Massive pneumoperitoneum immediately following initiation of mechanical ventilation has been previously reported. The air may dissect forward to the anterior abdominal wall and/or rupture into the peritoneal cavity. On rare occasions a scrotal pneumatocele (pseudoscum) occurs, as air enters directly from the peritoneal cavity. However, our patient did not have evidence of pneumoperitoneum on radiographic studies.

Although physically deforming, the presence of a pseudoscum has no clinical consequences and requires no immediate treatment. This unusual complication of pulmonary barotrauma should be recognized by physicians who care for artificially ventilated patients.

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Tracheobronchomegaly

To the Editor:

In the September 1991 issue of Chest, Boomsma and Schraufnagel reported a case of tracheobronchomegaly (Mounier-Kuhn syndrome). In their discussion of the findings, they mentioned that other conditions, such as diffuse inflammatory tracheomalacia, relapsing polychondritis, Ehlers-Danlos syndrome, and cutis laxa, can also rarely cause diffuse tracheal widening. The authors failed to refer to a quite frequent cause of an enlarged tracheal diameter, namely, diffuse pulmonary fibrosis.

Acquired tracheomegaly as a cause of diffuse pulmonary fibrosis has been reported by Woodring et al. These authors studied chest radiographs of 34 consecutive patients with diffuse pulmonary fibrosis and measured the internal transverse diameter of the trachea 2 cm above the top of the aortic arch, considering greater than 25 mm in men and 21 mm in women as indicative of tracheomegaly. Tracheomegaly was present in ten of their patients, including four with fibrosing alveolitis, four with sarcoidosis, and two with chronic progressive histoplasmosis. In seven of these patients, serial radiographs documented that the tracheal dilatation had progressed with time.

These data and our own experiences suggest that tracheobronchomegaly can occur as a complication of diffuse lung fibrosis. Fibrotic lung diseases should therefore be mentioned as a cause of increased size of the trachea.

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2 Woodring JH, Barrett PA, Rehm SR, Nurenberg P. Acquired tracheomegaly in adults as a complication of diffuse pulmonary fibrosis. AJR 1989; 152:743-47

Cardiac Dysfunction and Pulmonary Edema following Scorpion Envenomation

To the Editor:

In a report of cardiac dysfunction and pulmonary edema following scorpion envenomation, which appeared in the October 1991 issue

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1 Boomsma JD, Schraufnagel DE. A man with a large trachea. Chest 1991; 100:909-10
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of Chest, Abroug et al. state that the normal values of systemic blood pressure and vascular resistance observed in their patients contradict the hypothesis attributing a preponderant role to catecholamine discharge. They conclude that the mechanism of "left ventricular dysfunction" remains unclear and speculate that involvement of superimposed alveolar capillary lesions in the genesis of pulmonary edema is not excluded, without pathologic or experimental evidence of the existence of such lesions.

The authors also propose "a primary scorpion myocarditis" as the possible mechanism of the abnormalities observed. We also suspected a direct myocardial effect of scorpion venom based only on ECG changes, although such direct myocardial involvement is observed only in the isolated working guinea pig heart and in isolated atrial and papillary muscles, but not in the intact animal. We recently observed enzymatic myocardial injury in a group of symptomatic children without heart failure, however, we were unable to substantiate the hypothesis of a direct myocardial effect of scorpion venom.

Furthermore, there is ample evidence in humans of the existence of catecholamine cardiomyopathy due to excessive release of catecholamines, as seen in scorpion envenomation with characteristic myocardial pathology similar to that described in phereschromocytoma. Although the authors believe that the mechanism of pulmonary edema is not clear, there is sufficient evidence of left ventricular diastolic dysfunction, and recent echocardiographic observations emphasized reversible depressed systolic function ("myocardial stunning") after envenomation.

It should be emphasized that hemodynamic monitoring is quite difficult in small children with central nervous system dysfunction with or without respiratory failure. Hypovolemia due to excessive perspiration, increased insensible loss, and vomiting due to pancreatitis may obscure the hemodynamic changes. Echocardiography is quite sensitive in assessing myocardial compromise after envenomation without subjecting the patient to invasive evaluation except in the presence of pulmonary edema and hypovolemia, as seen in our patients, in order to facilitate the regulation of afterload reduction therapy:

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REFERENCES

To the Editor:

Our own experience and published studies on scorpion envenomation do not provide the certitudes of Gueron and Sofer about the mechanisms of pulmonary edema (PE) following scorpion envenomation and the underlying cardiac dysfunction.

Although most clinical and experimental studies and even our series give strong evidence of the hemodynamic type of PE following scorpion envenomation, we find we cannot conclude that this is the exclusive mechanism. Indeed, in an experimental model of scorpion envenomation, Rossi et al. found severe structural damage of the alveolocapillary barrier, suggesting a possible role of high permeability in the pathogenesis of PE. Furthermore, Bahav and Weiss, reported on one patient suffering from scorpion envenomation-induced PE with a pulmonary capillary wedge pressure within the normal range. Moreover, in some of the patients we recently treated for scorpion envenomation-induced PE, we found bronchial aspirations with an increased albumin content (unpublished data). All of these facts led us to be cautious in the conclusions of a report of five cases. Thus, as long as there are no definitive data, high-permeability PE should be kept in mind, and further studies are needed to search for ARDS indicators in this setting.

We believe that the mechanism of scorpion envenomation-induced cardiac dysfunction is still unclear. The catecholaminergic hypothesis of Gueron et al. needs to be more carefully substantiated in the scorpion envenomation setting. We agree that there is sufficient evidence of the existence of catecholaminergic cardiomyopathy in humans, but we lack precise information about the time course and the reversibility of such cardiomyopathy, and any comparison with scorpion envenomation cardiomyopathy appears hazardous. There is also a documented catecholaminergic discharge following scorpion envenomation. But should scorpion envenomation-induced cardiomyopathy be categorized as a catecholaminergic cardiomyopathy? The histopathologic changes observed at autopsy in scorpion envenomation (interstitial edema, increased cellularity, cardiac cell necrosis) should not be considered as specific for catecholaminergic cardiomyopathy.

In our study, we reported on early hemodynamic data neither supporting nor excluding the catecholaminergic hypothesis. We do not exclude other hypotheses, especially a possible "scorpionic cardiomyopathy" due to a direct myocardial effect of scorpion venom.

We believe that there are still many dark areas in the comprehensive approach to scorpion envenomation-induced cardiopulmonary disturbances, and any conclusion should be cautious and rigorous.

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