often delayed. Large sclerotic ant volume, high concentration, and advanced liver disease have been associated with increased risk of perforation.\(^3\) The specific complication of esophageal-pleural fistula has not been previously documented (to our knowledge), and was clearly necessary for the formation of this patient's empyema.

Studies performed by the food industry have shown \(S\) \textit{cerevisiae} to be virtually nonpathogenic.\(^4\) Our review of the medical literature found only 15 cases of serious infection in humans, including six episodes of fungal sepsis and one of empyema.\(^5^{14}\) Many of these patients had underlying debilitating diseases and a portal of entry for yeast. Two patients ingested brewer's yeast daily,\(^5\) and one case of \(S\) \textit{cerevisiae} peritonitis followed an abdominal procedure performed by surgical personnel who were also amateur bakers.\(^6\) Our patient prepared a chicken pot pie using brewer's yeast two weeks before hospital admission.

In summary, we present a patient with an esophageal-pleural fistula as a complication of prophylactic variceal sclerotherapy. Esophageal-pleural fistula should be considered a potentially fatal complication in any patient after sclerotherapy who presents with a pleural effusion. Organisms normally considered harmless must be considered potential pathogens in patients compromised by such a fistula.

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REFERENCES


\textbf{Pacemaker-Lead Puncture of the Tricuspid Valve*}

\textbf{Successful Diagnosis and Treatment}

\textbf{Pedro A. Rubio, M.D., Ph.D., F.C.C.P.; and Mohdi S. Al-Bassam, M.D.}

During insertion, a transvenous pacing lead pierced the tricuspid valve in a 66-year-old man, causing progressive tricuspid insufficienty and congestive heart failure. The defect remained undiagnosed for ten years and was then repaired. To our knowledge, this is the first case in which this problem has been successfully treated rather than being diagnosed at autopsy. (\textit{Chest} 1991; 99:1519-20)

Pacemaker-lead puncture of the tricuspid valve is an extremely rare complication of cardiac pacing. The four cases reported so far have all been diagnosed at autopsy.\(^1\)\(^4\) We describe a tricuspid puncture that occurred during lead implantation but was not diagnosed until ten years later. Surgical repair resulted in a successful outcome.

\textbf{CASE REPORT}

A 76-year-old man was admitted to our hospital with severe shortness of breath, sharp, mostly right-sided chest pain, lower abdominal pain, and massive edema of both lower extremities that had worsened over a seven-year period and had not responded to treatment. At age 66 years, the patient had sustained an extensive myocardial infarction with heart block, for which a permanent ventricular-inhibited transvenous pacemaker was implanted. One year before the present hospital admission, he had undergone repair of an atrial septal defect and aortocoronary bypass to the right coronary artery.

At the time of hospital admission, the patient had a blood pressure of 130/80 mm Hg, respirations of 20/min, and an irregular pulse of 82 beats/min. He also had an irregularly irregular cardiac rhythm, a grade 3/6 holosystolic ejection murmur heard best at the apex, and 3+ pedal edema. Other physical findings were unremarkable. Electrocardiography showed atrial fibrillation with a controlled ventricular rate and right bundle-branch block. Right- and left-sided cardiac catheterization disclosed severe coronary artery disease and severe tricuspid insufficiency.

The patient underwent sequential aortocoronary bypass from the ascending aorta to the anterolateral obtuse marginal and left anterior descending arteries with a reversed saphenous vein. The previous right coronary bypass was found to be functioning well.

Once the distal coronary anastomoses had been created, the right atrium was opened and the tricuspid valve was found to be dilated to a width of 7 cm. The pacemaker lead traversed and immobilized the posterior leaflet, to which it was attached by scar tissue (Fig 1).

The lead was divided as low as possible in the right ventricle, and the left-chest segment was removed, but the right ventricular portion remained inexcitable. Nevertheless, enough of the embedded wire was removed to allow free motion of the affected leaflet. The perforated leaflet and the anterosetal commissure were then repaired, and the tricuspid insufficiency was corrected with a de Vega annuloplasty. The pericardium was closed with a patch (Gore-Tex). An epicardial pacemaker was inserted, and circulatory support was initiated with an intraventricular balloon pump.

Postoperatively, the patient did well, except for a right-sided pleural effusion that was resolved with pleurocentesis. He was discharged from the hospital on the 16th postoperative day. Although

\*From the Departments of Surgery (Dr. Rubio) and Cardiology (Dr. Al-Bassam), HCA Medical Center Hospital, Houston.
Fatal Airway Obstruction Caused by a Mucous Ball from a Transtracheal Oxygen Catheter*

George G. Burton, M.D.; F.C.C.P.;† Fred A. Wagshal, M.D.;‡ Diannah Henderson, R.N.,§ and Samuel Wesley Kime, M.D.¶

A 50-year-old man with pulmonary fibrosis and COPD presented with worsening cough, dyspnea, chest pain, and hypoxemia of no readily apparent etiology, approximately four weeks after insertion of a transtracheal oxygen therapy catheter. Despite vigorous bronchial hygiene therapy, the patient died. Autopsy revealed obstruction of the trachea by a large mucous ball. We point out the nonspecificity of physical and radiologic findings associated with this condition and suggest that serial flow-volume loop analysis or earlier use of fiberoptic bronchoscopy might have been of assistance in premortem diagnosis of the mucous plug. (Chest 1991; 99:1520-23)

The use of percutaneously inserted TTO₂T catheters in hypoxic patients has been reported to result in less dyspnea, greater acceptance by the patient of oxygen dependency, and physiologic, aesthetic, and financial benefits (the latter due to variable reductions in gas flow necessary to maintain adequate tissue oxygenation). Few serious and no fatal complications of TTO₂T have yet been reported. We report a case of fatal obstruction of the trachea by a 7.0 × 3.0 × 3.0-cm mucous ball around a TTO₂T catheter.

CASE REPORT

A 50-year-old man was found to have pulmonary fibrosis at open lung biopsy in February 1987. In addition to his work as a metal grinder in a foundry, he had a 70-pack-year smoking history, and pulmonary function tests had shown obstructive and restrictive disease. At that time, he had significant exercise desaturation and was discharged to his home on oxygen therapy (1.0 L/min at rest and 2.0 L/min with exercise).

On May 26, 1989, the patient was admitted with increasing dyspnea, fever, chills, and dusky cyanosis. His resting values on room air were PaO₂ of 46 mm Hg and PaCO₂ of 24 mm Hg. A ventilation-perfusion lung scan suggested low probability for pulmonary emboli. No pneumonia was found. His COPD was optimally treated, and his condition improved. On June 2, 1989, the stent of a TTO₂T catheter (SCOOP; Transtracheal Systems) was inserted percutaneously, with careful instructions being given to the patient and his wife regarding its maintenance, including twice daily cleaning of the stoma. The TTO₂T device was inserted by one of us, a physician formally trained in its use. One week later, on the basis of exercise saturation, an oxygen flow of 2 L/min at rest and 3 L/

*From the Respiratory Services Department, Kettering Medical Center, Kettering, Ohio, and Wright State University School of Medicine, Dayton.
†Chief, Respiratory Services, Kettering Medical Center, and Clinical Professor of Medicine and Anesthesiology.
‡Assistant Clinical Professor of Medicine.
§Supervisor, Pulmonary Rehabilitation Program, Kettering Medicine Center.
¶Associate Clinical Professor of Pathology.

Reprint requests: Dr. Burton, Kettering Medical Center, 3535 Northern Blvd., Kettering, Ohio 45429

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