

Editor's Note

With this issue, *The Journal* is reinstating a feature which was included for many years and which, apparently, fell out of favor. Perhaps this may have been due, in part, to the marked decline in autopsy rates, as well as the growth and availability of sophisticated laboratory procedures including analyses of tissues obtained by biopsy via a percutaneous needle or endoscope. We expect that many of our interesting case presentations will be based on biopsy material and laboratory analyses. These developments and the evolution of reliable laboratory procedures have been a great boon to the quality of patient care, medical practice and medical education.

However, many cases remain in which these procedures have not always clarified the diagnoses and in which no post-mortem examinations had been performed. The anguish and emotional stress imposed on families, next of kin, and attending physicians associated with the procedure of obtaining consent for a post-mortem examination are understandably significant hurdles to be overcome. Surely, the post-mortem examination of tissues remains critical in evaluating the differential diagnoses and the effects of therapy. Any view that the post-mortem examination of tissues is not worthwhile, despite its expense, is just plain wrong. Without doubt, the results of any post-mortem examination must be incorporated into the assessment of the quality of care delivered to the specific patient, by personnel of the institution. Can we, as physicians, be sure that the history and physical examination provided the necessary clues? Or was an important detail overlooked? This exercise remains an important tool in our learning and in teaching our students. This feature will be directed towards these ends.

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The Mount Sinai Hospital Clinicopathological Conference

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**A 50-Year-Old Male with Diabetes Mellitus, Peripheral Neuropathy, Hypothyroidism,
Obesity, Sleep Disorder, Pickwickian Syndrome, and Heart Failure**

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Presentation of a Case

Dr. Noelle Mann¹: NR is a 50-year-old male with a medical history significant for diabetes mellitus, peripheral sensory neuropathy, difficulty in sleeping, chronic daytime somnolence, exertional dyspnea, two pillow orthopnea, early satiety, and hypothyroidism. He denied tobacco use, excessive alcohol consumption, and recent travel. He was admitted to The Mount Sinai Hospital in early January 1997 for symptoms of increasing shortness of breath, lower extremity edema, and increasing abdominal girth which had failed to respond to increasing doses of oral furosemide. Four years previously, he had been admitted to another hospital, with similar symptoms, and was diagnosed with heart failure. He was treated with diuretics at that time and had lost more than 100 pounds. Despite therapy with diuretics, he noticed weight gain, and recurrence of increasing shortness of breath, lower extremity edema, and abdominal girth since April 1996.

On admission, his medications included: glipizide 20 mg per day by mouth, furosemide 400 mg per day by mouth, digoxin 0.25 mg per day by mouth, aspirin one-half tablet (162.5 mg) per day by mouth, and oral potassium supplements 40 mEq in the morning and evening, and 20 mEq at bedtime. Levothyroxine replacement therapy had been stopped in November 1996.

On admission, the patient was found to be an obese male in no acute distress. He was afebrile

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with a blood pressure of 130/90 mm Hg. The pulse and respiratory rates were 80 and 18 per minute respectively. Physical examination was notable for jugular venous distention at 8-10 centimeters above the angle of Louis (read with the patient sitting at a 45-degree angle to horizontal). Auscultation of the lungs revealed no abnormal breath sounds. Examination of the heart revealed audible first and second heart sounds with a loud P₂. The abdomen was distended and tense with shifting dullness. Examination of the extremities showed pitting edema to both mid-thighs. Initial laboratory values are found in Table 1.

TABLE 1
Initial Laboratory Values

White blood count	6.7x10 ³ cells/mm ³
Hemoglobin	12.8 g/dL
Hematocrit	38%
Platelet count	1.74x10 ⁵ cells/mm ³
Mean corpuscular volume	84.3 μm ³
Red cell distribution width	14.9%
Prothrombin time	14.8 seconds
Partial thromboplastin time	30.6 seconds
Erythrocyte sedimentation rate	25 mm/hr

SERUM CHEMISTRIES

Sodium	136 mmol/L
Potassium	4.4 mmol/L
Chloride	100 mmol/L
CO ₂ content	31 mmol/L
Urea nitrogen	39 mg/dL
Creatinine	1.5 mg/dL
Glucose	347 mg/dL
Protein, total	7.4 g/dL
Albumin	4.2 g/dL
Calcium, total	9.4 mg/dL
Magnesium	2.1 mEq/L
Bilirubin, total	0.5 mg/dL
Arterial blood sample:	
pH	7.44
P _{O₂} (on room air)	99 mm Hg
P _{CO₂}	44 mm Hg

SERUM ENZYMATIC ACTIVITIES

Phosphatase, alkaline	117 IU/L
Alanine aminotransferase (ALT, SGPT)	17 IU/L

Table 1(cont.)

SERUM HORMONE VALUES AND THERAPEUTIC AGENTS

Digoxin level	1.1 $\mu\text{g/L}$
Thyroid Stimulating Hormone	4.7 $\mu\text{U/mL}$

The chest roentgenogram (Fig. 1) on admission revealed a normal cardiac silhouette and pulmonary vasculature, a tortuous aorta, sharp costophrenic angles, no infiltrates, and a slight prominence of interstitial lung markings at both lung bases.



Fig. 1. PA and lateral chest roentgenograms. Cardiac silhouette and pulmonary vasculature are normal. Interstitial lung markings at both bases are slightly increased.



Fig. 2 - Electrocardiogram showing normal axis and sinus rhythm at a rate of 81 per minute. ST-T wave changes are non-specific.

The electrocardiogram (Fig. 2) showed regular sinus rhythm at a rate of 81 per minute with nonspecific ST-T wave changes and a normal axis.

A PPD test was found to be negative. The patient underwent a trans-esophageal echocardiogram on the day after admission. This was a technically difficult study, but showed normal left ventricular size and function, paradoxical septal motion, and mild pulmonary hypertension. Also noted were mild right ventricular dilatation, moderately decreased right ventricular function, an estimated right ventricular systolic pressure of 36 mm Hg, a patent foramen ovale, minimal mitral and pulmonic regurgitation, and aortic sclerosis. No aortic or intra-cardiac thrombi were found. Three days later, the patient underwent ventilation and perfusion scans of the lungs, which were reported to be normal. A duplex Doppler examination of the lower extremities was negative for venous occlusion. The patient next underwent a nocturnal polysomnography study, which evidenced episodic hypoventilation and apnea (with hypoxemia by pulse oximetry), mild periodic limb movements, and frequent arousals with severely fragmented sleep architecture; these are all consistent with moderate obstructive sleep apnea syndrome. Studies of pulmonary function earlier that day demonstrated probable borderline restrictive impairment with severely reduced maximal inspiratory and expiratory pressures, and a normal diffusing capacity for carbon monoxide. A computed tomogram of the chest showed no evidence of a thickened pericardium (Fig. 3). Right heart catheterization on the 7th hospital day revealed increased pressures in the right atrium, right



Fig. 3 - One slice of a computed tomogram of the chest showing a normal pericardium without any evidence of thickening.

TABLE 2
Cardiac Catheterization Data

	RA mm Hg	RV mm Hg	PA mm Hg	PCWP mm Hg
Without NO	19	45/12 (33)	45/20 (28)	16
With NO			32/16 (22)	16

Right heart percutaneous catheterization performed via the internal jugular vein. Values in parentheses are the mean.

NO = Nitric oxide; PA = Pulmonary artery; PCWP = Pulmonary capillary wedge pressure; RA = Right atrium;

RV = Right ventricle

ventricle, and pulmonary artery (Table 2). A modest decrement in pressure occurred after the administration of aerosolized nitric oxide (Table 2). Abdominal ultrasound on the 14th day revealed ascites with increased echogenicity of the liver and an enlarged spleen.

Concurrently, the patient was treated with intravenous diuretics as well as sodium and fluid restriction. This resulted in a 60-pound weight loss and symptomatic improvement.

On the 15th day, the patient underwent a diagnostic investigation. Three days later, a therapeutic procedure was performed.

Differential Diagnosis

Dr. Michael Kim²: In summary, NR is a 50-year-old male with a history of diabetes mellitus with resultant neuropathy, hypothyroidism, and right-sided heart failure. His peripheral edema was resistant to outpatient diuresis.

First, I will discuss the potential source of this patient's edema. Specifically, edema derives from either a cardiac or non-cardiac source. Could this patient have primary kidney or liver disease? An albumin of 4.2 g/dL goes against both nephrotic syndrome and primary liver disease as the causes of edema. Other findings which argue against liver disease in this patient include normal values for the serum total bilirubin and alanine aminotransferase. The increased prothrombin time, though modestly elevated, does not point strongly towards primary liver disease and could be due to passive congestion of the liver. Finally, jugular venous distention on physical examination is not a specific finding for either the nephrotic syndrome or primary liver insufficiency and is often caused by cardiac pathophysiology.

TABLE 3

Etiology of Pulmonary Heart Disease

I. DISEASES AFFECTING THE PULMONARY VASCULATURE

A. Primary Disease of the arterial wall

- (1) Primary Pulmonary Hypertension
- (2) Granulomatous pulmonary arteritis
- (3) Toxin-induced pulmonary hypertension
 - a. Aminorex fumarate
 - b. Intravenous drug abuse
- (4) Chronic liver disease
- (5) Peripheral pulmonic stenosis

B. Thrombotic disorders

- (1) Sickle cell diseases
- (2) Pulmonary microthrombi

C. Embolic disorders

- (1) Thromboembolism
- (2) Tumor embolism
- (3) Other embolism (amniotic fluid, air)
- (4) Schistosomiasis and other parasite diseases

II. PRESSURES ON PULMONARY ARTERIES BY MEDIASTINAL TUMORS, ANEURYSMS, GRANULOMATA, OR FIBROSIS

III. DISEASES OF THE NEUROMUSCULAR APPARATUS AND CHEST WALL

Table 3 (cont.)

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- A. Neuromuscular weakness
- B. Kyphoscoliosis
- C. Thoracoplasty
- D. Pleural Fibrosis
- E. Sleep apnea syndrome
- F. Idiopathic hypoventilation

IV. DISEASES AFFECTING AIR PASSAGES OF THE LUNG AND ALVEOLI

- A. Chronic obstructive pulmonary diseases
- B. Cystic Fibrosis
- C. Congenital developmental defects
- D. Infiltrative or granulomatous diseases
 - (1) Idiopathic pulmonary fibrosis
 - (2) Sarcoidosis
 - (3) Pneumoconiosis
 - (4) Scleroderma
 - (5) Mixed connective tissue disease
 - (6) Systemic lupus erythematosus
 - (7) Rheumatoid arthritis
 - (8) Polymyositis
 - (9) Eosinophilic granuloma
 - (10) Malignant infiltration
 - (11) Radiation
- E. Upper airways obstruction
- F. Pulmonary resection
- G. High-altitude disease

Adapted with permission. In: Rubin LJ. Introduction. Pulmonary heart disease. Boston: Martinus Nijhoff; 1984. p. 4.

Table 3 lists the many possible causes of cor pulmonale. Chronic obstructive pulmonary disease and pulmonary thromboembolism are common causes of chronic and acute cor pulmonale. This patient denied the use of tobacco and had no chronic pulmonary complaints. The findings on ventilation and perfusion scanning of the lungs were normal. One consideration was that he might have α_1 antitrypsin deficiency, which leads to both liver disease and emphysema. However, the pulmonary function tests did not demonstrate an obstructive pattern, and the chest roentgenogram (Fig. 1) was not consistent with emphysema.

Severe hypothyroidism can cause neuromuscular weakness sufficient to present as cor pulmonale. However, no other symptoms associated with hypothyroidism were mentioned and his serum thyrotropin hormone concentration was normal upon presentation.

Obesity, male gender, daytime somnolence, and difficulty in sleeping are suggestive of obstructive sleep apnea syndrome. Shortness of breath and cor pulmonale are recognized consequences of this syndrome. Polysomnography was consistent with moderate obstructive sleep

apnea syndrome, but certain inconsistencies point away from this diagnosis as the principal problem. In particular, ventilation and perfusion scanning of the lungs typically demonstrate mismatch. Arterial blood gases during the daytime are usually consistent with hypercapnia, and secondary erythrocytosis might be expected.

In an effort to explain the patient's presenting signs and symptoms, I will now focus on possible cardiac etiologies. The hallmark of right ventricular failure is systemic venous congestion, as evidenced in this patient by jugular venous distention. Dependent edema, which is very common, is one of the early signs of right ventricular failure. Organ dysfunction from right ventricular failure usually involves the liver first (1). Right upper quadrant abdominal pain, abnormal liver function tests, prolongation of the prothrombin time, and even coma can occur (1). Splenomegaly is possible secondary to right ventricular failure, but it is much less frequent than hepatomegaly and always accompanied by hepatomegaly. Ascites is a late manifestation of heart failure, appearing more frequently in tricuspid regurgitation, restrictive cardiomyopathy, or constrictive pericarditis (1).

Notably, an important clue in this case rests with the admitting chest roentgenogram (Fig. 1). The examination typically reveals impressive cardiomegaly when heart failure is caused by valvular disease and shunts. In contrast, this picture usually is not seen in pericardial and myocardial disease. In this case, the chest roentgenogram reliably excludes a large pericardial effusion. The echocardiogram in this case eliminates the possibility of valvular disease such as tricuspid or pulmonic regurgitation, and supports the logic of focusing on primary myocardial and pericardial disease. The echocardiogram also shows no evidence of either dilated or hypertrophic cardiomyopathy, thereby narrowing the differential in this case to restrictive cardiomyopathy and constrictive pericarditis. As a general rule, one always considers the diagnoses of restrictive cardiomyopathy and constrictive pericarditis when evaluating a patient in heart failure who has a normal heart size on chest roentgenogram.

It should be noted that an important clue regarding restrictive cardiomyopathy or constrictive pericarditis is missing. An abnormal pulsus paradoxus is often appreciated on the physical examination (Table 5).

Cardiomyopathies are classified into three major functional categories (Table 4). Restrictive cardiomyopathy is the least common of these three categories in the Western hemisphere; idiopathic disease and amyloidosis are the most common etiologies worldwide (2). Endomyocardial fibrosis is most prominent in tropical and subtropical Africa, causing 10-20% of deaths due to cardiac disease (2).

TABLE 4
Classification of the Restrictive Cardiomyopathies

MYOCARDIAL

Noninfiltrative

Idiopathic
Scleroderma

Infiltrative

Amyloid
Sarcoid
Gaucher's disease
Hurler disease

Storage disease

Hemochromatosis
Fabry's disease
Glycogen storage disease

ENDOMYOCARDIAL

Endomyocardial fibrosis
Hypereosinophilic syndrome
Carcinoid
Metastatic malignancies
Radiation

Reprinted with permission from Wynne J and Braunwald E. The cardiomyopathies and myocardites: Heart disease A textbook of cardiovascular medicine. 5th ed. Vol. 2, Philadelphia: W.B. Saunders Co.; 1997. p. 1427. Table 41-13.

The physiological hallmark of restrictive disease is diastolic dysfunction with rigid ventricular walls that impede filling. Contractile (systolic) function is usually normal. Hemodynamically, the "square root sign" (3) is a classical feature. This represents the dip and plateau caused by the rapid and deep early decline in ventricular pressure at the onset of diastole, with a rapid rise to a plateau in early diastole. Both pulmonary and systemic venous pressures are elevated, though a larger gradient does point towards a restrictive pattern. Left-sided pressures can be more than 5 mm Hg greater than that of the right side, and the pulmonary artery systolic pressure is usually greater than 50 mm Hg (1).

Symptoms and signs are a direct consequence of the altered physiology. Exercise intolerance is common because tachycardia cannot increase ventricular filling in the face of abnormally stiff ventricles. Chest pain, however, is usually absent. Central venous pressure is elevated, resulting in peripheral edema, liver congestion, ascites, and anasarca. Physical examination reveals an elevated jugular venous pressure and an S3, S4, or both. Kussmaul sign may be seen (3). The

TABLE 5
Constrictive Pericarditis versus Restrictive Cardiomyopathy

	CONSTRICTIVE PERICARDITIS	RESTRICTIVE CARDIOMYOPATHY
S ₃ gallop	Absent	May be present
Pericardial knock	May be present	Absent
Palpable systolic apical impulse	Absent	May be present
Pericardial calcification	Present 50%	Absent
Pulsus paradoxus	May be present	May be present
Equal RV and LV diastolic pressures	Usually present	LV>RV
Rate of LV filling	80% in first half of diastole	40% in first half of diastole
CAT scan, echo, MRI	Thickened pericardium	Normal pericardium

Modified with permission from Fowler NO: Constrictive Pericarditis. In Fowler NO, editor. The pericardium in health and disease. Mt. Kisco (NY): Futura Publishing Co.; 1985. p. 319.

apical impulse remains palpable. The electrocardiogram may show low voltage, atrial fibrillation,

and conduction abnormalities (2). The image pattern on echocardiogram may show a "ground glass" appearance suggestive of infiltrative disease, or it may show thickening of the left ventricular wall and an increase in left ventricular mass. Doppler studies may show an expiratory augmentation of hepatic vein diastolic flow reversal (4). The prognosis is indeterminate, though potentially carrying a high mortality rate. No specific therapy is available, but it has been speculated that calcium antagonists may be of some value (1).

Constrictive pericarditis also restricts diastolic filling. The mechanism is limitation of relaxation by a fibrotic, thickened, and adherent pericardium. It usually begins with an initial episode of acute pericarditis, often complicated by a pericardial effusion (5). This effusion undergoes fibrous scarring and thickening, obliterating the pericardial space (5). The most common etiology was formerly tuberculosis, but most cases are now diagnosed as idiopathic (42 percent) (6). Constrictive pericarditis after cardiac surgery and after radiation therapy are well described and have been increasing in frequency over the last decade (6). Rheumatoid arthritis and uremia are also known causes of constrictive pericarditis (6).

Clinical features result from increased systemic venous congestion as in restrictive cardiomyopathy. Common findings are edema, ascites, and passive hepatic congestion along with exertional dyspnea, cough, and orthopnea. Anginal chest pain can occur and is thought to result from underperfusion of coronary arteries that are compressed by the thickened pericardium (5). Kussmaul sign may be appreciated and elevation of the jugular venous pressure is typically observed (3). The arterial pulse may be diminished, but pulsus paradoxus is uncommon (3). A diastolic pericardial knock along the left sternal border may be auscultated (3). Hepatomegaly is usually palpated and anasarca may be found. The chest roentgenogram may show a small, normal, or enlarged heart size. A calcified pericardium may be noted on a lateral film but does not necessarily imply constrictive physiology (7).

Electrocardiogram findings include low QRS voltage and P mitrale. Atrial fibrillation is found in fewer than 50 percent of patients (2). Echocardiogram may show pericardial thickening and abrupt displacement of the interventricular septum during early diastole (septal bounce). Doppler studies may also show expiratory augmentation of hepatic-vein diastolic flow reversal (4). Notably, computed tomography and magnetic resonance imaging may show a thickened pericardium (8). Cardiac catheterization of both left and right ventricles will show near equalization of diastolic filling pressures in all chambers and, usually, a right ventricular systolic pressure of greater than 50 mm Hg. A classical "square root" sign, as with the restrictive pattern, may also be present (3).

Constrictive pericarditis occasionally reverses spontaneously (7). Relief of symptoms may require surgical stripping and removal of both layers of the adherent and constricting pericardium. In such cases, elevated venous pressure readings should normalize within the first three months following surgery (9).

The differentiation between restrictive cardiomyopathy and constrictive pericarditis can be very difficult because they both present with overlapping features (Table 5). The exact diagnosis is often found only in the operating room (2). In this particular case, no physical, hemodynamic, or Doppler findings were presented which would aid in the diagnosis. Potential diagnostic investigations include left heart catheterization, endomyocardial biopsy, or pericardial imaging -- all to differentiate primary

myocardial disease from pericardial disease.

I believe the ultimate diagnosis will be constrictive pericarditis for which a therapeutic procedure was performed. The likely procedure was a thoracotomy with pericardiectomy.

Clinical Diagnosis:

Right-sided heart failure, ? Dilated cardiomyopathy, and ? Obstructive sleep apnea syndrome.

Dr. Michael Kim's Diagnosis:

Constrictive pericarditis.

Pathological Discussion

Dr. Dushyant Purohit³: An endomyocardial biopsy of the right ventricle was performed, followed by a pericardiectomy. Specimens of myocardium, endocardium, and pericardium were submitted for examination.

Histological examination of the endomyocardial biopsy revealed normal myocardium with no evidence of inflammation, granuloma or necrosis of myocytes, fibrosis of the interstitial tissue, neoplastic infiltration, intracellular storage of abnormal metabolic products, or amyloid deposits. The endocardium was histologically unremarkable.

The surgical specimen of the pericardium consisted of three pieces, together measuring 21 x 13 cm and up to 0.6 cm in thickness (normal thickness of pericardium is 0.1 cm). There were no other grossly visible abnormalities, such as hemorrhage, fibrinous deposits, or any focal lesions. Histological examination of the thickened pericardium (Fig. 4) showed marked fibrosis and fusion of the two layers (parietal and epicardial) of the pericardium. Focally the fused surfaces of the pericardial layers showed a thick band of granulation tissue with fibrovascular proliferation, loose cellular stroma and very little mature collagen, suggesting an ongoing repair process. There was no acute or chronic inflammatory infiltrate, fibrinoid necrosis, granuloma, calcification or neoplastic infiltration.

The histopathological findings suggest fibrous pericarditis, consistent with the clinical diagnosis of constrictive pericarditis.

Fibrous pericarditis may develop as a result of varied pericardial abnormalities, including tuberculosis or similar types of granulomatous inflammation, septic pericarditis, trauma, cardiac

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surgery, irradiation, collagen/autoimmune disease and viral infection (5, 10, 11). More often, however, the etiology is unknown (12). The pericardium did not show histological features of any definitive etiology. While there was no clinical history of pericardial disease, a previous and resolved episode of pericarditis may have occurred. In the past, a common cause of constrictive pericarditis had been tuberculosis. More recently, viral pericarditis has been the major cause. It has been suggested that in some cases of constrictive pericarditis when there is no morphologic evidence of viral infection or any other specific cause, as in this case, there may have been a prior viral pericarditis that was relatively mild or subclinical (13).

Final Pathologic Diagnosis:

Fibrous pericarditis of unknown etiology, consistent with the clinical diagnosis of constrictive pericarditis.

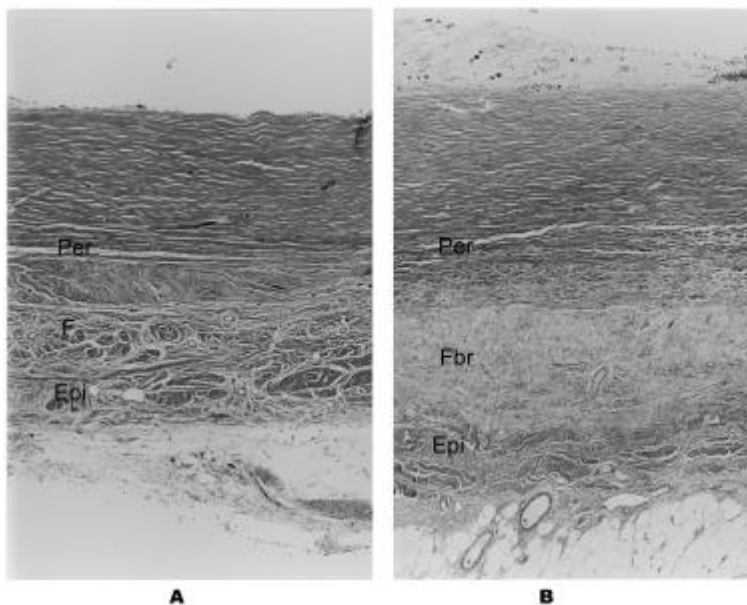


Fig. 4 - Fibrous pericarditis. (A): Parietal pericardial (Per) and epicardial (Epi) layers showing thickening, marked sclerosis, and fusion (F) of both layers. (B): In some areas of the fused pericardium, focal fibrovascular proliferation (Fbr) is seen with loose cellular stroma and paucity of mature collagen, suggesting an ongoing repair process. (Hematoxylin and eosin stain and magnification 40x).

Clinical Follow-up:

The patient tolerated the pericardiectomy without complications. One month after this procedure, the patient was readmitted for emergency repair of a ruptured umbilical hernia. The patient tolerated this surgery without any cardiac complications. Over the subsequent months, the patient had no evidence of heart failure. Diuretics and digoxin had been discontinued. The patient recently has become morbidly obese and at the last office visit weighed 306 pounds. He has sustained vertebral lumbar compression fractures and his diabetes has become more difficult to control.

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