Depends on Cause

sured on a sample of plasma by a commercially available oncometer. Physical signs include distention of the neck veins—which suggests an increase in right ventricular filling pressure — and alveolar edema, indicated by rales and generally confirmed by auscultation of the lungs. The clinician must, however, regard these signs with some caution, for several reasons. An increase in central venous pressure is contingent on right-sided failure, which is usually delayed following acute myocardial infarction. This is the very reason why the Swan-Ganz catheter—the flotation pulmonary artery catheter by which wedge pressure is measured — has been a boon.

Failure Without the Signs

The clinician should remember that right heart failure, although very common following left heart failure, follows only after substantial lag. Therefore, central venous pressure and neck veins may sometimes be normal during the earlier stages of left heart failure. Thus, pulmonary edema may develop as a result of left heart failure without the patient having signs of left heart failure.

Secondly, pulmonary edema in its earlier stages is interstitial edema, which doesn't involve the alveoli of the lungs. Consequently, the assumption that rales, heard in patients after onset of alveolar edema, would be reliable as a clear indication has to be rejected. The clinician must use more sophisticated measurement.

Now it may be that we have to accept the lesser efficiency of clinical signs and clinical measurements, under circumstances in which cost and volume of patients present obstacles to the more sophisticated techniques. But it would be really quite wrong to even remotely imply that this is sufficient, if the physician wants to do the optimal job.

For the management of these patients, the most effective drugs we have are a loop diuretic, furosemide, and ethacrynic acid. Occasionally, restoring of fluid volume is indicated. It is important, under those circumstances, to be aware of the colloid osmotic pressure in order not to dilute the volume with noncolloid-containing fluids.

Very often, volume buildup is necessary for a patient who is in cardiogenic shock, but that volume probably should not be entirely made up of noncolloid-containing fluids, but rather of albumin-containing fluids.

Pulmonary edema is the end result of multiple factors. In earlier years, it was thought that the condition was caused by failure of the left ventricle, and the hydrostatic forces, which raise the pressure in the pulmonary capillaries, resulting in leakage of fluid. We now know that the colloid contained in the plasma is also an important variable, and may, in fact, greatly modify the extent to which fluid would or would not leak out in the presence or absence of left ventricular failure.

The physician should keep in mind that in some patients following acute myocardial infarction, the colloid osmotic pressure is reduced and the left ventricular filling pressure may in fact be not very high. Under these conditions, the decrease in colloid osmotic pressure may be the primary cause of pulmonary edema. In these patients, not only the hydrostatic pressure but also the colloid osmotic pressure must be considered in efforts to reverse the edema. □

Practice Procedures

Pulmonary Edema—
Osmotic Pressure—Hydrostatic Pressure

Chief Signs: (Must be confirmed by more sophisticated measures)

- Moist rales.
- Pulmonary overload evident on chest x-ray.
- Distention of neck veins.

Measure:

- Colloid osmotic pressure.
- Pulmonary artery wedge pressure.

Management:

- If colloid osmotic pressure is critically reduced in a volume-depleted patient, consider fluid volume buildup with 5 per cent human serum albumin. Be cautious in use of noncolloid-containing fluids. Loop diuretics increase colloid osmotic pressure.