There was also no associated chest pain. Serum chemistry study revealed increased total CPK (1,952 U/L), CPK-MB (221 U/L), glutamic oxaloacetic transaminase (51 IU/L), and lactate dehydrogenase (781 IU/L) and normal electrolyte levels, suggesting the occurrence of acute myocardial infarction. The patient died 5 days after admission, and the elevation of the ST segment persisted until immediately before his death.

An autopsy showed that bilateral acute pyelonephritis and perirenal abscess were the cause of death. No evidence of acute myocardial infarction, sclerosis, or obstruction of coronary arteries or recent occurrence of cerebrovascular diseases was observed at postmortem investigations.

Although elevation of the ST segment, except in ischemic heart disease, appears in acute pericarditis, with high serum potassium level, and after DC cardioversion, these conditions were clearly excluded by the clinical and autopsy findings. I assume that mechanical damage of the heart that occurred during a general convulsion may have been the primary cause of the remarkable and vast elevation of the ST segment on the ECG in this patient.

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Complications of Central Venous Catheterization

To the Editor:

I read with interest the article by Duntley et al., which appeared in the June 1992 issue of Chest. The authors presented clinical data on eight patients with complications caused by central venous catheterization. The authors attributed the respiratory and cardiovascular symptoms observed to vascular erosion and secondary perforation caused by these catheters and offered radiographs to support their hypothesis. I would like to discuss whether another mechanism might be more probable.

In at least five of the eight patients, multilumen catheters were inserted. In such patients, extrusion of central lines can be caused by inadequate fixation of the catheters and/or movements of the patients resulting in extravascular positioning of at least one lumen. Pleural effusions and mediastinal widening can also occur by this mechanism. I reviewed 469 of our ICU patients with central lines (218 double- or triple-lumen catheters) and detected 3 secondary dislocations of the multilumen catheters with similar radiographic signs. In our intensive care patients, diagnosis of outward displacement was confirmed by extravasation of radiographic contrast media infused through proximal ports. Contrast infusion through the distal port demonstrated an adequate intravascular position of the catheter tip in these three patients.

The authors did not give detailed information concerning which port was used to infuse the contrast media in their patients. Our findings suggest that the migration of at least one catheter lumen into an extravascular position, not vascular erosion and perforation, could be the cause of the symptoms described in the patients reviewed by Duntley et al.

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To the Editor:

We appreciate Dr. Bach's suggestion of an additional mechanism for the formation of hydrothoraces in patients with central venous catheters. We do not believe, however, that catheter withdrawal
with tissue infiltration of fluid from the proximal port was more probable than catheter-tip erosion through central venous structures in our patient series. All catheters were secured to the skin to prevent migration, and seven of the eight patients had radiographic evidence of sufficient catheter insertion to avoid positioning of the proximal port within chest-wall tissues. In the remaining patient, the right subclavian catheter tip was positioned just below the clavicular head. This position may have contributed to pleural effusion formation by the infiltration of fluid infused through a proximal port. Radiographic contrast material infused through the distal port of this catheter, however, demonstrated extravasation into the mediastinum and erosion of the catheter tip through the medial wall of the right brachiocephalic vein. In all four patients who underwent contrast infusion, the contrast was instilled through the distal port of the catheter and demonstrated catheter erosion through central venous structures.

Dr. Bach’s recent report lends support to another mechanism of pleural fluid formation in patients with poorly secured central venous catheters undergoing migratory withdrawal. His observations, however, do not exclude the importance of catheter-tip erosion when catheters move in the opposite direction.

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Causes of Pleural Effusion in 75 HIV-infected Patients

To the Editor:

In HIV-infected patients, infectious, malignant, or aspecific etiologies have been frequently reported as the origin of bronchopulmonary lesions. However, there are very few data about the types and frequency of different causes of pleural effusion in such patients. We have retrospectively scrutinized the causes of pleural effusion in 75 HIV-infected patients diagnosed in the east Paris hospitals between 1986 and 1989. Criteria for pleural effusion of infectious origin were as follows: (1) identification of an opportunistic pathogen or Mycobacterium tuberculosis by direct examination and/or culture of pleural fluid specimens; (2) presence of pleural lesions specific for tuberculosis; or (3) identification of a pathogen by blood culture and/or culture of protected bronchial brushing products followed by a favorable outcome on appropriate anti-infectious treatment for bacteria. Criteria for pleural effusion due to Kaposi’s sarcoma were as follows: (1) typical macroscopic pleurapulmonary lesions seen during thorascopy or thoracotomy with confirmation on biopsy specimen; or (2) histologically confirmed skin lesions associated with characteristic macroscopic endobronchial lesions and bilateral lung abnormalities suggestive of Kaposi’s sarcoma on chest radiography and computed tomography. Biopsies were also diagnostic for malignant causes other than Kaposi’s sarcoma.

The underlying cause of pleural effusion was definitely established in 61 cases (81 percent). Table 1 shows the causes of pleural effusions in these 61 cases. Pleural effusions caused by aerobic bacteria were usually associated with pneumonia (8 of 11 cases, of which 6 were due to Streptococcus pneumoniae). Their frequency was probably underestimated because of possible negative culture results related to empirical antibiotic therapy prior to investigation. Pleural effusions due to opportunistic agents were caused by Cryptococcus neoformans (n = 4), Leishmania donovani (n = 1), and Pneumocystis carinii (n = 1). Tumors other than Kaposi’s sarcoma were lymphoma (n = 2) and squamous cell carcinoma (n = 1).

Although pleural effusions are observed in 50 percent of patients with pulmonary Kaposi’s sarcoma, Kaposi’s sarcoma and infections were nearly equally responsible for pleural effusion in our series. In an HIV-infected patient with pleural effusion, clinical and radiologic data that are highly suggestive of Kaposi’s sarcoma are highly useful in the selection of the diagnostic and therapeutic approach.

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Management of Massive Subcutaneous Emphysema

To the Editor:

In the August 1992 issue of Chest, Herlan et al1 presented several cases of massive subcutaneous emphysema and advocated subclavicular incisions as the treatment of choice. Although placement of multiple incisions in the skin and subcutaneous tissue has had some advocates in the past, this form of treatment has not proved generally successful or acceptable. Fortunately, subcutaneous emphysema rarely adversely affects the patient’s physiologic functioning, is usually self-limited, and rarely requires surgical treatment.

As the authors noted, the source of air is usually from the respiratory tract; more rarely, it is from a hollow organ, such as the esophagus. The emphysema is usually caused by air being forced from a reservoir in the chest into the interstitial tissue by the cough mechanism. The effectiveness of the latter depends upon the generated pressure and its duration. Whether the air is from a leaking alveolus, the bronchial tree, the esophagus, or the pleural cavity, it is forced into the subcutaneous tissue by the force of the cough, which is preceded by a rise in pressure against the closed glottis. In severe cases, it is most important to control the cause by limiting intrathoracic pressure. Venting the interstitial air is only locally effective. However, tracheostomy and emergency intubation limit the buildup of pressure by preventing closure of the glottis. Leaving the neck wound open will aid in venting the neck and mediastinum. This was advocated by Lindskog many years ago, as