Pulsating Varicose Veins in Functional Tricuspid Insufficiency

Case Report and Venous Pressure Tracing

By Philip W. Brickner, M.D., W. Tracy Scudder, M.D., and Michael Weinrib, M.D.

Tricuspid insufficiency may be caused by organic changes in the tricuspid valve, usually rheumatic, or may be relative to other forms of heart disease. This secondary or functional form of tricuspid insufficiency is particularly common when the pulmonary artery pressure is highly elevated, as in mitral stenosis. The tricuspid valve dilates because of the increase in pressure and size of the right ventricle and thereby becomes incompetent.

Tricuspid insufficiency results in the flow of blood from the right ventricle into the right atrium during ventricular systole. As a consequence the central venous pressure is elevated and the prominent physical signs follow: enlarged liver and distended veins, both of which may be pulsatile; peripheral edema; and ascites. Rarely the spleen may also be pulsatile.

Pulsatile veins in cases of tricuspid insufficiency have been observed throughout the body, largely in the neck, forearm, and forehead. Less commonly other veins may pulsate and cause symptoms. Hildebrand cites the case of a 54-year-old woman with the problem of disturbing pulsations in the buttocks when sitting, which were secondary to tricuspid insufficiency. Contardi reports an instance of pulsating bilateral varicocele in the same condition.

The purpose of this article is to present the unusual case of a patient with pulsating varicose veins of the legs associated with tricuspid insufficiency. In five large series reviewing 204 cases of tricuspid insufficiency and in several well-known textbooks of cardiology no mention is made of this finding. Only 19 similar cases have been reported to date. Of these, 16 are recorded by Teuff, whose case histories are not supplemented by venous pressure tracings. Other cases with this finding are reported by Hildebrand, Sensenbach and Hutzler, and Hallock and Clarke. Table 1 records a summary of these cases.

Case Report

A.S., a 59-year-old Virgin Island-born Negro man with known heart disease, was admitted to the First (Columbia) Medical Division, Bellevue Hospital, on October 26, 1960, for evaluation and possible ligation of varicose veins.

The patient gave a past history of “Spanish influenza” with fever and transient polyarthritis in 1918 and systemic vascular hypertension in 1936, with the onset of atrial fibrillation 2 years later. Severe cardiac decompensation first occurred in 1950, when the patient presented with hypertension, congestive heart failure, and a right-sided cerebral thrombosis. He recovered but had a residual left homonymous hemianopsia. In 1956 the patient was again hospitalized in congestive heart failure for a suspected myocardial infarction. Since that time his blood pressure has remained at normal levels.

The patient was first admitted to Bellevue Hospital in 1957 in severe heart failure with congested lungs, an enlarged heart, atrial fibrillation, a protodiastolic gallop, and mitral and tricuspid insufficiency. He had pulsating neck veins and liver, ascites, and edema of the legs. An electrocardiogram was interpreted as compatible with left ventricular hypertrophy and diffuse myocardial disease. Chest x-rays and fluoroscopy revealed marked right and left heart enlargement. The patient responded well to a cardiotonic and diuretic regimen.
Again in 1958, when a diagnosis of Paget's disease of bone was made, and in 1960 the patient presented in severe right and left ventricular failure with the physical signs of tricuspid insufficiency. A month later he was rehospitalized for thrombophlebitis of the left greater saphenous vein, with subsequent progressive enlargement of varicose veins in both legs.

In October 1960 the patient was admitted from the outpatient department, where he was being maintained on digitalis and diuretics, for surgical evaluation of his varicosities. Physical examination revealed a tall, well-built, slightly dyspneic Negro man with an irregular pulse rate of 82 per minute and a blood pressure of 132/80 mm. Hg. Positive physical findings included extreme myopia, left homonymous hemianopsia, an enlarged heart with atrial fibrillation, a loud, harsh, apical systolic murmur transmitted to the left axilla, and a softer systolic murmur at the lower left sternal border. There were prominent systolic pulsations of the cervical veins and a pulsatile liver with the edge felt four fingerbreadths below the right costal margin. Examination of the lower extremities showed moderate brawny edema of the legs and ankles with stasis dermatitis and pulsating varicosities. The Perthe and Trendelenburg tests revealed occlusion of the deep venous system with incompetency of the communicating veins bilaterally.

Blood count and urinalysis were within normal limits, as were the blood urea nitrogen and electrolytes. A blood Wassermann test was negative. On fluoroscopy a greatly enlarged left ventricle and right atrium were seen, with a moderately enlarged right ventricle and pulmonary outflow tract. An electrocardiogram showed atrial fibrillation and was compatible with left ventricular hypertrophy and diffuse myocardial disease.

Because of the lack of patency of the deep venous system, surgical correction of the varicose veins was deemed inadvisable.

A study was performed (see below) and the patient was discharged to clinic follow-up on digitalis and diuretic therapy. He steadily improved on this regimen, and the overt manifestations of tricuspid insufficiency including the pulsating varicose veins, disappeared.

The etiology of this patient's heart disease remains obscure. Rheumatic heart disease is suspected, with organic mitral insufficiency.

To confirm the finding of pulsating varicose veins of the legs related to tricuspid insufficiency simultaneous electrocardiographic and venous pressure tracings were recorded on a multichannel electronic recorder with a needle in a large varix in the right calf. The resulting phlebogram (fig. 1) showed a larger-than-normal positive wave.

Table 1

<table>
<thead>
<tr>
<th>Author</th>
<th>Age</th>
<th>Type of heart disease</th>
<th>Atrial fibrillation</th>
<th>Tricuspid insufficiency</th>
</tr>
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<tbody>
<tr>
<td>Teufl 42 F</td>
<td></td>
<td>Postendocarditic; mitral stenosis and insufficiency; died</td>
<td>Yes</td>
<td>Functional</td>
</tr>
<tr>
<td>Teufl 71 F</td>
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<td>Postendocarditic; mitral stenosis and insufficiency</td>
<td></td>
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<tr>
<td>Teufl 68 F</td>
<td></td>
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<td>No</td>
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<tr>
<td>Teufl 48 F</td>
<td></td>
<td>Postendocarditic; mitral stenosis and insufficiency; tricuspid insufficiency</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Teufl 56 F</td>
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<td>Postendocarditic; mitral stenosis and insufficiency</td>
<td></td>
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<tr>
<td>Teufl 65 M</td>
<td></td>
<td>Degenerative myocardial disease; died; autopsy</td>
<td>No</td>
<td>Functional</td>
</tr>
<tr>
<td>Teufl 49 F</td>
<td></td>
<td>Mitral stenosis and insufficiency</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Teufl 70 F</td>
<td></td>
<td>Mesoartitis; aortic insufficiency; died; autopsy</td>
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<td>Functional</td>
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<tr>
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<td></td>
<td>Hypertension</td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>Teufl 53 F</td>
<td></td>
<td>Mitral stenosis and insufficiency; tricuspid insufficiency; hypertension</td>
<td>No</td>
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<tr>
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<td></td>
<td>Degenerative myocardial disease</td>
<td></td>
<td>No</td>
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<td></td>
<td>Coronary sclerosis</td>
<td></td>
<td>No</td>
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<tr>
<td>Teufl 59 M</td>
<td></td>
<td>Calcified pericardium</td>
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<tr>
<td>Teufl 61 F</td>
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<td>Mesoartitis; cor bovinum</td>
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<tr>
<td>Hildebrand 70 M</td>
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<td>Mitral stenosis and insufficiency; tricuspid insufficiency</td>
<td>Yes</td>
<td>Probably organic</td>
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<tr>
<td>Senesenbach &amp; Hutauff 52 F</td>
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<td>Arteriosclerotic heart disease</td>
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<td>Hallock 46 F</td>
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<td>Clarke Present case 59 M</td>
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<td>Unknown heart disease, possibly mitral insufficiency</td>
<td>Yes</td>
<td>Functional</td>
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</table>

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Simultaneous electrocardiographic and venous pressure tracings demonstrating atrial fibrillation with a constant relationship (0.44 sec.) between each ventricular systole and the following venous pulsation.

Discussion

The large venous wave obtained, presumably related to the exaggerated jugular "v" wave of tricuspid insufficiency, represents "ventricularization" of the venous pressure through an incompetent atroventricular valve. Because of atrial fibrillation the normally occurring "a" or atrial contraction wave, if indeed it could be transmitted to the leg, was absent. According to Wood this single broad "v" wave with loss of the normal systolic collapse (or "x" trough) is characteristic of tricuspid insufficiency with atrial fibrillation, in contrast to the "a" wave and positive systolic "v" wave of atrial filling found with normal sinus rhythm.

However, Ferrer et al. and Sepulveda and Lukas pointed out that in patients with rheumatic heart disease, tricuspid insufficiency is a very frequent accompaniment of atrial fibrillation. Ferrer hypothesizes that the atrial fibrillation may have a dynamic re-
lation to the tricuspid insufficiency and predisposes to insufficiency because of the poor valve closure that occurs when there is no atrial systole preceding ventricular systole. Trainito and Nazzi\textsuperscript{18} and Müller and Shillingford\textsuperscript{12} also noted that cardiac failure and atrial fibrillation can profoundly modify the character of the venous tracing in tricuspid insufficiency.

As the degree of cardiac compensation improved in this patient, the physical signs of tricuspid insufficiency disappeared. This has been observed in other cases as well, and has been interpreted to indicate the functional nature of the tricuspid insufficiency in this instance. The extent of Paget’s disease of bone in this case was not great and was not thought to contribute to the venous pulsations through augmented circulation.

Summary

To the 19 previously reported cases of pulsating varicose veins of the legs in tricuspid insufficiency, another case is added. Simultaneous electrocardiographic and venous pressure tracings of a pulsating varicose vein were recorded. A single broad “v” wave with loss of a conspicuous “x” descent, characteristic of tricuspid insufficiency with atrial fibrillation, was noted. The clinical manifestations of tricuspid insufficiency disappeared as the patient’s cardiac status improved, suggesting the functional nature of the insufficiency in this case.

Acknowledgment

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References

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