Pulmonary Artery-Bronchial Fistula
A New Complication of Bedside Pulmonary Arteriography

To the Editor:

We found the report by Rubin and Puckett (Chest 1979; 75:515-516) very interesting, as we have had a similar experience recently. In our case, a 48-year-old man with acute myocardial infarction had a 7F Swan-Ganz catheter placed in the pulmonary artery. Intracardiac knotting was discovered and the catheter was withdrawn slightly leaving the tip in the pulmonary artery. The second day, pericarditis was detected and on the fourth, a small left pleural effusion appeared on roentgenogram. To exclude a possible pulmonary thromboembolism, after the injection of 0.6 ml of saline solution in the balloon, a representative tracing of wedging was recorded,1 followed by an injection of 7 ml of contrast at a flow rate of approximately 2 ml/sec. The patient had an immediate attack of coughing and produced bloody sputum. The chest roentgenogram revealed a parenchymatous collection of contrast material near the tip of the catheter; bronchogram of the left bronchial tree was completely identical to the one shown in the report by Rubin and Puckett (Fig 1).

Several mechanisms have been proposed to explain the perforation of the pulmonary artery associated with the use of Swan-Ganz catheters, but the cause remains unclear.2 In our patient, the catheter tip was advanced deeply into a small pulmonary branch and perforation may have been caused by increasing pressure on the lateral walls of the balloon when the balloon was reinflated and/or the tip of the catheter deflected into the wall with the balloon inflated; the shearing effect of the tip against the wall, promoted by the rapid injection of contrast material, may have perforated the wall of the vessel, which could be a different mechanism than the one postulated by Rubin and Puckett. However, we were unable to demonstrate any increase in the PCP with the balloon inflated in three patients.

To prevent this complication we recommend placing the catheter tip into a main pulmonary artery and to avoid intracardiac knotting to prevent spontaneous wedging. Reinfusion of the balloon placed into a small branch should be done slowly while carefully observing the contour of the pulmonary artery pressure until the change to PCP is observed. If less than 0.8 ml is necessary to wedge the catheter, we suggest withdrawing it into a larger artery before injecting the contrast material. The injection of contrast medium is made by hand and good angiograms are obtained with 15 ml or less with the segmental pulmonary artery blood flow arrested.

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Pseudo Kussmaul’s Sign
An Observation in Severe Respiratory Insufficiency and COLD

To the Editor:

The observation of venous distention during inspiration has been classically described as Kussmaul’s sign3 and associated with compressive cardiac disorders characterized by limitation of diastolic filling.4 In respiratory insufficiency and chronic lung disease a mechanical obstruction of external jugular flow can visually produce a similar finding.

CASE REPORT
An 86-year-old white man with known severe emphysema was hospitalized following a right hip fracture after a fall. Medical history included treatment for pneumococcal pneumonia six months prior to admission. No jugular venous distention was noted. Chest examination disclosed markedly decreased breath sounds bilaterally with scattered rhonchi and wheezes and an increased anteroposterior diameter. Results of heart and abdominal examinations were normal; no hepatic enlargement was noted and no dependent edema was seen.

After transfer to the medical service, Kussmaul’s sign was noted in the absence of pulsum paradoxus. Echocardiography was performed to rule out pericardial effusion and features of constrictive pericarditis.6 Inspiratory external jugular venous distention was recorded (Fig 1).

Three hours after fixation of the hip, he developed respiratory distress and was transferred to the medical service for treatment of aspiration pneumonia. Despite vigorous therapy, a right lower lobe abscess evolved. Mixed flora was ultimately unresponsive to antibiotics, chest tube drainage and endotracheal intubation following episodes of CO2 retention. A tracheostomy was unable to provide respiratory toilet, and following several respiratory arrests, the patient could not be resuscitated.
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.  In ten patients studied by Matalon et al the murmur was attributed to functional aortic insufficiency. Of note the fact that the murmur tended to be associated with significant systemic diastolic hypertension. Barratt et al 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 rise to diastolic murmurs. Falicov et al 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 39 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 Ronan 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 reports describe these

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COMUNICATIONS TO THE EDITOR

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