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Interpretation of the pulmonary artery occlusion pressure in mechanically ventilated patients with large respiratory excursions in intrathoracic pressure

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Abstract Objective: To assess the reliability of the pulmonary artery occlusion pressure (Ppao) when respiratory excursions in intrathoracic pressure are prominent.
Design: We studied 24 critically ill patients who had 15 mm Hg or more of respiratory excursion in their Ppao tracing. Large respiratory excursions resulted from respiratory muscle activity that persisted despite sedation and mechanical ventilation in the assist-control mode. From the Ppao tracing, the end-expiratory and mid-point values were recorded; the latter was measured halfway between end-expiration and the nadir due to inspiratory triggering. The Ppao was then re-measured after administration of a non-depolarizing muscle relaxant.
Setting: Medical intensive care unit of a university-affiliated teaching hospital.
Measurements and results: The difference between the pre-relaxation end-expiratory Ppao and the relaxed Ppao was larger than the difference between the pre-relaxation mid-point Ppao and the relaxed Ppao (11 ± 5 vs 3 ± 3 mm Hg, $p < 0.01$). In 21 of 24 (88 %) cases, the relaxed Ppao was more closely approximated by the mid-point Ppao than by the end-expiratory Ppao. The difference between the end-expiratory Ppao and the relaxed Ppao increased as the amount of respiratory excursion increased ($r = 0{,}51$; $p < 0.01$).
Conclusions: In mechanically ventilated patients whose respiratory muscles produce large excursions in the Ppao, the end-expiratory Ppao is often much higher than the Ppao measured after muscle relaxation. The pre-relaxation mid-point Ppao and the relaxed Ppao are usually similar, but this may not be true in individual patients. In this setting, the Ppao measured after muscle relaxation probably provides the most clinically reliable estimate of left heart filling pressure.

Key words Swan-Ganz · Pulmonary wedge pressure · Neuromuscular blocking agents

Introduction

Pulmonary artery occlusion pressure (Ppao) is used as a bedside indicator of the adequacy of left ventricular preload, and as an estimate of the pulmonary capillary pressure [1, 2]. For this reason, the Ppao often greatly influences clinical decisions regarding the manipulation of intravascular volume and the use of inotropic drugs. Unfortunately, errors in the measurement and interpretation of the Ppao are not uncommon [2–4]. One potential source of error is vigorous respiratory muscle activity, which results in large fluctuations in intrathoracic pressure [2, 3]. Transmural pressure (Ppao-pleural pressure), the pressure that influences left ventricular preload and fluid filtration from pulmonary capillaries, is conventionally estimated by recording the Ppao at end-

expiration, because the pleural pressure at end-expiration is normally only slightly less than atmospheric pressure and is most constant during the normal respiratory cycle [5, 6]. The activation of expiratory muscles during vigorous breathing may result in positive pleural pressure at end-expiration, causing the end-expiratory Ppao to overestimate transmural pressure significantly [2, 7].

Two approaches have been used to estimate transmural pressure in the face of large respiratory excursions in intrathoracic pressure. The first employs an esophageal ballon to estimate pleural pressure. A study in which simultaneous measurements of Ppao and esophageal pressure were made during exacerbations of COPD found that the end-expiratory Ppao greatly exceeded the transmural pressure [8]. In contrast, the mean Ppao provided a reasonably good estimate of transmural pressure in most cases [8]. A second approach, applicable only during mechanical ventilation, employs temporary paralysis of the respiratory muscles to obtain an estimate of transmural pressure. In a study involving seven patients who had large respiratory excursions in the Ppao while being supported in the intermittent mandatory ventilation (IMV) mode, the end-expiratory Ppao was found to be significantly greater than the post-paralysis Ppao [7]. The respiratory rates of the latter group of patients were much higher than the IMV rate [7], and the large amount of unsupported ventilation may have accounted in part for the vigorous activity of respiratory muscles [9]. In this study, there was good agreement between the post-paralysis Ppao and the pre-paralysis "mid-point" Ppao, measured halfway between the end-expiratory and end inspiratory values [7].

In the present study, we recorded Ppao tracings before and after muscle relaxation in 24 critically ill patients who had demonstrated large respiratory fluctuations in the Ppao despite being supported in the assist-control mode of mechanical ventilation. Although no Ppao value precisely measures transmural pressure, we assumed that the latter would be reasonably approximated by the Ppao measured after muscle paralysis [7]. The specific goals of the study were twofold: 1) to define the magnitude of error associated with use of the end-expiratory Ppao relative to the relaxed Ppao and 2) to determine whether the mid-point Ppao might provide a better estimate of the relaxed Ppao when breathing efforts are vigorous.

Methods

We studied 24 critically ill, mechanically ventilated adults who had at least a 15 mm Hg respiratory excursion in their Ppao tracing [7]. In all cases, elimination of large respiratory excursions was deemed to be clinically indicated, due to concern regarding the validity of the hemodynamic data. All patients were supine and were actively

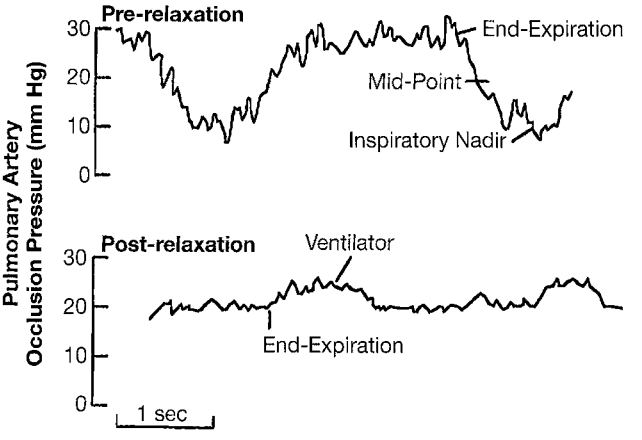


Fig. 1 Pulmonary artery occlusion pressure (Ppao) tracings showing the end-expiratory, end-inspiratory (nadir), and the mid-point values

triggering the ventilator in the assist-control mode of mechanical ventilation despite intravenous sedation. In addition, expiratory muscle activity was typically apparent, as determined by palpation of the upper abdomen during expiration. Three patients had airflow obstruction. The remaining 21 patients, none of whom had a history of COPD or clinical evidence for airflow obstruction, underwent mechanical ventilation and hemodynamic monitoring because of the adult respiratory distress syndrome, sepsis, hepatic failure, pneumonia, renal failure or pancreatitis, or a combination of two or more of these conditions.

Intravascular catheters were connected to standard bedside pressure transducers that had been zeroed at the level of the mid-axilla line with the patient supine. Before patient use, transducers (Medex Model Mx 860, Hilliard, Ohio) were compared to a Volumetrix standard (DPM-1, Volumetrix, Inglewood, Calif.) to ensure accuracy within ± 2.5 %. Of the various components of the pressure monitoring system, the bedside monitor (Merlin, Hewlett Packard, Andover, Mass.) had the lowest frequency response (DC-12.5 Hz). Prior to muscle relaxation, strip-chart recordings of the Ppao were obtained and measurements were made of the end-expiratory Ppao, the inspiratory nadir Ppao, the mid-point Ppao and the inspiratory-expiratory excursion. End-expiration and the inspiratory nadir were defined in ralationship to the patient rather than the ventilator. End-expiration was the point in the Ppao tracing just before the onset of patient inspiratory effort that triggered the ventilator, and the inspiratory nadir was the lowest Ppao during the inspiratory effort; the mid-point Ppao was defined as the pressure value halfway between the end-expiratory and nadir Ppao (Fig. 1). Respiratory fluctuations in the Ppao were often similar from cycle to cycle. When pressures varied with different respiratory cycles, several consecutive respiratory cycles were examined and the most representative values were recorded. Subsequently, the respiratory muscles were rendered inactive and the "relaxed" Ppao was recorded at end-expiration. Pressure tracings were obtained retrospectively in 5 cases and prospectively in 19 cases. Respiratory muscle inactivity was achieved by the administration of either vecuronium or atracurium. The dose of vasoactive drugs and ventilator settings remained unchanged.

Records of blood pressure, heart rate and minute ventilation before and after paralysis were available in 17 cases. Thermodilution cardiac output was measured pre- and post-paralysis in the last three patients studied. In two cases, bolus injections of iced saline were given at end-inspiration and the cardiac output was taken

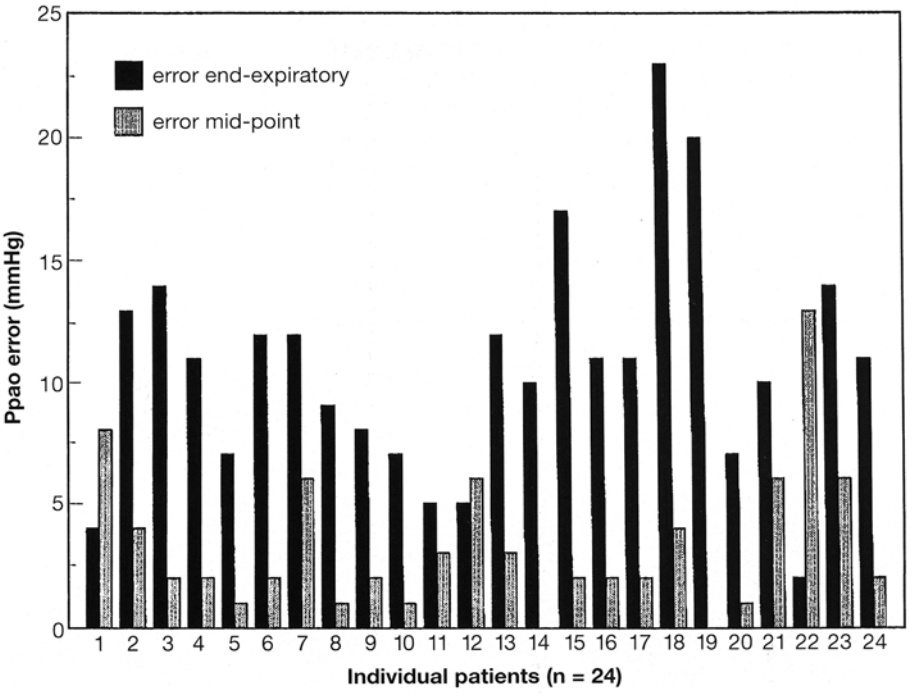


Fig. 2 Error associated with the use of the end-expiratory and mid-point pulmonary artery occlusion pressure (Ppao) for each of the 24 patients

as the average of 3–5 measurements. In the last instance, a continuous cardiac output catheter (Intellicath CCO/VIP, Baxter Edwards, Irvine, Calif.) was used.

A comparison of the difference between the end-expiratory Ppao and the relaxed Ppao and the difference between the mid-point Ppao and the relaxed Ppao was made using paired Student's *t*-test. Comparison of the y-intercept of relaxed Ppao versus end-expiratory Ppao and the y-intercept of relaxed Ppao versus mid-point Ppao was made using linear regression. The institutional review board approved data collection and analysis without the need for informed consent.

Results

Prior to muscle relaxation, the respiratory excursion in the Ppao was 22 ± 7 mm Hg, with a range of 15–40 mm Hg. The end-expiratory Ppao during vigorous breathing was 24 ± 8 mm Hg (range, 14–43 mm Hg), and the mid-point Ppao was 13 ± 7 mm Hg (range, 2–28 mm Hg). The post-relaxation Ppao was 14 ± 5 mm Hg (range, 5–30 mm Hg).

The difference between the end-expiratory Ppao and the relaxed Ppao was significantly greater than the difference between the mid-point Ppao and the relaxed Ppao (11 ± 5 vs 3 ± 3 mm Hg; $p < 0.001$). Using the relaxed Ppao as the reference value, the mean difference between the end-expiratory Ppao error and the mid-point Ppao error was 7.3 mm Hg (95 % CI: 4.6, 10.1). The errors associated with the use of the end-expiratory Ppao and the mid-point Ppao for individual patients are shown in Fig. 2. In most instances, the end-expiratory Ppao was associated with a large error, exceeding 10 mm Hg in half of the cases. In contrast, the mid-point Ppao differed from the relaxed Ppao by more than 5 mm Hg in only six instances. In 21 of 24 (88 %) cases, the mid-point Ppao provided a closer estimate of the relaxed Ppao than did the end-expiratory Ppao.

The relationship between the relaxed Ppao and the end-expiratory during assist-control ventilation is shown in Fig. 3 (left panel). The consistent overestimation of the relaxed Ppao by the end-expiratory Ppao is apparent. In contrast, the relationship between the mid-point Ppao and the relaxed Ppao was such that most individual data points fell reasonably close to the line of identity (Fig. 4, right panel). The y-intercept of the relaxed Ppao versus the end-expiratory Ppao relationship was significantly greater than the y-intercept of the relaxed Ppao versus mid-point Ppao relationship (8.8 vs–1.4 mm Hg; $p < 0.01$).

The error associated with the use of the end-expiratory Ppao increased as respiratory excursions increased; however, there was only a moderate correlation between these two variables ($p = 0.01$) (Fig. 4, left panel). In contrast, the error associated with use of the mid-point Ppao was independent of the degree of respiratory excursion ($p = 0.24$) (Fig. 4, right panel).

Paralysis did not result in a change in systolic blood pressure (108 ± 25–107 ± 25 mm Hg) or heart rate (105 ± 17–104 ± 16) and minute ventilation decreased only minimally (17 ± 5–15 ± 5 l/min). In the three patients studied, cardiac output did not change significantly with paralysis (5.6 ± 0.4–5.9 ± 1.6 l/min).

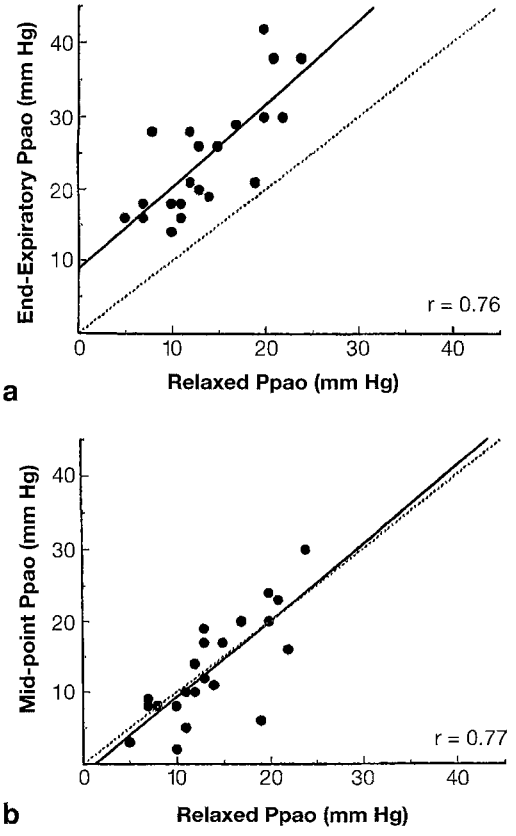


Fig. 3a,b Relationship between the relaxed pulmonary artery occlusion pressure (Ppao) and the end-expiratory Ppao (**a**), and between the relaxed Ppao and the mid-point Ppao (**b**). The y-intercept of the former is significantly greater than the y-intercept of the latter (8.8 vs–1.4 mm Hg; $p < 0.01$)

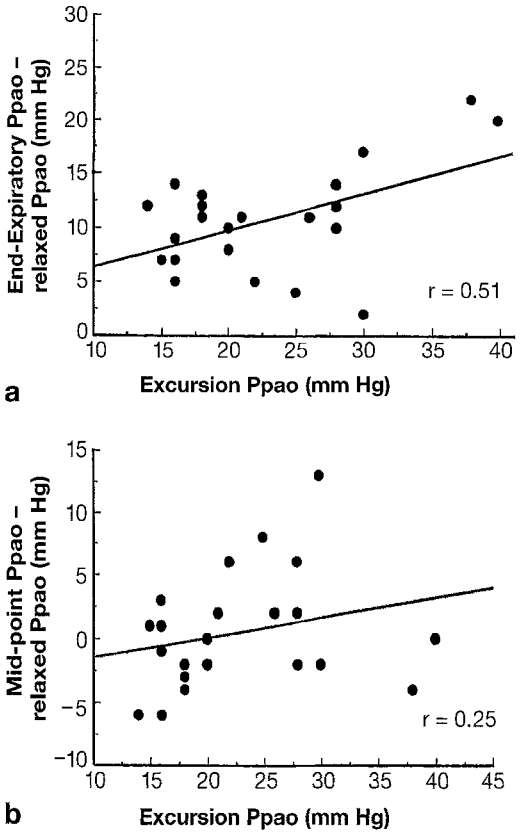


Fig. 4a,b Relationship between the degree of respiratory excursion and error associated with the measurement of the pulmonary artery occlusion pressure (Ppao) at end-expiration (**a**), and between the degree of respiratory excursion and the error associated with use of the mid-point Ppao (**b**)

Discussion

Errors in acquisition and interpretation of hemodynamic data are not uncommon [2–4]. In this study, we examined one potential source of error: large fluctuations in the Ppao due to vigorous respiratory muscle activity. Consistent with the findings of Schuster and Seeman [7], our findings show that large respiratory excursions in the Ppao usually predict that the end-expiratory Ppao will significantly overestimate the relaxed Ppao. The patients in our study were mechanically ventilated in the assist-control mode, while the patients described by Schuster and Seeman were supported with intermittent mandatory ventilation [7]. Nonetheless, the error associated with the use of the end-expiratory Ppao in the latter study (10 ± 5; range, 3–23 mm Hg) was remarkably similar to that found in the present study (11 ± 5; range 4–20 mm Hg). Errors of this magnitude may adversely influence clinical diagnosis and therapy.

The presence of significant respiratory muscle activity in the assist-control mode of mechanical ventilation is not surprising in the light of previous studies [9–13]. Marini and co-workers have documented considerable inspiratory muscle effort during machine-assisted breaths in the assist-control mode [9, 10]. Furthermore, the same investigators have shown that carbon dioxide-driven hyperpnea results in a similar degree of end-expiratory pleural pressure during machine-assisted and spontaneous ventilation [13]. Therefore, significant inspiratory and expiratory muscle activity may persist despite machine-assistance of each inspiratory effort.

Previous studies have found a high degree of correlation between changes in abdominal pressure and intrathoracic pressure during active exhalation [14, 15]. Positive abdominal pressure generated by expiratory muscle activity is not attenuated by the pulmonary parenchyma and may be fully transmitted across the relaxed diaphragm to the juxtacardiac region [7]. This probably accounts for the fact that active expiration typically has a much greater impact on the end-expiratory Ppao than does the therapeutic application of PEEP.

In contrast to the end-expiratory Ppao, we found the mid-point Ppao to be a reasonable estimate of the relaxed Ppao in most instances. Excursions in the Ppao

tracing due to respiratory muscle activity primarily reflect breathing-related excursions in pleural pressure [8]. The close relationship between the mid-point Ppao and relaxed Ppao suggests that our patients had similar degrees of inspiratory and expiratory excursion in pleural pressure. Rice and colleagues [8] made a similar observation in non-intubated patients with COPD. In their study, the mean Ppao and the transvascular pressure (Ppao-esophageal pressure) were within 3 mm Hg in 16 of 19 cases. In the report by Schuster and Seeman [7], the authors did not comment on the mid-point Ppao. However, their study included sufficient data to compute the mid-point Ppao before paralysis in six cases; the difference between the mid-point Ppao and the post-paralysis Ppao ranged from 1 to 4 mm Hg. Although perhaps somewhat surprising, these three studies suggest that many patients who are breathing vigourously will exhibit a roughly equal degree of negative and positive excursion in pleural pressure, regardless of whether breathing is spontaneous or is supported in the IMV or assist-control mode of mechanical ventilation.

As did Schuster and Seeman [7], we made the assumption that the post-relaxation Ppao would closely approximate transmural Ppao when respiratory muscles were active. This assumption would not be correct if the venous return were significantly different before and after muscle relaxation. Active breathing may influence venous return by its effect on intrapleural pressure or by increased metabolic activity of the respiratory muscles, and the effect on venous return will depend on the abdominal vascular zone conditions [16, 17]. The lack of a paralysis-related decrease in cardiac output was demonstrated in only three of our patients, but blood pressure was shown to be unaffected by paralysis in a larger number of cases ($n = 17$). Although inconclusive, this would suggest that a decrease in venous return did not make a significant contribution to the large discrepancy between the end-expiratory and relaxed Ppao. It also seems unlikely that increased end-expiratory lung volume before relaxation contributed to elevation in the end-expiratory Ppao. While the presence of respiratory muscle activity precluded measurement of the intrinsic PEEP by the method of airway occlusion, only three of our patients had COPD and minute ventilation decreased only minimally after paralysis.

An estimate of transmural pressure can be made by the simultaneous recording of Ppao and esophageal pressure [8]. Although esophageal pressure faithfully tracks changes in the intrathoracic pressure, obtaining a reliable estimate of absolute juxtacardiac pressure in the supine, mechanically ventilated patient may be problematic [18–23]. A precise bedside measurement of left ventricular filling pressure is not available. However, like others [7], we believe that, for clinical purposes, the Ppao measured after muscle relaxation may provide a reasonable estimate of transmural pressure. Even if the latter requires temporary paralysis, avoidance of potentially serious mistakes in clinical management would seem to outweigh the minimal risk of temporary paralysis in most instances. As an illustration of this principle, in several of our cases a high end-expiratory Ppao led to the use of diuretics or inotropes, or both, because of hypotension and oliguria. Once an accurate (and lower) Ppao was obtained after muscle relaxation, aggressive fluid administration led to a normalization of blood pressure, increased urine output and the discontinuation of vasoactive drugs.

Even though a brief period of paralysis carries minimal risk, continuous use of neuromuscular paralysis solely for the purpose of measuring the Ppao is undesirable [24]. In our study, we found that the mid-point Ppao before paralysis often closely approximated the post-paralysis Ppao. If these two values are found to be similar, then it may be appropriate to use the mid-point Ppao for subsequent clinical decision-making, unless clinical deterioration dictates a re-evaluation of its reliability. It is crucial to appreciate that the mid-point Ppao may not accurately reflect the relaxed Ppao in an individual patient. Therefore we advise against using the mid-point Ppao for clinical purposes until it has been correlated with the Ppao measured after muscle relaxation, provided that the patient is mechanically ventilated.

Alternative methods to eliminate expiratory muscle activity include patient reassurance and coaching, the use of intravenous sedatives and the suppression of respiratory activity by increasing the ventilator rate or tidal volume, or both. The former method is usually of minimal benefit in severely ill patients with respiratory failure. Likewise, sedation alone may be ineffective in eliminating respiratory activity unless very large, hemodynamically significant doses are given. Nonetheless, sedation must be given prior to paralysis to ensure amnesia and the sedative may sometimes provide adequate muscle relaxation. Lastly, the combination of sedation and respiratory overdrive sometimes suppresses spontaneous respiratory muscle activity, but one must ensure that the increase in minute ventilation does not lead to the development of intrinsic PEEP [22]. High levels of intrinsic PEEP may lead to hypotension and cause the end-expiratory Ppao to greatly overestimate the left ventricular filling pressure [25].

Large respiratory excursions due entirely to inspiratory muscle activity would not affect the reliability of the end-expiratory Ppao. Indeed, in three of our cases there was a large inspiratory excursion in the Ppao when the patient triggered the ventilator, but the end-expiratory Ppao and the post-paralysis Ppao were similar (Fig. 5). Of note, is the fact that clinical evidence of expiratory muscle activity in these three patients was equivocal. In most of the other cases, palpation of the

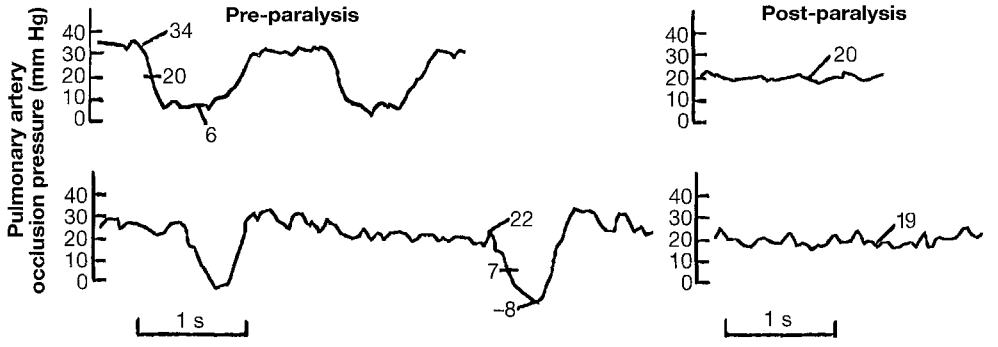


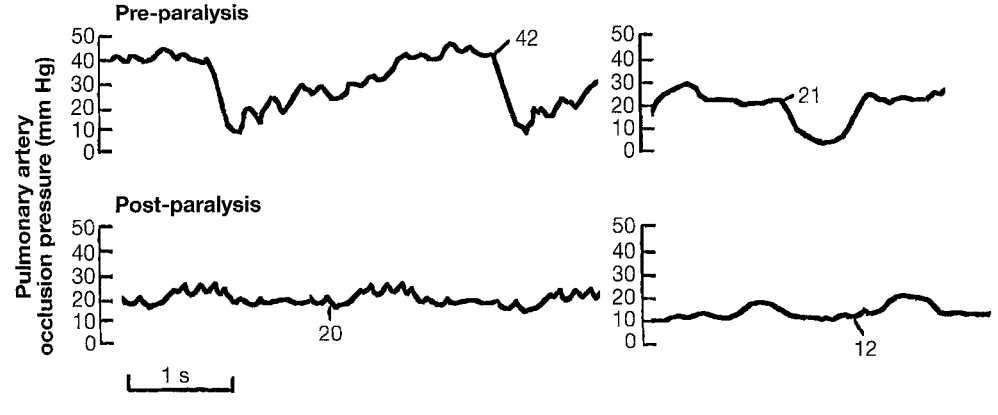
Fig. 5 Tracings of pulmonary artery occlusion pressure (Ppao) before and after muscle paralysis in two cases. Patient A had considerable expiratory muscle activity, as evidenced by the large discrepancy between the end-expiratory Ppao before paralysis and the post-paralysis Ppao. Patient B also had large respiratory excursions, but the end-expiratory Ppao before paralysis was reasonably close to the post-paralysis Ppao. This indicates that expiratory muscle activity was minimal

lateral upper abdomen revealed unequivocal evidence of expiratory muscle contraction that persisted until the end of expiration. In a recent report that demonstrated a significant error in the measurement of intrinsic PEEP due to active exhalation, the authors also commented on the uniform detection of expiratory muscle activity by abdominal palpation [14]. At the bedside, clinical examination that reveals tensing of the abdominal muscles at end-expiration indicates that measurements of end-expiratory Ppao (and intrinsic PEEP) will probably be misleading.

Fig. 6 **A** Pulmonary artery occlusion pressure (Ppao) tracing showing an upward slope throughout the latter part of expiration, indicating the presence of persistent expiratory muscle activity. **B** Pulmonary artery occlusion pressure (Ppao) tracing showing a plateau in pressure during the latter part of expiration. Despite this plateau, the presence of tonic expiratory muscle activity was detected by clinical examination, the degree of which was demonstrated by temporary muscle paralysis (post-paralysis Ppao = 12 mm Hg)

The respiratory-related contour of the Ppao tracing may also provide a clue to the presence of expiratory muscle activity. A Ppao tracing that slopes upward during the latter part of expiration is a clear indication of persistent expiratory activity (Fig. 6 A). However, the presence of a plateau in the Ppao at end-expiration does not necessarily indicate that the expiratory muscles are relaxed. Tonic muscle activity may result in positive end-expiratory pleural pressure, even though the Ppao tracing may reveal a stable plateau in pressure (Fig. 6 B). Similarly, in a study focusing on the measurement of intrinsic PEEP, Lessard and colleagues [14] found that a plateau in airway pressure during airway occlusion did not exclude tonic expiratory muscle activity. Again, bedside examination may prove invaluable in detecting abdominal muscle activity at end-expiration.

Accurate assessment of the Ppao in the non-intubated patient with active exhalation is problematic. Although our data was obtained from mechanically ventilated patients, certain principles derived from our study might be applied cautiously to the spontaneously breathing patients. Temporary cessation of large respiratory excursions, even if only for a few seconds, may be beneficial. One approach is to have the patient sip water through a straw to reduce or eliminate large respiratory excursions. Notwithstanding the potential pitfalls or esophageal pressure monitoring, this technique might be considered, preferably avoiding measurements with the patient supine [19, 21, 22]. If efforts to eliminate ex-



piratory muscle activity are unsuccessful and esophageal pressure is not measured, then the mid-point Ppao should probably be recorded for clinical use. Perhaps most importantly, when weighing the risk versus benefit of inserting a pulmonary artery catheter into a spontaneously breathing patient who demonstrates vigorous respiratory muscle activity, the likelihood that the Ppao will be difficult to interpret must be considered.

In summary, conventional measurement of Ppao at end-expiration will probably overestimate the transmural filling pressure in critically ill patients who have large respiratory excursions in the Ppao tracing and clinical evidence of expiratory muscle activity. When such patients are mechanically ventilated, temporary elimination of respiratory muscle activity provides a more clinically useful assessment of the Ppao and usually reveals that the mid-point Ppao, measured before relaxation, is a reasonable approximation of the post-relaxation Ppao.

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References

1. O'Quin R, Marini JJ (1983) Pulmonary artery occlusion pressure: clinical physiology, measurement and interpretation. Am Rev Respir Dis 128: 319–326
2. Leatherman JW, Marini J (1993) Pressure monitoring. In: Sprung C (ed) The pulmonary artery catheter: methodology and clinical applications, 2nd edn. Closter, NJ: Critical Care Research Associates, pp. 119–156
3. Raper R, Sibbald WJ (1986) Misled by the wedge? Swan-Ganz catheter and left ventricular preload. Chest 89: 427–434
4. Morris AH, Chapman RH, Gardner RM (1984) Frequency of technical problems encountered in the measurement of pulmonary artery wedge pressure. Crit Care Med 12: 164–170
5. Berryhill RE, Benumof JL, Rauscher A et al. (1978) Pulmonary vascular pressure reading at the end of exhalation. Anesthesiology 49: 365–368
6. Maran AG (1980) Variables in pulmonary capillary wedge pressure: variation with intrathoracic pressure, graphic and digital recorders. Crit Care Med 8: 102–105
7. Schuster DP, Seeman MD (1983) Temporary muscle paralysis for accurate measurement of pulmonary artery occlusion pressure: Chest 84: 593–597
8. Rice DL, Awe RJ, Gaasch WH et al. (1974) Wedge pressure measurement in obstructive pulmonary disease. Chest 66: 628–632
9. Marini JJ, Rodriguez R, Lamb VJ (1986) The inspiratory workload of patient-initiated mechanical ventilation. Am Rev Respir Dis 134: 902–909
10. Marini JJ, Capps J, Culver BH (1985) The inspiratory work of breathing during assisted mechanical ventilation. Chest 87: 612–618
11. Marini JJ, Smith TC, Lamb VJ (1988) External work output and force generation during synchronized intermittent mechanical ventilation: effect of machine assistance on breathing effort. Am Rev Respir Dis 138: 1169–1179
12. Guiliani R, Mascia L, Recchia F, Caracciolo A, Fiore T, Ranieri VM (1995) Patient-ventilator interaction during synchronized intermittent mandatory ventilation: effects of flow triggering. Am J Respir Crit Care Med 151: 1–9
13. Chandra A, Coggeshall JW, Ravenscraft SA et al. (1994) Hyperpnea limits the volume recruited by positive end-expiratory pressure. Am J Respir Crit Care Med 150: 911–917
14. Lessard MR, Lofaso F, Brochard L (1995) Expiratory muscle activity increases intrinsic positive end-expiratory pressure independently of dynamic hyperinflation in mechanically ventilated patients. Am J Respir Crit Care Med 151: 562–569
15. Gorini M, Misuri G, Duranti et al. (1997) Abdominal muscle recruitment and PEEPi during bronchconstriction in chronic obstructive pulmonary disease. Thorax 52: 355–361
16. Takata M, Robotham JL (1992) Effects of inspiratory diaphragmatic descent on inferior vena caval venous return. J Appl Physiol 72: 597–607
17. Takata M, Wise RA, Robotham JL (1990) Effects of abdominal pressure on venous return: abdominal vascular zone conditions. J Appl Physiol 69: 1961–1972
18. Bar-Yishay E, Hyatt RE, Rodarte JE (1986) Effect of heart weight on distribution of lung surface pressures in vertical dogs. J Appl Physiol 61: 712–718
19. Baydur A, Behrakis PK, Zin WA et al. (1982) A simple method for assessing the validity of the esphageal balloon technique. Am Rev Resp Dis 126: 788–791
20. Marini JJ, O'Quin R, Culver BH et al. (1982) Estimation of transmural cardiac pressures during ventilation with PEEP. J Appl Physiol 53: 384–391
21. Mead J, Gaensler EA (1959) Esophageal and pleural pressures in man, upright and supine. J Appl Physiol 14: 81–83
22. Milic-Emili J, Mead J, Turner JM (1964) Topography of esophageal pressure as a function of posture in man. J Appl Physiol 19: 212–216
23. Dhainaut JF, Devaux JY, Monsallier JF et al. (1986) Mechanisms of decreased left ventricular preload during continuous positive pressure ventilation in ARDS. Chest 90: 74–80
24. Hansen-Flaschen J, Cowen J, Raps EC (1993) Neuromuscular blockade in the intensive care unit: more than we bargained for. Am Rev Resp Dis 147: 234–236
25. Pepe PE, Marini JJ (1982) Occult positive end-expiratory pressure in mechanically ventilated patients with airflow obstruction. Am Rev Resp Dis 126: 166–170