

citator must be vigilant to a sudden deterioration in compliance. When the Ohio hand resuscitator is used without the Blount Percent adaptor, the vented O<sub>2</sub> nipple should be permanently bonded to the resuscitator housing or have standard threads of a design to preclude its interchange with incompatible fittings. The nonvented nipple on the Blount adaptor should be permanently fixed in place to avoid any possibility of removal and interchange with the vented nipple. A pressure-limiting system should be incorporated with an audible alarm. This system should safely limit

the peak pressure within the system to some arbitrary limit and warn the operator when that pressure is reached. The manufacturer should provide an indelible warning label on the resuscitator body if the resuscitator valve is likely to jam beyond a certain flow rate of O<sub>2</sub>. As with any type of anesthetic apparatus, close inspection prior to its use is mandatory.

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### Pulmonary Vascular Pressure Reading at the End of Exhalation

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Intermittent mandatory ventilation (IMV) with and without positive end-expiratory pressure (PEEP) is a commonly used ventilatory pattern in the management of patients who need mechanical ventilatory assistance.<sup>1</sup> Similarly, the balloon-tipped pulmonary arterial catheter is frequently used in assessing the cardiovascular status of critically ill patients.<sup>2</sup> Often IMV and pulmonary-artery catheters are utilized simultaneously in patient care. Presently it is recommended that pulmonary vascular pressures be obtained while the patient continues with the ventilatory pattern existing during treatment, because a different ventilatory pattern (*i.e.*, apnea and no PEEP) may cause irrelevant, misleading physiologic changes.<sup>3</sup> The recommended moment to read pulmonary vascular pressure during the ventilatory pattern existing during treatment is at the end of exhalation.<sup>4</sup>

We demonstrate and discuss a previously unreported problem in reading pulmonary arterial and pulmonary arterial wedge pressures in patients who have rapid wide swings in pleural and airway pressures. Such patients include those managed with IMV who have rapid spontaneous and mechanical ventilatory rates as well as decreased pulmonary compliance. Under these circumstances pulmonary vascular pressure patterns have wide fluctuations, and it is often difficult to determine the end of exhalation by the vascular

pressure pattern alone or by trying to time the pattern with observation of the patient. We therefore simultaneously recorded airway and pulmonary vascular pressures.

#### METHODS

Ten critically ill patients with endotracheal tubes in place, needing mechanical ventilatory assistance utilizing IMV, were studied consecutively. These patients also needed positive end-expiratory pressures (PEEP) ranging from 3 to 10 torr and F<sub>I</sub>O<sub>2</sub> 0.3-0.5 to maintain Pa<sub>O<sub>2</sub></sub> 67-123 torr.

Pulmonary arterial and pulmonary arterial wedge pressures were measured with a triple-lumen balloon-tipped catheter (Edwards #93A-113-7F) introduced into the pulmonary artery by the modified Seldinger technique via the internal jugular or subclavian vein. Airway pressures were measured directly by a T-connector interposed between the endotracheal tube and the ventilator hose. Both vascular and airway transducers (Hewlett-Packard 1280C) were calibrated with standard mercury manometers and were then referenced to atmospheric pressure at the level of the left atrium. Simultaneous pulmonary vascular and airway pressure patterns were recorded by a two-channel recorder.

During each patient's clinical course many simultaneous recordings of pulmonary vascular and airway pressure patterns were made. The pulmonary vascular pressure values at the end of exhalation did not vary more than 2 torr throughout any particular recording, each recording being continuous for at least 0.5 to 2 minutes. Therefore, all exemplary pulmonary vascular pressure values given for each patient are absolute values taken from arbitrarily selected but

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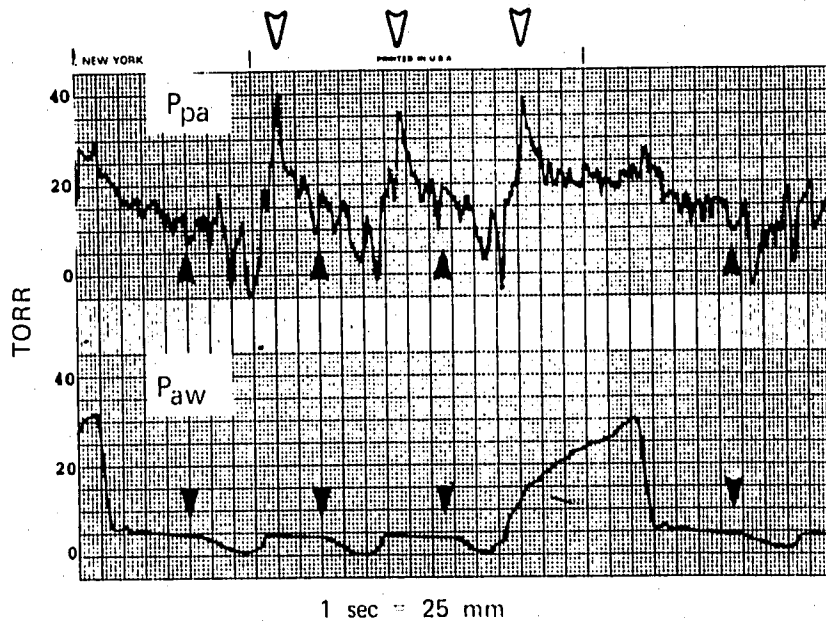


FIG. 1.  $P_{pa}$  = pulmonary arterial pressure;  $P_{aw}$  = airway pressure; closed arrows = end exhalation; open arrows = first heartbeat following a spontaneous inspiration.

typical pressure patterns during one of these short periods.

#### RESULTS

Representative examples of simultaneous pulmonary vascular and airway pressure pattern recordings in two patients are shown in figures 1 and 2. Figure 1 was recorded from a patient with a spontaneous ventilatory rate of 36/min, an IMV of 12/min and PEEP 5 torr. By using the phasic pulmonary arterial pressure pattern alone, the pulmonary arterial ( $P_{pa}$ ) diastolic pressure cannot be determined at the end of exhalation. However, with the simultaneously recorded airway pressure ( $P_{aw}$ ) pattern it is clear that the  $P_{pa}$  diastolic value at the end of exhalation (*solid arrows*) was consistently 8–10 torr. Notice the single large pulse at the end of each spontaneous inspiration (*open arrows*). Figure 2 was recorded from a patient with a spontaneous ventilatory rate of 36/min, an IMV of 8/min, and a PEEP of 10 torr. It demonstrates that even mean pulmonary arterial wedge pressure ( $P_{paw}$ ) can fluctuate widely (8–18 torr) with the ventilatory cycle, and that the simultaneously recorded airway pressure pattern is necessary to determine the  $P_{paw}$  value at the end of exhalation (*solid arrows*). Notice the patient-ventilator asynchrony, as shown by a spontaneous breath during the ventilator cycle.

For all ten patients, the widely fluctuating values of  $P_{aw}$ ,  $P_{pa}$  diastolic, and  $P_{paw}$  during the ventilatory cycle and the relatively static values at the end of exhalation are shown in table 1. We were able to measure at end exhalation the pulmonary vascular pressures in all patients consistently within a 2-torr range.

#### DISCUSSION

The effects of various airway pressure patterns upon the pulmonary vasculature have been well documented.<sup>5-7</sup> Therefore, it has been recommended that measurement of pulmonary arterial and pulmonary arterial wedge pressures should occur at the end of exhalation.<sup>4</sup> There is no air flow at that moment, so that intrapleural pressure is considered a static baseline influence upon pulmonary hemodynamics. When only the vascular pressure pattern is recorded, determining end exhalation becomes a difficult, if not impossible, task in patients who have rapid wide swings in pleural and airway pressures. However, when the airway pressure pattern is simultaneously recorded, the end-exhalation moment can be easily determined, allowing for temporal consistency in vascular pressure measurement. In our patients, changes in pulmonary vascular pressure values at end exhalation were evaluated in light of other measurements (*e.g.*, cardiac output, systemic arterial pressure, arterial blood gases, urine production) and the patients' overall clinical conditions, with the appropriate alterations in therapy then being effected. It is obvious that when reading pulmonary vascular pressures, when incorrect values are chosen, inappropriate fluid management or use of cardiotoxic or vasoactive drugs can occur.

In most circumstances of intravascular pressure monitoring the pressure pattern is displayed on an oscilloscope, but the values chosen for  $P_{pa}$  diastolic and  $P_{paw}$  are often taken from a digital panel printout. The digital panel meter, when switched to diastolic mode, displays data that are selected randomly every two seconds from the pressure pattern,

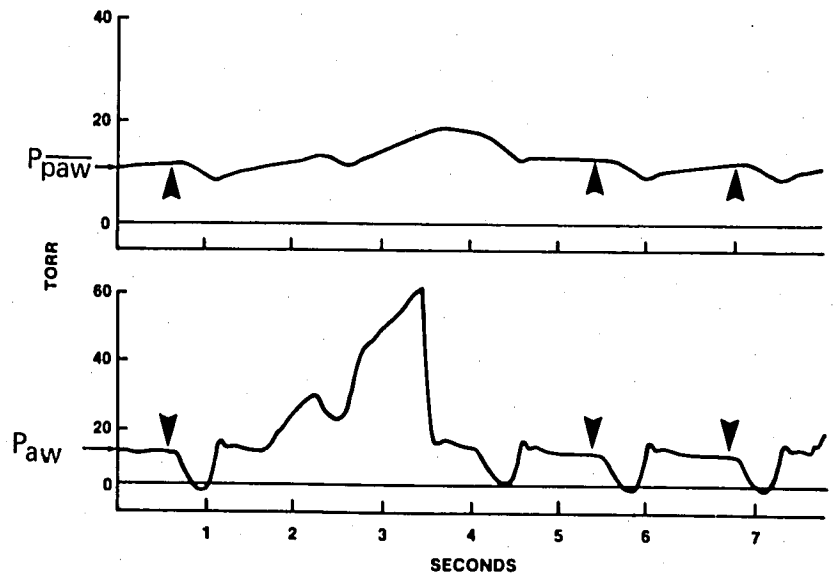


FIG. 2. See figure 1;  $P_{\overline{paw}}$  = mean pulmonary arterial wedge pressure.

which can have great variations as demonstrated by our examples. When switched to venous mode ( $P_{paw}$  and  $P_{\overline{paw}}$  are generally measured in this mode), it displays a value that is an integrated mean of the pressure pattern and therefore will be influenced by both the rate and mode of ventilation, whether positive-pressure or spontaneous. Also, in the venous mode, there is a time delay in the digital printout (signal-to-90 per cent response delay of  $6.9 \pm 0.1$  (SD) seconds, Hewlett-Packard 78205 B digital panel meter), which is necessary for the integration process, and therefore it is impossible visually to synchronize the appropriate number with the ventilatory pattern. Thus, the digital printout presently has no advantage over an oscilloscope trace and is not a solution to the problem. It may be possible in the near future to interface a micro-processor containing an algorithm that allows the digital

meter to display only the end-exhalation vascular pressure.

Of interest is the single large pulse of the heartbeat immediately following each spontaneous inspiration that is seen in the phasic  $P_{pa}$  pattern (fig. 1). This large pulse is probably due to volume loading of the right ventricle during the spontaneous inspiration. The clinical utility of observing this large pulse is that it can serve as a respiratory pattern marker of a beginning end-exhalation period provided an IMV breath does not occur. When only an oscilloscopic trace of the phasic  $P_{pa}$  pressure pattern is available, recognition of this large pulse may allow determination of a reasonably correct end-exhalation moment and thus the  $P_{pa}$  diastolic at that moment.

Generally, vascular pressures are presently measured relative to atmosphere, whereas the true distend-

TABLE 1. Airway, Pulmonary Arterial Diastolic and Pulmonary Arterial Wedge Pressure Values—Maxima, Minima, and at End Exhalation\*

	Pressure (Torr)											
	$P_{aw}$				$P_{pa}$ Diastolic				$P_{paw}$			
	Max	Min	$\Delta P$	EE	Max	Min	$\Delta P$	EE	Max	Min	$\Delta P$	EE
Patient 1	32	0	32	4	22	-4	26	8-10	15	-8	23	5-6
Patient 2	56	-2	58	10	34	2	32	18-19	30	-6	36	13-15
Patient 3	35	3	32	8	31	10	21	25-27	28	8	20	20-22
Patient 4	42	5	37	21	35	-5	40	30-32	32	-8	40	24-26
Patient 5	40	-3	43	5	24	-4	28	15-17	17	0	17	12-14
Patient 6	50	0	50	10	31	6	25	22-24	28	-3	31	16-18
Patient 7	37	5	32	10	22	5	17	14-16	18	-1	19	10-12
Patient 8	32	3	29	3	24	15	9	15-16	23	7	16	7-9
Patient 9	20	2	18	5	21	9	12	14-16	15	3	12	10-12
Patient 10	32	1	31	4	33	11	22	16-18	26	7	29	13-15

\*  $P_{aw}$  = airway pressure;  $P_{pa}$  Diastolic = pulmonary arterial diastolic pressure;  $P_{paw}$  = pulmonary arterial wedge pressure; Max = maximum pressure recorded; Min = minimum pressure

recorded;  $\Delta P$  = difference between maximum and minimum pressures; EE = pressure measured at end exhalation.

ing pressure in the pulmonary vasculature and heart is the transmural pressure, with the intrapleural pressure as reference. A recent report describing a technique for direct measurement of intrapleural pressure brings us one step closer to the true value for this transmural pressure.<sup>8</sup> However, the ultimate solution is obtaining the left ventricular end-diastolic transmural pressure as it relates to intravascular volume and the inotropic state of the myocardium. The pressure immediately surrounding the heart (pericardial) may be different from the intrapleural pressure<sup>9</sup>; however, the clinical significance of this is yet to be shown.

We offer this report as a partial solution to the problem of accurate measurement of pulmonary vascular pressures. Simultaneous airway pressure and pulmonary vascular pressure recording can resolve the difficulty in choosing the correct pressure value during the end of exhalation.

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## Induction of Anesthesia in a Patient with an Undiagnosed Pheochromocytoma

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The responses of a patient with pheochromocytoma to three identical anesthetic inductions are reported. The first was performed prior to diagnosis, the second after treatment with  $\alpha$ -methylparatyrosine, and the third after resection of the tumor.

### REPORT OF A CASE

A 54-year-old black man was scheduled for transsphenoidal hypophysectomy for resection of a pituitary adenoma. He had had one episode of severe, throbbing headache, blurred vision, diaphoresis, and dizziness. The patient had a ten-year history of mild diabetes, and had been treated intermittently for hypertension. Roentgenograms of the skull revealed a large sella turcica with erosion of the posterior wall and floor. Results of physical examina-

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tion were unremarkable. The patient weighed 72 kg. Hemoglobin was 12.4 g/100 ml, and T4 was 3.4  $\mu$ g/100 ml (normal 4.2-11.5  $\mu$ g/100 ml). Blood pressure was 135  $\pm$  14/85  $\pm$  11 torr. The hypothyroidism was treated with liothyronine, and the patient was also given dexamethasone preoperatively.

The patient was premedicated with morphine, 5 mg, and scopolamine, 0.5 mg. An arterial catheter was placed. Blood pressure was 160/80 torr, and pulse rate was 55/min. Meperidine, 100 mg, was given iv, followed a minute later by droperidol, 5 mg. Blood pressure increased to 200/100 torr, and pulse rate increased to 90/min. Thiopental, 250 mg, was then given. Ventilation was controlled by mask. Blood pressure abruptly increased to 280/140 torr, and pulse rate increased to 170/min. S-T segment depression developed, followed by bigeminy. The arrhythmia responded to iv infusion of lidocaine, 50 mg. The blood pressure was unresponsive to trimethaphan, but was reduced to 160/100 torr by sodium nitroprusside infusion. The tachycardia was treated with propranolol, 0.75 mg, which reduced the heart rate to 80/min. During this acute episode arterial blood gases were PaO<sub>2</sub> 269 torr, PaCO<sub>2</sub> 52 torr, pH 7.33, and base excess +1.0.

The operation was postponed, and the patient, now alert, was transferred to the surgical intensive care unit. The sodium nitroprusside infusion was withdrawn over several hours, and no further episode of hypertension or tachycardia occurred. Mean 24-hour urinary metanephrine excretion was 15 mg (normal, less than 1 mg). The patient was treated with  $\alpha$ -methylparatyrosine, 1,500 mg/day, in three doses. The 24-hour metanephrine excretion decreased to 11.3 mg, and blood pressure was reduced to 126  $\pm$  19/74  $\pm$  15 torr.

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