patients leads us to believe that percutaneous catheter fragmentation and dispersion of the thrombus using conventional cardiac catheters is important in the emergency management of patients who have collapsed or are seriously compromised because of a massive pulmonary embolus. While catheter pulmonary embolectomy by experienced operators undoubtedly has saved lives, clinicians in hospitals without angiographic equipment but with access to x-ray screening facilities should still consider attempting percutaneous catheter fragmentation and dispersion in such patients.

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References

1 Timsit JR, Reynaud P, Meyer G, Sors H. Pulmonary embolectomy by catheter device in massive pulmonary embolism. Chest 1991; 100:655-58
2 Greenfield LJ. Catheter pulmonary embolectomy [editorial]. Chest 1991; 100:593-94
3 Brady AJB, Crake T, Oakley CM. Percutaneous catheter fragmentation and distal dispersion of proximal pulmonary embolus. Lancet 1991; 338:1186-89
4 Coon WW, Coller FA. Clinicopathologic correlation in thromboembolism. Surg Gynecol Obstet 1959; 109:259-69
5 Donaldson GA, Williams C, Scannell JG, Shaw RS. A reappraisal of the application of the Trendelenburg operation to massive fatal embolism. N Engl J Med 1963; 268:171-74

To the Editor:

We appreciate the interest expressed by Brady and colleagues in our article and fully agree that only an experienced physician can carry out pulmonary embolectomy using a catheter device. We read with great interest the experience at the Hammersmith Hospital with breakdown of pulmonary emboli using conventional catheters.[1]

We recently undertook this procedure in an 82-year-old woman who had suffered from a massive acute pulmonary embolism with collapse and iterative cardiac arrests. Fragmentation of the thrombi was attempted via the femoral vein with an 8F pigtail catheter mounted on a J wire. Despite significant angiographic revascularization of the left pulmonary artery, the mean pulmonary arterial pressure remained elevated (27 mm Hg before and after the procedure), and inotropic support could not be reduced until 48 h after the procedure.

Even if promising results were obtained in the three patients reported by Brady et al,[1] the present case suggests that percutaneous catheter fragmentation could have variable success in improving hemodynamic and clinical status. One possible explanation is that dispersal of a proximal thrombus into the more distal branches would not significantly increase pulmonary blood flow if the smaller vessels are initially obstructed, so that catheter fragmentation would be less effective in this situation.

In our opinion, isolated proximal emboli are probably infrequent in massive pulmonary embolism. Therefore, there is need for further experience, on a more large-scale basis, to assess the efficacy of this attractive and simple technique.

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Reference

1 Brady AJB, Crake T, Oakley CM. Percutaneous catheter fragmentation and distal dispersion of proximal pulmonary embolus. Lancet 1991; 338:1186-89

Another Complication of Barotrauma

To the Editor:

Pressure-induced injury, commonly known as barotrauma, occurs in 1 percent to 20 percent of patients receiving mechanical ventilation.[1] The presence of subcutaneous air in the neck or upper part of the thorax is pathognomonic of pulmonary barotrauma.[2] We recently cared for a patient who developed an unusual complication of barotrauma.

A 45-year-old man with a past medical history significant for traumatic C5-6 quadriplegia was admitted to the hospital with mental status changes. His initial examination was significant for clouding of the sensorium, but the findings were otherwise unchanged from those during previous hospital visits. While being examined, the patient's condition deteriorated rapidly; respiratory failure developed, necessitating assisted ventilation and prompting his admission to the intensive care unit. His chest radiograph showed bilateral diffuse opacities, and arterial blood gas analysis revealed significant hypoxemia. Blood cultures obtained on admission showed *Candida parapsilosis* infection, which was treated with intravenous amphotericin B.

Six days after admission the patient's clinical condition continued to deteriorate with worsening hypoxemia unresponsive to increasing levels of supplemental oxygen and positive end-expiratory pressure. Subsequently the patient developed a right-sided pneumothorax, which was managed by tube thoracostomy. Within hours, a left-sided pneumothorax occurred, which was treated similarly. His oxygenation status remained marginal.

On day 8 after admission it was noted that the patient's scrotal size had increased rapidly (Fig 1). The scrotum was distended and tense. However, no scrotal masses were found on palpation or transillumination. No changes in skin color were noted. A chest radiograph obtained at the same time revealed significant subcutaneous emphysema and a loculated left-sided pneumothorax. No evidence of pneumoperitoneum was found on multiple abdominal radiographs. Unfortunately, multisystem failure ensued, and the patient died on day 10.

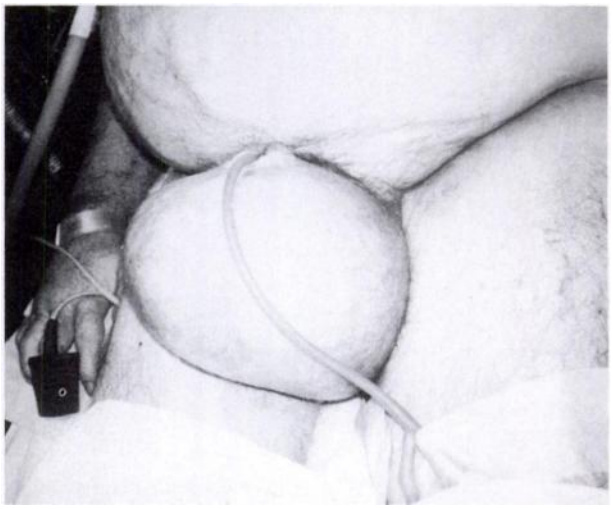

FIGURE 1. Scrotum was distended and tense, and size had increased rapidly.

Massive pneumoperitoneum immediately following initiation of mechanical ventilation has been previously reported.[3] The air may dissect forward to the anterior abdominal wall and/or rupture into the peritoneal cavity. On rare occasions a scrotal pneumatocele (pneumoscrotum) 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 pneumoscrotum 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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REFERENCES

1 Haake R, Schlichtig R, Ulstad DR, Henschen RR. Barotrauma: pathophysiology, risk factors and prevention. Chest 1987; 91:608-13
2 Steier M, Ching N, Roberts EB, Nealon TF. Pneumothorax complicating continuous ventilatory support. J Thorac Cardiovasc Surg 1974; 67:17-23
3 Beilin B, Schulman DL, Weiss AT, Mogle P. Pneumoperitoneum as the presenting sign of pulmonary barotrauma during artificial ventilation. Intensive Care Med 1986; 12:49-51

Tracheobronchomegaly

To the Editor:

In the September 1991 issue of *Chest*, Boomsma and Schraufnagel[1] reported a case of tracheobronchomegaly (Mounier-Kuhn syndrome). In the discussion of their 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.[2] 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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REFERENCES

1 Boomsma JD, Schraufnagel DE. A man with a large trachea. Chest 1991; 100:809-10
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

To the Editor:

Dr Russi has raised an important point that we did not have space to discuss in our "Roentgenogram of the Month" case report of a patient with Mounier-Kuhn syndrome.[1] The series by Woodring and colleagues[2] emphasizes that the trachea can enlarge in patients with lung fibrosis. Other acquired lung conditions may also cause increased tracheal diameter, but the Mounier-Kuhn syndrome, which is considered congenital tracheobronchomegaly, is generally associated with a much larger trachea. In the article by Woodring and colleagues[2] on acquired tracheomegaly, the average transverse diameter of the trachea was 23.9 mm (SD 6.3). In another series of patients with congenital tracheobronchomegaly also reported by Woodring and co-workers,[3] that diameter was 32.6 mm (SD 7.7). A *t* test shows that these samples are likely to be from different populations ($p<0.01$). One can further analyze their data by considering the values from women, who have smaller tracheas, separately by analysis of covariance. This test shows that the gender of the patient is not important ($p=0.23$), but whether they have acquired or congenital tracheomegaly is ($p=0.005$). Another consideration is that the series of patients with acquired tracheomegaly was from consecutive cases that included patients with normal tracheal size, whereas the series of patients with congenital tracheomegaly was not. However, inspection of the distribution curves of the two populations shows that only one of the 34 patients with lung fibrosis had a tracheal diameter larger than 30 mm, whereas only three of the ten patients with congenital tracheomegaly had tracheal diameters less than 30 mm.

Another important aspect of our case was that the chest radiograph had thin semicircular shadows. These sharp and narrow lines appear as if drawn by an accountant's pencil and represent the walls of bullae. They are distinguishable from the coarser brush strokes of interstitial fibrosis and should cause the physician to search for causes of lung disease other than interstitial fibrosis.

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REFERENCES

1 Boomsma JD, Schraufnagel DE. A man with a large trachea. Chest 1991; 100:809-10
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
3 Woodring JH, Howard RS II, Rehm SR. Congenital tracheobronchomegaly (Mounier-Kuhn syndrome): a report of 10 cases and review of the literature. J Thorac Imaging 1991; 6:1-10

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