Pulmonary Edema—Treatment

By Max Harry Weil, M.D.

Dr. Weil is Director and Clinical Professor of Medicine and Biomedical Engineering, University of Southern California Center for the Critically Ill, Los Angeles.

Pulmonary edema may follow shock, respiratory distress, lung injuries and infections, and a wide range of heart disorders. Although a common condition, it is not completely understood and should not be regarded as simply a result of pressure backed up from the left ventricle. Recent treatment of a specific group of patients shows that plasma colloid osmotic pressure and its relationship to pulmonary capillary pressure is intimately involved in the development of pulmonary edema. The difference between the two pressures, determined by measuring each, may indicate a leakage of plasma fluid into the pulmonary interstitium. Following massive fluid loss, replenishing of plasma volume with albumin-containing fluids may restore colloid osmotic pressure to normal and thereby reduce the risk of pulmonary edema.

Pulmonary edema may seem simple enough to diagnose and perhaps equally simple to treat, but, in fact, it presents a dire threat to the patient and a considerable challenge to the clinician.

Multiple factors contribute to its cause, and they bear directly on the choice of therapy. Signs and symptoms don't always mean what they seem to mean, and can lead the physician astray. Mortality is high—a survival rate of less than 50 per cent for patients who have pulmonary edema after active myocardial infarction. To successfully meet this combination of problems, the workup must be meticulous and management extremely selective.

Pulmonary edema often develops in the seriously ill patient, particularly after acute myocardial infarction. It can appear, too, after any type of left ventricular failure, aortic stenosis, myocardiopathy, mitral insufficiency, and congenital heart diseases. It may be related to lung injury (e.g., from aspiration), shock, or adult respiratory distress syndrome; or it may be superimposed on pulmonary infections. The so-called hydrostatic pulmonary edema is caused by a rise in the left ventricular filling pressure, which in turn results from a decrease in the ability of the left ventricle to eject a sufficient volume of blood. The pressure is retroconducted to the left atrium and then to the pulmonary veins, thereby increasing capillary hydrostatic pressure.

Measure the Pressures

In some patients, however, the problem stems from a reduction in the plasma colloids and therefore a lowering of colloid osmotic pressure. Since it is the connection between colloid osmotic pressure and hydrostatic pressure which has recently been related to the appearance of pulmonary edema, colloid osmotic pressure should be measured. At the same time wedge pulmonary artery pressure or pulmonary diastolic pressure should be measured as an indication of hydrostatic forces. In many cases, both decreased colloid osmotic pressure and increased hydrostatic pulmonary capillary pressure are involved.

The chief sign of pulmonary edema may be the early appearance of moist rales or evidence of pulmonary interstitial and alveolar fluid accumulation on the chest radiograph. To this extent the chest x-ray is a very useful qualitative guide. Quantitative determination of conditions in which pulmonary edema develops, however, is best gauged by hemodynamic measurements — of left ventricular filling pressure and colloid osmotic pressure. The left ventricular pressure can be recorded in a hospital, by putting a catheter directly into the left ventricle retrograde, from the axillary artery or from the femoral artery. The colloid osmotic pressure can be mea-