Coronary Bypass-graft Stenosis Causing Diastolic Murmur in a Patient on Hemodialysis

To the Editor:

Diastolic murmurs are said to be common in patients on longterm hemodialysis. The murmur is typically decrescendo in type, is high-pitched and is heard along the left sternal border or over the cardiac apex.1,3 In ten patients studied by Matalon et al1 the murmur was attributed to functional aortic insufficiency. Of note was the fact that the murmur tended to be associated with significant systemic diastolic hypertension. Barratt et al2 performed aortic root angiography in eight patients and none had demonstrable regurgitation of the aortic valve. They suggested that the sound may have been of pericardial origin.

Stenosed coronary arteries have been reported to give a rise to diastolic murmurs.3 Falicov et al4 recently described a patient on longterm hemodialysis who had a decrescendo diastolic murmur. Aortic root injection demonstrated no aortic valvular insufficiency, but coronary arteriography revealed stenosis of the left anterior descending coronary artery. The authors postulated that the murmur was caused by relatively high and turbulent flow across the stenosed artery, and that this might be the source of diastolic murmurs in other hemodialysis patients.

A 26-year-old white man was hospitalized on February 10, 1979 for repeat coronary artery bypass surgery. He was a juvenile-onset diabetic who had been on hemodialysis for two years. Because of disabling angina pectoris and coronary artery disease, he underwent coronary artery bypass graft surgery in 1976 to the left anterior descending and left circumflex coronary arteries.

Recurrent angina pectoris brought him back to Hartford Hospital where a grade 2/6 high-pitched decrescendo diastolic murmur was heard along the left sternal border in the second and third intercostal spaces. In previous records, the absence of murmurs was specifically documented. The intensity of the murmur had no relation to dialysis treatments. The patient had a cervical venous hum which was obliterated with cervical venous compression, but such compression did not change the intensity of the murmur. His hematocrit was 33 percent. He was normotensive.

Coronary angiography demonstrated severe, proximal stenosis in both vein grafts. No aortic insufficiency was observed. Because of continued angina, the patient underwent bypass surgery a second time, with the original grafts being resected and replaced. After surgery, the diastolic murmur was no longer heard.

The disappearance of the murmur in our patient after bypass surgery supports Falicov's theory that in at least some cases, a decrescendo diastolic murmur in hemodialysis patients may originate in a stenosed coronary artery. In this case, the murmur appears to have been caused by a stenosed vein graft. To our knowledge, this is the first such reported case.

Cervical venous hums are common in dialysis patients and may even radiate to the thoracic area. Danshy and Ronan5 reported the presence of cervical venous hums in 70 of 80 dialysis patients. In seven patients, the hum was heard below the level of the second rib. Because of the disappearance of the diastolic murmur after surgery, and its persistence during cervical venous compression, a cervical venous hum with radiation to the thoracic area was excluded in our patient.

It is worth noting that previous reports5,6 describe these
diastolic murmurs as being loudest over the cardiac apex or in the fourth intercostal space. The murmur in our patient was heard only in the second and third intercostal spaces. This is most likely because of the cephalic anatomic position of the vein graft in relation to the left anterior descending coronary artery.

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REFERENCES

Exacerbation of Respiratory Failure by Paregoric

To the Editor:

Narcotic agents are known to be associated with deterioration of respiratory status in patients with or without pre-existing lung disease. We observed two patients with exacerbation of chronic respiratory insufficiency following routine administration of paregoric for ampicillin-induced diarrhea.

CASE REPORTS

A 64-year-old man with COPD and hypercapnea was admitted for treatment of tracheobronchitis and received oral ampicillin, 250 mg qid. Although pulmonary symptoms improved, he developed diarrhea and was treated with 5 ml oral paregoric. An hour later, he was noted to be somnolent and with worsened hypercapnia as evidenced by arterial blood gas levels. Over the next two hours, his general condition spontaneously improved. However, the diarrhea persisted and he received an additional 5 ml of paregoric with similar somnolence and deterioration of arterial blood gas levels. This episode again corrected itself spontaneously with return of PO2 to "pre-paregoric" levels.

A 58-year-old man with a similar history and clinical presentation also received paregoric 5 ml for ampicillin-induced diarrhea. His somnolence and acute respiratory depression from paregoric was rapidly reversed with intravenous naloxone 0.8 mg.

COMMENT

Although paregoric-induced acute respiratory depression has not been reported, both of these patients illustrate the need to avoid pharmacologic blunting of cortical drive. Paregoric USP contains 50 mg of powdered opium (equivalent to 2 mg of anhydrous morphine) per 5 ml. Administration of even small doses of narcotic alkaloids should be avoided in the patient with chronic hypercapnia. Fortunately, paregoric-induced respiratory depression reverses administration of naloxone, a method preferred to oxygen administration which may depress hypoxemic drive. Antibiotic-induced diarrhea may be treated with alternative antibiotics and/or kaolin-pectin suspension, prophylactic administration of a Lactobacillus preparation when starting ampicillin therapy, or use of an antidiarrheal agent with low respiratory depression potential such as the non-narcotic loperamide.

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REFERENCES

Cause of Chyloous Pleural Effusion

To the Editor:

Even though the occurrence of chyloous ascites is a well recognized complication of pancreatitis,1-5 most reviews of chylothorax, including the recent extensive clinical conference by Hughes et al (Chest 76:212-219, 1979) have not included pancreatitis as a cause of chyloous pleural effusion. Pleural effusions due to pancreatitis may vary greatly in their gross characteristics, ranging from hemothorax to serous effusion. Rare instances of chylothorax associated with pancreatitis have also been reported.4,5

In 1960, Evans4 observed left-sided pleural effusion three days after a fall in an elderly woman with diabetes mellitus and depression. She had pain in the right hip and inability to move her right lower extremity. Two thousand ml of milky-creamy fluid was aspirated from the left pleural cavity. This fluid "contained much fat when stained with Sudan IV" and had 690 mg/ml of total lipids (separate values for cholesterol and triglycerides are not available). The creamy opacity partially cleared with ether extraction. She succumbed and at autopsy was found to have opaque milky fluid in the peritoneal cavity (6,000 ml) and in the left pleural cavity (3,000 ml). Fibrosis of the pancreas, particularly in the head, was noted. This dense fibrosis extended posteriorly involving the periaortic tissue and also obstructed the lower part of the cisterna chyli and its tributary chyle radicles. The upper part of the cisterna chyli and the thoracic duct were normal.

In a second case,4 an acute left pleural effusion developed after drainage of a posttraumatic pseudocyst. Pancreatic