that mild degrees of this alteration are relatively frequent, but are not detected by study of routine histologic sections, from which the cholesterol is removed during tissue processing. Polarized light microscopy of frozen sections is the method of choice for the demonstration of these cholesterol deposits.

ACKNOWLEDGMENT: We are grateful to Dr. Jeffrey M. Hoeg of the Molecular Disease Branch, National Heart, Lung, and Blood Institute, for performing the cholesterol analyses.

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Left Ventricular Function in Peripartum Cardiomyopathy*
Daniel Cepin, M.D.; Frank James, M.D.; and Blase A. Carabello, M.D.

Although many cases of peripartum cardiomyopathy have been reported in the literature, detailed data from left heart catheterization and contrast ventriculography are unavailable. A patient with peripartum cardiomyopathy had catheterization performed to clarify the diagnosis. Hemodynamics at rest and with exercise, angiographic volumes, and indices of left ventricular function are presented. The data indicate no serious abnormality of global systolic left ventricular function in this patient although a segmental wall motion abnormality was noted. Volume overload or diminished diastolic compliance may explain the symptoms of heart failure in some patients with this disease.

CASE REPORT
A 34-year-old black woman was admitted to Temple University Hospital with acute onset of shortness of breath. She had been well until one hour prior to presentation to the emergency room, when she developed acute shortness of breath while resting at home. She denied a history of chest pain, dyspnea on exertion, paroxysmal nocturnal dyspnea, edema, or syncope. There was no history of antecedent heart disease.

The patient was gravida 5 para 5 ab 0. Her four previous pregnancies were uncomplicated. Two weeks prior to admission, she had a spontaneous normal vaginal delivery of a 3.6 kg male child. There were no maternal complications, and mother and child were discharged two days after delivery. Blood pressure was normal during and after this pregnancy.

She denied alcohol abuse. She was not taking any medications at the time of admission. Previous chest x-ray films and ECGs were within normal limits.

Physical examination upon arrival in the emergency ward revealed a well-developed, well-nourished woman in respiratory distress. The temperature was 37.0°C, pulse rate, 90; respirations, 28 per minute; and the blood pressure was 130/100. Examination of the head, ears, eyes, and throat was unremarkable. Funduscopic examination revealed no hypertensive changes. The central venous pressure

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FIGURE 1. Chest x-ray film on admission showed borderline cardiomegaly and pulmonary edema (upper). A repeat chest x-ray film, five days later, showed resolution of the pulmonary edema (lower).

estimated from jugular vein examination was 9 cm of water. The carotid pulses were normal. The thorax was of normal configuration; rales were heard over the lower half of both lung fields. The heart was not enlarged, and the rhythm was regular; third and fourth heart sounds were heard prominently at the left ventricular apex. A grade 3/6 holosystolic murmur was heard at the cardiac apex and radiated to the back and axilla. No diastolic murmur was heard. The peripheral pulses were normal. The abdomen was normal. There was 1+ bilateral ankle edema.

Admission Laboratory Data

The total white blood cell count was 8,100/cu mm, and the hematocrit was 32 percent. Electrolyte levels and BUN value were normal.

An arterial blood sample while breathing room air showed a PaO$_2$ of 41 mm Hg, PaCO$_2$ 32 mm Hg, and the pH, 7.39. The ECG revealed normal sinus rhythm (85 per minute) within normal limits. The creatinine kinase MB fraction was 0.6 percent. An x-ray film of the chest showed mild cardiomegaly and a cardiothoracic ratio of 0.51 and pulmonary edema (Fig 1, upper).

Hospital Course

The patient received oxygen by nasal prongs and furosemide, 40 mg, intravenously. She developed a brisk diuresis and rapidly improved. She required no further diuretics and did not receive digitals. Serial ECGs and cardiac enzyme tests excluded the diagnosis of acute myocardial infarction.

An echocardiogram performed on the second hospital day demonstrated a mildly enlarged left ventricle (3.3 cm/m$^2$). There was no evidence of left ventricular hypertrophy or left atrial myxoma. The shortening fraction was 0.24, the lower limit of normal in our laboratory. No abnormality of the mitral valve was noted. Asymmetric septal hypertrophy was excluded. A second echocardiogram five days later showed no change. A repeat chest x-ray film showed resolution of the pulmonary edema (Fig 1, lower).

Despite marked clinical improvement, the patient’s murmur persisted. The diagnoses of peripartum cardiomyopathy versus acute mitral regurgitation were entertained. Cardiac catheterization was performed on the seventh hospital day to resolve the diagnosis.

CATHETERIZATION RESULTS

Hemodynamic data obtained during cardiac catheterization at rest and with isometric exercise are displayed in Table 1. Cardiac output was determined by dye dilution technique. Stroke work index was plotted against pulmonary capillary wedge pressure. With handgrip, exercise stroke work index rose while pulmonary capillary wedge pressure fell slightly as shown in Figure 2.

Ventriculography displayed only trivial mitral regurgitation (+1 Grossman’s criteria). The regurgitant fraction was small (0.21). Angiographically determined volumes and indices of left ventricular function are displayed in Table 2. Volumes were determined as previously described. The mean velocity of circumferential fiber shortening (Vcf) was calculated using previously described methods. Left ventricular end systolic wall stress was calculated using the method of Mirsky. Although our data indicate that global left ventricular systolic function was within the lower limit of normal, the left ventriculogram did clearly demonstrate an area of segmental apical hypokinesis. There was no pressure gradient across the aortic valve. Coronary arteriograms

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patient at Rest</th>
<th>With Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrial pressure (mean)</td>
<td>7 mm Hg</td>
<td></td>
</tr>
<tr>
<td>Pulmonary artery pressure (mean)</td>
<td>26 mm Hg</td>
<td></td>
</tr>
<tr>
<td>Mean pulmonary capillary wedge pressure</td>
<td>12 mm Hg</td>
<td>11 mm Hg</td>
</tr>
<tr>
<td>Left ventricular end diastolic pressure</td>
<td>20 mm Hg</td>
<td></td>
</tr>
<tr>
<td>Cardiac output</td>
<td>5.7 L/min</td>
<td>7.4 L/min</td>
</tr>
<tr>
<td>Cardiac index</td>
<td>3.2 L/mm/M$^2$</td>
<td>4.2 L/min</td>
</tr>
<tr>
<td>Stroke volume index</td>
<td>41 ml/M$^2$</td>
<td>46 ml/M$^2$</td>
</tr>
<tr>
<td>Heart rate</td>
<td>78/min</td>
<td>91/min</td>
</tr>
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</table>

Table 1—Hemodynamic Data Obtained by Cardiac Catheterization

Peripartum Cardiomyopathy (Cepin, James, Carabello)
congestive heart failure develops or co-existent heart disease. While hypertension is frequently associated with this entity, it need not be present for congestive heart failure to occur. Most typically, the woman is black, has had three or more pregnancies, is often over the age of 30, and develops the symptoms of congestive heart failure during the first month after delivery. The natural history appears to take two distinct courses. In the United States where the disease is rare, approximately one half of patients will have amelioration of their symptoms and return of heart size to or toward normal. These patients have a good prognosis. The other half continues to have symptoms of heart failure and cardiomegaly. In this latter group, the mean survival is less than five years. In Africa (particularly Nigeria), the disease is common and may occur in as many as 1 percent of all pregnancies. There, the disease follows the benign course in a large majority of women. Thus, Fillmore and Parry reported only 18 cardiac deaths in 193 women (9 percent) followed four to seven years after the diagnosis of peripartum cardiomyopathy was made. Cardiothoracic ratio fell from an average of 0.62 to 0.51 from onset of the illness to one year of convalescence and most became asymptomatic.

While the etiology of the disease is unknown, a recent report by Sanderson et al of echocardiographically derived indices of left ventricular function in 43 Nigerian patients suggests that systolic function is not significantly depressed in the majority of African women with this disease. Ejection fraction and mean velocity of fiber shortening were normal in many patients in this report. These findings are consistent with the benign course of the disease in Africa. However, more severe left ventricular dysfunction was noted in some patients, and nine had ejection fractions of less than 40 percent. It would be anticipated that prognosis in this latter group is poor.

Our findings indicate adequate systolic left ventricular function in our patient with peripartum cardiomyopathy. They are consistent with Sanderson's report. Although catheterization occurred seven days after presentation, there was no objective change echocardiographically in left ventricular function from the first day after presentation to catheterization. During isometric handgrip, there was a 33 percent increase in stroke work index without an increase in left ventricular filling pressure. This normal response suggests adequate ventricular reserve. Ejection phase indices of left ventricular function-ejection fraction and Vcf were in the low range of normal. The end systolic stress-end systolic volume index ratio was also normal. Thus, global left ventricular systolic dysfunction cannot account for our patient's symptoms, signs, and roentgenographic evidence of heart failure. Left ventriculography was, however, abnormal. A small but definite segmental apical area of hypokinesis was noted but is unexplained. The patient's coronary arteries were normal, and her cardiac enzyme levels and ECGs were not consistent with the syndrome of infarction with normal coronaries or coronary spasm.

Since no significant defect in systolic muscle function is implicated in our patient's heart failure, then a diastolic compliance abnormality or volume overload are possible explanations. Our patient's left ventricular end-diastolic pressure of 20 mm Hg, and increased end-diastolic volume index after diuresis (which may have been even higher prior to diuresis), are consistent with either hypothesis. The causes of such a diastolic compliance abnormality or volume overload remain unknown. Previous authors have speculated that the disease in Africa is in part due to the custom of heavy postpartum salt ingestion and volume retention. However, such a history of excessive salt ingestion was not present in our patient.

The diagnosis of peripartum cardiomyopathy rests on the documentation of the absence of previous or concomitant heart disease and the development of congestive heart failure in the peripartum period. Prior to admission, the patient's history, physical examination, chest x-ray film, and ECGs detected no cardiovascular abnormality. However, physical examination and the chest x-ray film at the time of admission were consistent with pulmonary edema. The findings of third and fourth apical heart sounds and elevated central pressure

**Table 2**—Angiographically Derived Left Ventricular Volumes and Indices of Function

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>End-diastolic volume index</td>
<td>105 ml/M²</td>
</tr>
<tr>
<td>End-systolic volume index (ESVI)</td>
<td>42 ml/M²</td>
</tr>
<tr>
<td>Left ventricular ejection fraction</td>
<td>0.60</td>
</tr>
<tr>
<td>Mean velocity of fiber shortening</td>
<td>1.2 cm/sec</td>
</tr>
<tr>
<td>End-systolic wall stress (ESWS)</td>
<td>168 dynes x 10⁶/cm²</td>
</tr>
<tr>
<td>ESWS/ESVI</td>
<td>4.0 dynes x 10⁶/cm²/ml/M²</td>
</tr>
<tr>
<td>Heart rate</td>
<td>65</td>
</tr>
</tbody>
</table>
support a cardiac etiology for this case of pulmonary edema, as does the postdiuresis left ventricular end-diastolic pressure of 20 mm Hg. Cardiac catheterization disclosed only trivial mitral regurgitation which was probably the cause of this patient's apical holosystolic murmur. Thus, the absence of previous significant heart disease and the presence of cardiogenic pulmonary edema support the diagnosis of peripartum cardiomyopathy in our patient. Additionally, our patient's age, race, multiparity, and time of onset of symptoms are quite typical for peripartum cardiomyopathy and further strengthen the diagnosis.

We wish to emphasize that our findings do not extend to all patients with this illness. Most likely, our patient is reflective of the benign course which is followed by some patients with peripartum cardiomyopathy. Severe left ventricular dysfunction may well be present in those patients who have persistent heart failure and cardiomegaly.

In summary, we present the hemodynamic and angiographic data of a patient with peripartum cardiomyopathy. Global systolic function was normal, although an unexplained segmental wall motion abnormality was noted. An elevated left ventricular end-diastolic pressure in association with adequate systolic function at rest and with exercise suggests that reduced ventricular compliance or volume overload may be implicated in the pathophysiology of this entity.

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Adverse Effects of Nifedipine Therapy on Hypertrophic Obstructive Cardiomyopathy*

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A 62-year-old man with obstructive hypertrophic cardiomyopathy was given sublingual nifedipine, 10 mg, during invasive hemodynamic monitoring. After 15 minutes, his left ventricular outflow gradient increased from 22 to 80 mm Hg while arterial pressure fell from 152/70 to 122/64 mm Hg. Left ventricular end-diastolic pressure increased from 15 to 22 mm Hg. These adverse hemodynamic responses may have been a result of vasodilation of the peripheral circulation induced by nifedipine. Thus, some patients with hypertrophic obstructive cardiomyopathy may develop serious hemodynamic compromise when treated with nifedipine.

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