Communications

noticed after the forceps had been used for over 50 cases, the second broke after 30 biopsies, and the third forceps jaws broke after only ten biopsies. No unusual force had been used in attempting the biopsy during these procedures. Figure 1 shows a normal alligator biopsy forceps and two showing the line of breakage (left to right). In one patient the broken piece was coughed out after the procedure. Chest x-ray (PA and lateral) films taken on separate dates in all three patients showed no metallic foreign body. The post-procedure course was uneventful in all.

Breakage of the jaws of the alligator biopsy forceps is rare and has not been reported. We have been unable to sort out the exact reasons for the possible breakage of the biopsy forceps heads. Metal fatigue during repeated sampling and suboptimal tensile strength due to fenestrations in the head of the alligator forceps are probable causes. We do not know if tropical climate affects the properties of the forceps metal.

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Reference
4 Sanders DM. Needle in a haystack. Chest 1983; 83:935-36

Noncardiac and Cardiac Pulmonary Edema
To the Editor:

Sibbald et al (Chest 1983; 84:452-61) are to be complimented for their most useful article on noncardiac (NCPE) and cardiac pulmonary edema (CPE). I would like to take exception, however, to their statement (page 454) that "the initial clinical presentation in both NCPE and CPE is uniquely similar." I do not know of any clinical-physiologic study comparing the major respiratory symptom, dyspnea, and its mechanical correlates in CPE and NCPE.

In my experience, the acute respiratory distress syndrome (ARDS) of traumatic or infectious etiology and without left ventricular failure is seldom, if ever, associated with orthopnea. The conscious patient with ARDS (NCPE) who can choose his position in bed, many times lies flat using only one pillow to support his head. I have asked 12 patients with ARDS and a pulmonary capillary wedge pressure (PCWP) less than 15 mm Hg if their dyspnea was improved by elevating the head of the bed to 25-30" or 45". At 25-30" inclination, their dyspnea was slightly improved; at 45" the additional improvement was barely perceptible. In contrast with the ARDS patient, I would still have to see a conscious patient with acute or "chronic" pulmonary edema and extensive lung infiltrates without orthopnea.*

For reasons related to the mechanics of breathing, any dyspneic patient, including the bronchitis or asthma patient, may feel more comfortable sitting up than lying down. However, it seems to me that the patient with ARDS has a higher tolerance for dorsal decubitus than the patient with acute cardiogenic pulmonary edema. One would not expect orthopnea to allow a clear-cut separation of ARDS and acute cardiogenic pulmonary edema in all patients, regardless of the degree of pulmonary whitening, PCWP, concentration of serum albumin, etc. This positional and ventilatory response to fluid overload and hemodynamic changes in CPE and NCPE needs to be carefully defined. Until then, I believe that the obvious presence of orthopnea, or alternatively its definite absence in conscious patients, may help separate CPE from NCPE.

If the ] receptors have a determinant role in the pathogenesis of tachypnea and orthopnea produced by pulmonary edema,1,2 it is intriguing that the presence of excess water in the lung interstitium and alveoli is not invariably associated with orthopnea. The patients with ARDS are tachypneic, a fact suggesting that both the interstitial sensors ( ] receptors) and the central nervous system (CNS) can react to pulmonary fluid overload. If so, would the orthopneic response of NCPE be blunted in the acutely ill or alternatively, would the (reflex?) pathways of tachypnea and orthopnea be entirely different in CPE and NCPE? For instance, the rate of fluid accumulation, the composition of this fluid, and the degree of distension of pulmonary capillaries and veins are ostensibly different in NCPE and CPE. As suspected by Braunwald,3 for entirely different reasons, and as suggested in this letter, the distension of the pulmonary capillaries and/or veins may constitute an important signal for orthopnea associated with pulmonary edema. Should such a clinical concept be validated physiologically, one could easily understand the biologic significance of this symptom and sign. In CPE, orthopnea is both a mechanically convenient position for patients with increased work of breathing, and a hemodynamically protective mechanism, sparing the upper lung zones (zones with increased venous circulation in this condition) of pulmonary edema. The ARDS patients with "mild" or no orthopnea stand to benefit hemodynamically much less from orthopnea than the patients with CPE; in ARDS the extravasation of fluid is primarily due to diffuse capillary damage, and the venous blood flow is apparently not redistributed to upper zones.

There may be other differences in clinical presentation and respiratory physiology between CPE and NCPE. For instance, paroxysmal nocturnal dyspnea is usually not seen in NCPE, although many of these patients without left ventricular impairment, especially in the early phase of their illness, are fluid overloaded in the hospital; part of the (increased) nocturnal venous return would be expected to filter into the lungs through altered capillaries and lead to acute, nocturnal orthopnea. Then, the bronchial secretions are many times foamy in CPE, but not so in NCPE, in spite of a large protein content of the bronchial secretions of the latter. (Would the

*Observations made during my affiliation with the Department of Medicine, Medical College of Ohio at Toledo.

838 Communications to the Editor
Intracavitary Aminocaproic Acid for Massive Pulmonary Hemorrhage

To the Editor:

Medical therapy for massive endobronchial hemorrhage from an intracavitary mycetoma has been very disappointing at best. Surgical therapy is limited to the minority of patients who have significant cardiopulmonary reserve and are therefore candidates for partial lung resection. This case demonstrates the successful and quick control of life-threatening pulmonary hemorrhage by intracavitary injection of aminocaproic acid.

Case Report

A 48-year-old man was admitted for treatment of pulmonary aspergilloma with hemoptysis. He had left superior sulcus carcinoma treated with resection and radiation therapy 13 years prior to admission, with a residual cavity at the left apex. Nine months prior to admission, cavitary aspergilloma was diagnosed by needle puncture. Because of symptoms (fever and hemoptysis) thoracotomy was performed, but lung resection could not be accomplished due to marked radiation fibrosis. The cavity was explored, confirming the presence of Aspergillus fumigatus, and a large 30 ml balloon Foley catheter was placed in the cavity. The patient was treated for several weeks with both intracavitary and systemic amphotericin B, and he improved temporarily. On the day of admission, he noted increasing hemoptysis plus bloody drainage from the catheter. He promptly developed acute massive intracavitary hemorrhage and hemoptysis that required intubation with a double lumen endotracheal tube to isolate his left lung. He received 13 units of packed red blood cells, 6 units of fresh frozen plasma and 14 liters of saline solution. Bronchoscopic examination ruled out bleeding from the right lung, and attempts to tamponade the bleeding with the Foley catheters’ balloon were unsuccessful. Despite transfusions and intravenous vasopressors, he remained markedly hypotensive and continued to bleed briskly from the cavity into the intracavitary catheter. Surgery was not considered possible in this patient. Five grams (20 ml) of aminocaproic acid was injected into the cavity via the intracavitary catheter and the tube was clamped for several minutes. The bleeding slowed down immediately and within a few hours it had completely stopped. The patient was stabilized, and weaned off the ventilator a few days later. Unfortunately, the patient developed nosocomial Pseudomonas pneumonia in his right lung and died from this three weeks after admission. Cultures from the left cavity continued to grow Aspergillus.

Discussion

This is the first report of the use of aminocaproic acid for control of intracavitary pulmonary hemorrhage. Commonly, patients with fungus balls have significant pulmonary dysfunction and surgical therapy for significant hemothorax involves unacceptable risks. Mild-to-moderate hemothorax can usually be managed with conservative measures. Massive hemothorax, of course, is a highly lethal condition, but frequently the underlying condition makes surgical risk extremely high. In cases of massive hemothorax from cavitary fungal infections, the high-risk patient may benefit from a trial intracavitary aminocaproic acid.

Aminocaproic acid inhibits fibrinolysis principally via inhibition of plasminogen activating substances and through its antiplasmin activity. It readily penetrates red blood and tissue cells, and is rapidly excreted in the urine mostly unmetabolized. Five or more grams by intravenous infusion are usually required to control bleeding from systemic fibrinolysis. It has been postulated that extravascular clots formed in vivo with incorporated aminocaproic acid may not undergo spontaneous lysis as do normal clots.

We cannot prove that the drug led to the cessation of bleeding, but our observation in this particular case strongly suggests aminocaproic acid played an important role in controlling the bleeding. Obviously, more information and investigation of this therapeutic modality is needed before it can be recommended, except in extreme cases such as the one reported here.

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References


Pulmonary Artery Catheterization

To the Editor:

We read with interest the article of Boyd et al (Chest 1983; 84:245-48) regarding pulmonary artery catheterization. Boyd et al should be commended for their study, as it represents one of the largest prospective investigations of complications of pulmonary artery catheterization. It is unfortunate that the true incidence of the most common serious complication of pulmonary artery catheterization, that of ventricular arrhythmia, was not determined. Only 11 percent of their patients developed premature ventricular contractions, and only 2 percent had evidence of ventricular tachycardia. This low incidence may be a serious underestimation secondary to two factors. First, more than one half of their patients were "good risk" patients undergoing preoperative catheterization. These patients might not be expected to have as high an incidence of ventricular arrhythmia as more critically ill patients. However, it