Reply: Will the Real Pulmonary Pressure Please Stand Up?

From the Authors:

The authors Soto and Mehmood described several important factors that could explain the variability in the hemodynamic measurements noted in our study. We thank them for their interest in our work and we now provide additional insights into our study methodology.

1. Sedation: as per our standard right heart catheterization (RHC) protocol, intravenous sedation or analgesia is hardly ever used for the procedure. All patients in our study received local anesthesia using 5 ml of 1% lidocaine (1), which is unlikely to have affected our hemodynamic measurements.
2. Overdamping: all precautions were taken to avoid this phenomenon that particularly affects the systolic and diastolic pressures but less so the mean pulmonary pressure. All pressure lines were flushed before measurements, paying careful attention to ensure the system was free of bubbles, blood residues, or kinks. A square wave (fast flush) test was routinely done to assure an adequate dynamic response of the hemodynamic system and therefore detect under- or overdamping. Pressures were obtained from waveform tracings, showing the characteristic waveform patterns, including the presence of dicrotic notch on the pulmonary artery waveform.
3. Fluid administration: hemodynamic determinations were obtained prior to any additional fluid or exercise challenge (2). Nevertheless, we did measure cardiac output by thermodilution between the first and second set of hemodynamic determinations. For thermodilution, we injected a total of 30–50 ml of dextrose 5% in water, as we averaged three cardiac output estimates that had $<$15% variation (3). In addition, we flushed the system one or two times (2–4 ml) between the two sets of measurements. The small amount of fluid injected over several minutes is insufficient to explain the degree of pressure variation noted in our study. In fact, D'Alto and colleagues showed that the rapid administration of 7 ml/kg of fluid (average $\pm$ SD of 478 $\pm$ 87 ml) led to an increase in mean pulmonary artery pressure and pulmonary artery wedge pressure (PAWP) of 6 $\pm$ 2 and 3 $\pm$ 1 mm Hg in patients with pulmonary arterial hypertension and 6 $\pm$ 3 and 7 $\pm$ 2 mm Hg in patients with postcapillary pulmonary hypertension (4). Assuming a linear relationship to facilitate calculations, 30–50 ml of fluid would produce changes in mean pulmonary artery pressure and PAWP of $<$1 mm Hg.
4. PAWP determination: We routinely seek to obtain blood from the distal port of the pulmonary artery catheter while in the wedge position. A blood oxygenation $>$90% (5) supports the PAWP determination (6). When we questioned the validity of the PAWP measurement, we reduced the amount of air in the pulmonary artery balloon or advanced the pulmonary artery catheter to a different branch or the contralateral pulmonary artery (5). Rarely, wedge angiography using 5 ml of iohexol (Omnipaque 350; GE Healthcare, Inc.) was performed to confirm the validity of the measurement (5). After all these interventions, no patient in the present study required a determination of left ventricular end-diastolic pressure to confirm the PAWP measurement. Wedge blood was not routinely obtained during the second PAWP determination; unless the hemodynamic measurement was markedly different from the first one or at least one of the other four remaining criteria (PAWP is less than the diastolic PAP, tracing is compatible with the atrial pressure waveform, fluoroscopic image demonstrates a stationary catheter after inflation, and free flow is present within the catheter) (5) for an adequate PAWP, determination was not met.

Soto and Mehmood accentuate many contributing factors that, if not considered, could potentially influence the accuracy and repeatability of pressure measurements during RHC. These key points emphasize the importance of having an experienced team for obtaining and interpreting the RHC results. Hence, it is especially important to be critical of the results of any RHC, particularly when measurements are discordant with the clinical presentation, ancillary studies, or prior hemodynamic determinations.

Author disclosures are available with the text of this letter at www.atsjournals.org.

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