Volume 67, No. 3
September 1987

THE JOURNAL OF Anesthesiology
THE AMERICAN SOCIETY OF ANESTHESIOLOGISTS, INC.

EDITORIAL VIEWS

Anesthesiology
67:289-291, 1987

Measuring Central Vascular Pressures: A Surprisingly Complex Problem

CENTRAL VASCULAR PRESSURES, *e.g.*, central venous or right atrial (RAP), pulmonary artery (PAP), pulmonary capillary wedge (PCWP), and left atrial (LAP) pressure, are frequently measured in patients undergoing anesthesia, cardiac catheterization, and intensive care. However, there are clinically important differences in the way these pressures are measured in the presence of the variations that occur during the cardiac cycle and with respiration.

Regardless of the ventilatory mode, *e.g.*, spontaneous, controlled, or intermittent mandatory ventilation, anesthetists usually measure central vascular pressures between the end of exhalation and the beginning of inspiration (termed, for simplicity, end-expiration), whereas many cardiologists and internists average central pressures over respiratory cycles. Those who use the end-expiratory period disagree about whether atrial and wedge pressures should be determined at specific times during the cardiac cycle or averaged throughout the end-expiratory period.

These discrepancies in measurement practices exist for several reasons. First, the potential errors in interpreting central pressures averaged during respiration are not appreciated. Second, no commercially available pressure monitor can automatically determine end-expiratory pressures reliably without utilizing additional measurements to detect end-expiration.[1] Third, the reasons for measuring central vascular pressures often are not clearly defined.

Although measured central vascular pressures increase during a positive pressure breath, end-diastolic volumes (EDV) usually decrease. Conversely, during a spontaneous breath, these pressures decrease, while EDVs increase. Consequently, inferences about EDVs made from the RAP, PCWP, or LAP measured during respiration can be misleading. EDV depends upon the transmural pressure, which is the difference between the pressures in the ventricular cavity and the pericardium. However, the pressure usually measured is the difference between ventricular cavitary and atmospheric pressures, which equals the transmural pressure plus the intrathoracic pressure (more correctly, plus the pericardial pressure for the RAP, PCWP, and LAP). Therefore, the RAP, PCWP, and LAP measured during a respiratory cycle are poor indicators of the transmural EDP, and, thus, the EDV. In contrast, measured and transmural pressures are almost identical at end-expiration in the absence of positive end-expiratory pressure (PEEP) because intrathoracic and atmospheric pressures are nearly equal.

The RAP, PCWP, and LAP measured during a respiratory cycle also are imprecise, because their relation to transmural pressure varies with changes in ventilator settings, respiratory patterns, and thoracic and lung compliance, as well as continuously during a respiratory cycle. Averaging these pressures over respiratory cycles helps solve these problems only if these respiratory parameters remain constant. Otherwise, averages can be a source of important clinical misinterpretation. For example, if a patient hyperventilates, mean central vascular pressures will decrease because of lower mean intrathoracic pressures, whereas transmural pressures and, thus, EDVs will tend to increase. End-expiratory values of the PAP also should be used in estimating right ventricular load and pulmonary vascular resistance or if the PA diastolic pressure is used to estimate the PCWP.

Given that central vascular pressures should be measured at end-expiration, should the RAP, PCWP, and LAP be averaged over the cardiac cycle or measured at

Accepted for publication April 27, 1987.

Address reprint requests to Dr. Teplick: Department of Anesthesiology, Massachusetts General Hospital, Boston, Massachusetts 02114.

Key words: Blood pressure: left atrial; pulmonary artery; right atrial. Equipment: computer. Measurement techniques: computerized monitoring.

specific times during the cycle? The answer depends on the purpose of the measurement. In estimating EDV or changes in EDV, the time during the cardiac cycle when the measurement is made is important, because atrial pressure approximates the EDP only during that brief portion of the cardiac cycle following atrial contraction preceding the onset of ventricular systole. Thus, because the magnitude of the "A," "C," or "V" waves may vary without any change in the EDP, averaging these pressures over the cardiac cycle can be misleading and imprecise.

In contrast, if central vascular pressures are measured as indicators of the systemic or pulmonary capillary pressures that affect fluid transdation, it may be more appropriate to average these pressures over the cardiac cycle than to measure them at the onset of systole, because high mean pressure, due to, for instance, large "V" waves, may cause transudation, even if the EDP is not markedly elevated.

Even if central vascular pressures are measured during end-expiration and at the onset of systole, they still may differ from transmural pressures, because intrathoracic and atmospheric pressures may not be equal. This issue arises commonly for patients receiving PEEP, because PEEP increases intrathoracic pressure. Fortunately, a constant error in estimating transmural pressure is relatively unimportant in assessing EDV. For patients receiving PEEP, it suffices to recognize that transmural pressures are lower than the measured pressures by an amount that depends on the properties of the lung and thoracic cage. Thus, a patient receiving PEEP who has high measured central vascular pressures and a low stroke volume may actually be hypovolemic, because the PEEP has increased the intrathoracic and pericardial pressures so that transmural EDPs actually are low. This may be tested clinically by judiciously administering volume.

Although little effort has been devoted to developing monitors that can automatically measure central pressure at specific times during the cardiac cycle, there have been sporadic attempts to recognize automatically the end-expiratory periods of pressure waveforms. This is relatively straightforward when patients are either fully ventilated or breathing entirely spontaneously, because the end-expiratory pressures are usually the lowest or highest values, respectively. However, end-expiration cannot be detected in this manner when patients both breathe spontaneously and receive mechanical ventilation (*e.g.*, intermittent mandatory ventilation), because end-expiratory pressures are neither the highest nor the lowest values. Furthermore, even accurate determination of the end-expiratory period by placing a hand on the patient's chest is not helpful in reading end-expiratory pressures from a digital display, because the displayed pressures are derived using various averaging techniques, and such averages may include pressures determined during respiratory cycles.[2]

Virtually all current monitors attempt to display end-expiratory central pressures by using some variant of an algorithm developed by Ellis.[3] This method is based on the assumption that the pressure at end-expiration will be constant for longer time periods than any pressure measured during respiratory cycles. Therefore, the most stable or commonly occurring pressure is sought and displayed as the end-expiratory value. This method becomes inaccurate or fails completely under some conditions, such as high respiratory rates.[3] However, the monitor gives no indication of such failures. In a prospective test, central pressures determined using this algorithm varied around the true end-expiratory values as much as, and were no more accurate than, those obtained using simple averages.[4] Thus, although this method does work in some circumstances, it fails unpredictably. Unless a calibrated analog display is used, these failures cannot be detected. In addition, monitors made by different manufacturers will display disparate pressures for the same waveform because of differences in the implementation of Ellis' algorithm.

The method described in the article by Mitchell *et al.*[5] in this issue of ANESTHESIOLOGY is important, because the authors have shown, rigorously and prospectively, that it can remove respiratory variation from the PAP waveform to yield both end-expiratory pressures and a display of the waveform with respiratory variations removed. Their technique relies on the fact that, usually, fluctuations in pressure due to respiration occur much more slowly than the heart rate. Therefore, by filtering, a PAP waveform can be separated into a low-frequency component consisting of the end-expiratory mean pressure with superimposed respiratory variations, and a high-frequency component that is primarily the PAP waveform, with the mean pressure and respiratory variations subtracted out.

The authors, like Ellis, assume that the end-expiratory mean pressure is the most commonly occurring pressure in the low frequency component of the filtered waveform. In effect, this pressure is continuously calculated from this low frequency component, and the mean of the high frequency phasic PAP waveform is continuously added to this value to yield the correct absolute pressures. This method is easier to understand than Ellis', and its behavior under different conditions is easier to predict. As indicated by the authors, their method also will fail at high respiratory rates. However, it is less sensitive to arrhytmias and more accurate and precise than Ellis' method.

As yet, the authors have not provided systematic evidence that their method performs as well with RAP,

PCWP, or LAP waveforms. Although they assert that "cardiac features" (presumably the "A," "C," and "V" waves) of these waveforms are preserved, this is not evident in figures 2 or 3 of the paper, or clearly substantiated elsewhere. Because these features may contain both high- and low-frequency components, it is not obvious that they would be preserved in the high-frequency component and eliminated in the low-frequency component of the filtered waveform. Consequently, they may be distorted or obliterated. The authors contend that mean values should be used because, "in conditions which result in large "A" or "V" waves, the mean relates well to effective ventricular preload and cardiac performance." As discussed above, this is incorrect, and detracts from their assertion that these features are well-preserved. If these features are lost, and only a mean RAP, PCWP, or LAP can be obtained, the utility of this method for tracking changes in EDVs is limited whenever the magnitudes of the "A," "C," or "V" waves are changing. However, this situation can be detected by observing the unfiltered waveform.

Even if this method turns out not to yield all of the desired information from the RAP, PCWP, or LAP, it is a major advance in monitoring central vascular pressures, because it eliminates respiratory-induced pressure variations in an understandable manner. Therefore, its limitations are predictable, and failures can be detected by comparing the displayed waveforms before and after being processed by this algorithm, a comparison that is not possible with Ellis' method. This paves the way for a uniform, physiologically sensible, and meaningful method to measure central vascular pressures.

Before this method becomes widely adopted, additional data must be obtained to confirm the reported performance with the RAP, PCWP, and LAP. Meanwhile, clinicians should be aware that the central pressures displayed digitally on their monitors may have an unpredictable and variable relation to end-expiratory pressures, and that spurious changes in central pressures may occur because of both differences in equipment and in the way the pressures are measured (for example, when a patient is moved from a catheterization laboratory to the operating room).

RICHARD S. TEPLICK, M.D.
Massachusetts General Hospital
Boston, MA 02114

References

1. Oden R, Mitchell MM, Benumof JL: Detection of end-exhalation period by airway thermistor: An approach to automated pulmonary artery pressure measurement. ANESTHESIOLOGY 58:467–471, 1983
2. Gengiz M, Crapo RO, Gardner RM: The effect of ventilation on the accuracy of pulmonary artery and wedge pressure measurements. Crit Care Med 11:502–507, 1983
3. Ellis DM: Interpretation of beat-to-beat blood pressure values in the presence of ventilatory changes. J Clin Monit 1:65–70, 1985
4. Fish DJ, Teplick R: Clinical value of variable weight filter algorithm equipped monitors for invasive pressure measurement. (Abstract) ANESTHESIOLOGY 65:A156, 1986
5. Mitchell MM, Meathe EA, Jones BR, Donch TE, Ricks WG, Benumof JL, Saidman LJ. Accurate, automated, continuously displayed pulmonary artery pressure measurement. ANESTHESIOLOGY 67:294–300, 1987.

Anesthesiology
67:291–293, 1987

Do Premature Infants Require Anesthesia for Surgery?

THE PAST DECADE HAS SEEN a tremendous increase in the survival of premature infants, particularly those weighing less than 1000 grams at birth. Many of these patients require surgery to ligate a patent ductus arteriosus; to treat necrotizing enterocolitis, or to insert ventriculo-peritoneal shunts, chest tubes, and feeding gastrostomies. They frequently have concomitant respiratory and cardiac failure, sepsis, and fluid and electrolyte problems. When surgery is required the anesthesiologist is presented with a very ill infant who, more than likely, has undergone fluid restriction and been treated with potent diuretics to reduce lung water and improve oxygenation and ventilation.

In the early 1970s, our knowledge of how to safely anesthetize these patients was rudimentary at best, and the monitoring equipment available was inadequate. There were no oscillotonometers, oximeters, infusion pumps, or even EKG monitors that consistently worked. Since few anesthesiologists had any experience anesthe-

Accepted for publication May 22, 1987.

Address reprint requests to Dr. Berry: Department of Anesthesiology, University of Virginia Medical Center, Box 238, Charlottesville, Virginia 22908.

Key words: Pediatrics: premature. Potency, anesthetic: age factors; MAC.