in the United Kingdom had originally documented pulmonary changes in 28 of 32 cases, but Spelman found only eight of 111 patients with Q fever pneumonia. Marrie et al recently reported an incidence of acute pulmonary changes in three of 16 cases.

In Millar's series, the most common pulmonary abnormality was single or multiple round pneumonia, usually in the basal segments of the lung. Lobar consolidation also occurred, but was less frequent. During resolution, the pneumatic consolidations have resembled coin or mass lesions. The histologic findings of the pneumonia is usually that of a monocytic infiltration. In the case of a resected inflammatory pseudotumor of the lung, the histologic findings demonstrated a mixture of mononuclear cells obstructing the bronchioles and infiltrating the alveoli and septa. In inflammatory pseudotumor, the composition of monocytic cellular infiltrate is highly variable but similar to the monocytic infiltration in acute Q fever pneumonia. An infiltrative component was also previously noted by Millar who observed the common presence of atelectasis in some cases of Q fever pneumonia.

The similarities of inflammatory pseudotumor of the lung and Q fever rounded pneumonia are not surprising, the key importance of pseudotumor is its roentgenographic mimicry of a neoplastic lesion, resulting in resection as reported by Janigan and Marrie.

The case we are reporting had the roentgenographic features of a malignant neoplasm, identical to the pseudotumor described by Janigan and Marrie. Bronchoscopy was performed to confirm the diagnosis of malignancy, but no abnormalities or malignant cells were revealed. The detection of Q fever antibody established the correct diagnosis, and with the institution of appropriate antibiotic treatment, the pseudotumor resolved completely in one month, both roentgenographically and serologically.

We believe that Q fever pneumonia and pseudotumor are probably variations of the pulmonary changes in Q fever. The treatments are the same and a pseudotumor will make a rapid response to antibiotic therapy, as was illustrated with our case. With a high index of suspicion and good serologic findings, correct diagnosis and treatment, thoracotomy for these pseudotumors can be avoided.

REFERENCES
7 Mushes DM. Q fever: a common treatable cause of endemic nonbacterial pneumonia. JAMA 1965; 204:863-66

Pneumomediastinum after Self-Dilation of the Esophagus

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Pneumomediastinum following esophageal perforation is a known complication of Eder Puestow dilation for esophageal stenosis. This is the first reported case of esophageal perforation and pneumomediastinum occurring after instrumental self-dilation of a stenotic esophageal lesion. The observed 0.02 percent perforation rate in this patient (compared to the reported 0.3 percent in Eder Puestow "hetero"-dilation) makes the Eder Puestow autodilation procedure seem justifiable in a well-trained and well-informed patient.

E der Puestow dilation (EPD) of the esophagus has been utilized for many years as a treatment for esophageal stenosis and relief of dysphagia. Esophageal perforation after EPD occurs in about 0.3 percent of procedures. Blind dilation is thought to have a greater rate of perforation than with fluoroscopic guidance. We report a patient who developed pneumomediastinum after self-dilation of a postcricoidal esophageal stenosis, a procedure that was carried out by the patient for almost 30 years without complications.

CASE REPORT

A 65-year-old man was admitted because of intense intrathoracic pain and dyspnea. The pain started 12 h earlier rather insidiously, a few minutes after self-dilation of a stenotic esophageal lesion with metal olives (Eder Puestow technique, dilation sequence 31-35 Ch olives). The patient performed this procedure by himself three times a week for 30 years, after a one-year supervised training period. The stenosis had occurred after caustic esophageal damage due to accidental drinking of lye.

At the time of admission, blood pressure was 130/90 mm Hg; heart rate was 100 beats per minute; and temperature was 36.7°C. The patient was dyspneic, polyneic (30 breaths per minute), and complained of stabbing precordial chest pain. There were no signs of subcutaneous emphysema. Heart sounds were normal. Careful auscultation of the lungs revealed a few crackling rales over the left lung base.

The electrocardiogram revealed sequelae of an inferior myocardial infarction. Standard chest roentgenogram showed mediastinal widening and vertical lines of radiolucency confirming the presence of pneumomediastinum. A Gastrografin swallow study showed stenosis of the esophagus and a prestenotic diverticulum, with extravasation of the contrast medium.

Laboratory values were normal except for the white blood cell count (11,000/cu mm, with 90 percent polymorphonuclear neutrophils). The diagnosis of postdilation esophageal perforation and pneumomediastinum was withheld, and the patient was transferred to the Intensive Care Unit. Treatment consisted of withdrawal of all oral intake analgesic agents (morphine sulfate, pentazocine), antibiotics (penicillin G, netilmicin, metronidazole), continuous esophagogastric aspiration, total parenteral nutrition and intravenously given fluids, according to the current conservative ap-
proach to noncomplicated esophageal perforation. ¹ ² ³

A control Gastrografin swallow at day 13 showed absence of extravasation. Cautious oral administration of fluids and, later, food intake was started; total parenteral nutrition was discontinued. On day 18, the patient left the hospital.

**Discussion**

Perforation of the esophagus is the most common serious complication of instrumental esophageal dilation. The incidence of perforation is higher with pneumatic dilation for achalasia (1 to 5 percent), than with EPD with metal olives (0.3 percent) and mercury bougienage with Maloney or Hurst dilators (0.1 percent). ¹ ²

Dilation of the esophagus carries a risk for perforation because most of the time it is performed for stricture (reflux esophagitis, postoperative stenosis, achalasia). Perforations, therefore, generally occur in the diseased portion of the esophagus. ³ The risk of perforation is increased: (1) after hasty dilation, consisting of either too rapid progression through the dilation sequence or too frequent dilation sequences; ³ (2) dilation of chronic lye strictures; ³ and (3) when luminal irregularities, Zenker’s diverticulum, severe esophagitis or ulceration, and malignant neoplasm with prestenotic diverticula are present. ³ ⁴

It is not clear why, after an uneventful 30-year period of self-dilation, perforation eventually occurred in our patient. He denied having changed his dilation sequence or technique. There were no concurrent illnesses or new conditions known to increase perforation risk. We presume that perforation occurred in the setting of the dilation of a chronic lye stricture associated with a prestenotic diverticulum, two conditions known to increase perforation risk in themselves.

The clinical and radiologic presentation of esophageal perforation and pneumomediastinum, as well as the diagnostic approach to this problem, are well documented elsewhere. ³ ⁴ ⁵

Eder Puestow dilation always carries a risk for perforation, its reported incidence being 0.3 percent. ³ ⁴ Our observation of a 0.02 percent perforation rate after 30 years of self-dilation is adequate testimony for the safety of EPD carried out by the patient. When considering cost-effectiveness and safety, these observations make self-dilation procedures seem justifiable in certain circumstances, provided the patient is well-instructed and well-trained. Although this is so, we now probably would prefer to instruct patients in a dilation technique other than the Eder Puestow method.

**References**


**Bigeminal and Trigeminal Distribution of Ventricular Extrasystoles as an Expression of “Atypical” Concealed Bigeminy**

**Giuseppe Orote, M.D.; Francesco Luzza, M.D.; Gaetano Satullo, M.D.; and Leo Schamroth, M.D., D.Sc., F.C.C.P.**

This report reflects a case of bigeminal and trigeminal ventricular extrasystoles where bigeminal extrasystoles are associated with short coupling intervals, while trigeminal extrasystoles manifest long coupling intervals. The arrhythmia is interpreted as an “atypical” form of concealed bigeminy.

Bigeminy and trigeminy are the two most common distributional patterns of extrasystoles and very often manifest in the same tracing. The transition from the bigeminal to the trigeminal rhythm can occur without any apparent reason and in such a way that the extrasystoles appear in a haphazard distribution. This presentation reflects a case where bigeminal and trigeminal ventricular extrasystoles are associated with remarkably different coupling intervals. The arrhythmia is interpreted as an “atypical” form of concealed bigeminy.

**Case Report**

The electrocardiogram was recorded from a 62-year-old man with coronary artery disease. Figure 1 reflects a basic sinus rhythm with frequent ventricular extrasystoles that manifest in bigeminal or trigeminal rhythm and occasionally in quadrigeminal rhythm. The ectopic ventricular complexes will be termed X, whereas the sinus beats will be termed R. The sinus beats contained in each interbeat interval are progressively numbered as R1, R2, and R3. Analysis of a long recording reveals the following:

First, the extrasystolic coupling intervals are variable. Bigeminal extrasystoles are associated with short coupling intervals, which range from 0.44 second to 0.57 second, whereas trigeminal extrasystoles manifest with longer coupling intervals, which range from 0.68 second to 0.76 second and often result in ventricular fusion beats. The extrasystoles in quadrigeminal distribution (ie, those preceded by three sinus beats) have relatively short coupling

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