patients with AEP, but also of any other patients with respiratory symptoms. In CS-AEP, it is especially important to know when the patient started smoking.

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Echocardiography in the Diagnosis and Management of Pulmonary Embolism

A Limerick

If PE you want to detect,
And ÉCHO is what you select,
Why did you not plan
CT or lung scan
To prove there’s a blood flow defect?
Once diagnosis is secure,
The ÉCHO itself is no cure.
For prognostication,
Risk stratification,
ÉCHO can guide treatment for sure!
You’re still hesitating, I see.
Not certain about the RV?

Figure 1. Graphs of data obtained during a challenge test. Top, WBC and eosinophil count (in count \(10^3/\mu\text{L}\) vs time. Middle, \(\text{PaO}_2\) and \(\text{PaCO}_2\). Bottom, diffusion capacity of lung for carbon monoxide (DLCO) and FEV\(_1\) as percent of FVC vs time. m-PSL = methylprednisolone sodium succinate.
Tracheobronchomalacia

A Cause of Flow Oscillations on the Flow-Volume Loop

To the Editor:

The visual inspection of the flow-volume loop can reveal the presence of flow oscillations. Flow oscillations are defined as a reproducible sequence of alternating decelerations and accelerations of flow.1 In a large retrospective survey of 2,800 flow-volume loops, the incidence of flow oscillations was 1.4%. These oscillations can be due to a variety of causes, such as obstructive sleep apnea syndrome, structural or functional disorders of the larynx, or neuromuscular diseases, but tracheobronchomalacia has not been mentioned.

A 58-year-old man with a diagnosis of COPD with chronic cough and exertional dyspnea was referred to the Respiratory Medicine Division because of a pulmonary nodule. The patient did not have symptoms of sleep disorders or neuromuscular disease. At fiberoptic bronchoscopy, the larynx was normal, but the trachea and main bronchus completely collapsed even during quiet expiration. Thoracic CT revealed a 1.5-cm nodule, and images obtained during expiration demonstrated the collapse of the trachea. Spirometry showed FVC of 3.96 L (118% of reference value) and FEV1 of 2.28 L (84%); the volume-time loops, the incidence of flow oscillations was 1.4%.

The prevalence of tracheobronchomalacia is relatively low; 1% and 4.5% in two large series of bronchosopies.2 The principal symptom is chronic dyspnea, but attacks like those in asthma are seldom seen, and response to bronchodilator treatment is scanty.3 In patients with tracheobronchomalacia, a typical notch (can be one or more) in the volume-time curve of the spirogram has been reported; this finding was not found in the patient described here. The notch is considered to reflect a sudden diminution of flow at the beginning of the expiration when the airway collapses. Nevertheless, alterations on the flow-volume loop have not been reported.

The finding of flow oscillations on the flow-volume loop should instigate extensive investigations primarily directed at the upper airway and its surrounding musculature.1 In view of the case described here, tracheobronchomalacia should be considered among the possible causes of flow oscillations.

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Catamenial Pneumothorax

An Example of Porous Diaphragm Syndromes

To the Editor:

I would like to comment on the letter of Funatsu et al (December 1999),1 concerning the relationship of catamenial pneumothorax to the peritoneal stomata of the diaphragm.

I do not believe that the peritoneal stomata have anything to do with catamenial pneumothorax, much less Meigs’ syndrome or the many other examples of transdiaphragmatic passage of gases, fluids, blood, tissue, chyle, and other substances from the abdominal cavity to the pleural space.

I have characterized these clinical phenomena as porous diaphragm syndromes because of their common feature, a defect in the diaphragm.

Certainly, peritoneal stomata do exist, having been demonstrated by silver stains by von Recklinghausen3 in 1862 and by many others, long before the observations of Allen4 in 1936 and more recently by Li and Yu5 in 1991. But these stomata are tiny, requiring electron microscopy and scanning electron microscopy to demonstrate them. They are hardly large enough to account for the massive and rapid transfer of gases (as occurs in catamenial pneumothorax and artificial pneumoperitoneum with pneumothorax), fluids (found in cirrhosis of the liver with hepatic hydrothorax and peritoneal dialysis), blood (occurring in hemoperitoneum with bloody pleural effusion), and even chyle.

Such conditions are best explained by the presence of gross defects in the diaphragm, which have been demonstrated repeatedly at thoracotomy, thoracoscopy,6 and autopsy.7 Strangely enough, such defects have yet to be observed in Meigs’ syndrome, although Drs. Edward Churchill and Richard Sweet, (eminent Harvard thoracic surgeons, both quoted by Meigs) assumed them to be present and, therefore, to account for the hydrothorax.2

Why the right-sided thoracic predominance? I believe that spikes of intraperitoneal pressure in the right subphrenic space accompanying respiratory activity and physical exertion, with the solid fixed liver acting as a piston, are responsible. In contrast, the loose arrangement and increased mobility of the left subphrenic organs minimize such pressure changes under the left hemidiaphragm.

Finally, if the peritoneal stomata theory were correct, every case of cirrhosis with ascites and every peritoneal dialysis should develop hydrothorax, and every case of pneumoperitoneum (spontaneous, as in catamenial pneumothorax, or induced, as in laparoscopy) should develop pneumothorax. Also, it would occur bilaterally with equal incidence. This is patently not the case.

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ECHOCARDIOGRAPHY.