Correspondence

Letter by Hanna and Smart Regarding Article, “Clinical Diagnosis of Pulmonary Hypertension”

To the Editor:

We read with great interest the article by Rich and Rich1 regarding the diagnosis of pulmonary hypertension. We particularly appreciate the fact that the authors alluded to pulmonary veno-occlusive disease (PVOD), a complex and frequently overlooked entity.

We would like to comment on the hemodynamic assessment of PVOD and the hemodynamic tracings of Figure 4. The authors state that, in PVOD, the pulmonary capillary wedge pressure (PCWP) is elevated in some pulmonary segments, exceeding the left atrial pressure and the left ventricular end-diastolic pressure. However, we argue that it is important to distinguish PCWP from pulmonary capillary pressure in this instance, as we described previously.2 In fact, pulmonary capillary pressure is elevated in PVOD, exceeding the left atrial pressure, but PCWP is normal.2–4 Wedging the pulmonary arterial catheter leads, in most cases, to a static column of blood between the catheter tip and an equally large pulmonary vein, even when the pulmonary venules are obstructed.2–4 Therefore, PCWP corresponds to the pressure in the large pulmonary veins that are not affected by PVOD, and consequently, to the left atrial pressure rather than pulmonary capillary pressure, which explains why PCWP is often normal in PVOD. PCWP may be damped and featureless with attenuated A and V waves, because the left atrial pressure gets retrogradely transmitted through the obstructed vasculature, but still correlates with the left atrial pressure rather than pulmonary capillary pressure and is usually normal.2,4 This damping of PCWP waveform explains why it is difficult to ascertain whether the tracing obtained is a true PCWP2,3; the aspiration of blood from the distal catheter and the demonstration of an arterial oxygen saturation is often necessary to prove successful wedging. We agree that the pulmonary arterial catheter should be wedged in multiple locations to avoid spurious tracings and obtain the best possible waveform.

In Figure 4, the presumed PCWP is larger than the left ventricular end-diastolic pressure. However, this presumed PCWP tracing is damped, with no clearly defined A and V waves. Thus, there are 2 possible explanations for this apparent gradient between PCWP and left ventricular diastolic pressure: (1) the tracing may not represent a true PCWP tracing (eg, catheter overwedging or catheter wedging in lung zone 1); and (2) the tracing may represent a true but damped PCWP tracing, with an attenuated V wave and attenuated Y and X downslopes, which falsely creates the impression of a gradient between PCWP and left ventricular diastolic pressure.

Unfortunately, given those limitations, PVOD is an entity that is difficult to distinguish from pulmonary arterial hypertension by cardiac catheterization. The diagnosis of PVOD is suggested by the triple combination of severe pulmonary hypertension, normal PCWP, and radiographic pulmonary edema. In addition, the lack of response to vasodilator therapy (or the deterioration with this therapy) should suggest PVOD.3,5

Disclosures

None.

Elias B. Hanna, MD
Frank W. Smart, MD
Department of Medicine
Cardiovascular Section
Louisiana State University
New Orleans, LA

References

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Elias B. Hanna and Frank W. Smart

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