Hepatic Vein Obstruction Due to Swan-Ganz Catheter Placement*

Michael S. Davis, M.D., F.C.C.P.

Complications from Swan-Ganz catheters during insertion, long-term placement, or removal have been known since its development. I describe the unusual presentation of a pacing Swan-Ganz catheter mispositioned into the hepatic vein producing vascular obstruction, yet with adequate cardiac pacing. (Chest 1994; 106:603-05)

Since the introduction of the flow-directed balloon-tipped catheter (Swan-Ganz) for continuous monitoring of hemodynamic function and cardiac pacing, a number of authors have reported complications associated with the routine use of this catheter.1-3 Arrhythmias, infection, perforation of the pulmonary artery, as well as vascular occlusion and pulmonary infarction have occurred. In addition, the catheter has injured the pulmonic and tricuspid valves and has been reported to knot within the ventricle. Recently, I had the opportunity to evaluate a patient with a Swan-Ganz pacing catheter with an unusual presentation that I have not seen reported.

CASE REPORT

This 73-year-old woman presented in October 1992 with a complaint of chest pain. Paramedics were called to her home and noted the patient to have a BP of 68/42 mm Hg, and an irregular pulse rate of 26 beats/min. A cardiac rhythm (lead 2) suggested a third-degree heart block with junctional escape rhythm. The patient was transported to the local emergency department where one of the staff physicians inserted a pacing Swan-Ganz catheter (Baxter model 93-200 H-7F, Edwards Swan-Ganz Pacing TD Catheter with AMC Thromboshield) with successful capture at a rate of 80 beats/min; vital signs improved (Fig 1). Cardiac enzymes subsequently became positive.

The patient had a history of high BP, type II diabetes mellitus, and peripheral vascular disease. She had a myocardial infarction in June 1991. Coronary angiograms at that time revealed 40 percent narrowing of the right carotid artery, 30 percent narrowing of the trunk of the circumflex, and 50 percent narrowing of the proximal anterior descending branch. The ejection fraction at that time was estimated to be 40 percent. An anterior apical wall aneurysm was also identified on the echocardiogram. A chest radiograph obtained at the time of this hospital admission revealed normal heart size and a light diffuse bilateral lower lobe infiltrate. A Swan-Ganz catheter was noted to loop in the right ventricle and terminate in what was reported as consolidated right lower lobe. The attending physician presumed the Swan-Ganz catheter was properly positioned because of appropriate pacemaker capture. The nurses reported either the “inability to ‘wedge’ the catheter” or “abnormal pulmonary artery pressures on monitoring” and appropriate hemodynamics were thus not obtained. Cardiology consultation confirmed the diagnosis of an acute inferior wall myocardial infarction with complications of complete heart block and congestive heart failure. They recommended observation for 7 days prior to the decision to insert a permanent pacemaker.

On the fourth hospital day, acute respiratory failure developed. Arterial blood gas indicated a pH of 7.41, PCO2 of 23.9 mm Hg, PO2 of 48.9 mm Hg, HCO3 of 50 mmol/L, and SaO2 of 86 percent (FiO2 of 45 percent). The patient had a WBC count of 19,400 cells per ml with 75 percent polymorphonuclear cells, 9 percent bands, 9 percent lymphocytes, and 6 percent monocytes. Liver enzyme levels were markedly elevated with respect to those at hospital admission (Table 1). A new chest radiograph revealed the heart size to be normal. Persistent basilar infiltrates were seen. The Swan-Ganz catheter appeared looped in the right ventricle with the tip of the catheter below the diaphragm and presumed in the right hepatic vein (Fig 2). Echocardiogram revealed an ejection fraction of 10 to 20 percent.

In view of the inappropriate placement of the Swan-Ganz catheter and suspected hepatic vein obstruction, the decision was made to replace the catheter. The patient was 100 percent dependent on the pacemaker as indicated by a trial of turning off the pacemaker resulting in the patient’s heart rate dropping to 30 beats/min with an occasional junctional escape rhythm and systolic BP dropping to 40 mm Hg. A new pacing Swan-Ganz catheter was inserted before removal of the old catheter with the use of fluoroscopy to ensure appropriate placement and to detect possible entanglement with the initial Swan-Ganz catheter. This was performed successfully with appropriate pacemaker capture. Hemodynamics were now appropriately recorded (Table 1).

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FIGURE 1. Electrocardiographic tracing subsequent to insertion (at the time of hospital admission) of Swan-Ganz catheter with ventricular pace leads showing successful capture.
DISCUSSION

Blood from the liver drains into the inferior vena cava via three large venous trunks, the right, middle, and left hepatic veins plus some branches.6 Obstruction of these venous drains may occur from multiple causes. Although trauma is the most common disorder, there are several other diseases that may involve the hepatic vein. Thrombosis of the hepatic vein (Budd-Chiari syndrome) commonly is due to abdominal trauma, the use of oral contraceptives, or with hematologic disorder (polycythemia, rubra vera, paroxysmal nocturnal hemoglobinemia, and myeloproliferative disease).5 Veno-occlusive disease, due to alcoholic hepatitis, and postchemotherapy with certain agents also may produce a Budd-Chiari-like syndrome. Thrombi, tumor, parasitic infections, or a membrane in the inferior vena cava may obstruct the hepatic vein and produce a similar clinical presentation. Signs and symptoms typically include abdominal pain, ascites, hepatomegaly, splenomegaly, and jaundice.6 Serum transaminases, bilirubin, and alkaline phosphatase values may rise mildly, but in acute illness may be significantly increased.

The balloon floatation catheter developed by Drs. H. Swan and W. Ganz permits the estimation of intrachamber pressure in the right atrium, pulmonary artery, and pulmonary capillary wedge position. Subsequent development and modification of the Swan-Ganz catheter also included the ability to insert either a pace wire or to have one incorporated into the catheter.

In general, the incidence of complications from Swan-Ganz catheterization is low. Reports mention frequent minor problems.13 These include the following: transient arrhythmias during passage, balloon rupture, catheter thrombosis, catheter coiling in the right ventricle, and local infection at the cutaneous insertion site. More serious complications have also been reported; rhythm disturbances (ventricular tachycardia, ventricular fibrillation, atrial arrhythmias, right bundle branch block) may occur. Embolization resulting in pulmonary embolism, pulmonary infarction, or pulmonary vascular thrombosis occurs particularly if the balloon is kept inflated too long. Aseptic thrombotic endocardial vegetations, subacute bacterial endocarditis, and endocardial mural thrombosis are reported to occur if the catheter remains in the central circulation for prolonged periods. Other complications such as fatal pulmonary artery perforation, intracardiac knotting by catheters, catheter suture to the right atrial wall, percutaneous placement of the catheter into the carotid artery, percutaneous placement of the catheters into the trachea, abscess at the venous cut-down/insertion site, and rupture cord of the tricuspid valve have all been reported. In this case, the possibility of hepatic vein obstruction due to an errantly placed Swan-Ganz catheter appears to exist. The rise of the serum transaminases and their subsequent fall after the replacement of the catheter favors this diagnosis (Table 1).

Although selective catheterization of the hepatic vein has been performed before hepatic vein shunt surgery for severe liver cirrhosis or hepatic vein thrombosis, it has not been reported as an accidental result of cardiac catheterization for hemodynamic monitoring or cardiac pacing with successful capture. Previously published hepatic vein pressures were not identified. The degree of hepatic injury appears related to the duration and degree of vascular flow

Table 1—Chronological Liver Function Values With Selected Hemodynamic Values*

<table>
<thead>
<tr>
<th>Date</th>
<th>ALT</th>
<th>AST</th>
<th>LDH</th>
<th>CPK</th>
<th>Bili</th>
<th>BP</th>
<th>HR</th>
<th>PAP</th>
<th>PCWP</th>
<th>CI</th>
<th>PVR</th>
<th>SVR</th>
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<td>38</td>
<td>235</td>
<td>159</td>
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<td>68/42</td>
<td>26</td>
<td>30/24</td>
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<td>91</td>
<td>273</td>
<td>645</td>
<td>1,419</td>
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<td>105/67</td>
<td>85</td>
<td>27/21</td>
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<tr>
<td>10-27-92</td>
<td>161</td>
<td>256</td>
<td>910</td>
<td>842</td>
<td>0.7</td>
<td>117/67</td>
<td>80</td>
<td>25/20</td>
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<td>177</td>
<td>835</td>
<td>311</td>
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<td>105/77</td>
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<td>49/21</td>
<td>18</td>
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<td>1,350</td>
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<td>S-G changed prior to laboratory draw</td>
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<td>625</td>
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<td>105/95</td>
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<td>38/19</td>
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<td>557</td>
<td>—</td>
<td>0.3</td>
<td>108/47</td>
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<td>41/22</td>
<td>18</td>
<td>2.1</td>
<td>225</td>
<td>942</td>
</tr>
</tbody>
</table>

*ALT=alanine transaminase (serum glutamic pyruvic-SGPT) (U/L); AST=aspartate transaminase (serum glutamic oxaloacetate-SGOT) (U/L); LDH=lactate dehydrogenase (U/L); CPK=creatine phosphokinase (U/L); Bili=total bilirubin (mg/dL); S-G=Swan-Ganz catheter; BP=blood pressure (mm Hg); HR=heart rate (beats/min); PAP=pulmonary artery pressure (mm Hg); PCWP=pulmonary capillary wedge pressure (mm Hg); CI=cardiac index (L/min); PVR=pulmonary vascular resistance (dynes-s/cm5); SVR=systemic vascular resistance (dynes-s/cm5).

Figure 2. Chest radiograph on first hospital day showing normal heart size, Swan-Ganz catheter looping in right ventricle and terminating in hepatic vein.
obstruction. Liver injury can be transient as demonstrated by the rapid normalization of transaminase levels as seen in this patient after repositioning of the Swan-Ganz catheter.

**CONCLUSION**

This patient, unfortunately, continued to extend her infarct. Her heart failure could not be reversed despite the judicious use of intravenous nitroglycerin, low-dose dopamine, dobutamine, furosemide, and metoprolol. On the sixth hospital day, the patient died. Laboratory studies performed before her death revealed a near normal to total resolution of her liver enzyme abnormalities (Table 1).

**REFERENCES**


**Miliary Mesothelioma***

*Michael Huncharek, M.D., M.P.H.*

Metastases in pleural mesothelioma usually occur late in the disease process. Diffuse involvement of the lung parenchyma is rare. A patient with miliary pulmonary parenchymal involvement with malignant mesothelioma is described. To our knowledge, this represents the first such case reported. (Chest 1994; 106:605-06)

Malignant pleural mesothelioma is a relatively rare tumor that is becoming increasingly common. The tumor is characterized as a highly malignant neoplasm, with a mean survival time from diagnosis of approximately 12 months. Metastases occur late in the disease process, with common sites of spread being the contralateral lung, liver, kidneys, and adrenal glands. Involvement of the contralateral lung is usually in the form of large nodules or pleural based lesions. Bilateral diffuse pulmonary involvement is unusual, and a miliary pulmonary parenchymal pattern of spread has not been previously reported to our knowledge. A case of pleural mesothelioma with a miliary radiographic presentation is described.

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**FIGURE 1.** Initial chest x-ray film showing left-sided pleural effusion and clear lung fields.

**CASE REPORT**

The patient is a 61-year-old white woman who presented complaining of 3 months of increasing pleuritic left lower chest pain, dyspnea on exertion, and fatigue. She had had no change in her chronic cough or production of white sputum. One week prior to admission, the patient had presented to the emergency ward, where a chest x-ray film (Fig 1) showed a left-sided pleural effusion, which was found to be exudative. The findings from physical examination were remarkable for decreased breath sounds at the lung bases bilaterally, with some dullness to percussion of the left base.

A tuberculin skin test was positive. A workup in the hospital included thoracentesis (X4) and two pleural biopsies, all negative for granuloma, acid-fast organisms, or tumor. A liver-spleen scan and mammograms were normal. A chest x-ray film (Fig 2) and chest computed tomogram (Fig 3) obtained 1 week following admission showed a miliary pattern believed to represent miliary tuberculosis. The patient was started on therapy with isoniazid, rifampin, and ethambutol. She was discharged from the hospital in stable condition.

The patient’s medical history included a total abdominal hysterectomy with bilateral salpingo-oophorectomy 15 years earlier for menorrhagia. She had a 40-pack-year smoking history and remote exposure to a brother with active tuberculosis. She denied any toxic exposures or exposure to asbestos.

Over the subsequent 3 weeks, the patient’s weight decreased by 2.7 kg (6 lb), and she complained of anorexia, morning nausea, dyspnea on exertion, and continued left-sided pleuritic chest pain. A repeat chest x-ray film showed the miliary pattern previously described, as well as a persistent moderate left-sided pleural effusion. The patient was admitted to the hospital shortly thereafter for bronchoscopy, which was not performed due to poor pulmonary function. The forced vital capacity was 1.06 L, and the forced expiratory volume in 1 s was 0.74 L. Arterial blood gas analysis with the patient breathing room air showed a pH of 7.43, PaO₂ of 68 mm Hg and PaCO₂ of 44 mm Hg.

A bone marrow biopsy and liver biopsy were performed and were negative for acid-fast organisms; pathologic findings was negative. Streptomycin and pyrazinamide were added to her...