Tricuspid Valve Erosion from Swan-Ganz Catheters*

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Two cases of histologically documented tricuspid valve ulcers caused by use of Swan-Ganz catheters are described. The implication of these clinically silent erosions is discussed.

Infrequent reports have been published of tricuspid valve damage caused by Swan-Ganz catheterization, with the spectrum of complications ranging from asymptomatic sterile thrombotic excrescences1 and infectious endocarditis2 to rupture of the chordae tendineae.3 We present two cases of histologically proved asymptomatic erosion of the septal leaflet of the tricuspid valve caused by a Swan-Ganz catheter, which to our knowledge has not been previously reported.

CASE REPORTS

CASE 1

A 78-year-old man with myelofibrosis and a history of heavy alcohol abuse was admitted to The New York Hospital five days antemortem for evaluation of jaundice. He had no prior history of cardiac murmurs, rheumatic heart disease, illicit intravenous drug abuse, or previous cardiac catheterization.

On admission, he was afebrile and normotensive. Physical examination revealed icterus, hepatosplenomegaly, and ascites. One day antemortem, fever, hypotension, and leukocytosis developed. Sepsis was suspected, and therapy with broad-spectrum antibiotics was begun. A Swan-Ganz catheter was introduced via the left subclavian vein in order to monitor his hemodynamic status. Coagulopathy and deteriorating renal function ensued, and the patient expired. Blood cultures taken before he died later grew Escherichia coli.

At autopsy, a 0.6-cm shallow, hemorrhagic ulcer was present on the atrial surface of the septal leaflet of the

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Figure 1. Photomicrograph of valvular erosion, case 1. Note small eosinophilic fibrin thrombi (T) with scant numbers of admixed red and white blood cells adherent to the ulcerated fibrosa (F) of the valve. Microscopically, the valvular erosions of Cases 1 and 2 are virtually identical (hematoxylin and eosin, original magnification ×80).

Figure 2. Gross appearance of the tricuspid valve (TV) ulceration (arrow), Case 2. The macroscopic appearances of the valvular ulcerations of cases 1 and 2 are similar.

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toptenia developed. Three days antemortem, yeast-like organisms were grown from the blood. Approximately 36 hours antemortem a Swan-Ganz catheter was placed for hemodynamic monitoring. Despite the addition of amphotericin B to the drug regimen, his respiratory function continued to deteriorate, and he died.

The postmortem examination showed a 0.5-cm, well-circumscribed erosion on the atrial surface of the septal cusp of the tricuspid valve (Fig 2). No pulmonary emboli were present. On microscopic section, the fibroa of the valve was focally ulcerated and covered by scant thrombotic material. Hematoxylin and eosin, Gram, and Comori methenamine silver stains of permanent sections of the erosion did not show microorganisms or leukocytic infiltrates. Postmortem blood cultures remained sterile.

**Discussion**

In a previous report on Swan-Ganz catheter-induced thrombotic verrucous lesions of the tricuspid valve, Greene and Cummins7 speculated that thrombi form on endothelial surfaces damaged by the heart's motion against the plastic catheter. No histologic evidence was provided, however, to demonstrate actual injury to the valve. Our microscopic finding of endocardial erosions of the tricuspid valve show that an early lesion predisposing to thrombus formation may occur with a Swan-Ganz catheter. Other histologically demonstrated intimal erosions with overlying thrombi have been documented in the superior vena cava with a central venous pressure catheter9 and in the right pulmonary artery with Swan-Ganz catheters.5 Recently an editorial pointed out that the symptomatic and often fatal complications of flow-directed pulmonary artery catheters are well known, but the exact incidence of complications is uncertain.6

Our two cases of this clinically silent finding suggest that the overall incidence of valvular damage is indeed higher than that previously expected.

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**References**

1 Greene JF, Cummings KC. Aseptic thrombotic endocardial vegetations: a complication of indwelling pulmonary artery catheters. JAMA 1973; 225:1525-26
2 Greene JF, Fitzwater JE, Clemmer TP. Septic endocarditis and indwelling pulmonary artery catheters. JAMA 1974; 233:891-92

**Bronchial Collapse in Obstructive Lung Disease**

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A 57-year-old man who died suddenly with severe bilateral mainstem bronchial collapse is described, and an alteration of the elastic tissue in the membranous portion of the bronchi is identified. The morphologic abnormalities, physiologic dynamics, and potential clinical consequences of such an alteration are discussed.

The motion of the mainstem bronchi during respiration is well known, and cases of increased pliability of the membranous septum have been observed.1-4 We describe a severe case of bronchial collapse and demonstrate a specific structural alteration of the membranous septum of the bronchial wall. The potential etiology, physiologic consequences, and clinical significance are discussed.

**Case Report**

In 1972 a 57-year-old man had membranoproliferative glomerulonephritis, which was medically managed for six years until progressive renal failure necessitated regular hemodialysis. He had a 60 pack-year history of cigarette smoking and chronic productive cough. Pulmonary function tests done in 1975 were indicative of obstructive lung disease with hyperinflation (Table 1). In February 1979 treatment for a nonresolving left lower lobe pneumonia included flexible fiberoptic bronchoscopy, which demonstrated easy collapsibility during expiration of both mainstem bronchi in the absence of any endobronchial masses or apparent extrinsic compression. This pneumonia subsequently clinically improved with antimicrobial therapy, but roentgenograms indicated persistent scarring.

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**Table 1—Pulmonary Function Tests**

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<tr>
<th>Test</th>
<th>Result</th>
<th>Predicted Values</th>
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<tr>
<td>Vital capacity, L</td>
<td>2.37</td>
<td>64</td>
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<tr>
<td>Forced vital capacity, L</td>
<td>2.19</td>
<td>59</td>
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<tr>
<td>Functional residual capacity, L</td>
<td>4.28</td>
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<tr>
<td>Residual volume, L</td>
<td>3.78</td>
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<tr>
<td>Total lung capacity, L</td>
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<td>One-second forced expiratory volume</td>
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<td>46</td>
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<tr>
<td>Maximum mid-expiratory flow, L/sec</td>
<td>0.70</td>
<td>24</td>
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