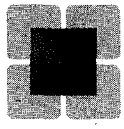

Arterial pressure monitoring: what are we really measuring?

NOWHERE in clinical medicine is accurate assessment of systemic blood pressure as important as in patients undergoing surgical procedures requiring cardiopulmonary bypass (CPB). Despite general agreement that blood pressure by itself is a poor indicator of overall cardiovascular performance, its measurement in the peri-CPB period guides both anesthetic and surgical management.[1,2]

Errors in invasive blood pressure measurement are derived from three sources: inaccurate calibration of the monitoring system, distortion of the physiologic signal by the pressure tubing-transducer system, and modification of the pressure waveform as it traverses the arterial vasculature.[3,4]

■ *See Bazaral et al (pp 448–457)*

With this as background, it is relatively easy to see the importance of the work by Bazaral et al[5] at The Cleveland Clinic Foundation. Applying an elegant experimental design, they have demonstrated the contribution of the hydraulics, as well as the site of monitoring (central aorta *v* radial artery), to waveform distortion. Using the subclavian arterial waveform values as the standard, they compared the "true" radial artery pressure and the radial artery pressure obtained from the standard (long) length of pressure tubing employed in clinical practice. An important part of the experimental design was bench testing of the equipment before clinical use. Although many readers may skip this section of the methodology, it is important for the clinician. In vitro laboratory testing has demonstrated that commonly used vascular monitoring systems can cause significant distortion of a given signal. The CCF team demonstrated that a similar degree of distortion also occurs in vivo. However, the site of monitoring (radial *v* subclavian) appeared to be the more significant determinant of arterial waveform distortion.

Unfortunately, for logistical reasons, their protocol could not allow assessment of the effects of tubing length (transducer-tubing system) on pressure waveform distortion following CPB. However, their paper, as well as a number of other recent reports, emphasized that radial arterial pressure is *lower* than central aortic pressure following CPB.[6,7] The hypothesis is that forearm vasodilation following CPB creates a "forearm steal" or a physiologic arteriovenous fistula. This inversion of the radial arterial to central aortic gradient persists for approximately 60 minutes after CPB. By increasing systemic vascular resistance, radial artery pressure was again 8–10 mmHg higher than central aortic pressure.

The data of Bazaral et al[5] suggest that mean arterial pressure (MAP) measurements should be used, since they appear to be least affected by waveform distortion. However, they and other investigators[6–8] have shown that even this parameter (mean radial artery pressure) may also be subject to significant error. For example, in the study by Mohr et al,[6] the gradient between the mean femoral and mean radial artery pressure following CPB was greater than 10 mmHg in eight of 48 patients. Increase in the systemic vascular resistance abolished the gradient.

How does all this information help clinicians manage patients better? First, as emphasized by the CCF group, meticulous attention must be paid to the transducer-tubing system to minimize distortion by using monitoring systems with high resonant frequencies and normal damping coefficients. Second, if the radial artery is used for intra-arterial monitoring, the clinician must be on guard for waveform distortions that could cause initiation of unnecessary therapy. Perhaps other anatomic sites for arterial catheterization should be routinely used, including the femoral or brachial arteries. Previous studies in a large number

of patients have documented that these sites are not associated with increased morbidity.[8] Finally, for those physicians who use radial artery catheterization, any evidence of waveform distortion or spuriously low radial arterial pressure readings mandates central aortic pressure measurement to confirm the hemodynamic abnormality.

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