A 78-year-old man with longstanding hypertension, diabetes, and coronary artery disease was referred to a multidisciplinary heart failure clinic for consideration of pericardial stripping after coronary artery bypass surgery. The patient had taken medications for hypertension for 45 years. He developed palpitations that were diagnosed as atrial fibrillation 18 months before this presentation. At the same time, he received a new diagnosis of diabetes and had begun therapy with pioglitazone.

The patient’s exertion was increasingly limited by dyspnea without chest pain. Coronary angiography showed triple vessel disease for which he underwent cardiac surgery for placement of 3 vein grafts, an internal mammary artery graft, and ablation of atrial fibrillation. After surgery, the patient remained limited by dyspnea, which increased over the next 6 months. Peripheral edema increased progressively. CT scan showed changes consistent with prior cardiac surgery and no evidence for pulmonary emboli. Repeat catheterization showed an occluded vein graft for which a stent was successfully placed. At that time, pressures in the right ventricle (RV) and left ventricle (LV) were noted to be equal at baseline and to change together in response to respiration.

The patient’s exertional dyspnea continued to worsen, with new onset of orthopnea and ascites. He was hospitalized twice for intravenous diuretic therapy in the 2-month period before referral to a cardiac surgeon with presumed constrictive pericarditis related to his previous surgery. The patient had no history of tuberculosis, collagen vascular disease, or symptoms of pericarditis. He was referred by the cardiac surgeon to an advanced heart disease center for a second opinion regarding the diagnosis of constrictive pericarditis and indications for repeat surgical intervention. Medications at the time of referral were sotalol 80 mg BID, diltiazem 240
mg QD, furosemide 60 mg QD, lisinopril 5 mg QD, metoprolol succinate 100 mg BID, warfarin 10 mg QD, clopidogrel 75 mg QD, pioglitazone 15 mg QD, and pravastatin 40 mg QD.

**Clinical Assessment**

The initial assessment after referral to the cardiomyopathy clinic revealed jugular venous pressure of 18 cm H₂O with V waves and a markedly positive Kussmaul sign. Cardiac examination revealed a prominent RV impulse without LV impulse. A harsh murmur consistent with aortic valve disease and a blowing murmur without respiratory increase were heard in systole. A prominent crisp sound was heard early in diastole of higher pitch than the usual third heart sound. The chest examination showed dullness at the right base without rales. The abdominal silhouette was consistent with nontense ascites, and the liver edge could not be palpated nor percussed. The patient’s skin showed some brawny changes and superficial ulceration over the feet and calves. Edema was pitting to the knees.

The patient had moderate renal insufficiency, with a creatinine level of 1.8 mg/dL and normal sodium level of 141 mmol/L. Cardiac enzyme levels were negative, hematocrit level was 32%, and liver function tests were within the normal range. ECG showed atrial fibrillation and normal voltage. Echocardiography (Figures 1 to 3) revealed a LV ejection fraction of 45% to 50%; LV wall thickness of 1.2 cm; LV end-diastolic dimension of 4.2 cm; septal and inferior hypokinesis; mild aortic stenosis; moderate tricuspid regurgitation with estimated pulmonary arterial systolic pressure of 48 mm Hg; restrictive Doppler mitral inflow pattern; and a dilated, severely dysfunctional RV. His peak oxygen consumption was 9.7 mL/min despite unusually vigorous effort, reaching an respiratory exchange ratio of 1.63.

**Hospitalization for Evaluation and Therapy**

The patient was admitted for hemodynamic assessment and intravenous diuretic therapy, with consideration of a possible diagnosis of pericardial constrictive disease. Initial simultaneous RV and LV end-diastolic pressures were...
both raised at 25 to 28 mm Hg and did not seem to separate with respiration (Figure 4). Coronary angiography showed the previously placed stent to be patent.

Over the next 12 days, he received therapy with continuous intravenous furosemide. Because of its potential contribution to peripheral edema, pioglitazone was stopped, and glycemic control was achieved with glipizide. The patient underwent a gradual diuresis with 13 lb of weight loss. His jugular venous pressure was reduced to 7 cm H₂O, and peripheral edema had resolved. His exercise tolerance had advanced to allow him to walk around the ward and climb 1 flight of stairs. Left- and right-side heart catheterization was repeated. Right atrial pressure was 8 mm Hg; pulmonary pressure, 30/15 mm Hg; and pulmonary capillary wedge pressure, 16 mm Hg (Figure 5). Simultaneous RV and LV end-diastolic pressures were 15 and 9 mm Hg, respectively, with clear separation throughout diastole (Figure 6). Repeat echocardiography (Figures 7 and 8) showed resolution of septal flattening and a less restrictive pattern of mitral inflow.

The patient was discharged feeling well and is followed in heart failure clinic regularly in conjunction with the heart failure nurse practitioner, requiring intermittent adjustments in diuretic dosing. The Table summarizes his clinical findings over the course of this case. His discharge medications are sotalol 80 mg BID, diltiazem 120 mg QD, torsemide 20 mg BID, lisinopril 5 mg QD, metoprolol succinate 50 mg BID, warfarin 10 mg QD, clopidogrel 75 mg QD, glipizide 5 mg QD, pravastatin 40 mg QD. The
patient follows strict sodium and fluid restrictions and manages well in his day-to-day life with New York Heart Association functional class II–III symptoms. This case highlights the complexity of the interactions between the RV and LV and the dynamic relationship of these interactions with fluid status in the intravascular and extravascular compartments.

**Acknowledgment**

We thank Lynne W. Stevenson, MD, Brigham and Women’s Hospital, Boston, MA.

**Disclosures**

None.

**KEY WORDS:** heart failure, hemodynamics, ventricles, constriction
Table. Changes in Physical Findings and Renal Function

<table>
<thead>
<tr>
<th></th>
<th>On Admission</th>
<th>At Discharge</th>
<th>At 1-Month Follow-Up</th>
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<tbody>
<tr>
<td>Weight, lb</td>
<td>189</td>
<td>177</td>
<td>178</td>
</tr>
<tr>
<td>BP, mm Hg</td>
<td>118/70</td>
<td>112/64</td>
<td>110/82</td>
</tr>
<tr>
<td></td>
<td>(8-mm paradox)</td>
<td>(no paradox)</td>
<td></td>
</tr>
<tr>
<td>JVP, cm H2O</td>
<td>18</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>Leg edema</td>
<td>2+</td>
<td>Trace</td>
<td>Trace</td>
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<tr>
<td>Creatinine, mg/dL</td>
<td>1.8</td>
<td>2.0</td>
<td>1.9</td>
</tr>
<tr>
<td>Blood urea nitrogen, mg/dL</td>
<td>60</td>
<td>79</td>
<td>58</td>
</tr>
</tbody>
</table>

BP indicates blood pressure; JVP, jugular venous pressure.
Equalization of Right- and Left-Sided Intracardiac Pressures: Is it Constriction?
Patricia Campbell, Jane A. Leopold, Andrew P. Selwyn and Donato Sisto

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