Ruptured Chordae of the Tricuspid Valve*

The Consequence of Flow-Directed Swan-Ganz Catheterization

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A case of autopsy-documented ruptured chordae tendineae of the tricuspid valve, secondary to flow-directed Swan-Ganz catheterization of the right side of the heart is presented. A possible mechanism is discussed and safeguards to prevent this complication are suggested.

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References

Since first introduced by Swan et al, the flow-directed balloon-tipped catheter has become a valuable aid in management of critically ill patients. It has also been useful in the cardiac catheterization laboratory.

Although the most frequent complication of Swan-Ganz right heart catheterization is minor arrhythmias, other complications have been reported. To our knowledge, ruptured chordae tendineae of the tricuspid valve has not been previously described.

Case Reports

This 63-year-old woman had rheumatic fever in early childhood and known mitral insufficiency for 15 years. Approximately three days prior to transfer to Orange County Medical Center, she had an episode of severe precordial squeezing pain of two hours' duration, with associated diaphoresis, nausea, and dyspnea. She was hospitalized at a local facility where pressor agents were used to maintain her blood pressure. Urinary output was less than 5 ml per hour. After two days, she was transferred to our facility.

Initial physical examination showed blood pressure of 80/64 mm Hg, pulse rate 120 per minute and regular, respirations 30 per minute, and oral temperature 37°C (98.6°F). She was moderately dyspneic. Cardiovascular evaluation revealed the point of maximal impulse to be hyperdynamic in the sixth intercostal space at the anterior axillary line. A grade 4/6 holosystolic regurgitant murmur was loudest at the apex, but well heard over the entire precordium and radiated to the axilla and left subscapular area. There was no increase in the intensity of the murmur during inspiration. A prominent third heart sound was also present. End inspiratory rales were heard throughout both lung fields. She had no urinary output.

Admitting laboratory data showed an ECG consistent with an extensive anterolateral infarction. Serum glutamic oxaloacetic transaminase (SGOT) was 1555 mU/ml (normal 8-40); BUN was 137 mg percent, creatinine 3.4 mg percent, serum potassium 6.8 mEq/L and white blood cell count 21,000/mm3 with a normal differential smear. Chest radiograph was compatible with cardiogenic pulmonary edema.

Hospital Course

Hypertonic peritoneal dialysis was begun to correct her fluid and electrolyte status and she was subsequently digitalized. Multiple arrhythmias, including recurrent episodes of ventricular tachycardia and ventricular fibrillation, responded poorly to antiarrhythmic agents. During the early hours of

Table 1—Swan-Ganz Right Heart Pressures (all pressures in mm Hg)

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<tr>
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<th>Before*</th>
<th>After**</th>
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<tr>
<td>Right atrium</td>
<td>mean 20</td>
<td>mean 32; CV/Y, 50/22</td>
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<tr>
<td>Right ventricle</td>
<td>80/20</td>
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<td>Pulmonary artery</td>
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<td>80/50</td>
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<tr>
<td>Pulmonary artery wedge</td>
<td>mean 24; CV/Y, 30/20</td>
<td>mean 24; CV/Y, 30/20</td>
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*Before = pressures on entry of the Swan-Ganz catheter into the various right heart chambers
**After = pressures after Swan-Ganz catheter had been advanced and withdrawn several times with an inflated balloon.
her second hospital day, a No. 5 Swan-Ganz catheter (Edwards Laboratories) was inserted by a member of the house staff. Several times the catheter was advanced through the right heart chambers to the pulmonary artery wedge position and withdrawn with the balloon inflated. Initial pressures were consistent with biventricular failure and mitral insufficiency (Table 1). Figure 1 shows the initial right atrial pressures which are elevated but without a CV-wave. Pressures after the catheter had been advanced and withdrawn (with the balloon inflated) were essentially unchanged except in the right atrium (Table 1) where mean pressures had increased by 50 percent and a prominent CV-wave had become evident. Figure 2 shows the record of this right atrial pressure, which is consistent with tricuspid insufficiency, ie, prominent CV-wave with a rapid Y-descent.

The patient continued to have arrhythmias resistant to medical therapy and expired early the next day.

Autopsy Data

The heart weighed 500 grams. The mitral valve was 12.5 cm in circumference with fibrosis and calcification of the leaflets and short, thickened fibrotic chordae. The tricuspid valve was 13.5 cm in circumference and normal except for recent rupture of a single chorda to the anterior leaflet. Extensive recent anterior infarction of the left ventricle was also present, but without involvement of the right ventricle.

The examining pathologist did not specifically look for incompetence of the tricuspid or mitral valves by pressure filling the ventricles with water. Therefore, it is not possible to judge the competency of these valves from our autopsy data.

Discussion

Swan et al1 warned of possible torn chordae to the tricuspid valve as a consequence of withdrawing their catheter with the balloon inflated.

In our patient, a prominent CV-wave was not noted on initial entry into the right atrium although pressures were elevated. The subsequent recording of a tall CV-wave with a steep Y-descent (after the balloon-inflated catheter had been advanced and withdrawn several times) suggests that tricuspid insufficiency resulted from this procedure.

Probably during its advancement, the catheter entangled in the tricuspid apparatus, and subsequent withdrawal, with an inflated balloon resulted in the torn chorda found at autopsy.

Almost certainly this patient's cardiac output decreased with the acute tricuspid insufficiency. However, because of the extent of myocardial infarction, it is unlikely that the tricuspid insufficiency that developed in this patient contributed significantly to her demise. In some patients this acute process could be devastating and require open heart surgery. Almost certainly, open heart surgery would not have benefited this patient.

Tricuspid insufficiency may accompany severe left ventricular failure. Rupture of a chorda to the tricuspid valve in our patient could have resulted from severe left-sided failure alone, although this seems unlikely.

In conclusion, we want to emphasize that Swan-Ganz flow-directed catheters, once inserted, should never be withdrawn with the balloon inflated.

References

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Figure 1. Right atrial pressure tracing before the No. 5 Swan-Ganz catheter had been advanced and withdrawn several times with an inflated balloon. Pressures are in mm Hg.

Figure 2. Right atrial pressure tracing after the No. 5 Swan-Ganz catheter had been advanced and withdrawn several times with an inflated balloon. Pressures are in mm Hg.
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Changing P Wave Polarity
Intermittent Posterior Internodal Conduction
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Spontaneous changes of P-wave polarity without change in heart rate were noted on the resting electrocardiogram of a patient admitted because of chest pain. Intracardiac and His bundle electrograms were compatible with a sequence of activation from high to low right atrium with both positive and negative P waves in lead aVR. The decrease in the P-His interval with positive P waves in lead aVR is consistent with selective conduction via the posterior internodal pathway.

The P wave polarity in the normal surface electrocardiogram is generally positive in frontal-plane leads other than lead aVR. Deviations of the P-wave axis from the normal have been ascribed to translocation of the pacing site to areas remote from the sinoatrial node. This approach resulted from the original studies of Sir Thomas Lewis, who proposed that atrial activation

spread in wave-like fashion from the sinoatrial node. Although Lewis was aware of the earlier work suggesting specialized internodal connections, his experiments did not prove a functional role for these pathways, which led him to believe they were not of electrophysiologic significance.

Descriptions of rhythms associated with abnormal P-wave morphology were based on the wave-spreading theory proposed by Lewis; however, this concept could not be substantiated by intracardiac pacing studies which translocated the pacing site but did not produce the expected P wave morphology based on the classic wave-spread theory. Recent studies have considered the role of the specialized intra-atrial pathways and their functional significance in the production of normal and abnormal P wave morphologies. Spontaneous changes in P wave polarity have not been studied by intracardiac recordings in the intact human subject. The present case demonstrates changes in P wave polarity without any change in heart rate, suggesting an intermittent change in the internodal conduction rather than translocation of the pacing site.

Case Report
A 65-year-old white woman was admitted for evaluation of retrosternal distress on exertion and an "abnormal" ECG. The retrosternal distress was relieved with rest. The blood pressure was 130/70 mm Hg, and the pulse was 64 beats per minute and regular. There was a systolic ejection click and a grade 2 systolic ejection murmur. Findings from the remainder of the physical examination were normal. A chest x-ray film was unremarkable.

The ECG showed a normal QRS pattern with no resting ST segment or T wave abnormalities (Fig 1). Spontaneous changes in P wave morphology were observed without change in the basic rate or P-R interval. Studies of the altering P wave morphology were then performed.

Methods
Results were recorded on photographic paper on a recorder (Electronics for Medicine DR 8) at a paper speed of 150 mm/sec with 0.1 second time lines. A standard tripolar His-bundle catheter was positioned across the tricuspid valve. A No. 4.5 French pacing catheter was inserted into the right atrium and coronary sinus from the right antecubital vein. Pacing and recording were performed with the pacing catheter, and recordings were obtained from the His bundle catheter in the usual fashion.

Results
With normal P wave polarity the P-His (P-H) interval was 129 msec, and the His-ventricle (H-V) time was 42 msec (Fig 2 and 3). The values were similar in leads aVR and aVF, except for a slightly longer P-H interval due to earlier appreciation of the P wave activity in lead aVF. When positive P waves occurred in lead aVR, the P-H interval shortened to 102 msec (Fig 4). The H-V interval was unchanged. The sequence of activation was

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