Literature Briefs

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Literature Briefs were submitted by Drs. G. Battit, M. Laver, T. Caldwell, and J. Jacoby. Briefs appearing elsewhere in this issue are part of this column.

Circulation

POSTTRANSFUSION PULMONARY EDEMA WITHOUT OVERLOAD Overload of the circulation is a frequent cause of pulmonary edema following blood transfusion. Two cases are described; overload was definitely ruled out in one, and unlikely in the other. Additional reported cases are cited. Characteristically the patient is anemic or hypovolemic, has received one to two units of blood, and develops chills, fever, cough, tachypnea, frothy sputum, cyanosis, and sometimes urticaria. The chest x-ray reveals patchy infiltrates not perihilar in distribution, generally considered to be indicative of interstitial edema. Possible sources of this reaction include: a hypersensitivity response analogous to urticaria but appearing in the pulmonary tissue; intravascular coagulation, a mechanism reminiscent of the postperfusion lung; pulmonary vascular vasoconstriction attributed to products of hemolysis or microemboli. Treatment is empirical and includes, according to the authors, support with a volume-controlled respirator, oxygen, dextran 40, and steroids. Heparin and isoproterenol may also be of value. (*Byrne, J. P., and Dixon, J. A.: Pulmonary Edema Following Blood Transfusion Reaction, Arch. Surg. 102: 91–94, 1971.*) EDITOR'S COMMENT: Although respiratory failure secondary to these factors remains a possibility, further case reports without the benefit of hemodynamic assessment of the pulmonary circulation and sophisticated analysis of pulmonary blood flow distribution will not resolve the dilemma.

Respiration

PULMONARY CIRCULATION IN ALPHA$_1$ ANTITRYPSIN DEFICIENCY The status of the pulmonary vasculature was assessed in five patients with pulmonary emphysema secondary to alpha$_1$ antitrypsin deficiency. Studies included pulmonary wedge arteriograms (radioopaque dye injected with the pulmonary arterial catheter in the wedge position), pulmonary arteriograms, right-heart hemodynamics, and pulmonary scintiscans. All patients were severely disabled by dyspnea on exertion or at rest, and all had severe airway obstruction and hypoexmia (P_{O_2} range: 39 to 51 mm Hg). The hemodynamic studies were carried out with the patients breathing ambient air, breathing O_2 by nasal cannula, and after 30 minutes of a constant intravenous infusion of aminophylline. The pulmonary wedge arteriograms revealed a diminution in the number of small vessels (diameters 0.2 to 1.0 mm) in the lower lung zones compared with normal. Middle and upper lung zones showed fewer abnormalities. The lower zones were underperfused as demonstrated by a slowness of arterial filling, a diminished capillary phase on the arteriogram, and diminished radioactivity on scintiscans. Although cardiac index was normal at rest, both mean pulmonary arterial pressure (PAP) and pulmonary vascular resistance (PVR) were markedly elevated. Nasal O_2 and aminophylline given intravenously reduced PAP and PVR. These findings suggest both structural and functional changes as the source of pulmonary hypertension in patients with alpha$_1$ antitrypsin deficiency. Involvement of the lower lung fields in this condition differs from that in chronic obstructive lung disease, in which alterations in pulmonary perfusion are patchy and often unilateral, with