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The effect of ventilation on the accuracy of pulmonary artery and wedge pressure measurements

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A comparison was made of automated versus manual measurement of pulmonary artery (PA) and wedge (WP) pressures. The manual pressure measurements were taken at end-expiration whereas the automated measurements were taken using existing monitor and computer algorithms. A total of 40 critical care patients were divided into groups according to the ventilatory mode used (spontaneous, intermittent mandatory ventilation [IMV], or assist/control). In patients who were breathing spontaneously, the automated method underestimated mean PA pressure (MPAP) ($p < 0.01$), WP ($p < 0.001$), and PA diastolic ($p < 0.001$) pressure but not PA systolic pressure. In patients on IMV, the automated method underestimated MPAP ($p < 0.05$), WP ($p < 0.001$), and PA diastolic ($p < 0.001$) pressure and overestimated PA systolic pressure ($p < 0.05$). In patients on assist/control, the automated method overestimated WP ($p < 0.001$) and PA systolic ($p < 0.005$) pressure, underestimated PA diastolic ($p < 0.001$) pressure and did not affect MPAP. The error was not affected by respiratory rate, thoracic compliance, or level of PEEP.

The errors in automated pressure measurements believed to be clinically important varied with the ventilatory mode used. Patients breathing spontaneously had the largest measurement error, with 42% of these patients having a clinically important error in WP and 99% having a clinically important error in PA diastolic pressure. Patients on assist/control had the fewest errors in automated pressure measurements. In all ventilatory modes used, automated measurement of PA diastolic pressure had the largest amount of error.

Since the advent of the PA balloon flotation catheter in the early 1970s,[1] PA and WP have been used in the evaluation and treatment of critically ill patients. The adequacy of intravascular fluid volume, ventricular function, and pulmonary and systemic vascular resistance can be assessed by measuring these 2 pressures together with cardiac output.[2-7] These pressure measurements must be consistent and reliable because inaccuracies could lead to serious errors in subsequent treatment of the patient. One potential source of error in the measurement of pulmonary pressures results from the change in intrathoracic pressure during ventilation.

Transmural pressure determines ventricular inflow and stroke volume. In the majority of blood vessels, extravascular pressure is atmospheric; therefore, when the measurement is referenced to atmospheric pressure, the recorded intravascular pressure equals transmural pressure. If the extravascular pressure becomes positive, transmural pressure is reduced by the amount of extravascular pressure. Negative extravascular pressure would increase transmural pressure and tend to dilate the vessel and increase flow. In both situations, transmural pressure cannot be measured correctly by a pressure transducer that is referenced to atmospheric pressure.

Normally, intrathoracic pressure is slightly negative but very close to atmospheric pressure at the end of a passive expiration. During spontaneous breathing, inspiration lowers intravascular pressure in the chest, but transmural pressure remains the same or increases slightly.[8-13] PA pressure and WP measured relative to atmospheric pressure increase after a positive-pressure breath, whereas transmural pressures remain unchanged.[10] PEEP also causes intravascular pressure to rise relative to atmospheric pressure, but it has no effect on transmural pressure.[14-17]

Monitoring equipment measures pressure by the use of transducers which are referenced to atmospheric pressure. The pressure measurements displayed by monitors will reflect, therefore, changes in intrathoracic pressure. Systolic and diastolic pressures are averages of the peak and lowest pressures measured over several seconds. Mean pressure is the average of all pressures measured by the transducer for the same time interval. Fluctuations due to changes in intrathoracic pressure are averaged into these pressure measurements.

One method of minimizing the effects of variation in intrathoracic pressure is to measure PA pressures and WP at end-expiration when intrathoracic pressure is closest to atmospheric pressure. These measurements can be made manually from strip chart recorders. In this way, the expiratory phase can be identified on the tracing and pressures can be measured at that point.

The purpose of this study is to determine if PA pressure and WP measurements by automated means differed from the manually measured pressures, made at end-expiration for a variety of ventilatory modes.

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MATERIALS AND METHODS

Clinical Series

Data were obtained from 40 consecutive patients who had a functioning PA catheter. Bentley M800 (Bentley Laboratories Inc., Irvine, CA 92705) and Statham P23ID (Gould Inc., Medical Products Division, Oxnard, CA 93030) pressure transducers were used for all pressure measurements. All transducers had a fixed sensitivity which was calibrated and checked for accuracy (±1%) between use on subsequent patients.

Protocol

A fixed-sensitivity 2-channel recorder (Gould Inc., Instrument Systems Division, Cleveland, OH 44114) was used for pressure tracings. A zero reference point was recorded with every pressure tracing. Computerized measurements of PA pressure and WP were made by sampling the waveforms and the ECG at 200 times/sec with a carefully calibrated 10-bit analog-to-digital converter (Resolution 0.3 mm Hg) for a 10-sec interval. The ECG signal was used as a trigger, and a "composite" waveform for the 10-sec interval was derived.[19] Then the systolic, diastolic, and mean pressures were determined from the composite waveform. No special respiratory compensation was incorporated in the computer algorithm at that time. In some patients, PA and WP also were measured by Tektronix 414 monitor (Tektronix Inc., Beaverton, OR 97077). This device identifies the peak and valley pressures and determines systolic and diastolic pressures by averaging them, weighing the more recent pressures more heavily than those occurring at an earlier time. The pressure waveforms are filtered at a much lower frequency in order to derive the mean pressure. Most manufacturers utilize this same technique in their pressure monitoring systems (Table 1).

Before each measurement, patients were placed in a supine position, and the transducer was placed near the midaxillary line. A stopcock connected to the transducer was then opened to the atmosphere at the midaxillary line, and the strip recorder and automated monitoring devices were zeroed. Dynamic response of the monitoring system was then checked using a Sorenson Intraflo to produce a step-function change as described by Gardner.[20]

TABLE 1. Filter characteristics for mean pressure measurement used in bedside monitors

Manufacturer	Bandwidth Hz[a]	Number of poles in filter[b]
1 LDS Hospital	0.08	1
2 Hewlett-Packard	0.16	2
3 Tektronix/Vitatek	0.03	4
4 Mennen	0.12	2
5 Spacelabs	0.11	2
6 E for M/Honeywell	0.16	1
7 Datamedix	0.09	2

[a] Bandwidth = frequency at which the signal is reduced by 3 db (0.707 of the amplitude at DC).

[b] Poles = measure of how quickly the signal is attenuated. A 1-pole system is equivalent to a simple RC low-pass filter—20 db attenuation for each 10 times (decade) increase in frequency; 2 poles—40 db/decade; 4 poles—80 db/decade.

Using the step-response method, the natural frequency (fn—frequency at which the system oscillates when disturbed) and damping coefficient (ζ—a measure of how quickly the system stabilizes after being disturbed) of the catheter-transducer plumbing system can be determined. The natural frequency of the PA plumbing system we used had to be greater than 12 Hz and the damping coefficient less than 0.6 to reproduce faithfully the PA pressure waveform.[20] For a typical setup, the natural frequency was 18 Hz and the damping coefficient 0.3. If the system showed a poor dynamic response (fn <12 Hz or $\zeta > 0.6$), the cause was determined and was corrected before taking a pressure measurement. If the patient was coughing or otherwise agitated, pressure measurements were delayed until the situation stabilized. Pressures were measured simultaneously with the automated monitoring device(s) and the strip recorder.

Manual PA pressure and WP measurements were determined from tracings of at least 3 complete ventilatory cycles. End-expiration was identified on the strip recording, and PA systolic and diastolic pressures were measured from the phasic waveform. Pressures from 3 ventilatory cycles were compared to ensure a consistent reading. Pressures were measured independently by 2 investigators (MC and ROC).

MPAP mean was obtained electronically by low-pass filtering the pressure waveform with a filter cutoff at 0.08 Hz. (See Table 1 for description of and comparison of this filter with those generally available in commercially available monitors.) The low-pass filter smooths out the beat-to-beat pulsatile variations but allows the respiratory variations in pressure to be recorded.

Age, sex, diagnosis, ventilatory mode, blood pressure, pulse, respiratory rate, PEEP, thoracic compliance

$$\left(\frac{\text{tidal volume}}{\text{plateau pressure-PEEP}}\right),$$

and current therapy were recorded with each set of pressure measurements. Data were collected on a daily basis during the time that the catheter was in place.

PA pressure and WP measurements were divided into 3 groups according to ventilation mode (spontaneous, IMV, or assist/control). To avoid sample bias, no more than 2 measurements were taken on a given patient in any one ventilation mode.

In each ventilatory mode, MPAP and WP measured by the computer and the Tektronix 414 monitor were compared with measurements calculated from a strip recording using a paired Student's *t*-test.

TABLE 2. Spontaneous group[a]

	MPAP		WP		MPAP		WP		PA sys		PA dia	
	M	R	M	R	C	R	C	R	A	R	A	R
Mean ± SD	25 ± 10	29 ± 9	11 ± 5	14 ± 6	26 ± 10	29 ± 9	11 ± 5	14 ± 5	41 ± 15	40 ± 12	12 ± 8	22 ± 7
n	24		24		25		25		22		22	
$\bar{\Delta}$	−2.2		−3.3		−2.5		−3.7		0.6		−10.2	
p	<0.01		<0.001		<0.001		<0.05		NS		<0.001	

[a] PA pressure and WP in patients breathing spontaneously. Statistics for mean pulmonary artery (MPAP), wedge (WP), pulmonary artery systolic (PA sys), and pulmonary artery diastolic (PA dia) pressures; monitor (M) vs strip recorder (R), computer (C) vs strip recorder (R), and automated (A) vs strip recorder measurements. $\bar{\Delta}$ = mean difference between measurements; NS = not significant.

The number of PA systolic and diastolic pressure measurements available for statistical analysis was decreased due to excessive catheter whip artifact making it impossible to measure manually these pressures in some patients, and in other patients the computer displayed only MPAP. Because the method for measuring PA systolic and diastolic pressures was similar with both automated systems, computer and monitor measurements were combined into a single automated group in order to increase the number of measurements available for statistical analysis.

RESULTS

Thirty-one patients had pressure measurements in 1 ventilation mode only, 7 patients had measurements in 2 modes, and 2 patients had pressure measurements in 3 modes. In the entire study, no patient had more than 4 PA pressure and WP measurements.

In spontaneously breathing patients, both MPAP and WP which were measured by automated methods (computer and monitor) were significantly lower ($p < 0.01$ and < 0.001) than those made with the strip recorder (Table 2). The mean differences were small, but the range of difference was large. More than 20% of MPAP measurements had a 4-mm Hg or greater difference in pressure, and over 40% of WP measurements had a 4-mm Hg or larger difference (Fig. 1). PA systolic pressure was not affected by the method of measurement, but PA diastolic pressure measured with automated devices was significantly lower ($p < 0.001$) than with hand measurements (mean difference 10 mm Hg).

In patients supported with IMV, differences between automated measurements of PA pressure and WP, and from those made with a strip recorder were smaller than for the spontaneous group, but automated measurements were still significantly lower ($p < 0.05$ and $p < 0.001$, respectively), particularly for WP (Table 3). There was no correlation between the amount of variation in the WP and spontaneous respiratory rate, assisted respiratory rate, thoracic compliance, or level of PEEP. Automated measurement of PA systolic pressure was significantly higher ($p < 0.05$) than hand measurements (mean difference 4.8 mm Hg), and PA

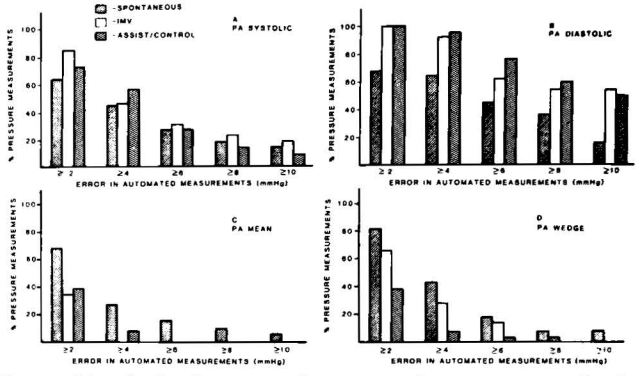

FIG. 1. Magnitude of error seen in automated measurements of pulmonary artery systolic (A), diastolic (B), mean (C), and wedge (D) pressures. The magnitude of error in PA diastolic pressure has particular importance for the coronary care patient.

diastolic pressure was significantly lower ($p < 0.001$) when measured with the automated method (mean difference 12 mm Hg).

In patients requiring total mechanical support (assist/control group), MPAP and WP were not significantly affected by the method of measurement (Table 4). However, 2 measurements had considerable spontaneous ventilatory effort before each ventilator-assisted breath. When these 2 measurements were excluded, the automated WP measurement was significantly larger ($p < 0.001$) than the manually measured WP. The difference was, however, small (0.7 mm Hg). There was no correlation between the variation in WP and the respiratory rate, thoracic compliance, or level of PEEP. The automated measurement of PA systolic pressure was significantly higher ($p < 0.005$) than the manual measurement (mean difference 4 mm Hg). Automated PA diastolic pressure was significantly lower ($p < 0.001$) than that for the manual method (mean difference 6 mm Hg).

DISCUSSION

The contrast between the large degree of error in automated WP measurements of patients with spontaneous ventilation, and the lack of error in patients requiring total mechanical support, was unexpected. No spontaneously breathing patient complained of hav-

TABLE 3. IMV group[a]

	MPAP		WP		MPAP		WP		PA sys		PA dia	
	M	R	M	R	C	R	C	R	A	R	A	R
Mean ± SD	33 ± 1	33 ± 10	16 ± 7	18 ± 7	31 ± 8	32 ± 8	13 ± 6	16 ± 6	45 ± 14	41 ± 11	10 ± 11	23 ± 8
n	6		9		20		20		13		13	
$\bar{\Delta}$	0.4		1.9		−0.9		−3.1		4.8		−12.4	
p	<0.05		<0.05		NS		<.0001		<0.005		<0.001	

[a] PA pressure and WP in patients on IMV. Statistics for mean pulmonary artery (MPAP), wedge (WP), pulmonary artery systolic (PA sys), and pulmonary artery diastolic (PA dia) pressures; monitor (M) vs strip recorder (R), computer (C) vs strip recorder (R), and automated (A) vs strip recorder measurements. $\bar{\Delta}$ = mean difference between measurements; NS = not significant.

TABLE 4. Assist/control group[a]

	MPAP		WP		MPAP		WP		PA sys		PA dia	
	M	R	M	R	C	R	C	R	A	R	A	R
Mean ± SD	26 ± 9	25 ± 9	10 ± 5	9 ± 4	29 ± 9	30 ± 9	12 ± 6	11 ± 6	43 ± 12	39 ± 12	17 ± 10	24 ± 8
n	12(10)		12(10)		26(24)		26(24)		22		22	
$\bar{\Delta}$	0.7(1.1)		1(1.7)		−0.38(−0.08)		−1.7		4.3		−6.2	
p	<0.05		<0.05		NS		<0.001		<0.005		<0.001	

PA pressure and WP patients requiring mechanical ventilation. Statistics for mean pulmonary artery (MPAP), wedge (WP), pulmonary artery systolic (PA sys), and pulmonary artery diastolic (PA dia) pressures; monitor (M) vs strip recorder (R), computer (C) vs strip recorder (R), and automated (A) vs strip recorder measurements. $\bar{\Delta}$ = mean difference between measurements; NS = not significant. Values in parentheses calculated excluding 2 patients with considerable spontaneous ventilatory effort.

ing dyspnea, or any significant respiratory distress, nor did they appear to the investigator to be having significant respiratory difficulty. It was impossible to predict which of the spontaneously breathing patients would exhibit an important error in their automated WP measurements. Patients on ventilators, with small differences in automated and manual WP, had thoracic compliances as low as 10 ml/cm H_2O and required up to 25 cm H_2O of PEEP. The large magnitude of measurement error in patients breathing spontaneously is especially important because the variation in intravascular pressures (due to ventilation) is believed to be small in this group and might be overlooked in the clinical setting. The clinical implications are given further import in the light of recent findings on the effects of ventilation on left ventricular afterload and left ventricular dysfunction.[21-23]

The respiratory variation seen in WP in a patient breathing spontaneously and in a patient on a mechanical ventilator is shown in Figure 2. Ventilation causes much less variation in WP in mechanically ventilated patients than in patients breathing spontaneously. It is more likely that the variation is a result of a smaller change in pleural pressure occurring in mechanically ventilated patients rather than the change in pleural pressure resulting from inspiration not being transmitted as effectively to the intrathoracic vessels.

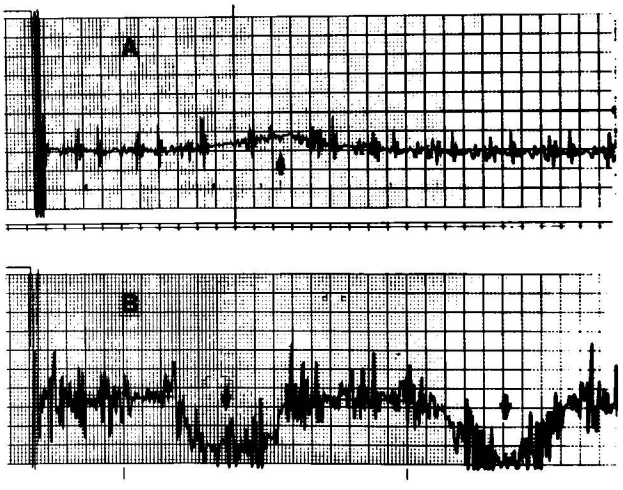

FIG. 2. Example of respiratory variation seen in a patient on a mechanical ventilator (A) and a patient breathing spontaneously (B). *Arrows* mark inspiration in each tracing. Paper speed 25 mm/sec.

The expected change in pleural pressure with mechanical and with spontaneous ventilation can be estimated by using the formula for compliance ($C=\Delta V/\Delta P$). Pleural pressure during inspiration while on mechanical ventilation would be +4.8 cm H_2O in a patient who has a thoracic compliance of 30 ml/cm H_2O, a normal chest wall (compliance=200 ml/cm H_2O), a pleural pressure during expiration of zero, a tidal vol-

ume of 900 ml, and a continuous positive airway pressure (CPAP) or PEEP of 10 cm H_2O. While breathing spontaneously, the patient would have a pleural pressure during inspiration of −25.2 cm H_2O. It is apparent that inhalation could have a significant impact on the measurement of intravascular pressure in patients breathing spontaneously.

What magnitude of difference in WP would be necessary to cause a clinically significant error? A 1- or 2-mm Hg difference is the expected measurement error. A 4-mm Hg or larger difference in a pressure with such a small range could prove important. A measured WP of 4 mm Hg when it is actually 8 mm Hg would make little difference because both measurements are within normal limits. However, if the WP were measured as 16 mm Hg when it was actually 20 mm Hg or more, the result could have an important influence on the recommended treatment of the patient because 20 mm Hg or greater is often associated with pulmonary edema.

Many authors believe that trends in WP are more important than absolute pressure and, therefore, use monitor-determined pressures or a WP averaged over 1 complete ventilatory cycle.[24-26] Following trends in WP is important; however, the pressure being monitored must have a consistent relationship to the absolute pressure. Because pulmonary pressures are known to change with ventilation and because ventilatory patterns are variable, it is unreasonable to accept pulmonary pressures measured with this unknown and large variability. Not correcting for respiratory variability in pulmonary pressures is equivalent to measuring the same pressures without concern for transducer position and zero values. Where possible, all known errors and artifacts must be eliminated in the pressure measurement.

By reading PA pressures and WP from a strip recording at end-expiration, the clinician is able to: (1) measure pressure closest to transmural or absolute pressure, and (2) avoid artifactual error in pressure trends due to ventilatory pattern and catheter whip. A clinical case study illustrates the importance of correct measurement. One patient in our study had been admitted with a myocardial infarction and later required surgery for a bowel abscess. After surgery, he was sedated and intubated but he was breathing spontaneously. His WP, using automated measurement methods, was 4–8 mm Hg; however, when measured manually from a strip recording, it was 22 mm Hg. He had a marked decrease in WP during inspiration and a respiratory rate of 40/min. This rapid rate apparently was not diagnosed as respiratory distress but as postoperative discomfort. That the patient was probably in early pulmonary edema is evidenced by the fact that the following day he was on a ventilator, having developed frank pulmonary edema during the night. It is likely that his treatment was affected by the erroneous underestimation of his WP while he was breathing spontaneously.

The large magnitude of error in PA diastolic pressure (Fig. 1) resulting from measurements with automated methods is clinically important because this measurement is sometimes used as a substitute for WP. Automated systems identify peaks as systolic pressure and valleys as diastolic pressure. Unfortunately, the peaks and valleys frequently are caused by artifact or catheter whip, rather than by actual changes in pressure. The automated system does not differentiate between artificial and actual pressure fluctuations and so records artifact as systolic and diastolic pressure. The whip artifact is unique to PA pressure measurement and is caused by the catheter moving as the right ventricle contracts against it and the tricuspid and pulmonic valves close around it. The artifact is amplified further by the underdamped catheter-transducer system.[20]

Many monitoring systems have low pass filters designed to minimize the whip artifact in PA pressure. Unfortunately, these filters make it impossible to assess the dynamic response characteristics of the system, a capability considered essential by the authors as well as others.[20, 27] At present, we are aware of no pressure monitoring system which provides a means for evaluating dynamic response as well as eliminating the whip artifact.

If PA pressures and WP are to be used for clinical treatment decisions, it is the responsibility of those caring for these patients to make sure the measurements are as accurate as possible. In this age of advanced technology, we tend to believe that any automated device produces faster and more accurate results, especially when they are so impressively displayed. However, if we are to use these devices, it is important to understand how they work, their advantages, and their limitations before we unconditionally accept the information we obtain from them.

Elimination of artifacts from the PA pressure and WP signal which are caused by ventilation is mandatory if accurate assessments of these pressures are used to make clinical decisions. Bedside monitors and computer algorithms developed before 1982 do not eliminate these artifacts. We believe that such algorithms can and will be developed and encourage such development. However, for the present time, for those using available monitors, a strip recorder should be available for manual pulmonary pressure measurement.

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