A more plausible explanation for this early and rapid cardiac failure is related to the low cardiac output syndrome, which is seen in up to 28% of patients with chronic constrictive pericarditis after undergoing pericardiectomy [9, 10]. It is believed that the postoperative myocardial dysfunction in this setting is related to myocardial atrophy. The chronic external support of the heart by the tight pericardium and fluid when released may allow the heart to overdilate rapidly, leading to systolic dysfunction and heart failure. If this pathophysiologic mechanism also occurs in some patients with acute tamponade, then the suggestion by Neelakandan and associates [6] for gradual decompression of the pericardial effusion may have some merit, although the practical implementation of this suggestion may be difficult. Presumably pericardiocentesis with gradual removal of pericardial fluid with an indwelling catheter might be a feasible approach. However, this syndrome, no matter how lethal, is relatively rare and recommendations on prevention will await characterization of the patients at risk. Therefore, at present until more information is available, we recommend only that anesthesiologists, cardiologists, and thoracic surgeons be aware of the possibility that their patient might develop transient or even fatal heart failure after relief of a benign or malignant pericardial tamponade, similar to that seen in our patient. Treatment for this problem should be supportive with appropriate invasive monitoring and inotropic support, which will result in recovery in some patients.

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Surgical Management of Necrotizing *Candida* Esophagitis

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Invasive esophageal candidiasis produced transmural necrosis leading to perforation in 2 patients aged 10 and 27 years. Both patients survived after esophageal resection and complete diversion. One patient with acute leukemia and neutropenia experienced systemic candidiasis, which resolved after esophagectomy. Esophagectomy and diversion for yeast-induced necrosis may lead to complete recovery and resolution of disseminated candidiasis when combined with systemic antifungal therapy.

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Candida colonization of the gastrointestinal tract is the most important source of opportunistic dissemination. The esophagus is the most common site of colonization [1]. Although cases of ulceration, perforation [2], and stricture attest to the potential for panmural involvement, recent evidence suggest that ulceration and deep tissue invasion are rare in patients without neutropenia [3]. To our knowledge, no one has correlated the depth of esophageal invasion with the likelihood of disseminated candidiasis; however, normally functioning polymorphonuclear leukocytes likely limit invasion and concomitant dissemination [3]. Systemic antifungal therapy remains the best available treatment, and more aggressive intervention, including operation, is rarely considered. The prognosis is poor in disseminated candidiasis, and half of all neutropenic patients with disseminated disease die [4].

We report 2 patients who required surgical intervention for complications of panmural esophageal candidiasis. Immediate resection, indicated for perforation of the esophagus in one and total necrosis in the other, resulted in complete recovery and freedom of fungal colonization during a 1-year period of observation. These cases demonstrate that esophageal candidiasis may progress insidiously to necrosis, perforation, and mediastinitis, and they support the notion of invasive esophageal candidiasis as a nidus for systemic infection. Heightened awareness of fungal esophagitis as a risk factor for necrosis and prompt intervention may improve the outcome of patients with this condition.

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Patient 1

A 27-year-old autistic man had a history of recurrent urinary tract infections and was undergoing chronic hemodialysis for obstructive uropathy. Stool and urine cultures revealed colonization with vancomycin-resistant *Enterococcus.* In August 1996, an endoscopic examination to evaluate coffee-ground emesis demonstrated severe inflammation with white plaques in the lower esophagus. A biopsy sample indicated necrosis of the esophageal wall extending into muscle. Yeast were identified on a periodic acid-Schiff stain, and esophageal brushings grew *Torulopsis glabrata.* The patient was treated with oral fluconazole. One month later, he was admitted with recurrent vomiting and fever. An endoscopy without biopsy suggested improved, but persistent, esophagitis. A percutaneous jejunostomy tube was placed for enteral nutrition. One week later, the patient became profoundly hypotensive and required intubation. A new right pleural effusion was noted, and a meglumine diatrizoate study demonstrated perforation of the thoracic esophagus into the right chest. Exploration through a right thoracotomy exposed a 5-cm longitudinal tear in a thin lower esophageal wall denuded of mucosa and muscle. The esophagus was resected and a cervical esophagostomy was created. Histologic sections of the specimen showed diffuse inflammation and necrosis, and a silver methenamine stain revealed budding yeast indicative of candidiasis. Reconstruction of the swallowing passage was deferred owing to the patient's permanent disability.

Torulopsis glabrata grew from a urine culture done 1 month after endoscopic diagnosis of esophagitis and from pleural fluid after perforation. However, preoperative blood and postoperative blood and urine cultures did not grow yeast. Stool cultures obtained 2 months after removal of the esophagus also grew *T. glabrata*.

Patient 2

A 10-year-old boy with Down syndrome and repair of an atrioventricular canal defect at 3 months of age was found to have acute B-cell lymphocytic leukemia in November 1996. One week after beginning induction chemotherapy with cytosine arabinoside, vincristine, and asparaginase, the patient began to complain of a sore throat and he had difficulty swallowing. He was admitted 2 weeks after induction therapy with fever, neutropenia, and streptococcal bacteremia. Several days later, while thrombocytopenic, the patient was observed to have copious oral secretions and emesis with streaks of blood. One week later, bright red hematemesis and melena occurred. Endoscopic examination demonstrated necrotic tissue lining the wall of the thoracic esophagus. A biopsy of the lining disclosed lung tissue, and periodic acid-Schiff as well as silver stains later showed fungal hyphal elements. The contrast examination of the esophagus is depicted in Figure 1. On exploration through a right thoracotomy, the posterior mediastinum was empty except for necrotic debris and approximately 3-cm-long stumps of cervical and abdominal esophagus. The swallowing conduit was made up of the mediastinal surface of both lungs, the prevertebral fascia, and the posterior pericardium. The mediastinum was drained, the gastric cardia closed with sutures, and a cervical esophagostomy was created. A gastrostomy and a jejunostomy were placed. Esophageal tissue grew *Candida* organisms. Immunohistochemical stains for cytomegalovirus and herpes simplex virus were negative. The patient had disseminated candidiasis, and *Candida albicans* grew from blood, urine, external auditory canal, sputum, and stool cultures. He also received systemic antifungal therapy. After complete recovery, consolidation chemotherapy with teniposide and arabinoside was resumed. Four months later, a substernal gastric conduit with cervical esophageal anastomosis was constructed. The patient returned to an unrestricted oral diet and continues to receive chemotherapy. A recent bone marrow aspirate contained less than 1% blastocytes.

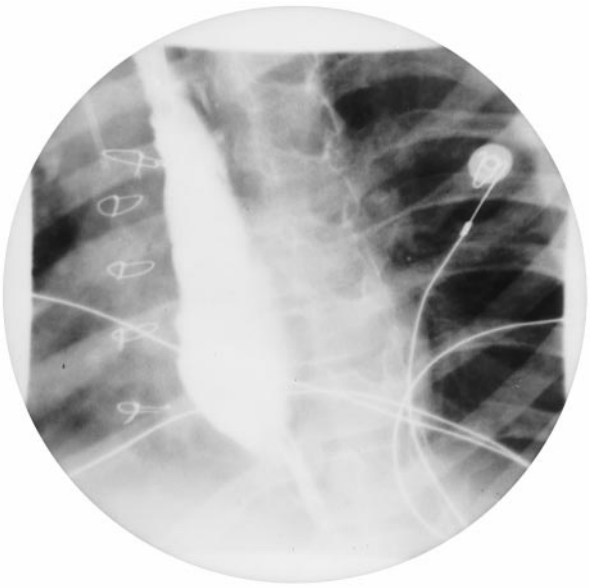

Fig 1. Esophagogram (patient 2). Note pear-shaped contour of mediastinal contrast with tip of nasoesophageal tube terminating in lower esophagus of normal caliber.

Stool cultures grew *Candida* organisms nearly 1 year after esophagectomy, although repeated postoperative blood, sputum, and urine cultures did not grow yeast.

Comment

Candidiasis resulted in transmural inflammation and was advanced at the time of diagnosis. In patient 1, biopsy confirmed loss of mucosa and exposed muscle. Three months after this biopsy, perforation through a weakened wall resulted in gross contamination of the right pleural space. Esophageal necrosis occurred in patient 2 within 1 month after diagnosis of leukemia. A similar case of rapidly evolving, invasive esophageal mycosis was reported in a 30-year-old patient who disgorged an infarcted segment of esophagus 1 month after diagnosis of acute myeloid leukemia [5]. The patient had more extensive disease with severe medullary and ex-

tramedullary infiltrates. No specific therapy was instituted and he died. Leukemia infiltrates in the esophagus could have mimicked our second patient's presentation; however, this was not likely to have caused necrosis. Not only is esophageal leukemia involvement rare [6], but in our patient the residual esophagus did not show leukemia infiltrates, and the disease was well controlled after induction chemotherapy. Our patients therefore are remarkable for advanced esophageal candidiasis and for their ability to recover from a life-threatening illness.

The standard therapy of *Candida* esophagitis is fluconazole [7]. Itraconazole combined with flucytosine or amphotericin B is preferred for fluconazole-resistant strains [8]. The species of yeast isolated from patient 1 is known to be relatively resistant to fluconazole, and treatment with amphotericin B may have prevented progression to esophageal perforation. In both patients, yeast were cultured from multiple body sites before esophagectomy, but stool was the only site of colonization after recovery from operation. Both patients remained free of fungal infection during a 1-year follow-up after operation despite persistence of risk factors and enteric colonization with yeast.

Resection was indicated in our patients for life-threatening complications of esophageal mycosis. Clinical deterioration in disseminated fungal disease, particularly in neutropenic patients, is not commonly attributed to a single organ but to systemic disease. The favorable outcome in our patients points to resection as a potential alternative in the management of patients with disseminated candidiasis and transmural esophagitis, but without perforation, who are failing medical therapy. There is no reliable way to assess the severity of esophageal disease because endoscopy or radiographic study may underestimate the depth of inflammatory involvement. Future efforts should target better diagnostic tests, perhaps endoesophageal ultrasound, to elucidate the extent of esophageal disease in the hopes that more aggressive medical and surgical management would prevent catastrophic complications.

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Aortopulmonary Window With Anomalous Origin of the Right Coronary Artery

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We report a case of an aortopulmonary window with a right coronary artery arising from the pulmonary trunk. This exceedingly rare anomaly with anomalous coronary artery presented without myocardial ischemia owing to the aortopulmonary window. The correct diagnosis was made by angiography and a successful surgical correction was performed.

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Aortopulmonary window is a rare but well-described congenital heart disease. However, the combination of aortopulmonary window with an anomalous origin of the right coronary artery from the pulmonary trunk is extremely rare and only about 8 cases with the same anomaly have been published worldwide [1–8]. The purpose of this paper is to present a literature review (Table 1) and a new case of this rare abnormality with the physiologic effects and its surgical correction.

A 3-week-old male infant was admitted with history of intermittent tachypnea and failure to thrive. On angiography there was a 4-mm aortopulmonary window between the inner curvature of the ascending aorta and the pulmonary trunk (Fig 1). The mean arterial pressure in the aorta was 45 mm Hg and 31 mm Hg in the main pulmonary artery with an O_2 saturation of 100% and 96%, respectively. A patent ductus arteriosus was present as well as a small atrial septal defect. The left coronary artery was normal, but the right coronary artery (RCA) originated anomalously from the right anterior aspect of the main pulmonary artery proximal to the window (Fig 2).

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