative wave, due to collapse of the venous pulse in the latter part of ventricular systole, when the veins empty into the thoracic venous reservoirs.

2. Tricuspid regurgitation replaces this negative wave with a positive wave as the veins distend from backflow, and it is followed by an abrupt diastolic venous collapse.

3. When tricuspid regurgitation is combined with tricuspid stenosis there is a delay in the appearance of this positive wave following the c wave, the rise of the wave is slower, the peak occurs later, and the diastolic collapse is slower.

4. These differences in the venous records in "functional" tricuspid regurgitation as contrasted with those from patients with organic tricuspid stenosis are accounted for by impedance effects due to the stenosis.

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